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Editorial

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To 'D' or not to 'D' in the older person, that is the question

John Agens

In anticipation of new recommendations from the Institute of Medicine and others, it behooves physicians and healthcare providers to review their knowledge base concerning adequate vitamin D intake for fall and fracture prevention in the elderly. There is enough new data for the Institute of Medicine to consider a new Dietary Reference Intake, or DRI, for vitamin D.1 A recent review by Bischoff-Ferrari et al, of numerous randomized controlled trials of vitamin D supplementation in older persons, concluded that both falls and fractures could be prevented. In addition, a dose-response relationship suggested that the optimal supplementation dose is 700 IU to 1000 IU per day.2 Epidemiologic associations between low vitamin D status and various cancers has led some to recommend balancing risk and benefit of moderate ultraviolet light (UV) exposure against complete UV protection for prevention of skin cancer.3 Others have reviewed the epidemiologic evidence for vitamin D supplementation in treatment of hypertension and prevention of cardiovascular disease.4 These epidemiologic studies are tantalizing, yet the evidence is not sufficient to support a causal relationship in making decisions about vitamin D supplementation for the prevention of cancer and cardiovascular disease. I will limit my editorial comments to preventing falls and fractures.

I would suggest looking at potential short- and long-term risks as well as the benefits of any intervention. What evidence do we have for the risks of vitamin D use for prevention? One recent study using a single dose of 500,000 IU of vitamin D daily showed an increased relative risk of fractures,5 but the dose of vitamin D in that study was far higher than other randomized controlled trials. Bischoff-Ferrari et al reviewed documented cases of hypercalcaemia in the randomized controlled trials;2 those authors add that only one trial reported nephrolithiasis, the Women's Health Initiative.6 It is noteworthy that only the self-reported vitamin D and calcium dose was determined in that study, not the vitamin D status of the subjects. My opinion is that hypercalcaemia is uncommon and its complications are rare.

Many interventions that are routinely recommended for the older person probably have higher risks than the 700 IU to 1000 IU of vitamin D per day suggested by the evidence. Medications for hyperlipidaemia are one case in point; antihypertensives are another. Both are considered relatively safe and effective in primary and secondary prevention of cardiovascular disease. The long-term risks of the supplementation of 700 IU to 1000 IU of vitamin D are not well known compared to those long-term risks associated with lipid-lowering drugs or antihypertensives. On the other hand, some older persons at increased fall risk have more immediate threats to their health from a fall or fracture than any long-term risks of vitamin D supplementation. Given the detrimental consequences of falls and fractures in the elderly, the risks of vitamin D supplementation may be worth it.

Competing Interests
None declared

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Research Article

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Prevalence of Psychiatric Co morbidities in Traumatic Amputees-A cross sectional study from Kashmir (Indian Part)

Imtiyaz Mansoor, Mushtaq A Margoob, Nasseer Masoodi, Huda Mushtaq, Tayzeen Younis, Arshad Hussain, Shabir Dhar, and Parvez Chowdary

ABSTRACT

Background and objectives: Loss of a limb for any a reason is a major event with profound implications on the psychological health of an individual involved. Due to prevailing sociopolitical disturbances in Kashmir Valley (Indian administered) and lack of epidemiological data, a study of amputation and its co-morbid psychiatric conditions seems crucial for planning care management for these patients. The aim of our current study was to study various socio-demographic variables of amputees and to find prevalence of psychiatric disorders in amputees from the out-patient population.

Methods: A total of 100 consecutive cases of amputation were studied. Patients who had an amputation were identified and diagnosed according to DSM-IVcriteria for psychiatric co morbidities. Epidemiological and demographic data obtained from the interview of the subjects was analysed and simple percentages were obtained. Prevalence of psychiatric co-morbidities and indication for the amputation were calculated.

Results: In our study we found that, majority (45%) of the amputees were males in the age group of 15-30 years from rural areas (81%) with low literacy rates. Motor vehicle accident accounts for majority (53%) of amputations followed by 21% from ongoing sociopolitical disturbance (landmines, blast, firearms). The most common co-morbid psychiatric condition in our study was major depressive disorder (63%). 40% of patients were suffering from anxiety disorders which included 20% as PTSD (Post Traumatic Stress Disorder), 4% as ssPTSD (sub syndromal PTSD), 10% as GAD (Generalized Anxiety Disorder), and 6% as panic disorder.

Conclusion: Most of the patients with psychiatric co-morbidities were males of younger age group from rural areas. Major depressive disorder was the most common co-morbidity.

Keywords: Psychiatric co-morbidities, traumatic amputation, major depressive disorder, PTSD

Background:

Loss of a limb for any a reason is a major event with profound implications on the psychological health of an individual involved. It has been seen that 20-60% of the amputees attending surgical or rehabilitation clinics are assessed as being clinically depressed[1-3]. Individuals suffering traumatic limb loss at any age are likely to suffer subsequent difficulties with their body image, but these relationships are more striking in the younger age groups who have experienced traumatic injuries. The psychological reactions to amputation are clearly diverse and range from severe disability at one extreme; determined and effective resumption of a full and active life at other end. Indeed, among adults the age at which an individual receives the amputation is also an important factor. The investigation of psycho-social adaptation to amputation has generated a plethora of clinical and empirical studies [4-7]. An amputation is typically equated with loss of once perception of wholeness [8], loss of spouse [9], symbolic castration and even death [10, 11]. The individual's response to a traumatic event is influenced by personality traits, psychiatric premorbid state, gender, peri-traumatic dissociation, prolonged disability of traumatic events, lack of social support and inadequate coping strategies [12-15]. Even though the previous research on consequences of amputation has focused primarily on relationships among demographic variables, coping mechanisms, and outcome measures; there is lack of literature on prevalence of various specific psychiatric disorders post-amputation [16, 17]. Most of the literature and research on prevalence of specific psychiatric morbidity has largely focused on symptoms of depression[18].

To the best of our knowledge there has been very little published about the psychiatric co-morbidity in the victims of amputation. In view of paucity of studies in this field, especially due to prevailing sociopolitical disturbances in Kashmir valley (Indian administered), study of amputation and its co morbid psychiatric conditions seems crucial for planning care management of these patients. Such a study seems justified for more than one reason, as the present state of affairs is in sharp contrast to the traditional circumstances that people of valley used to live in. The aim of our current study is:

1. To study various socio-demographic variables of amputees.
2. To find prevalence of psychiatric disorders in amputees from the out-patient population of the bones and joint surgery hospital, Srinagar which also has an artificial limb rehabilitation centre attached with it.

Materials and methods:

The study was conducted in the Post Graduate Department of Orthopaedics, Govt. Medical College, Srinagar. This 200 bedded hospital is the sole orthopaedic hospital in the Kashmir valley and Ladakh and caters to the needs of all districts of the valley and Ladakh region and some areas of Jammu province. It is affiliated to Govt. Medical College, Srinagar as the teaching hospital, for both under and post graduate studies. A total of 100 patients were studied. The sample comprised of 100 consecutive cases of amputation. Patients who had an amputation were identified and diagnosed according to DSM-IV [19] lead criteria for psychiatric co morbidity. After patient consent, a detailed history was taken, and a general physical examination was performed to identify any medical problems. A detailed semi structured interview with all relevant items from MINI [20] (mini international neuro psychiatric interview) was administered to all the cases included in the study. The cases were selected on the basis of inclusion and exclusion criterion.

Inclusion criteria:

1. Informed consent from the patients under study
2. Amputation of more than one year duration
3. Age more than 14 years and less than 60 years
4. Patients were included in the study irrespective of their sex

Exclusion criteria:

1. Those who do not give consent
2. Those persons who have history of any DSM-IV axis I or axis II disorder before the development of amputation.
3. Presence of disabling medical or neurological conditions like motor neuron disease, Parkinson disease, etc.
4. Age less than 14 years
5. Age more than 60 years

Observations and results:

The data was categorised according to age, sex, residential address, education etc. Data obtained from the interview of the subjects was analysed and simple percentages were obtained. Besides socio-demographic profile, prevalence of psychiatric co-morbidities and reason for amputations were calculated. Results are shown in tables 1-3.

Table-1: Socio-demographic characteristics of the amputees

Characteristic		Number (n)	Percentage
Age	15-30	45	45
	31-45	30	30
	46-60	25	25
Sex	Male	79	79
	Female	21	21
Education	Illiterate	61	61
	Literate	39	39
Marriage	Married	55	55
	Un-married	45	45
Residence	Rural	81	81
	Urban	19	19
Occupation	Domestic workers	42	42
	Unskilled laborers	19	19
	Students	17	17
	Businessmen	16	16
	Govt. employees	06	06
Religion	Islam	95	95
	Sikhism	03	03
	Hinduism	02	02

Table-2: Indication/cause of amputation

Indication/cause	Number (n)	Percentage
Motor vehicle accident	53	53
Blast	11	11
Land mine	06	06
Fire arm injury	04	04
Others*	26	26

*The others include fall from tree, electrocution, machinery mishap, fall from hillock.

Table-3: Prevalence of psychiatric co-morbidities in amputees

Co morbidity	Number(n)	Percentage
Major depressive disorder	63	63
Post traumatic stress disorder	20	20
Impulse control disorder	19	19
Phantom limb phenomenon	14	14
Generalized anxiety disorder	10	10
Panic disorder	06	06
Sub syndromal PTSD	04	04
None	16	16

Discussion

Socio-demographic profile: In our study we found that males out-numbered females by approximately 4:1 ratio (79% males, 21% females). The majority (45%) of the amputees were males in the age group of 15-30 years, followed by 30% in the age group of 31-45 years and 25% in the age group of 46-60 years. The most likely explanation for this observation is that younger people are known to have higher exposure to the violence as

compared to older people. In addition younger patient readily seek help for their psychological problems in comparison to older people. The results are consistent with the study conducted by Ebrahimzadeh et al [21] and Shukla [1] et al. Male predominance could be derived from the reason that ours is a patriarchal type of society where the men are the bread earners of the family and the women usually prefer to stay at home. Another reason could be that men report for rehabilitation and also seek help for their psychological problems more readily. Similar findings have also been reported by Cavanagh et al where they reported 75% of patients were male [22]. 55% of our patients were married which could be due to the reason that majority of our sample were of adults in the marriageable age group. The findings of our study are consistent with the earlier reported studies by Margoob et al [23]. We also observed that majority (81%) of our cases were from rural areas with low literacy rates. Most likely explanation for this observation is that the majority (74.9%) of the population in our state is from rural back ground. Low literacy rates is explained on the basis that most of the people who visit government hospitals of our valley are from poor background where it is very difficult for people to achieve and afford formal education. The other reason could be that Jammu & Kashmir is one of the states of India where literacy rates are low (54.46%) than average in India (65.38%) [24]. Shukla et al in their study of amputees reported that majority of their patients were uneducated [1]. In our study majority of the patients (95%) were Muslim. This is explained by the demographic profiles of the valley of Kashmir-Muslims are the majority community and other communities like Hinduism, Sikhism form part of minority. The greater percentage of Muslims is also substantiated because of mass exodus of minority community in early nineties with start of armed conflict in Kashmir whereby non Muslims migrated amass to different parts of the country.

Reason for amputation: In our study motor vehicle accident account for majority (53%) of the amputations. Most plausible explanations include overwhelming increase of traffic with road being in dilapidated conditions, narrow lanes, lack of driving skills by the motorists, lack of road signs and poor judgment while crossing the road by the pedestrians across the valley [25]. The lawlessness and violence in valley also contribute to reckless driving and negligence of law enforcement agencies. The other collective percentage of 21% which includes 11% for blast injuries, 6% for land mine explosions and 4% for fire arm injury is significant by all means because of the ongoing sociopolitical disturbance in Kashmir since 1990s. The above findings are in accordance with high prevalence of traumatic events in Kashmir as observed by Margoob et al [23]. The study revealed that 59.51% of adult men and 57.39% of women have lifetime prevalence of exposure to traumatic events.

Prevalence of psychiatric co-morbidities:The most common co morbid psychiatric condition in our study was major depressive disorder. 63% of patients were suffering from it. Our results are in accordance with the study conducted by Shukla et al (70.2%). Similar findings have also been reported by Rendal et al [3] and Kashif et al [26].In our study 40% of patients were suffering from anxiety disorders which included 20% as PTSD (Post Traumatic Stress Disorder), 4% as ssPTSD (sub syndromal PTSD), 10% as GAD (Generalized Anxiety Disorder), and 6% as panic disorder. The higher prevalence of PTSD in our study sample is because of higher rate of PTSD in this part of the world as reported in a series of studies by Margoob et al [23]. The results of our study are also in agreement with those reported by Fukunishi[26] (33.9%), and Grieger et al [27]. 19 % of patients in our study reported impulse control disorder in the form of crying spells and outbursts of anger. The lower prevalence of phantom phenomenon (14%) in our sample could be attributed to the fact that the time duration since amputation was variable and usually of longer duration. This is in agreement with the study by Ebrahimzadeh et al [21] where it is reported that 40% of the patients had phantom sensation and 32% were suffering from phantom pain. In another study by Lacorix et al [29], 90% had phantom sensation and 29% had phantom pain. Our observation is further substantiated by Melzack [30], Sherman et al [31], and Pezzin Et al [32] who in their respective studies reported that phantom limb sensation and pain gradually decreases with time. In our study 16% of the patients reported to have no psychiatric co morbidity. This could be due to various coping strategies adopted by the patients with primarily religious and spiritual involvement and obedience to local clergy, Imams (person who leads daily worship services at mosques) and spiritual healers [33]. This observation is in agreement with the study by Margoob et al [34]. In another study Huda et al [33] and Margoob et al [36] found that resorting to religious practices happens to be most often used coping method for dealing with problems and intense emotions of trauma in Kashmiri society. Similar observations have been made by studies in internally displaced people of Chechnya by Jong Kde et al [35].

In light of the above observations of our study, spreading awareness about the co-morbid psychiatric disorders in amputees can be very helpful in diagnosing and proper treatment of such cases and further to prevent chronic debilitating course associated with amputation. More intensive physical and psychiatric rehabilitation with the attention to the provision of prosthesis, retraining, and financial support packages may improve the quality of life of these patients.

Limitations of our study include a small sample size (100). Also results can't be generalised for rest of India or Asia because of socio-political and religious practice differences.

Conclusion:

Psychiatric co-morbidities are very common in amputees in our study. Most of the patients were married males of younger age group from rural areas. The majority of the sample population comprised of unemployed people and those less educated. Major depressive disorder is the most common co-morbidity followed by anxiety disorders in which PTSD subjects were predominant followed by impulse control disorder and phantom phenomenon respectively. A significant number reported no symptoms of mental health illness.

Competing Interests
None declared
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Research Article

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The impact of the provision of extended laboratory service of Troponin T assay

S.M. Coughlin , I. Walker and W.S. Wassif

ABSTRACT
The impact of extending the cut-off time for the provision of Troponin T assay from 4:00 to 7:00 pm, focusing specifically on same-day patient discharge was studied over a four-month period. The number of patients discharged on the same day, who would have otherwise been admitted overnight, was determined. The fiscal benefit of the extended laboratory service was then calculated. Of the 140 patients included in the study, 36 (26%) patients were discharged on the day of hospital presentation based on a negative Troponin T concentration; all except one had a Troponin T <0.03ug/L. Based on the cost of overnight stay of £657 we concluded that the extended service would save the hospital £70,956 annually.
Extending the provision of Troponin T assay for 3 hours daily is cost effective and reduces the number of unnecessary hospital admissions of patients presenting with chest pain of non-cardiac origin.
KEYWORDS Troponin T, lipid profile, cost-effectiveness, hospital admission

Introduction

Troponin T is a protein component of cardiac muscle. When death or damage of the myocardium occurs, it is released in to the circulation and can be detected by immunoassays [1]. Troponin T is a sensitive and specific marker of myocardial damage when taken at least 12 hours after a suspected cardiac event and can be detected up to 7-10 days after myocardial damage [1,2]. When used in conjunction with clinical history, electrocardiograms (ECGs) and cardiac imaging it is effective in excluding acute coronary syndrome (ACS) and myocardial infarction (MI). The cost of a Troponin T assay is £3.75 per sample inclusive of staff time.

Troponin concentrations have been incorporated in up to date definitions of acute MI. One of the criteria for diagnosis of acute MI is the detection of rise and/or fall of cardiac biomarkers (Troponin) with at least one value above the 99thpercentile of the upper reference limit (URL) together with evidence of myocardial ischaemia with at least one of the following: ischaemic symptoms, new ischaemic ECG changes, pathological Q waves on ECG, or imaging suggesting loss of viable myocardium or new regional wall abnormality [3]. Other criteria include unexpected cardiac death involving cardiac arrest, Troponin concentrations associated with percutaneous coronary intervention (PCI) and coronary bypass grafting (CABG) and pathological findings of acute MI [3]. Troponin T is an important component of the risk stratification of patients with acute myocardial ischaemia and can be used to predict 30-day mortality [4,5].

Detection of a rise and/or fall in Troponin T concentration is important when diagnosing acute MI [3,6]. It is the rise and fall that differentiates individuals who have sustained myocardial damage from other causes such as chronic kidney disease (CKD) [3, 7]. In these other conditions the elevated Troponin T concentrations are sustained. To establish the diagnosis of MI, one elevated value above the decision level is required. The demonstration of a rise and/or fall in Troponin T levels assists clinicians in distinguishing elevated background Troponin T concentrations from elevations in the same patients suggestive of MI. Detection of rise and/or fall also identifies those patients with re-infarction within a short time period after an acute MI [8].

It is important to remember however that if the patient presents 24 hours after the onset of symptoms this rise and fall of Troponin T concentration is not necessary to make the diagnosis of MI. Troponin T levels must be interpreted in the light of the clinical presentation. An elevated concentration of Troponin T in the absence of clinical evidence of ischaemia should prompt a search for other aetiologies, such as CKD, congestive heart failure, myocarditis, aortic dissection, or pulmonary embolism [3, 6].

Risk stratification also includes the measurement of lipid profile in those presenting with suspected ACS or MI. To ensure that a cholesterol level representative of the patient's normal baseline the blood sample must be organised within 24 hours of the event. In those with delayed presentation or where cholesterol is omitted on admission clinicians should wait until 3 months after the event to obtain a reliable cholesterol level, although

most would be expected to have started lipid-lowering medications [9,10,11].

Method

We studied Troponin T requests made between 4pm and 7pm for a four-month period. Request cards were retrieved and the Troponin T result for each request was obtained. Any other Troponin T results obtained at any time relating to that event were noted as well as any rise and fall of the Troponin T concentrations. Review of the hospital notes for each patient established the working diagnosis, whether any other appropriate investigations had been carried out during admission, co-morbidities that were present and current relevant medications.

The final patient outcome was noted. The number of patients discharged on the same day, who would have otherwise been admitted overnight, based on Troponin T concentration was determined. Those patients with a Troponin T concentration above the 99th percentile of the upper reference limit (URL) used in the local laboratory (Troponin T$\leq$0.03ug/L) who were not discharged on the day of Troponin T measurement were identified and the reason for admission determined. The fiscal impact of the extended laboratory service was calculated.

Results

Of 162 Troponin T requests received during the four-month period, 140 (86%) were included in the study; 22 (14%) were excluded (12 haemolysed, 1 unlabelled, 2 not on computer system, 7 clinical notes unavailable).

The study population comprised of 74 (53%) male and 66 (47%) female patients. The age range was 21 – 101 years; mean (±SD) 67.6 (±16.8).

Half of Troponin T requests were received from the Acute Assessment Unit (AAU), 20% from the Emergency Department, 14% from inpatients, 8% from the Critical Care Complex (CCC) and 8% from the Coronary Care Unit (CCU).

Clinical notes indicated that 97 (69%) of Troponin T requests were taken appropriately at least 12 hours after the onset of the event, 19 (14%) were taken less than 12 hours after the event, in the remaining 24 (17%) the time of sample in relation to the event was not known. Interestingly only 30% of request cards had documented that sample was taken at least 12 hours after the event.

The indication documented on each request card is detailed in Figure 1. Indications detailed under other included: trauma, sepsis, collapse, cold & clammy, oesophageal cancer with hypercalcaemia, poor complex tachycardia, post-operative after abdominal aortic aneurysm repair, respiratory infection, sweating, palpitations, fall and repeat bleed because of previously unsuitable sample.

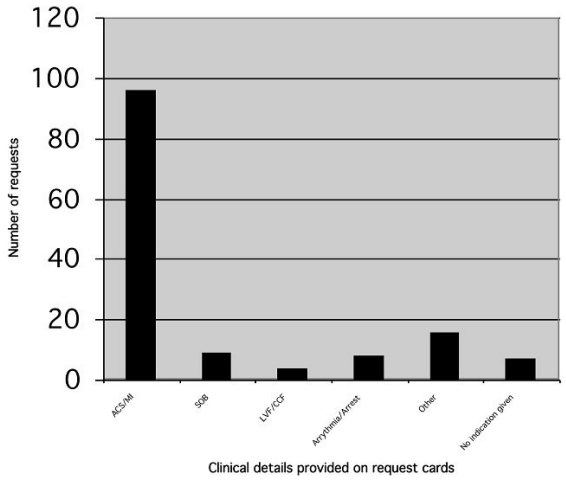

Figure 1: Indication noted on request card for Troponin T. ACS: acute coronary syndrome, MI: myocardial infarction, SOB: shortness of breath, LVF: left ventricular failure, CCF: congestive cardiac failure.

One hundred and two (73%) patients had a non-elevated Troponin T concentration of $\leq$0.030ug/L and 38 (27%) had an elevated Troponin T concentration >0.03ug/L. Only 5 (4%) patients had the rise and fall of Troponin T documented.

Eighty-three (59%) patients had no lipid profile measured during the attendance/admission. Of the remaining 57 patients, 31 (54%) had cholesterol assayed within 24 hours of the event, in 16 (28%) the cholesterol was taken between 2 and 17 days after the event and in 10 (18%) patients the time of cholesterol assay in relation to the event was not known. Overall only 1 in 5 patients had a lipid profile obtained within 24 hours of the event.

Interestingly of the 38 patients with raised Troponin T concentration of >0.03ug/L only 13 (34%) had a lipid profile organised. Only 7 of the 13 (54%) were obtained within 24 hours of the event, 4 were taken between 2 and 10 days after the event and in 2 patients it was not known when the lipid profile was obtained in relation to the event.

Overall no correlation was noted between cholesterol and Troponin T concentrations in all patients who had an elevated Troponin T concentration and cholesterol measured. Interestingly in those where cholesterol was measured within 24 hours of the suspected cardiac event there was some correlation, but the numbers involved were small.

The working diagnosis as stated in hospital notes is documented in Table 1.

Table 1: Working Diagnosis

Working Diagnosis	Number (%)
ACS/MI	62 (44.3%)
Arrythmia/Arrest	8 (5.5%)
Fast AF/atrial flutter	6 (4.3%)
CCF/LVF	5 (3.6%)
Myocarditis	1 (0.7%)
Musculoskeletal chest pain	7 (5.0%)
Respiratory complaint	18 (12.9%)
GORD put in legend	4 (2.9%)
Other	11 (7.9%)
No diagnosis	18 (12.9%)
Total	140 (100%)

ACS, acute coronary syndrome; MI, myocardial infarction; AF, atrial fibrillation; CCF, congestive cardiac failure; LVF, left ventricular failure; GORD, gastro-oesophageal reflux disease.

Table 2: Reason why those patients with non-elevated Troponin T concentration of ≤0.03 (ug/L) were not discharged on the same day by the clinician.

Reason for admission	Number of patients
Trop T assayed <12hrs	5 (8%)
Ongoing chest pain	10 (15%)
ECG changes	3 (5%)
High CAD risk patient	2 (3%)
Monitoring and cardiology review	2 (3%)
Already inpatient	7 (10%)
Repeat attendance in 24hrs	1 (1%)
Other medical (non-cardiac) problem	28 (42%)
No reason documented	6 (9%)
Outcome not available	1 (1%)
Self discharge	2 (3%)
Total	67 (100%)

All of the 36 (26%) patients except one who were discharged on the day of Troponin T assay had a negative Troponin T concentration of ≤0.030ug/L. This patient had CABG one month previously and presented with chest pain and associated cough, although his Troponin T was 0.14ug/L, this was deemed not significant in view of a previous Troponin T concentration of 0.16ug/L assayed two days earlier.

Sixty three (45%) patients remained in the AAU or were admitted to a medical ward, 15 (11%) were admitted to CCU, 4 (3%) to CCC and 18 (13%) were already inpatients. Of the remaining 3 patients, 2 self-discharged and in 1 the final destination was not available.

Of those patients with a raised Troponin T concentration of >0.03ug/L 5 died during this attendance.

The majority (60/102) of patients in whom Troponin T was not raised (≤0.030ug/L) still required hospital admission (Table 2). Another 6 patients with a non-elevated Troponin T concentration had no obvious reason for admission documented.

Based on an overnight stay cost of £657 we conclude that the laboratory's extension of Troponin T service of 3 hours would save the hospital £70,956 annually. No additional manpower was required to provide the extended laboratory service as Biomedical Scientists are already providing urgent out of hour on-call service for other biochemical analysis. No additional laboratory costs were incurred, as the same number of samples would have been analysed during working hours the following day.

Discussion

There was sufficient demand for Troponin T assay to justify extension of the laboratory service for 3 hours each day. As expected most requests for Troponin T came from the AAU and the Emergency Department where the majority of patients with chest pain of potential cardiac origin would initially present. In those patients presenting with suspected myocardial damage 3 out of 4 had chest pain of non-cardiac origin.

In those patients where the time of event was known the majority had an appropriate Troponin T assay taken at least 12 hours after the event suggesting that most of the medical and nursing staff were well informed. In contrast it appears that only few of the medical profession were aware of the need to measure lipid profile soon after admission as only 1 in 5 patients had their lipid profile organised within 24 hours of the event.

The majority of requests had appropriate clinical details to justify Troponin T request. However one in four requests were deemed inappropriate (Fig. 1). Since Troponin T may be raised in other conditions the assay should be reserved for those patients where myocardial damage is suspected. Inappropriate testing is potentially hazardous and may expose patients to further unnecessary invasive investigations e.g. cardiac catheterisation with associated morbidity and mortality.

In patients presenting with chest pain, Troponin T assayed appropriately >12 hours after onset of the event can be used effectively to exclude myocardial damage and discharge can be made on the basis of this result without the need for admission. A small proportion (6%) of patients with non-elevated Troponin T concentrations who had no obvious reason for admission, were deemed unnecessary.

Dyslipidaemia plays an important role in the risk stratification of patients with suspected ACS or MI, yet only one in five patients with myocardial damage had a lipid profile organised within 24 hours of the event. Cholesterol measurements organised between 2 and 17 days after the event would not have been representative of the true concentration and were deemed inappropriate. Too few lipid profiles were assayed within 24 hours of the event in patients with an elevated Troponin T concentration to determine whether there is any correlation between cholesterol and Troponin T concentrations.

Similarly only a small number of patients had the rise and fall of Troponin T documented. The lack of serial measurements of Troponin T concentrations may have resulted in failure to recognise some patients with other conditions, which may cause elevated Troponin T concentrations and potentially subject them to unnecessary further invasive investigations.

The provision of the extended laboratory service had a positive impact; it enabled earlier discharge of patients with chest pain of non-cardiac origin, resulted in fewer unnecessary overnight hospital admissions and reduced the demand on hospital beds. Extending the service did not result in extra work for junior doctors, on the contrary by improving the efficiency of the process has not only speeded the patient journey but has improved junior doctors' time-management.

We have shown that extending the provision of Troponin T assay for 3 hours daily has both fiscal and management benefits and reduces the number of unnecessary hospital admissions. Further extension to incorporate a 24-hour laboratory service for this assay would potentially reduce hospital admissions further with more potential savings.

Learning Points

- Extending Troponin T service has a fiscal benefit.
- Rise and fall of Troponin T values should be documented.
- Lipid profile should be organised within 24 hours in all patients presenting with chest pain of potentially cardiac origin.
- Measuring Troponin T where myocardial damage is not clinically suspected is potentially hazardous and may expose patients to further inappropriate and invasive investigations with associated morbidity and mortality.
- In the current climate of litigation detailed documentation is necessary.

Conclusion

Extending the provision of Troponin T assay for 3 hours daily has fiscal and management benefits and reduces the number of unnecessary hospital admissions of patients presenting with chest pain of non-cardiac origin.

Competing Interests
None declared

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Research Article

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Patients and Professionals attitude towards postoperative recovery: Academic Competency Assessment versus Patients Real Time Experience

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ABSTRACT

Open and laparoscopic surgery is evolving fastest ever; however the professional advice regarding patient's postoperative care and transient lifestyle changes remains historical. This study aimed to evaluate the knowledge based advice from surgical trainees and general practitioners about accumulative postoperative care after routine surgical operations in comparison to patient's journey back to routine life activities.

Materials and Methods:

Patients aged 65 years or less, who had routine surgical procedures over a six-month period completed a self-devised questionnaire with regards to time taken to return to normal activities following surgery. A further questionnaire was distributed to GPs and surgeons, including trainee doctors.

Results:

Varicose vein surgery: Patients take a shorter time to return to heavy work, driving and normal activities than that advised by both sets of doctors.

Open hernia repair: Patients take longer to return to office work and normal activities than that advised by both sets of doctors.

Laparoscopic hernia repair: Patients take a shorter time to return to heavy work, than that advised by both sets of doctors.

Laparoscopic cholecystectomy: Patients take longer to return to office work, but a shorter period of time to return to heavy work than that advised by both sets of doctors.

Conclusion:

The advice received by the patients with regards to their post operative recovery robustly varies between surgeons and non surgical professionals, and does not reflect the real time experience of patients. A consensus among surgeons and primary care physicians is essential to streamline surgical care pathway.

Introduction

Patients, in both the pre and post-operative periods, seek and receive advice from a number of health professionals. The advent and subsequent increasing use of day case surgery has also meant that patients have a reduced exposure to the surgical staff. This subsequently results in patients increasingly seeking post-operative advice from their general practitioner and allied health care professionals. The development of innovative surgical techniques has meant that the traditional teachings with regard to time taken for convalescence following surgery are somewhat outdated. The aim of this study was to initially determine the exact time taken for patients to return to work, driving and daily routine for a number of routine general surgical procedures. Secondly we aimed to determine the advice that GPs and surgeons would give to patients following routine surgery.

Patients and Methods

Patients aged 65 years or less, who had routine surgical procedures (open unilateral inguinal hernia repair, laparoscopic cholecystectomy, laparoscopic hernia repair and unilateral varicose vein surgery) over a six month period (January – June 2004) were identified from the theatre database. A single page questionnaire was sent to each patient (Appendix 1). Each patient was questioned with regard to the following:

- Occupation
- Time taken to return to normal activities following surgery
- Time taken to return to driving following surgery and any advice given
- Expected and actual time off from work following surgery
- Distribution and length of a sick note
- Expectations following surgery
- Experience of day case surgery

Questionnaires were returned and data collected on a specially constructed database. Concurrent to this a further questionnaire (Appendix 2) was distributed to a number of differing groups of health professionals. These were namely:

- GPs – this included the GPs of all patients who had been identified as having undergone surgery in the specified six month period as well as all doctors on the vocational training scheme.
- Surgeons – this included all senior house officers on the Yorkshire School of Surgery Basic Surgical Training Scheme and all Higher Surgical Trainees (General Surgery)

within the Yorkshire Deanery including non-carrier grade doctors.

Replies were anonymous and each health care professional was asked with regards to the advice they would give to an "average" patient undergoing the four procedures with regard to time it would take to return to work (office or heavy), driving and return to normal activities. They were also asked whether they felt the procedure was suitable for day case surgery.

Statistical Analysis

Statistical analysis was undertaken using the Analyse-it statistical package (Leeds, UK.). Non-parametric analysis using either Kruskall 1- way ANOVA or the Mann-Whitney U test was used to test for a difference between the medians of independent samples. The Wilcoxon signed-ranks test was used to test for a difference between the medians of 2 related samples. Significance was determined as a p-value < 0.05.

Results

Nineteen of 48 patients who underwent varicose vein surgery (39%), 44 of 72 patients who underwent a laparoscopic cholecystectomy (61%), 23 of 35 patients who underwent a laparoscopic hernia repair (65%) and 12 of 23 patients who underwent an open inguinal hernia repair (52%) over the six month period returned a completed questionnaire. Of the health care professionals, 65 primary care physicians were identified and sent questionnaire, of which fifty three GPs (81.5%) replied. From the Yorkshire deaneries database sixty five trainees were identified (Spr, SHO, HO, non-carrier grades), of which 41(63.2%) surgically trained doctors returned a completed questionnaire. Among the responders, we also include four consultant surgeons who have performed the operations on patients in our hospital. Overall one hundred and thirty participants were sent study forms, of which 94 (72.3%) health professionals responded with completed questionnaire.

Varicose Vein Surgery (Table 1)

Of the 19/48 patients who returned a completed questionnaire, eleven (57.8%) were women with an overall median age 44 years (range 21-64 years). Seventeen of the 19 patients worked (89%), 11 of who undertook office work (57.8%). Patients tended to return to driving and normal activities quicker than that recommended by doctors. GPs and surgeons offered similar advice with regard to return to all activities following varicose vein surgery. Nine of the 19 patients were uncertain about whether they have received any advice or perhaps forgotten any information regarding when to return to driving. Five patients received no advice about when to return to work. No significant difference was observed between expected time off work and actual time off work experienced by the patients (2 weeks vs. 1 week – p=0.15 Wilcoxon Rank test). Fifteen of the 19patients (79%) said that their recovery was what they had expected with the reasons for not meeting expectations being wound infection in 2, bruising and a larger incision in one patient each. Seventeen patients had their surgery performed as a day case (89.4%). Fifteen patients stated that they would have surgery again as a day case (88.2%).

Laparoscopic Cholecystectomy (Table 2)

Of the 44/72 patients who returned a completed questionnaire 39 were women (88.6%) with an overall median age 47 years (range 20-63 years). Thirty-two of the 44 patients worked (72%), 25 of who undertook office work (56%). Patients returned to office work significantly later than that recommended by both groups of doctors. Overall, patients took a significantly shorter time to return to work that involved lifting heavy objects. Surgeons also recommended shorter times to return to work when compared with GPs. Of further interest is the observation that it took a shorter time for those patients undertaking heavy work to return to work when compared with the patients undertaking office work. There was no significant difference in the time taken to return to driving and normal activities experienced by the patients when compared to the advice given by both groups of doctors. Ten of the 44 patients (22%) stated that they had received no advice regarding when to return to driving or perhaps they may have no memory about driving instructions. Seven patients stated they received no advice about when to return to work (15%). Overall, patients expected a significantly shorter time off work than was actually experienced (2.5 weeks vs. 4 weeks – p<0.01 Wilcoxon Rank test). Twenty-one of the 44 patients (48%) said that their recovery was not what they had expected (47%). Of these 21 patients, 6 said that their recovery was better than expected (28%), 5 said that their recovery was longer than expected (23%), and the rest either complained of pain or wound infection. Seventeen patients had their surgery performed as a day case (38%). Of these 17, 11 said that they would have surgery again as a day case (64%). A significantly higher proportion of GPs felt that this procedure was suitable for day case surgery compared with the proportion of patients who actually underwent the procedure as a day case (p=0.02 chi squared test).

Laparoscopic Inguinal Hernia Rapair (Table 3)

Of the 23/35 patients who returned a completed questionnaire, the majority had bilateral hernias repaired. 22 were men (95%) with an overall median age 48 years (range 35-63 years). Twenty one of the 23 patients worked (91%), 10 of who undertook office work (43%).

No significant difference was found between the actual time taken to return to office work and the advice given by either group of doctors. Patients returned to heavy work significantly sooner than that recommended by both groups of doctors. There was no significant difference in the time taken to return to driving and normal activities experienced by the patients when compared to the advice given by both groups of doctors.

Activity		Time (IQR in Weeks)	Overall (K)	Surgeons vs. GPs (M)	Surgeons vs. Patients (M)	GPs vs. Patients (M)
Office Work	Surgeons	2 (1-2)	0.13	0.56	0.10	0.05
	GPs	2 (1-2)				
	Patients	1 (1-2)				
Heavy Work	Surgeons	3 (2-5)	<0.01	0.75	<0.01	<0.01
	GPs	4 (2-4)				
	Patients	1 (1-1.75)				
Driving	Surgeons	2 (1-2)	<0.01	0.24	<0.01	0.02
	GPs	2 (1-2)				
	Patients	1 (1-1)				
Normal Activities	Surgeons	2 (2-4)	0.05	0.57	0.04	0.02
	GPs	2 (2-4)				
	Patients	1.5 (1-2)				

Table 1: Time taken to return to work, driving and daily activities as experienced by patients and as suggested by both surgically trained doctors and GPs for unilateral varicose vein surgery. Time: Median time to return to activity (IQR - weeks) K: Kruskal Wallis ANOVA. M: Mann Whitney U test. P<0.05 deemed as significant.

Activity		Time (IQR - weeks)	Overall (K)	Surgeons vs. GPs (M)	Surgeons vs. Patients (M)	GPs vs. Patients (M)
Office Work	Surgeons	2 (1-2)	<0.01	0.02	<0.01	<0.01
	GPs	2 (2-3)				
	Patients	5 (3-7)				
Heavy Work	Surgeons	4 (2-4)	<0.01	<0.01	0.26	0.04
	GPs	4 (4-6)				
	Patients	2 (1.5-4)				
Driving	Surgeons	2 (1-2)	0.19	0.19	0.10	0.43
	GPs	2 (1-3)				
	Patients	2 (1-4)				
Normal Activities	Surgeons	2 (1-4)	0.19	0.20	0.09	0.47
	GPs	3 (2-4)				
	Patients	4 (2-6)				

Table 2: Time taken to return to work, driving and daily activities as experienced by patients and as suggested by both surgically trained doctors and GPs for laparoscopic cholecystectomy. Time: Median time to return to activity (IQR - weeks) K: Kruskal Wallis ANOVA. M: Mann Whitney U test.

Activity		Time (IQR - weeks)	Overall (K)	Surgeons vs. GPs (M)	Surgeons vs. Patients (M)	GPs vs. Patients (M)
Office Work	Surgeons	2(1-2)	0.73	0.56	0.48	0.714
	GPs	2 (1-2)				
	Patients	2 (1-2.75)				
Heavy Work	Surgeons	6 (4-6)	0.03	0.31	0.01	0.03
	GPs	4 (4-6)				
	Patients	3 (2-4)				
Driving	Surgeons	2 (1-4)	0.22	0.21	0.12	0.46
	GPs	2 (1-2)				
	Patients	1 (1-2.25)				
Normal Activities	Surgeons	2 (2-4)	0.41	0.87	0.31	0.17
	GPs	3 (2-4)				
	Patients	2.5 (1.25-3)				

Table 3: Time taken to return to work, driving and daily activities as experienced by patients and as suggested by both surgically trained doctors and GPs for laparoscopic hernia repair. Time: Median time to return to activity (IQR - weeks) K: Kruskal Wallis ANOVA. M: Mann Whitney U test.

Activity		Time (IQR - weeks)	Overall (K)	Surgeons vs. GPs (M)	Surgeons vs. Patients (M)	GPs vs. Patients (M)
Office Work	Surgeons	2 (2-2)	0.01	0.07	<0.01	0.05
	GPs	2 (1.25-3)				
	Patients	4 (3-4)				
Heavy Work	Surgeons	6 (4-6)	0.57	0.49	0.47	0.39
	GPs	6 (4-7.75)				
	Patients	5 (4.25-5.75)				
Driving	Surgeons	3 (2-4)	0.03	0.06	0.02	0.15
	GPs	2 (2-3)				
	Patients	2 (1-2)				
Normal Activities	Surgeons	2 (2-2)	<0.01	0.07	<0.01	0.01
	GPs	2 (1.25-3)				
	Patients	4 (2.5-5)				

Table 4: Time taken to return to work, driving and daily activities as experienced by patients and as suggested by both surgically trained doctors and GPs for open hernia repair. Time: Median time to return to activity (IQR - weeks) K: Kruskal Wallis ANOVA. M: Mann Whitney U test. P<0.05 deemed as significant.

Three (13%) patients were uncertain about receiving advice regarding when to return to driving or they might have no memory of information received. Six (26%) patients stated they cannot recall about receiving any advice regarding when to return to work. There was no significant difference seen in the time patients expected off work than was actually experienced (2 weeks vs. 2 weeks – p>0.05 Wilcoxon Rank test). Nine of the 23 patients (39%) said that their recovery was not what they had expected. Of these 9 patients, 2 (22%) said that their recovery was longer than expected, 4 (44%) said that they experienced more pain than they expected; one (11%) said that the recovery time was much shorter and one (11%) experienced some bleeding from the umbilical port.

Twenty patients (86%) underwent their surgery as a day-case. Of these 20, 16 (69%) said that they would have their surgery again as a day case.

Open Inguinal Hernia Repair (Table 4)

All 12/23 patients who returned a completed questionnaire were men with an overall median age 54 years (range 42-65 years). Nine of the 12 patients worked (75%), 5 of whom undertook office work (41%).

Patients took a significantly longer time to return to office work when compared to the advice given by either group of doctors. No significant difference was observed in the time taken for patients to return to manual work and the advice given by either group of doctors. Surgeons advised a longer period of abstinence from driving compared to that actually undertaken by the patients. Patients took a significantly longer time to return to normal activities when compared to the advice given by either group of doctors. Two patients (16%) replied that no information was given or may not recall in regards to when to return to driving and one patient (8.3%) stated that he cannot recall any professional advice he has received about return to work. There was no significant difference seen in the time patients expected off work than was actually experienced (3 weeks vs. 5 weeks – p>0.05 Wilcoxon Rank test). Five patients (41%) said that their recovery was not what they had expected. Of these 5 patients, 4 (80%) said that they experienced more pain than they expected and one (20%) experienced more bruising. Seven patients (58%) underwent their surgery as a day-case and of these, 5 (71%) said that they would have their surgery again as a day case.

Discussion

With the advent of day case surgery there is an increasing number of health professionals giving advice to patients about their post-operative course. Advocates of minimal access surgical techniquesand day case surgery claim that this is associated with a reduction in the periodof postoperative recovery[1, 2]. The proposed benefits,however, may never be seen if there is no concordance in the advice given by medical practitioners. The advice given to patients is still based upon personal experience rather than firm scientific evidence and indeed, there have been few studies that have analysed patients return to normal activities following surgery. Majeed *et al* questioned 59 general practitioners and61 surgeons with regard to the time taken for young (25 years old) and older (55 years old) patients to return to sedentary, light manual and heavy manual work following a number of common surgical procedures (including varicose vein surgery, unilateral open inguinal hernia repair and laparoscopic cholecystectomy) [3]. The moststriking finding was the enormous variation in opinion betweendifferent doctors. For example, a 55 year old heavy manual workerhaving a haemorrhoidectomy could be given between one and 16 weeks off work depending on which doctor he or she consulted. Such wide variation was not observed in our study and in general, the advice given by both GPs and surgeons was similar apart from the fact that surgeons advised a shorter period off office work for patients undergoing laparoscopic cholecystectomy. The end of the twentieth century has brought an exponential growth in new surgical techniques for standard general surgical procedures. Not only there has been an increase in the use of mesh for open inguinal hernia repairs but there has also been an increasing use of laparoscopic hernia repair, with the recent guidance by the national institute for health and clinical excellence (NICE) liable to further increase the role of laparoscopic repair[4]. Furthermore, there has been the widespread acceptance of laparoscopic cholecystectomy and an increased awareness of the role of general anaesthetic in increasing the number of procedures that can be undertaken as a day case. Given these continuing developments in surgical technique as well as in both pre- and post-operative care the present advice and experience of GPs could be seen to be somewhat out-dated.

Two surgeons within the unit perform laparoscopic hernia repair (one the transabdominal preperitoneal repair (TAPP) and one the totally extraperitoneal (TEP) repair) with three performing solely the open technique. Although our results based on small sample size but match with evidence based recommendation by NICE, suggests that laparoscopic repair does reduce the time taken for post operative recovery when compared to open repair. In fact, all patients returning to heavy work following laparoscopic hernia repair do so quicker than that advised by either GP's or surgeons although unlike the surgeons, GPs do tend to recognise the likely reduction in pain experienced following a laparoscopic repair and alter the advice given to those in heavy work accordingly. Restriction of activity on the advice of surgeons may be basedon their concern for tissue healing and strength, which may have arisen in the days when absorbablesutures such as catgut were used. The use of mesh should now change this thinking and it has indeed been shown that there is no increase in the recurrence of inguinal herniasafter early return to work [5]. Office workers undergoing an open inguinal hernia repair take a longer time to return to work (4 weeks) than that advised by both groups of doctors. Furthermore, patients undergoing laparoscopic cholecystectomy

take a shorter time to return to heavy work than office work. These results do require more evaluation. At face value it would appear that doctors underestimate the time taken for return to office work and in the case of the cholecystectomy overestimate the time it takes to return to heavy work. In fact the patients in office work took a significantly longer time to return to work following cholecystectomy than those in heavy work. Although only 20% of the working cohort of patients who underwent cholecystectomy were in "heavy work" this result probably represents the fact that a high proportion of people in heavy work are self-employed and time off work is money lost. Patients who are selfemployed return to work much sooner than those in salaried jobs [6]. Furthermore, there may well be an element of low job satisfaction in people in office work, which has also been shown to be a major predictor of delayed return to work [7]. The time taken to return to work, however, may be dependant on the patients' expectation of convalescencetime formed prior to surgery, which in many cases is based upon advice given by medical practitioners. Furthermore, the attitude of the medical profession in the post-operative period is important as they have to issue the certification necessaryto ensure financial compensation for the patient.

Patients undergoing varicose vein surgery returned to heavy work, driving and normal activities significantly sooner than that suggested by either group of doctors. This may well be down to a recently concerted effort to encourage patients to walk to reduce the risk of DVT. All patients had long saphenous vein (LSV) surgery by either the standard high tie, stripping of the LSV and multiple stab avulsions or by local ligation of the LSV. Overall it would appear that a one-week period of recuperation is all that is needed following unilateral varicose vein surgery. The advent of minimally invasive treatment for varicose veins may result in a shorter post-operative recovery period [8].

There are some shortcomings associated with this study. Questionnaire based studies always present methodological issues including problems with response rate. There is never an "average patient" and normal activities for one patient may be completely different from those of another patients and any advice given should be individually tailored. Furthermore, occupations were not classified as either manual or office based prior to the start of this study, but were classified on an individual basis during collation of the data. However, we hope that the data presented here will help medical practitioners advising their patients about postoperative routine life activities.

Conclusion

We believe that our overall practice is not different with regards to the pre, peri and post-operative management of patients when compared to the majority of units within the UK. However, there may well be some variation with regard to healing and time taken to return to work and we would encourage other units to undertake similar studies to determine convalescence times.

Appendix 1

Sex (Male / Female)	
Age at time of surgery.	
Do you work If yes, what job do you do?	Yes / No
How long did it take to you to return to your normal activities of daily living following your operation (weeks).	
If you drive, how long did it take for you to start driving again (weeks). What advice, if any, were you given about driving after your operation?	
The following questions are to be completed if you do work.	
Prior to your surgery, did you receive any information about how long you would be off work? If YES, what information was given to you?	Yes / No
How long did you expect to be off work following your surgery (weeks)? How long were you actually off work following your surgery (weeks)?	
If you are in employment: Did you get a sick-note: • From the hospital • From the GP How long was the sick note for (weeks)? Did the sick note need to be extended?	 Yes / No Yes / No Yes / No
Was the recovery after your operation as you had expected it to be? If no, why not?	Yes / No
Did you go home on the same day as you had your operation? If YES, would you do the same if you had the operation again or would you prefer to stay overnight after your operation? If you would prefer to stay overnight, why?	Yes / No

Appendix 2

Dear Doctor.

We at xxxxxx Hospital are undertaking a study to determine whether the information given to patients following routine general surgical procedures is consistent and compares to the actual recovery period experienced by the patients themselves. We would be grateful if you would consider the four general surgical procedures below and give us an average length of time (in weeks) that you would advise the patient to abstain from:

(a) office work
(b) heavy work
(c) driving

(d) to return to normal activities of daily living

The general surgical procedure to be considered are

1) mesh repair of an inguinal hernia (unilateral)
2) laparoscopic hernia repair
3) unilateral varicose vein surgery
4) laparoscopic cholecystectomy

	Office Work	Heavy Work	Driving	Normal Activities
Mesh repair inguinal hernia				
Lap. Repair inguinal hernia				
VV surgery				
Lap Chole				

Conflict of Interests
None Declared
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Review Article

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Prospects of Adult Stem cells therapy in Peripheral Vascular Diseases

Jayprakash Gopall , Wen Huang and Yu Zhao

ABSTRACT

Peripheral Vascular Disease (PVD) is a growing medical problem and presents itself mainly in two different clinical forms. Intermittent claudication is an early moderate manifestation, while patients with critical limb ischaemia suffer from severe muscle tissue loss or ulcers and are at high risk of limb amputation. Despite recent advances in surgical and radiologic vascular procedures, a large number of patients are not eligible for these revascularisation procedures. Recent evidence indicates that adult stem cells (ASC) are a potential new therapeutic target. This review discusses the potential of ASC in patients with PVD. The safety of stem cells must be scrutinised and assessed throughout the entire treatment and research process. Guidelines and strategies must also be developed to ensure that every aspect of stem cell use from identification and isolation of stem cells to stem cell transplant is stringently coordinated.

KEYWORDS

Adult stem cells, peripheral vascular disease, critical limb ischaemia, therapeutic neo-angiogenesis.

ABBREVIATIONS

Peripheral Vascular Disease (PVD),Adult Stem Cell (ASC),Intermittent Claudication (IC),Critical Limb Ischemia (CLI),Peripheral Arterial Occlusive Disease (PAOD), Vascular Endothelial Growth Factor (VEGF), Fibroblast Growth Factors (FGFs),Bone Marrow (BM), Endothelial Progenitor Cells (EPC), Bone Marrow Mononuclear cells (BM-MNC), Granulocyte Colony Stimulating Factor (G-CSF),Peripheral Blood Mononuclear Cells (PB-MNC), Therapeutic Angiogenesis using Cell Transplantation (TACT).

Introduction

Currently, peripheral vascular disease (PVD), causing an inadequate oxygen supply to the limbs, globally affects no less than 3–10% of the population[1]. Peripheral vascular disease, including diabetic foot, arteriosclerosis obliterans, and thromboangitis obliterans, commonly affect the arteries supplying the leg. Based on the severity of the symptoms, usually two clinical presentations are distinguished: intermittent claudication (IC) is characterised by pain upon walking while critical limb ischaemia (CLI) is a more severe form in which pain occurs at rest and which is accompanied by necrosis and ulceration.

Peripheral arterial occlusive disease (PAOD) is estimated to develop in 500 to 1000 individuals per million persons per year[2, 3]. The prevalence of all stages of PAOD in the general population is estimated to be 4.2% to 35%. Within this group, 4.3% to 9.6% will experience progression of the disease towards CLI, eventually resulting in amputation of the affected limb[4]. Diabetic PAOD patients are at the highest risk within this patient group: they are about 10 times more likely to come to amputation, and the prevalence of gangrene is 20 to 30 times higher[2]. CLI has important functional implications and a major impact on the quality of life. Quality of life indices of patients with CLI have been reported to be similar to those of terminal cancer patients[5]. In addition, CLI is associated with surgery and hospitalisation[6]. CLI is also associated with increased mortality (the 1-year mortality is approximately 25% and may be as high as 45% after amputation)[7] , and even asymptomatic PAOD by itself is a significant predictor of cardiovascular morbidity and death[8].While obstructive atherosclerotic disease is the most common cause of PVD, some forms of vasculitis, such as thromboangiitis obliterans or Buerger's disease, also result in peripheral ischaemia (in feet and/or hands), often progressing to tissue loss and major amputations[9,10].

Unfortunately, a significant proportion of patients (including both IC and CLI cases) are not eligible for or do not beneficially respond to these revascularisation procedures due to the widespread nature or the distal location of the obstructions or due to the presence of co-morbidities putting them at higher risk for peri-procedural death.

For these 'no-option' patients, non-invasive revascularisation strategies have been introduced, which fall into two categories: single gene/protein-based or cell-based strategies. Angiogenic growth factor (e.g., vascular endothelial growth factor (VEGF), fibroblast growth factors (FGFs), and hepatocyte growth factor) therapy has been tested clinically since more than 5 years. But the overall benefit for PVD patients has been disappointing[11].

Consequently, exploring new strategies for revascularisation of ischaemic limbs is of major importance.

What are stem cells?

Stem cells are defined as a cell population capable of self-renewal, proliferation and differentiation. They serve as a repair system for the body.

Stem cells are classified into two different types during the development of the organism: embryonic stem cells and adult stem cells (ASCs).

The use of adult stem cells in research and therapy is not as controversial as embryonic stem cells, because the production of ASC does not require the destruction of an embryo. Additionally, because in some instances ASC can be obtained from the intended recipient, (an autograft) the risk of rejection is essentially non-existent in these situations.

Where are adult stem cells found, and what do they normally do?

Adult stem cells (ASCs) have been identified in many organs and tissues, including brain, bone marrow, peripheral blood, blood vessels, skeletal muscle, skin, teeth, heart, gut, liver, ovarian epithelium, and testis. In HYPERLINK "http://en.wikipedia.org/wiki/Adult" \o "Adult"adult organisms, stem cells and HYPERLINK "http://en.wikipedia.org/wiki/Progenitor_cell" \o "Progenitor cell"progenitor cells act as a repair system for the body, replenishing specialised cells, but also maintain the normal turnover of regenerative organs, such as blood, skin or intestinal tissues. They are thought to reside in a specific area of each tissue (called a "stem cell niche"). In many tissues, current evidence suggests that some types of stem cells are pericytes, cells that compose the outermost layer of small blood vessels. Stem cells may remain quiescent (non-dividing) for long periods of time until they are activated by a normal need for more cells to maintain tissues, or by disease or tissue injury.

The concept of stem cell based revascularisation emerged in 1997, when Isner's group described circulating cells in adults called endothelial progenitor cells (EPC) with the capacity to differentiate into endothelial cells (EC) and incorporate into new vessels in ischaemic tissue[12]. Since then, the number of studies reporting on stem cell related revascularisation has exponentially increased. Bone marrow (BM) derived stem cells have been identified as a potential new therapeutic target. Most adult stem cells are lineage-restricted (multipotent) and are generally referred to by their tissue origin for example: mesenchymal stem cell, adipose-derived stem cell, endothelial stem cell etc[13, 14].

In the 1950s, researchers discovered that the bone marrow contains at least two kinds of stem cells. One population, called hematopoietic stem cells, forms all the different types of blood cells in the body. A second population, called bone marrow stromal stem cells (also called mesenchymal stem cells, or skeletal stem cells by some), were discovered a few years later. These non-hematopoietic stem cells make up a small proportion of the stromal cell population in the bone marrow, and can generate bone, cartilage, fat, cells that support the formation of blood, and fibrous connective tissue.

Adult stem cell treatments have been successfully used for many years to treat leukaemia and related bone/blood cancers through bone marrow transplant[15].

Relationship between neoangiogenesis and cell population.

Neoangiogenesis:

Three concepts of vascular growth have been described to date—angiogenesis, vasculogenesis, and arteriogenesis (collateral artery growth)—which represent different aspects of an integrated process. Stimulation of arteriogenesis seems clinically most relevant and has most recently been attempted using autologous bone marrow transplantation with some beneficial results, although the mechanism of action is not completely understood.

Cell population:

Hematopoietic stem cells may be CD34+ AC133+ or CD34- AC133+ or CD34+ AC133-. Vascular development is regulated by growth factors and their receptors such as vascular endothelial growth factor (VEGF) and VEGF tyrosine kinase receptors such as VEGFR-1 (flt-1) or VEGFR-2 (KDR or flk-1). Other growth factors such as angiopoietin-1 that bind a tyrosine kinase receptor Tie-2 may be involved in completing the vascular architecture by assembling pericytes and smooth muscle cells around endothelial cells[16].

Marrow or peripheral blood CD34+ hematopoietic stem cells express VEGFR and Tie.12 When cultured ex-vivo in fibronectin-coated flasks with VEGF, CD34+ AC133+ cells differentiate into endothelial cells by morphology, acetylated low-density lipoprotein incorporation, nitric oxide release, Von Willebrand factor expression, and lectin binding[17].

The unfractionated mixture of hematopoietic mononuclear cells includes more differentiated cells that are thought to provide angiogenic cytokines as well as stem cells that become incorporated into collateral vessels by a process of neoangiogenesis. In clinical trials, Tateishi-Yuyama et al.[18] injected autologous bone marrow mononuclear cells into patients with ischaemic PVD. Patients were selected for chronic ischaemic extremity pain or non-healing ischaemic ulcers or both and a resting blood pressure ankle-brachial index less than 0.6. Bone marrow cells were collected under general anaesthesia from the posterior superior iliac crest and with a 26-gauge needle injected into the gastrocnemius muscle of the ischaemic leg in multiple sites divided by a 3x3 cm grid. Significant improvement in the ABI, trans-cutaneous oxygen pressure, and pain-free walking occurred following treatment[18].

Several independent clinical studies have reported beneficial effects of the administration of bone marrow mononuclear cells (BM-MNC), Granulocyte Colony Stimulating Factor (G-CSF) mobilised Peripheral Blood Mononuclear Cells (PB-MNC), G-CSF-mobilised PB-MNC after ex vivo culturing, G-CSF mobilised CD34+ cells, and G-CSF mobilised CD133+ cells in patients with CLI. However, no direct comparisons have been performed and it is still unclear which cell types or subpopulations provide the best treatment results. The progenitor cells specifically involved in vascular repair and neovascularisation were initially thought to originate from the CD34+ hematopoietic progenitor cell population, analogous to the common hemangioblast precursor in embryonic development[19, 20].

Consistently, in the Therapeutic Angiogenesis using Cell Transplantation (TACT) study, legs that were injected with PB-MNC, containing approximately 500-fold less CD34+ cells than BM-MNC, showed much smaller increases in collateral perfusion as compared with BM-MNC-injected legs.[18,21] Furthermore, Saigawa et al demonstrated a correlation between the number of implanted CD34+ cells and the efficacy of bone marrow implantation[21].

However, several studies suggest that CD34- cell populations also play an important role in the beneficial effects of BM cell therapy. Asahara et al already showed that CD34- cells, added to CD34+ cells in culture, improved outgrowth of cells with an endothelial phenotype[12]. Co-culture of CD34+ cells with CD34-cells in an in-vitro 3-D matrix model using human microvascular endothelial cells significantly enhanced neovascularisation as compared with CD34+ cells alone[22].Other groups described that non-hematopoietic bone marrow mesenchymal precursor cells and myeloid/monocyte lineage cells (CD14+) can also differentiate into EPC or into cells with EPC characteristics[23-26]. Iba et al compared the angiogenic effects of the same numbers of BM-MNC and PB-MNC (containing 2.4% and 0.02% CD34+ cells, respectively) in a rat hind limb ischaemia model and showed that although there was no incorporation of PB-MNC, the angiogenic effect of PB-MNC was approximately 72% relative to that of BM-MNC[27]. Moreover, Tateno et al showed that there was no significant difference in stimulation of neovascularisation after infusion of PB-MNC and BM-MNC[10].

These data suggest that, apart from incorporation of EPC, EPC supply of angiogenic factors such as vascular endothelial growth factor (VEGF), basic fibroblast growth factor, and angiopoietin-1 plays an important role. This role of the paracrine effects of EPC on vascular growth have also been demonstrated by the group of Schaper[28, 29].

A recent report proposed that implanted cells stimulate muscle cells to produce angiogenic factors, thereby promoting neovascularisation[10]. Yang and co-workers reported a simple and effective therapeutic approach for diabetic limb ischaemia by autologous transplantation of G-CSF -mobilised peripheral blood stem cells[30].

Thus, different cell populations are involved in vascular repair and neovascularisation, and these cells may act via direct incorporation into the endothelial layer and endothelial differentiation, by supply of angiogenic factors, or by a combination of both[31].

The majority of studies on cell therapy for CLI have used whole MNC fractions and at this moment it is unclear whether administration of more selected cell populations or ex-vivo culture toward an endothelial phenotype would be more effective.

Although clinical studies showed promising results from both BM-MNC and G-CSF-mobilized PB-MNC, recent data suggest that functional activity of the G-CSF mobilised cells, as assessed by the migratory response to VEGF and stromal cell-derived factor1, is significantly reduced as compared with non-mobilised cells from the same patient. Also in in-vivo experiments in nude mice with hind limb ischaemia, G-CSF-mobilised EPC show a reduced capacity to augment blood flow recovery and to prevent necrosis as compared with the same EPC without G-CSF stimulation[32].

It is important to note that cell isolation protocols may also have a major impact on the functional activity of BM-derived progenitor cells[33].

Optimal Dosage

It is remarkable that all studies discussed above reportfavourable outcome, despite varying dosages, with an even so varying concentration of $CD34^+$ cells. In the studies involving BM cell administration, amounts of aspiratedBM cell ranging from 80 to 1000 ml, from which theinjected dosage of progenitor cells was retrieved, were reported.In the TACT Study[18] and in the study by Higashi etal.[34] approximately 1.6x 10^9 MNC were obtained from 500ml of BM, whereas Durdu et al.[9] retrieved a 50-fold of MNCfrom the same amount of BM (101x10^9 MNC from 653 mlof BM). Bartsch et al.[35]separated a 2.5 times smaller amountof MNC from the same amount of BM (0.1x10^9 MNC from80 ml of BM). The fraction of $CD34^+$ cells in the isolatedMNC population varies from 0.6% in the study by Kajiguchiet al.[36] to 2.4% in the TACT study[18].

Clinical Evaluation

Currently used measures for clinical evaluation, such as ankle-brachial pressure index, are subject to factors other than improvements in perfusion alone. In accordance with the Trans-Atlantic Inter-Society Consensus for the Management of Peripheral Arterial Disease (TASC-II) recommendations, future trials should ideally combine multiple measures for clinical improvement and quantification of the arterial flow to evaluate treatment success, which include ankle pressure, toe pressures,

TcPO2, microcirculation investigation methods like laser Doppler fluxometry, and anatomic imaging[1].

In addition, questionnaires addressing pain experience, pain "magnitude" (pain intensity, emotion, cognitive-evaluative, and sensitivity) and pain at rest (on a visual analogue scale), as well as quality of life questionnaires will provide patient-based parameters for the clinical effects of therapy.

Ulcer status should be assessed by measurement of the cumulative total ulcer area, with ulcer healing defined as healing of all ulcers of the treated leg. Limb status can be assessed using the criteria of Rutherford[37].

Contrast-enhanced high spatial resolution magnetic resonance angiography is a reproducible and robust modality for assessment and quantification of new vessel formation, detecting different sizes of collateral vessels, and determination of (changes in) tissue perfusion.

However, Choksy and Chan[38] pointed out that a major scientific weakness in angiogenesis research lies in the assessment of vascular growth.

Avenues to explore?

- How do adult stem cells evolve during development and how are they maintained in the adult? Are they "leftover" embryonic stem cells, or do they arise in some other way?
- If the beneficial effect of adult stem cell transplantation is a trophic effect, what are the mechanisms?
- What are the factors that control adult stem cell proliferation and differentiation?
- What are the factors that stimulate stem cells to relocate to sites of injury or damage, and how can this process be enhanced for better healing in PVD?
- Why do stem cells remain in an undifferentiated state when all the cells around them have differentiated? What are the characteristics of their "niche" that controls their behaviour?
- How can assessment of neo-angiogenesis be improved?

Conclusion

Clearly, stem cell safety must be scrutinised and assessed throughout the entire treatment or research process. Guidelines and strategies must also be developed to ensure that every aspect of stem cell use - from identification and isolation of stem cells to stem cell transplant - is stringently coordinated.

Although several clinical studies show promising results, larger randomised, blinded, placebo-controlled trials are needed to provide definite proof of the clinical effects of adult stem cell therapy in these patients. In addition, questions regarding the cell population(s) to be used, optimal dose, and routes of administration will have to be addressed. If doctors and scientists can establish safe protocols for stem cell use, everyone can benefit from the full potential of the remarkable and possibly life-saving stem cell therapies.

Competing Interests
None declared
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Review Article

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Irritable Bowel Syndrome (IBS) At a Glance

Rakesh Kumar Jha, Yanli Zou, Jin Li and Bing Xia and Bing Xia

ABSTRACT

Irritable bowel syndrome (IBS) is a common disorder characterized by abdominal pain and altered bowel habit. Hence, IBS is associated with a significantly impaired health-related quality of life (HRQOL) and reduced work productivity around the world. The incidence of IBS is rising dramatically worldwide. Currently, 7 – 10 % of people have IBS worldwide and it is 1.5 times more prevalent in younger women than in men. Much research has been undertaken during the past several decades, which has lead to deep understanding about IBS, particularly the pathogenesis and management. This review summarizes the epidemiology, underlying pathophysiology, diagnosis and treatment about IBS that has been published in recent years. We hope this review can help to provide some reference in clinical practice for physicians in the management of IBS.

KEYWORDS

IBS, Spastic Bowel syndrome, Nervous Bowel, Irritable colon, Splenic Flexure Syndrome, Functional Bowel Disease

Introduction :

Irritable bowel syndrome (IBS) is a common disorder characterized by abdominal pain and altered bowel habit for at least three months.[1]

IBS is further defined depending on the predominant bowel symptom: IBS with constipation (IBS-C) or IBS with diarrhoea (IBS-D). Those not classified as either IBS-C or IBS-D are considered as mixed IBS (IBS-M). Alternating IBS (IBS-A) defines patients whose bowel habits oscillate from diarrhoea to constipation and vice versa.

Synonyms: Spastic Bowel syndrome, Nervous Bowel, Irritable colon, mucous colitis, Splenic Flexure Syndrome, Functional Bowel Disease.[2]

Epidemiology:

IBS is a prevalent and expensive condition that is associated with a significantly impaired health-related quality of life (HRQOL) and reduced work productivity. IBS care consumes over $ 20 billion in both direct and indirect expenditures. Moreover, patients with IBS consume over 50% more health care resources than matched controls without IBS.[1]Based on strict criteria, 7 – 10 % of people have IBS worldwide. Community-based data indicate that diarrhoea-predominant IBS (IBS-D) and mixed IBS (IBS-M) subtypes are more prevalent than constipation-predominant IBS (IBS-C), and that switching among subtype groups may occur. IBS is 1.5 times more common in women than in men, is more common in lower socioeconomic groups, and is more commonly diagnosed in patients younger than 50 years of age. Prevalence estimates of IBS range from 1 % to more than 20% in North America(7%).[1] In Asia the prevalence is about 5%.[3,4,5]Recently, a School-Based Study in china reported the prevalence of IBS in adolescents and children was 13.25% and the ratio of boys to girls was 1:1.8.[6] Most patient with IBS in India are middle-aged men (mean age 39.4 years).[7]

Underlying pathophysiology:

Given the lack of definitive organic markers for IBS, the absence of a consolidated hypothesis regarding its underlying pathophysiology is not surprising. Nevertheless, important advances in research made during the past 50 years have brought us closer than ever to understanding the numerous existing aetiological factors involved in this multifaceted disorder, including environmental factors, genetic factors, previous infection, food intolerance, and abnormal serotonergic signaling in the GI tract.

Environmental factors :

The biopsychosocial model proposed by Engel[8] takes into account the interplay between biologic, psychological, and social factors. This model proposes that there is an underlying biologic predisposition for IBS that may be acted on by environmental factors and psychological stressors, which contribute to disease development, the patient's perception of illness, and impact on treatment outcomes. Different studies have shown that stress can result in release of stress-related hormones that affect colonic sensorimotor function (eg,

corticotropin-releasing factor [CRF] and inflammatory mediators [eg, interleukin (IL)-1]), leading to inflammation and altering GI motility and sensation.

Genetics factors :

Twin studies have shown that IBS is twice as prevalent in monozygotic twins than in dizygotic twins.[9,10,11] IBS may be associated with selected gene polymorphisms, including those in IL-10, G-protein GNb3, alpha adrenoceptor, and serotonin reuptake transporter (SERT).

Post-infectious IBS (PI-IBS):

Culture positive gastroenteritis is a very strong risk factor for IBS. Different prospective studies show IBS symptoms developed in 7% to 32% of patients after they recovered from bacterial gastroenteritis.[12,13,14] Specific risk factors for the development of PI-IBS have been identified, including younger age, female sex, presence of severe infectious gastroenteritis for a prolonged period, use of antibiotics to treat this infection, and presence of concomitant psychological disorders (eg, anxiety).[12,13,15,16]

Small Intestinal bacterial overgrowth

Pimentel and colleagues[17,18] have shown that, when measured by the lactose hydrogen breath test (LHBT), small intestinal bacterial overgrowth (SIBO) has been detected in 78% to 84% of patients with IBS. Hence, a higher than usual population of bacteria in the small intestine has been proposed as a potential aetiological factor in IBS. While another study involving a review for the presence of gastrointestinal-related symptoms (including IBS) has shown that asensitivity of the LHBT for SIBO has been shown to be as low as 16.7%, and specificity approximately 70% and the test alone for small intestinal bacterial overgrowth were poor. Hence, combination with scintigraphy resulted in 100% specificity to assess the treatment responce, because double peaks in serial breath hydrogen concentrations may occur as a result of lactulose fermentation by cecal bacteria. [19,20]

Food intolerance :

Approximately 60% of IBS patients believe and different studies show that allergy to certain foods could trigger IBS symptoms. Recent research involving exclusion of foods patients had immunoglobulin (Ig) G antibodies, which are associated with a more delayed response after antigen exposure than IgE antibodies, resulted in significantly better symptom improvement than in patients in the non-exclusion group.[21]

Serotonin signaling in Gastrointestinal (GI) tract:

Normal gut physiology is predicated to be an interaction between the GI musculature and the autonomic nervous system (ANS), and central nervous system (CNS) by the neurotransmitter serotonin (5-hydroxytryptamine [5-HT]) . Impairment in this interaction affects GI motility, secretion, and visceral sensitivity leading to the symptoms associated with IBS .[22]

Preliminary steps toward making a positive diagnosis of IBS:

A careful history and physical examination are frequently helpful in establishing the diagnosis. A variety of criteria have been developed to identify a combination of symptoms to diagnose IBS. Different guidelines from different studies help in making a positive diagnosis of IBS based primarily on the pattern and nature of symptoms, without the need for excessive laboratory testing. In 1978, Manning and colleagues [23,24] proposed diagnostic criteria for IBS that were found to have a reasonable sensitivity of 78% and a specificity of 72%.[1] In 1984, Kruis and colleagues developed another diagnostic criteria with a high sensitivity of 77% and a specificity 89%. Likewise, in 1990 Rome I [25] criteria came with a sensitivity of 71% and specificity of 85%. RomeII(1999)[26] and Rome III(2006)[27] have not been evaluated yet. None of the symptom based diagnostic criteria have been evaluated and ideal reliability found.[1]

Summary of diagnostic criteria used to define IBS:[1]

In 1978, Manning defined IBS as a collection of symptoms, given below, but did not describe their duration. The number of symptoms that need to be present to diagnose IBS was also not reported in the paper, but a threshold of three positive is the most commonly used:

a) Abdominal pain relieved by defecation
b) More frequent stools with onset of pain
c) Looser stools with onset of pain
d) Mucus per rectum
e) Feeling of incomplete emptying
f) Patient-reported visible abdominal distension

Kruis in 1984, defined IBS by a logistic regression model that describes the probability of IBS. Symptoms need to be present for more than two years. Symptoms are as follows:

a) Abdominal pain, flatulence, or bowel irregularity
b) Description of character and severity of abdominal pain
c) Alternating constipation and diarrhea

Signs that exclude IBS (each determined by the physician) :

a) Abnormal physical findings and/or history pathognomonic for any diagnosis other than IBS
b) Erythrocyte sedimentation rate >20 mm/2 h
c) Leukocytosis >10,000/cc
d) Anaemia (Hemoglobin < 12 for women or < 14 for men)
e) Impression, the physician could perform a PR and see blood or the patient may report it.

Again in 1990, Rome I defined IBS as abdominal pain or discomfort relieved with defecation, or associated with a change in stool frequency or consistency, PLUS two or more of the

following symptoms on at least 25% of occasions or days for three months:

a) Altered stool frequency
b) Altered stool form
c) Altered stool passage
d) Passage of mucus
e) Bloating or distension

Rome II, in 1999, redefined the criteria as abdominal discomfort or pain that has two of three features for 12 weeks (need not be consecutive) in the last one year.

a) Relieved with defecation
b) Onset associated with a change in frequency of stool
c) Onset associated with a change in form of stool

Recently , Rome III (2006) defined IBS as recurrent abdominal pain or discomfort three days per month in the last three months associated with two or more of:

a) Improvement with defecation
b) Onset associated with a change in frequency of stool
c) Onset associated with a change in form of stool

The role of routine diagnostic investigation in patients with IBS:

Routine diagnostic investigation is based on the age of the patient, family history of selected organic diseases including colorectal cancer, inflammatory bowel disease(IBD), coeliac sprue and the presence of 'alarm' features(table1), such as rectal bleeding, weight loss, iron deficiency anaemia and nocturnal symptoms.[1] In patient with typical IBS symptoms and no alarm features, routine diagnostic investigation (complete blood count, serum chemistry, thyroid function tests, stool for ova and parasites and abdominal imaging) is not recommended[1] because of a low likelihood of uncovering organic disease.

Table-1 Lists of alarm features:

Rectal bleeding
Weight loss
Iron deficiency anaemia
Nocturnal symptoms: abdominal pain
family history of of selected organic diseases: colorectalcancer, Inflammatory Bowel Disease(IBD), celiac sprue

Summary of diagnostic investigation in patient with IBS : [1,2]

Diagnostic Investigations :

Routine serologic screening for coeliac sprue for patients with IBS-D and IBS-M.

Lactose Breath test done in lactose maldigestion despite dietary modification.

Colonoscopic Imaging done in IBS patient (>50 yrs age) with alarm feature to rule out organic diseases and screening of colorectal cancer.

Colonoscopy with random biopsies taken in IBS-D to rule out microscopic colitis.

Management of IBS:

The goal of IBS management is to provide relief of symptoms and improve overall well-being.[28] Most studies use a combination therapy including patient education and psychological therapies, diet and fibre therapy along with different types of new emerging pharmacological therapies.

Patient education and psychological therapies:

The majority of patients with IBS have anxiety, depression and features of somatization. Psychological therapies, including cognitive behavioral therapy, dynamic psychotherapy, hypnotherapy[1] shed new light on the management of patients with IBS. The outcome of psychological therapies is improved when delivered by a trained professional (physician, occupational therapist, nurse).[29] A study by Guthrie[30] showed that psychological therapy is feasible and effective in two thirds of patients with IBS who do not respond to standard medical treatment.

Role of diet in IBS:

The concept of food intolerance and the consequent elimination of certain foods from the diet benefit symptoms of IBS. However, there is no sufficient evidence to support this.[1]

Therapeutic effect of dietary fibre, bulking agents and laxatives: The quality of evidence supporting the recommended use of dietary fibre or bulking agents to regularize bowel function is poor.[31] Ispaghula husk (Psyllium hydrophilic mucilloid) and calcium polycarbophil are moderately effective and can be given a conditional recommendation because of the weakest type of evidence.[1] Polyethylene glycol(PEG) laxative has a role in improving stool frequency but no effect on abdominal pain. Different clinical studies and expert opinion suggest that increased fibre intake may cause bloating, abdominal distension and flatulence.[32] So gradual adjustment of dose is advised for the use of these agents.

Therapeutic effect of antispasmodic agents including peppermint oil:

Certain antispasmodics (hyoscine, cimetropium,and pinaverium and peppermint oil) may provide short-term relief of abdominal pain/discomfort in IBS.[33,34]Evidence for safety and tolerability.

Agent	Mechanism of action	Targeted disorder	Clinical status
Crofelemer	CFTR	IBS-D	Phase2b complete
Linaclotide	Guanylate cyclase-c agonist	IBS-C	Phase 3
Arverapamil	Calcium channel blocker	IBS-D	Phase 3
Asimadoline	Kappa opioid agonist	IBS	Phase 2b complete
Mitemcinal	Motilin receptor agonist	IBS-C	Phase 2
Ramosetron	5-HT 3 antagonist	IBS-D	Phase 3
TD-5108	5-HT 4 agonist	IBS-C	Phase 2
DDP-773	5-HT 3 agonist	IBS-C	Phase 2
DDP-225	5-HT 3 antagonist and NE reuptake inhibition	IBS-D	Phase 2
BMS-562086	Corticotropin-releasing hormone antagonist	IBS-D	Phase 2
GW876008	Corticotropin-releasing hormone antagonist	IBS	Phase 2
GTP-010	Glucagon-like peptide	IBS pain	Phase 2
AGN-203818	Alpha receptor agonist	IBS pain	Phase 2
Solabegron	Beta-3 receptor agonist	IBS	Phase 2
Espindolol (AGI-011)	Beta receptor antagonist	IBS (all subtypes)	Phase 2
Dextofisopam	2,3 benzodiazepinereceptors	IBS-D and IBS-M	Phase 3

Table 1: Source: ACG Task Force on IBS(2009)

of these agents are very limited.The commonest adverse effects are dry mouth,dizziness and blurred vision.[34-36]

Therapeutic effect of anti-diarrhoeal medications:

The anti-diarrhoeal agent 'Loperamide' is effective at slowing down colonic transit and improving stool consistency for the treatment of IBS-D with no severe adverse effects.[37] But safety and tolerability datas are still lacking in many studies.

Therapeutic effect of antibiotics:

Many studies show well tolerance of a short term course of non-absorbable antibiotics (Rifaximin) is most effective for improvement of global symptoms in IBS-D and IBS patient with the predominant symptom of bloating and other associated symptoms, such as diarrhoea and abdominal pain.[38-40] While, the Unted States Food and Drug Administration (FDA or USFDA) approved Rifaximin for treatment of traveler's diarrhoea. Other antibiotics, Neomycin[41], Clarithromycin and Metronidazole[42] have been well evaluated for the management of IBS.

Therapeutic effect of Probiotics:

Probiotics have a large number of properties that can benefit IBS. Bifidobacteria is the active agent in probiotic combination therapy.Whereas many studies show Lactobacilli to have no impact on symptoms.[43] But one Korean study concluded that the composite probiotics containing Bifidobacterium bifidum BGN4, Lactobacillus acidophilus AD031, and other species are safe and effective, especially in patients who excrete normal or loose stools.(44) Recently, P Moayyedi and colleague in their systematic review recommend that probiotics appear to be efficacious in IBS patients ,but the magnitude of benefit and the most effective species and strain are uncertain. [45]

Therapeutic effect of the 5HT3 receptor antagonists:

Alosetron (5-HT3 receptor antagonists), with dosage of 0.5 to 1 mg daily, is more effective and the commonest drug used for treatment of patients with IBS-D in spite of serious side effects including constipation and colon ischemia.The balance model of benefits and harms for 'Alosetron' is most encouraging in women who have not responded to conventional therapies.[46,47]

Therapeutic effect of 5-HT4 receptor agonists:

Tegaserod (5-HT4 receptor agonist) is more effective for the treatment of IBS-C mostly in female and IBS-M. The side effects reported among the patient receiving Tegaserod are diarrhoea (commonest), cardiovascular events i.e. myocardial infarction, unstable angina, or stroke.[48,49] Currently Tegaserod is available from FDA through an emergency investigational new drug protocol. Other 5-HT4 agonists (Cisapride,Renzapride) have not demonstrated improvement compared with placebo.[50,51]

Therapeutic effect of the selective C-2 chloride channel activators:

Lubiprostone (selective C-2 chloride channel activator) is effective for relieving symptoms of IBS-C, mostly in women,

and has less frequent side-effects including nausea(8%), diarrhea(6%) and abdominal pain(5%).[52]

Therapeutic effect of antidepressants :

Patients with prominent symptom of abdominal pain in IBS that fails to respond to peripherally acting agents often are considered for treatment with antidepressants (TCAs and SSRIs), however, limited data on safety and tolerability of these agents is shown.[53] Antidepressants have the combined effect of both central and peripheral mechanism in IBS.[54] SSRIs are better tolerated than TCAs and have a prokinetic effect hence work better in IBS-C.[53,55] whereas TCAs are of greater benefit for IBS-D.

Therapeutic effect of herbal therapies and acupuncture:

Unique Chinese herbal mixtures show a benefit in IBS management.[56] Traditional Chinese herbal remedies are routinely used in China to treat the condition, but so far have not been generally accepted by conventional Western medicine.[56,57] Bensoussan and colleague in one randomized, double-blind, placebo-controlled trial concluded that the Chinese herbal formulations appear to offer improvement in symptoms for some patients with IBS.[57] A systematic review of different trials of acupuncture was inconclusive because of heterogenous outcomes.[58,59] Hence further work is needed before any recommendations on acupuncture or herbal mixtures therapy.

Emerging therapies :

The improved understanding of underlying mechanisms in IBS is beneficial for the development of new pharmacological treatment options.

A brief overview of emerging agents in IBS therapy summarized in Table 1 [1]

Conclusion:

IBS is a true medical disorder that has significant impact on those in agony with regard to symptom severity, disability, and impaired quality of life, which exceeds that of most GI disorders. Advances in research over the past several decades have paved the way for an ameliorable understanding of the underlying pathophysiology and standardized symptom-based approaches that can be implemented in making a positive diagnosis and development of innovative treatment options for multiple IBS symptoms. Although many unanswered questions remain, the progress is promising and it has equipped physicians better to efficiently diagnose IBS and choose from a growing armamentarium of treatment options.

Competing Interests
None declared
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Evidence based evaluation of syncope of uncertain origin

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ABSTRACT
Syncope is a common medical condition encountered in clinical practice. The pathophysiology can be complex and at times making a definitive diagnosis can be difficult. It can be associated with high rates of morbidity and mortality. Physicians' approaches to this condition are varied and at times, due to lack of a methodical approach, potential life threatening conditions are missed. Some patients are under investigated while other patients are over investigated. This increases the already high health care costs associated with managing this condition. This article discusses an evidence based methodical approach to diagnosis and treatment of this often complex condition.
KEYWORDS
Syncope, Collapse, Guidelines

Introduction

Syncope is a common condition encountered in acute medical practice. Many patients with syncope are initially labelled as having "collapse query cause". It is defined as transient loss of consciousness (T-LOC) due to transient global cerebral hypoperfusion characterized by rapid onset, short duration, and spontaneous complete recovery[1]. Incidence of syncope is difficult to determine accurately as many cases remain unreported. Some studies quote an overall incidence rate of a first report of syncope to be 6.2 per 100 person-years. Clearly this is age related and the incidence increases dramatically in patients over the age of 70 years[2]. Syncope accounts for 1-6% of hospital admissions and 1% of emergency department (ED) visits per year[3-5]. Hospital episode statistics from NHS hospitals in England reported a total of 119,781 episodes of collapse/syncope for the financial year 2008-09 which is about twice the number of episodes reported in the year 1999-2000. About 80% of patients were admitted and they have an average length of stay of 3 days accounting for over 269,245 bed days during that financial year[6].

Syncope is also associated with significant mortality and morbidity if left untreated. Literature reports a 6-month mortality of 10%, which can go up to 30% if cardiac syncope is untreated[7]. Non-cardiac syncope is associated with a survival rate comparable to people with no syncope[2]. Syncope is also a risk factor for fractures related to falls especially in elderly and can cause significant morbidity in this group[8]. In addition, there are significant health care related costs associated with management of syncope. Cost per diagnosis can vary from over £611 in the UK to €1700 in Italy. Hospitalisation alone accounted for 75% of cost in some studies[9,10]. Diagnosis of this condition can be difficult especially if there is a lack of structured approach. Over the last few years this topic has attracted enormous interest and several studies have been published, aiming at improving the approach to this condition. Standardised syncope pathways improve diagnostic yield and reduced hospital admissions, resource consumption and over all costs[10]. Recently the task force for the diagnosis and management of syncope of the European Society of Cardiology published guidelines for the diagnosis and management of syncope[1]. However, in spite of the available evidence very few hospitals have standardised syncope pathways for the management of this complex condition. Only 18% of EDs have specific guidelines and access to a specialist syncope clinic[11]. This article focuses on evidence based structured evaluation of syncope.

Current practice in the management of syncope

Due to the difficulty in diagnosis and mortality associated with this condition, a cautious approach may be taken by physicians resulting in hospitalisation of majority of patients presenting with syncope.

We recently audited the practice of syncope in our hospital, which is a tertiary centre in the north of Scotland. 58 patients admitted with this condition over a period of a month were included in the audit. It showed an average length of stay (LOS)

of 4.76 days in these patients. Due to a lack of methodical approach and standardised pathway for management of this condition many patients were subjected to several inappropriate inpatient investigations significantly prolonging the LOS and increasing the cost. Only 7 (12%) cardiac events were observed in this group and in retrospect a good methodical approach would have predicted these events. It should be noted that even in the geriatric population, reflex syncope that carries a benign prognosis is more common than cardiac syncope[2].

A systematic approach to the management of syncope (Figures 1 and 2).

Fig 1 – Approach to collapse query cause [1,39]

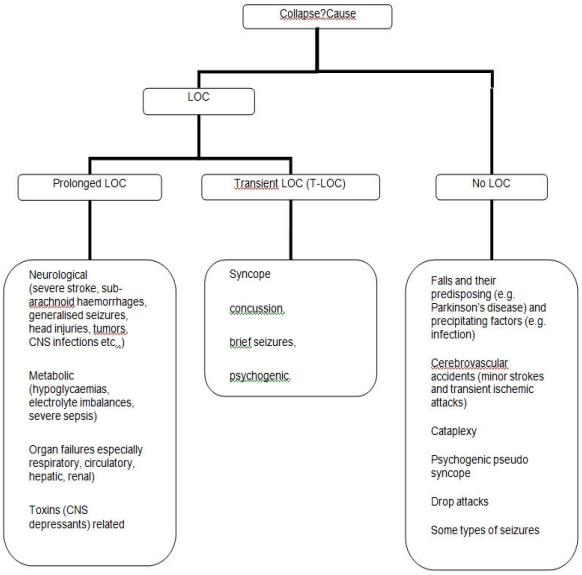
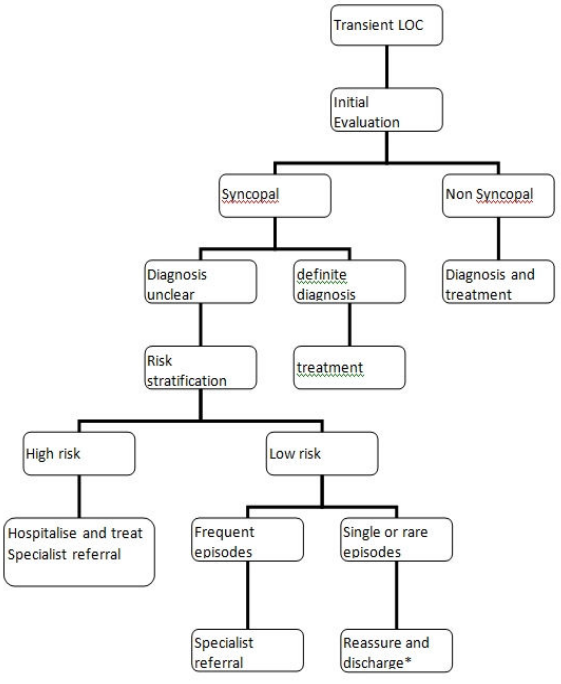


Fig 2 Approach to syncope (adapted from Parry, S.W et al) [40]



*see text for exceptions

The causes of syncope can be broadly divided in to cardiac causes and non-cardiac causes (Table 1). Initial evaluation leads to a diagnosis in less than 50% patients in most instances[4,12-14]. If there is uncertainty about diagnosis then the patient is risk stratified. High-risk patients are hospitalised, evaluated and treated whereas early discharge could be considered in low risk patients.

Aetiology of Syncope[41]

Neurally-mediated (Reflex) Syncope	**Cerebro vascular**
Vasovagal syncope Carotid sinus syncope Situational syncope e.g., Micturition, post prandial, defecation, cough	Vascular steal syndromes
Cardiac	**Orthostatic**
Structural cardio-pulmonary disease Obstructive valvular heart disease/cardiomyopathies Acute aortic dissection atrial myxoma pericardial tamponade pulmonary embolus pulmonary hypertension Cardiac arrhythmias Brady arrhythmias - Sinus node and AV node disease Tachyarrhythmias - Paroxysmal supraventricular and ventricular tachycardia, Inherited syndromes (prolonged QT, Brugada syndrome), Pacemaker or Implantable Cardioverter Defibrillator malfunction	Autonomic failure Primary autonomic failure syndromes e.g. Parkinson's disease, multi system atrophy, pure autonomic failure Secondary autonomic failure syndromes e.g. diabetic neuropathy, amyloid neuropathy Drug and alcohol induced orthostatic syncope Volume depletion

Initial evaluation (Table 2)

History Witness account Physical examination Vitals – heart rate, lying and standing blood pressure, oxygen saturation, blood glucose Standard 12 lead ECG Relevant blood tests (e.g. to rule out metabolic abnormality) Pacemaker check if appropriate

History

Many patients with syncope are initially labelled as having "collapse query cause". Loss of postural tone is termed "collapse". Indeed, the term "collapse query cause" does not give any useful information regarding the underlying condition.

A clear history from the patient and the bystander or witness (if available) is the key to the diagnosis. Firstly, determine if the collapse was associated with loss of consciousness (LOC). LOC can be transient (T-LOC) or prolonged. Categorising "collapse"

is important at this stage as the aetiology and approach to each category is different (Figure 1).

Secondly, establish if the collapse was syncopal. The LOC should be transient (e.g. did the patient regain consciousness in the ambulance, before or on arrival to hospital?), of rapid onset and associated with a spontaneous complete recovery. Also the mechanism should be due to transient global hypoperfusion. T-LOC secondary to other mechanisms such as trauma and brief seizures should be excluded. On occasions syncope could be associated with brief jerking movements mimicking seizures[15]. Also note that a transient ischemic attack (TIA), commonly listed as a differential diagnosis of syncope by physicians, is not a cause of syncope as this is not associated with global cerebral hypoperfusion.

The absence of a coherent history because patient had no recollection of events and there was no witness account available can make this distinction difficult. This is also particularly difficult in the elderly with cognitive impairment. Other useful information includes whether the syncope was associated with postural change. Orthostatic hypotension occurs after standing. If present it will be useful to check drug history (new vasodepressive drugs). Features suggestive of Parkinson's disease or amyloidosis may raise the possibility of autonomic neuropathy. A strong family history of sudden cardiac death may be of relevance. Table 3 summarises the features of neurally mediated and cardiac syncope.

Table 3 Features suggesting neurally mediated and cardiac syncope[42]

Neurally mediated	Cardiac
Preceded by prodrome Related to particular activity - e.g., Micturition, postprandial, prolonged standing, unpleasant situations Associated with nausea and vomiting After exertion	Absence of prodrome, no warning Associated with chest pain, breathlessness, palpitation During exertion or supine History of cardiac disease Family history of sudden cardiac death

Physical examination

The next step is a thorough physical examination. This should include an ABC approach if the patient is very ill and particular attention should be given to exclude immediate life threatening conditions such as pulmonary embolism, acute myocardial infarction, life threatening arrhythmias, acute aortic dissection, seizures *etc*… Recording the vital signs is important as it may give a clue to diagnosis (*e.g.*, acute hypoxia may indicate massive pulmonary embolism). Recording postural blood pressure when lying and during active standing for 3 minutes is useful to exclude orthostatic hypotension[1]. Recording a deficit in blood pressure in both arms may be a useful clinical finding especially if acute aortic dissection is suspected. Thorough cardio respiratory examination may reveal an obvious condition such as cardiac failure or aortic stenosis. Patients should also be examined for potential injuries as a result of syncope.

Standard ECG

A 12 lead ECG should be performed in all patients admitted with syncope. The abnormalities in table 4 would suggest a cardiac aetiology. The QT interval should always be measured, as it is a commonly overlooked abnormality.

Blood tests

Blood tests are usually unhelpful in establishing a diagnosis but can detect metabolic abnormalities such as hypoglycaemia, electrolyte abnormalities and other causes to explain LOC especially when witness account is not available. An acute drop in haemoglobin suggests blood loss. One recent study claims the usefulness of brain natriuretic peptide (BNP) for predicting adverse outcomes in syncope but it is not externally validated yet and it is too early to recommend for routine clinical practice[16].

Pacemaker check

It is not uncommon to see a patient with a pacemaker implanted, admitted to hospital with syncope. In these circumstances, it is essential to rule out a device malfunction although this is not a common cause of syncope. A preliminary and easy test will be interrogating the pacemaker if available. This should pick up any problems with the pacemaker in most instances.

With the above information establishing a diagnosis will be possible in a significant proportion of patients. Further investigations and management should be guided by the underlying diagnosis[1]. However in over half of patients the diagnosis may still be uncertain[12,13,17]. The following section explains the management of unexplained syncope.

Risk stratification in patients with unexplained syncope (Tables 4 and 5)

Table 4 ECG changes in 'high-risk' Syncope[41]

ECG changes favouring bradyarrhythmias

- High degree AV blocks – Mobitz type 2 second degree AV block, complete heart block, trifascicular block (first degree heart block with left bundle branch block (LBBB) or right bundle branch block (RBBB) with axis deviation)
- Bifascicular block (defined as either LBBB or RBBB combined with left anterior fascicular block or left posterior fascicular block) especially if new
- Other intraventricular conduction abnormalities (QRS duration >0.12 s)
- Asymptomatic sinus bradycardia (<50 bpm), sinoatrial block or sinus pause >3 s in the absence of negatively chronotropic medications

ECG changes favouring tachyarrhythmias

- Pre-excited QRS complexes (e.g. WPW syndrome)

- Prolonged QT interval
- Right bundle branch block pattern with ST-elevation in leads V1–V3(Brugada syndrome)
- Negative T waves in right precordial leads, epsilon waves and ventricular late potentials suggestive of arrhythmogenic RVD
- Q waves suggesting myocardial infarction
- Non sustained Ventricular Tachycardias

Table 5 – Clinical features of high-risk syncope[1,18-23]

- History of severe structural heart disease or heart failure, presence of ventricular arrhythmia
- Syncope during exertion or supine
- Absence of prodrome or predisposing or precipitating factors
- Preceded by palpitation or accompanied by chest pain or shortness of breath
- Family history of sudden cardiac death
- Examination suggestive of obstructive valvular heart disease
- Syncope associated with trauma
- Systolic blood pressure less than 90mm Hg
- Hematocrit less than 30% (acute drop in hemoglobin)

When the cause of syncope is uncertain it is essential to risk stratify patients to enable appropriate treatment and further investigation.

Risk stratification tools

There are several scoring systems for risk stratification of syncope. Syncope Evaluation in the Emergency Department Study (SEEDS), Osservatorio Epidemiologico sulla Sincope nel Lazio (OESIL score), Evaluation of Guidelines in SYncope Study (EGSYS score), San Francisco Syncope Rule (SFSR), The Risk stratification Of Syncope in the Emergency department (ROSE) and American College of Emergency Physicians clinical policy are the popular ones and each has its own advantages and disadvantages[1,16,18-23]. Discussing each scoring system is beyond the scope of this article and we shall restrict the discussion to the summary of these risk stratification tools (Table 5). It will be too early to include all the factors mentioned in the ROSE study, as it is not externally validated yet. It could be argued that taking all the risk factors described may increase admission rates but this approach may at least not miss the high-risk patient. This is a developing field and more evidence is likely to be published soon.

High-risk vs. low-risk syncope:

A high-risk syncope patient is the one where a cardiac cause is likely and where the short-term mortality is high due to major cardiovascular events and sudden cardiac death. High-risk syncope is said to be present if **any** of the features in the table 4 or 5 are present.

Management of low-risk syncope

Patients with a single or very infrequent syncope are usually reassured and discharged, as the short-term mortality is low[1,2]. Tilt table test is not usually required where a single or rare episode of neurally mediated syncope is diagnosed clinically. One exceptional circumstance where single rare episodes are investigated further with a tilt table test is when there could be an occupational implication (e.g. aircraft pilot) or if there is a potential risk of physical injury. Patients with recurrent unexplained syncope need to be further investigated (see below).

Management of high-risk syncope / suspected cardiac syncope

High-risk patients usually require hospitalisation and inpatient evaluation. Other high-risk patients who may be considered for admission are vulnerable patients susceptible to serious injuries, for example, elderly patient or a patient with multiple co-morbidities.

Further investigations (Table 6)

Non invasive	Invasive
Echocardiography ECG monitoring Telemetry Holter monitoring External loop recorder* Carotid sinus massage Cognitive testing (in elderly) Ambulatory blood pressure monitoring Tilt table test* Exercise stress test	Implantable loop recorder* Coronary angiography* Electrophysiology*

* Specialist Investigation

Echocardiography

Echocardiography is a relatively inexpensive and non-invasive investigation. It should be performed if there is a clinical suspicion of a significant structural abnormality of heart such as ventricular dysfunction, outflow tract obstruction, obstructive cardiac tumours or thrombus, pericardial effusion *etc*… The yield of this test is low in the absence of clinical suspicion of structural heart disease. However in the presence of a positive cardiac history or an abnormal ECG, one study detected LV dysfunction in 27% of patients and half of these patients had syncope secondary to an arrhythmia. In patients with suspected obstructive valvular disease 40% had significant aortic stenosis as a cause of syncope[24].

ECG monitoring

These tests have utility in identifying arrhythmogenic syncope. If a patient has syncope correlating with a significant rhythm abnormality during the monitoring period with the device, then the cause of syncope is due to the underlying rhythm abnormality. On the other hand, if no rhythm abnormality is recorded during a syncopal attack, then an underlying rhythm problem as a cause of syncope is excluded. Therefore, these tests are meaningful only if there is a symptom-rhythm correlation, which is the working principle of

these devices. In the absence of syncope, during the monitoring period, these tests may pick up other abnormalities that may be relevant. For example, rapid prolonged supra-ventricular tachycardias, ventricular tachycardias, periods of high degree AV blocks (mobitz type 2 or complete heart block) or significant sinus pauses >3seconds (except during sleep, negatively chronotropic therapy and trained athletes), which will require further investigation or treatment.

Telemetry

Telemetry can be used in inpatients. Although the diagnostic yield of this investigation is only 16%, given the high short-term mortality, this test is indicated in the high-risk group [1]. Usually patients are monitored for 24 to 48 hours although there is no agreed standard period for monitoring[25].

Holter monitoring

This involves connecting the patient through cutaneous patch electrodes. It records the ECG activity conventionally over 24-48 hours or at times up to 7 days.

It is particularly useful only in patients who have frequent regular symptoms (≥1 per week). For this reason, the yield of this test can be as low as 1-2% in unselected population[1]. Long inpatient waiting lists in some hospitals can significantly prolong the length of stay and cost. Selecting patients carefully for this test based on risk stratification will reduce costs and waiting lists.

Carotid sinus massage

This simple bedside test is indicated in patients over the age of 40 years with syncope of unexplained origin after initial evaluation. A ventricular pause lasting >3 s and/or a fall in systolic BP of >50mmHg defines carotid sinus hypersensitivity (CSH) syndrome. It is contraindicated in patients with recent cerebrovascular accidents (past 3 months) or with carotid bruit except when a Doppler study has excluded significant stenosis[1].

Cognition test

If an elderly patient had forgotten about the events, in the absence of an obvious cause, it may be useful to test cognition. If cognitive impairment is present, common problems associated with cognitive dysfunction should be considered e.g. falls, orthostatic hypotension.

Other investigations

In spite of the above tests if a cause is not determined, early specialist input is recommended for further investigation and treatment. The following non-invasive and invasive investigations may be appropriate in these circumstances.

An external loop recorder

This is a non-invasive form of electrocardiographic monitoring. The principle is same as that of Holter monitoring. External loop recorders have a loop memory that continuously records and deletes ECG. When activated by the patient, typically after a symptom has occurred, 5 – 15 min of pre-activation ECG is stored and can be retrieved for analysis. Studies have shown that they have increased diagnostic yield compared to Holter[1]. They should be considered in patients who have symptoms on a monthly basis.

A Tilt table test

This is indicated in cases of recurrent unexplained syncope after relevant cardiac causes of syncope are excluded and a negative Carotid sinus massage performed in the absence of contraindications. It is also indicated when it is of clinical value to demonstrate patients susceptibility to reflex syncope and thereby to initiate treatment. Other less common indications are recurrent unexplained falls, differentiate jerking movements secondary to syncope and epilepsy, diagnose psychogenic pseudo syncope and differentiate orthostatic and reflex syncope. Indication of this test in the context of a single unexplained syncope is discussed above.

Ambulatory blood pressure monitoring

This may be useful in patients with unexplained syncope particularly in old age to check if there is an element of autonomic failure and if a single set of orthostatic blood pressure recording is not helpful. In one study, it has been shown that 25% of the elderly patients admitted with falls or syncope had postprandial hypotension especially after breakfast[26]. It may be more readily available than a tilt table test in some centres.

Exercise stress test

This may be useful in a rare entity called exercise induced syncope. Outflow tract obstruction should be excluded by echocardiography before subjecting a patient to this test especially in the presence of relevant signs. However there is no evidence for supporting this test in investigating syncope in general population.

Implantable loop recorders

These are implanted subcutaneously. It needs to be activated either by the patient or a bystander after a syncopal attack. It is indicated in high-risk patients where a comprehensive evaluation did not establish an underlying diagnosis. In the absence of high risk factors, it is also indicated in patients with recurrent unexplained syncope especially if infrequent. Conventionally it is used as a last resort in patients with recurrent unexplained syncope as the initial costs are high. It has been shown in one study to be more cost effective

than the conventional strategy and was more likely to provide a diagnosis in patients with recurrent unexplained syncope[27]. However patients with poor LV function and those at high risk of life-threatening arrhythmias were excluded from this study.

Coronary angiography or CT coronary angiography

This may be helpful in suspected myocardial ischemia or ischemia related arrhythmias. Electrophysiological study may be considered in certain circumstances by cardiologists. When a standardised pathway is used, diagnosis is ascertained in 21% patients on initial evaluation and further 61% patients with early investigations. Only in 18% patients the diagnosis was still uncertain[12]. Other studies have shown similar results[28]. Although these results are from a dedicated syncope unit following a standardised pathway, these could be extrapolated to any unit following these standardised pathways. Further management is dictated by the underlying diagnosis with early specialist input for appropriate treatment.

Treatments

Single or rare episodes of reflex syncope do not require treatment. However, recurrent troublesome reflex syncope may warrant treatment. Treatment modalities are primarily non-pharmacological such as tilt training, physical counter pressure manoeuvres (leg crossing, hand gripping) and ensuring adequate hydration[29]. If refractory to non-pharmacologic measures midodrine (alpha agonist) may be considered in patients with frequent hypotensive symptoms[30,31]. Fludrocortisone may be used in elderly but there is no trial evidence to support this. Betablockers have been presumed to lessen symptoms but are shown to be ineffective in several studies [32]. They may potentially exacerbate bradycardia in carotid sinus syncope and are not recommended in treatment of reflex syncope. Treatment with cardiac pacing in reflex syncope is controversial and may be considered in patients with predominant cardio inhibitory response on carotid sinus massage (in CSH syndrome) or on tilt test (in reflex syncope). It should be noted that cardiac pacing has no effect on the often-dominant vasodepressor component of reflex syncope.

In patients with orthostatic hypotension, non-pharmacologic measures like increased salt and water intake, head up tilt sleeping, physical counter pressure manoeuvres, abdominal binders and compression stockings may help reducing symptoms. Midodrine is an efficient alternative in these circumstances and fludrocortisone also can be used.[33,34] Syncope secondary to cardiac arrhythmias needs treatment if a causal relationship is established. Potential reversible causes such as electrolyte abnormalities and drug induced causes should be excluded. Cardiac pacing is a modality of treatment in significant bradyarrhythmias secondary to sinus node or advanced AV nodal disease such as mobitz type 2 block, complete heart block or tri-fascicular block. Catheter ablation and anti-arrhythmic drug therapy are the main modalities of treatment for tachyarrhythmias. Implantable cardioverter defibrillator may be indicated in patients susceptible to malignant ventricular tachyarrhythmias. Treatment of syncope secondary to structural cardio pulmonary abnormality will need surgical intervention if possible.

Driving and Syncope

Doctors are poor at addressing and documenting this issue[35]. Table 7 gives some useful information from the DVLA website (http://www.dft.gov.uk/dvla/medical/ataglance)[36]. This information is country specific and subject to change.

Table 7 – Driving and Syncope in the UK[36]

Type of Syncope	Group 1 entitlement (car, motorcycle etc.,)	Group 2 entitlement (Large goods vehicle, passenger carrying vehicle)
Simple faint	No restrictions	No restrictions
Unexplained syncope with low risk of recurrence*	Allowed to drive 1 month after the event	Allowed to drive 3 months after the event
Unexplained syncope with high risk of recurrence** and cause identified and treated	Allowed to drive 1 month after the event	Allowed to drive 3 months after the event
Unexplained syncope with high risk of recurrence** and cause not identified	Licence is refused or revoked for 6 months	Licence is refused or revoked for 12 months

*Absent clinical evidence of structural heart disease and normal ECG

** Abnormal ECG, clinical evidence of structural heart disease, syncope causing injury, recurrent syncope

Syncope units

Syncope units aim to evaluate syncope (and related conditions) in dedicated units consisting of generalists and specialists with an interest in syncope. A sufficient number of patients are required to justify such a unit. They are well equipped with facilities for recording ECG, blood pressures, tilt table, autonomic function testing, ambulatory blood pressure monitoring, and invasive and non-invasive electrocardiographic monitoring. It has been shown to be cost effective and reduces health care delivery costs by reducing admission rates, readmission rates and event rates. Examples include the Newcastle model, Manchester model and the Italian model.[12,18,37,38]

Conclusions

The incidence of syncope is increasing in the UK with an aging

population. There is significant cost incurred in the delivery of health care for this condition. The approach to syncope varies widely amongst practising physicians due to lack of a methodical approach. A thorough initial evaluation yields a diagnosis in less than half of the patients. When the cause of syncope remains unexplained after initial evaluation, the patients should be risk stratified. While a patient with a single episode of low risk syncope can be reassured and discharged, those with high-risk features should be hospitalised for further management. Outpatient evaluation could be offered for low risk patients if recurrent. Early specialist input should be sought in high-risk syncope and recurrent unexplained syncope. This standardised approach or pathway will reduce cost by reducing hospitalisation, inappropriate investigations and length of stay.

Key Facts

- Collapse associated with transient loss of consciousness is called syncope if it is due to transient global cerebral hypoperfusion and characterized by rapid onset, short duration, and spontaneous complete recovery
- Standardised syncope pathways improve diagnostic yield and reduce hospital admissions, resource consumption and over all costs
- A thorough initial evaluation yields a diagnosis in less than half of patients. If the cause of syncope is undetermined after initial evaluation, patients should be risk stratified.
- Early discharge should be considered in low risk patients while high-risk patients need urgent evaluation.
- Early specialist referral is recommended in patients with high risk syncope and recurrent unexplained syncope

Future Interests

Syncope had been known for several decades and still remains a complex condition, as the exact mechanisms are poorly understood especially in non-cardiac syncope. Mechanism of syncope in the elderly patients may be different from those of young patients and studies should focus in understanding the mechanics. Further research is needed in risk stratifying syncope. It may enable us to develop more robust care pathways for management of syncope. The role of BNP in investigating and risk stratifying syncope need to be further clarified. In spite of sophisticated tests the cause of syncope in a proportion of patients remain uncertain. Studies should focus on the long-term outcome and management of syncope in this group. The role of implantable loop recorder in the investigation of syncope should be better defined and more studies should focus on when it should be offered in the pathway of management of syncope. Studies are also required to develop effective pharmacotherapies for this condition.

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Case Series

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Paediatric Symptom Falsification ('Munchausen Syndrome by Proxy') – Psychiatric Manifestations

Ciaran Clarke and Norbertas Skokauskas

ABSTRACT

Aims

The aim was to explore the importance and inherent challenges of child abuse presenting with spurious psychiatric manifestations ('Munchausen Syndrome by Proxy') through a case series.

Methods

Three cases of child abuse are described, each presenting with falsification of psychiatric features and symptoms by caregivers. Similarities and differences from classical 'Munchausen Syndrome by Proxy' are explored, and the nosological status of this entity is discussed.

Results

1. Caregiver characteristics resembled those in classical 'Munchausen Syndrome By Proxy'. 2. Cases differed from typical medical presentations in age at presentation, in course, and in prominence of educational consequences. 3. The relationship of the mother with the child's physician was less prominent than some suggest in classical 'MSBP'.

Conclusions

The incidence of Paediatric Symptom Falsification may be higher than generally believed, and psychiatric presentations differ from classical presentations. Reporting to child protection agencies is essential, but this is difficult and tends to be avoided for various reasons. There is a need for more education of mental health and allied professionals about this condition to prevent the suffering of many children. 'MSBP' involves psychological problems in the mother which are very difficult to manage, and which require understanding and compassion, but which should not be a barrier to protecting the child. In so far as it describes a situation and not a disease, 'MSBP' is a disorder only by analogy. Provided this is borne in mind, its difference from other forms of child abuse argues for the retention of a specific descriptive name, so that it can be better detected and prevented.

KEYWORDS

Munchausen Syndrome by Proxy; Paediatric Condition Falsification, Paediatric Symptom Falsification; Psychiatric

Introduction

'Munchausen Syndrome by Proxy' (MSBP) was first described by Meadow in 1977 [1], as a form of child abuse in which the caregiver (generally the mother) causes or simulates illness in the child for psychological gain. Three features are required for diagnosis: 1. The history, signs and symptoms of disease are not credible; 2.The child is receiving unnecessary and harmful, or potentially harmful, medical care; 3. If so, caregivers are instrumental in instigating the evaluations and treatment [2]. As has been emphasized, the motivation of the mother (her psychological needs), is important in distinguishing MSBP from other forms of Paediatric Condition Falsification (PCF) [3]. Initial reports described young children with acute, life-threatening events, such as sleep apnoea, epilepsy, diarrhoea, or bowel obstruction [4,5]. Sudden Infant Death Syndrome (SIDS) has an ominous association with MSBS which has survived controversy [6-9]. MSBP has been reported in children in learning disability settings;[10] whereas there have been few, if any, reports in general child psychiatry. Causal factors for this may be that it is perceived to be less exciting, of a multi-disciplinary nature, and, arguably, due to more humdrum circumstances which surround it. Initial descriptions, indeed, required that the child present with organic symptoms [11]. Psychiatric presentations may be more difficult to detect because of the paucity of objective diagnostic tests, with the consequent greater reliance on the history provided by caregivers. Caregivers who simulate school refusal in their children have been considered not to have MSB [12], (though they do show PCF). The exclusion of psychiatric presentations is noteworthy, because such psychiatric presentations may differ in important ways from the typical, dramatic, presentations seen in very young children. Since psychiatric diagnosis in children is so dependent upon parental history, such cases may also be more difficult to detect, and therefore more common than previously thought. Three cases are described here, which, though typical of MSBP in many ways, differ in their psychiatric symptoms, in their age, and in the prominence of school refusal.

Cases

Case X

This girl was born at 37 weeks gestation by emergency caesarean section because of a slowing of the foetal heart rate. She was the eldest of 4 children. Her father was severely disabled with a chronic degenerative disease. No distress was evident at birth, but she had short stature (3rd centile for height

and weight). At 9 years of age, she presented to a Child & Adolescent Mental Health service with low self-esteem and an eating problem. Her parents attributed this to bullying at school, which, the mother told doctors in a subsequent interview, took place when the child was 7, and was characterised by beatings so severe as to leave her with multiple bruises, and 'a voice which changed following this trauma'.

The girl was found to have an IQ in the borderline range. Her mother reported that she had 'allergies to milk and eggs'. At another interview she said the allergy was to 'shellfish and nuts', and that it had been discovered when she developed an anaphylactic reaction in another country at 18 months of age. The mother also claimed that eczema was diagnosed at that time. Her diet was restricted, and her mother commented that she 'needed to buy special foods', and that 'asthma developed at age 7'. The child was found to be unhappy, but not depressed. There was no evidence of weight loss, but an eating disorder was diagnosed on the basis of maternal reports.

X's brother had been diagnosed as having ADHD, and was prescribed psychostimulants. Aggression and odd behaviour had given rise to suspicion of an autism spectrum disorder. After a number of assessments, no diagnosis was made. Compliance with medication was a problem, the mother claiming that, although she personally administered tablets, he had somehow avoided swallowing them, and adduced this as a reason for their lack of efficacy. The mother later reported that 'he was doing extremely well off all treatment'.

The mother expressed dissatisfaction with the attention and care her daughter had received from one service. However, a letter, written shortly afterwards, gives an insight into her personality, and the relationship which had developed with professionals. She wrote: 'I did not realise I was so overpowering towards you all... I only feel constant pain & hurt at what has happened to our daughter. Let me know if you do not want to see us anymore.'

It was at this point that the ability of the parents to care for their children was questioned, when it was discovered that the mother had been leaving the children unsupervised overnight, so that she could help with a scout camp (with which none of her own children were involved). School authorities were also alarmed that the children were left unsupervised for long periods before school opening, and that they arrived hungry and dishevelled. The mother opposed involvement by social services, and, in the absence of any formal complaint, they were not notified.

Unhappy with the treatment she received from her psychiatrist, the mother sought a second opinion. To this doctor, the mother reported that there had been an increase in frequency of nocturnal enuresis at 7 years. She reported that her daughter was happy at school, but that there were problems at home. She reported that her daughter was 'unable to swallow solid food'; she was 'able to take liquidised food only', and 'she became increasingly anxious about solids'. The mother said she had contacted the Anorexia Nervosa Society 'who advised she had a phobia about food'. The mother reported that when she gradually re-introduced solids, eating recovered and that appetite returned.

At 14 years, X's mother told a paediatrician that her problems started at age 7, when she had been bullied by several children in the class. This doctor found her to have constitutional short stature. No allergies were reported or found. The mother resisted attempts to obtain copies of previous medical records. A neurologist found 'no evidence of regression'. The mother quoted a paediatrician to another professional as having said, 'It would be disastrous for her if she had a period', and she told another paediatrician that the referring doctor had said she was being referred, 'to sort out her psychological problems immediately'. She was referred to a third child psychiatrist, who found low mood, social withdrawal, and 'recent adjustment problems'. She was then referred again to local psychiatric services for follow-up. At this stage, her mother wrote: '... it is now confirmed that her short stature has a psychological basis, and that this started at 6 years of age because of bullying'.

At her mother's insistence, X was seen then by another psychiatrist, 'to discover the psychological cause of her short stature'. No psychopathology was found. Mother declared that she was 'astounded' and demanded to know how the doctor could explain 'the dramatic fall in IQ which had taken place at age 10'.

By the time of referral to social services, the child had been seen by 3 paediatricians, 4 psychiatrists, and numerous other mental health professionals.

Case Y

This boy was born without complication, developing normally up to middle childhood. When he was 3 years old, a younger sister was born. She died at the age of 3 months, and a coroner's verdict of SIDS was returned. His mother experienced a pathological grief reaction.

The family holidayed abroad periodically; such trips were frequently marked by attendance at the A&E department of the local hospital, where the boy presented with sundry somatic complaints. There were occasional brief hospitalisations, but no physical abnormality was ever diagnosed.

The child's mother made several unsubstantiated allegations of bullying at school. His father disputed these, though he did not oppose them. Thorough investigation failed to substantiate any such episode. The father complained to doctors of his powerlessness in the face of his wife's over-protective, domineering approach; he recognised the harmful effect this was having on their son.

Whereas the mother complained that her son had 'loss of interest', 'poor concentration', was 'not eating', and had suffered weight loss; the boy told doctors, 'I'm good at school'; and 'I'm always happy with my friends'.

His mother had a benign brain tumour, which had presented initially with epilepsy when she was in her teens, but was well controlled. During her mid-40's, she presented frequently with pseudo-seizures and other odd neurological symptoms. To each doctor she met, she presented herself has having a 'terminal brain tumour'. She told her neurologist that she was 'distancing herself from her son', as she was 'going to die soon', and 'he would have to be tough enough to get on without her'.

On one occasion, when brought to hospital because of suicidal ideation, Y commented that 'school was good', and that he had 'a few good friends'. He listed a few favourite subjects, and responded appropriately to wishes about the future. The conclusion was that he was euthymic. Notwithstanding his reports to doctors, he was kept at home for prolonged periods because of 'bullying'. The mother claimed that her husband 'can be abusive to the child when angry', though the impression of numerous psychiatrists was that he had a passive, dependent personality. He begged professionals for help in protecting his child, and when a referral to social services was eventually made, he was profuse in his gratitude.

Case Z

This boy's monozygotic twin brother presented to a private psychiatrist at 1 ½ years of age with 'behavioural problems', and a diagnosis of autism was made. No standard observational assessments were performed, nor neurodevelopmental history taken. He was referred to specialist autism services, who found no abnormality following a multi-disciplinary assessment. When the twins were 7 years of age, the mother brought the other twin, Z, to community child psychiatric services with vague concerns regarding 'development' and 'social skills'. By this she meant that he '[had eaten] his food off the floor when he was younger', and that he currently 'behaved in a silly, immature way'. She said that 'teachers complained of disruption', and she, in turn, complained of poor cooperation from the school. Independent contact with the school disclosed no such behavioural concerns. Her husband spent most nights away on business, and the twins slept in her bed. They 'would not settle for hours' and she 'had to sing to get them to sleep'.

His mother had been treated for depression in the past. She reported that she had given up her job in order to look after her children, whom she felt had 'special needs'.

At clinical interview she was extremely reluctant to leave her son, and her prolonged departure from the room was accompanied by exaggerated displays of affection, which clearly embarrassed her son. He, on the other hand, separated easily. On his own, he was initially reserved, but became talkative and happy when discussing his friends, school, interests, etc. There was no evidence of any autistic-type disorder, nor indeed any other psychiatric problem.

At the time of writing, she has refused to accept the assurances of two different services that there was nothing wrong with her son, and she was continuing with attempts to have him re-assessed.

Discussion

All three of these children presented with complaints from mothers, which, while perhaps credible in isolation, became more and more far-fetched when viewed together as a whole. In all cases harm resulted to the child from excessive investigation, social isolation, and absence from school. In all cases, mothers had narcissistic personality problems, and fathers had a subordinate role (one because of chronic illness, another because of a dependent personality). All children had siblings with dubious or unexplained illness. All cases involved many physicians and allied professionals, who became involved in what transpired to be, when they started to communicate with one another, a pattern of simulated illnesses and symptoms. There was a general reluctance to refer to social services, and a delay in their involvement, perhaps because of the absence of acute or marked abuse, and because the caregivers seemed the opposite of the 'typical' negligent, abusive mother. These children were somewhat older than most cases of factitious disorder by proxy, who also differ in presenting with acute, life-threatening events or surgical emergencies. Although probably less lethal, psychiatric presentations may offer more scope for abuse, due to the greater reliance on parental reports in child psychiatry. Unwitting collusion by schools, in part caused by an understandable sensitivity to bullying allegations, may have facilitated presentation to psychiatric services.

Most cases of MSBP have emerged from the U.S. The phrase 'Psychological needs' has been emphasised, and suggests vagueness, an impression strengthened by the initial psychodynamic terms in which these case were couched. It may be a useful term, nonetheless, in that it distinguishes motivations such as revenge, delusions, or poverty, from those in which the perpetrator behaves in this way to, for example, fool doctors or exhibit herself as an ideal parent [13]. Schreier and Libow [14] suggested that a key psychological factor may be an ingratiating relationship which the mother pursues with the child's physician, who tends to be male, isolated, and idealistic. The preponderance of mothers among perpetrators may be due to a satisfaction obtained by deceiving 'authority' or 'power' figures, but this (and any other explanation) has not yet been substantiated. Such manipulation was not present in any of these three cases. This may be due to the central role which the family doctor retains in many other countries, and to the multi-disciplinary nature of health care, particularly mental health services, in other health systems, which precludes the doctor from seeming a 'hero figure'.

'Munchausen Syndrome by Proxy' is clearly not a disease, and can be considered a disorder only by analogy. This, as well as the general tendency in medicine to abandon eponymous diagnostic labels, argues for use of the term 'Paediatric Condition Falsification', preferred by the American Professional Society on the Abuse of Children (APSAC) [15]; or 'Factitious Disorder by Proxy', as preferred by the Diagnostic & Statistical Manual of Mental Disorders (DSM) [16]. Notwithstanding these compelling reasons, the radically different profile of the 'caring mother', in cases like those under discussion, make it essential to distinguish the situation described in this case series from other circumstances in which mothers harm children. The mother's motivation is crucial if the child is to be protected: specific remedies in other circumstances may offer hope of an amelioration, but the rate of recidivism in 'Munchausen Syndrome by Proxy' [17,18], means that these children will require vigilance and protection for as long as they are in contact with their mother.

It is important to consider how these episodes might have been detected earlier. Good history-taking would have revealed the falsehood of an allergy to dairy products in the first case. A higher index of suspicion might have led to greater communication, and better detection, among disparate professionals as in the case of Munchausen Syndrome in adults. Formal channels for reporting concerns without fear of recrimination could be established in hospitals and out-patient settings. Greater institutional support for healthcare workers with concerns, as well as broader awareness among family doctors, nurses, psychologists, and social workers, is a prerequisite. In medical presentations, a bizarre, inconsistent history, a failure to cooperate with attempts to obtain medical records, features of a maternal histrionic or narcissistic personality, and any history of abuse towards other siblings, should raise the alarm. These apply also in the case of psychiatric presentations. In medical presentations, abuse can be detected by observing 'recovery' of the child when removed from the parent's reach. The chronicity and gradual nature of psychiatric symptoms make these cases appear less dramatic, and such a 'test' impractical, but certain other features may help. Unconvincing reports of bullying at school, despite thorough investigation, poor school attendance without an adequate explanation, and an incongruity between maternal reports and the child's mental state, may all be helpful.

Conclusion

Reports of Psychiatric presentations of Paediatric Symptom Falsification are rare, but there are good reasons for suspecting that the true incidence may be higher. Psychiatric presentations are probably not typical of Paediatric Symptom Falsification, and may for this reason be missed. Experience shows that reporting to child protection agencies is essential, but this is difficult and tends to be avoided for various reasons. There is a need for education of mental health and allied professionals in this condition so that much suffering of children can be avoided. Paediatric Symptom Falsification involves psychological problems in the caregiver (generally the mother), which are very difficult to manage. These require understanding and compassion, but should not be a barrier to protecting the child.

The authors declare that they have no conflict of interest.

Competing Interests
None declared
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Case Report

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Physical and psychological effects of the new legal high 'Ivory Wave': a case report

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ABSTRACT

Introduction 'Ivory Wave' is a designer drug that has been popular amongst young people since the ban of mephedrone in the United Kingdom (UK) last April. It is easily available from the Internet where it is advertised as a bath salt. Recently, a spate of Ivory Wave-related hospital admissions were reported around the UK, and this has raised concerns about the effects of the substance. However, limited information is available regarding the physiological and psychological effects of the drug.

Case presentation We report the case of a 26-year-old Caucasian male who presented to Accident and Emergency (A&E) after snorting a large amount of Ivory Wave. He presented with severe agitation, paranoid delusions, and auditory and visual hallucinations. He also complained of breathing difficulty and involuntary movements of his limbs. He was pyrexial and tachycardic but the rest of physical examination was unremarkable. The main laboratory findings included an elevated white cell count, C-Reactive protein (CRP) and creatinine kinase (CK). These markers gradually fell to their normal ranges within a week. The involuntary movements disappeared too. The patient required occasional lorazepam and regular diazepam for his agitation. This improved after approximately a week as the paranoid delusions and hallucinations wore off.

Conclusion The side effects of Ivory Wave include over-stimulation of the cardiovascular system and the nervous system with potential risk to heart and kidneys. Mental state can also be severely disturbed with agitation, paranoid delusions and/or hallucinations.

Introduction

The legal high 'Ivory Wave', also known as 'Ivory Coast', 'Purple Wave' or 'Vanilla Sky', is a designer drug that has become popular among clubbers in the United Kingdom (UK) after mephedrone was banned in April 2010.[1] Ivory Wave is advertised as a relaxing bath salt and has been freely available on the Internet for about £15 a packet (200mg).[2]Three different versions have been on the market, namely, Ivory Wave, Ivory Wave Ultra (also known as Ivory Wave 2), and Ivory Wave 3, although their differences are unknown.[3] Studies have shown that Ivory Wave contains cathinone-derived stimulants and, when snorted in high doses, bring similar effects to those of amphetamine and ecstasy.[4]

Recently, clusters of hospital admissions have been reported around the UK following the use of Ivory Wave. The majority of patients were described to have 'acute paranoid psychosis' with severe agitation, which wore off after a couple of days.[5] However, some patients had more serious physical complications and had to be monitored in the coronary care units for up to 12 hours.[2, 5]

Following the increase in the number of Accident and Emergency (A&E) admissions relating to Ivory Wave, healthcare professionals have expressed their concerns about the harmful effects of the substance. The Department of Health has issued advice on handling the users who may present to health services for help.[6] However, the literature is limited on the physical and psychological effects of the substance at present. Therefore, we report our case here to describe some of the clinical features of Ivory Wave misuse.

Case presentation

A 26-year-old Caucasian male, with a background history of obsessive-compulsive disorder (OCD) and depression, attended an Accident A&E after snorting approximately 700mg of 'Ivory Wave Version 3' in a day. He presented with severe agitation, persecutory delusions, and auditory and visual hallucinations. He stated that 'people' were trying to kill him and his mother with a knife, and he could hear their voices threatening to kill him. He also complained of mild/moderate breathing difficulty and involuntary movements of his arms and feet.

In recent years he had been 'experimenting' with several legal highs, including Ivory Wave, 'Charge' and 'Mojo'. Five weeks prior to this admission he had visited A&E with a similar presentation, but without persecutory delusions, after sniffing an unknown amount of 'Ivory Wave 2'. The hallucinations shortly disappeared and he was discharged home.

Otherwise, he was physically fit and well. He had a long history of severe OCD with borderline psychotic features/social anxiety where he was consistently worried about what other people may do to him. There was no personal or family history of psychosis. He was taking clomipramine (125mg) and olanzapine (12.5mg) for OCD and depression but his compliance had been erratic before the admission.

In the present admission he was very agitated and restless. He had non-goal-directed involuntary movements on both arms and feet: repetitive flexion of his elbows and dorsiflexion of his ankles. The physical examination showed that he was pyrexial with a temperature of 37.9°C and had bilaterally dilated pupils. The respiratory examination was normal with an oxygen saturation of 98% on air. The heart rate was slightly fast at 109 beats per minute (bpm) and blood pressure was 122/82 mmHg. The rest of examination was unremarkable with a normal electrocardiogram (ECG). Laboratory investigations revealed a raised white blood cell (WBC) count of 23.5 ´ 10^9/L and C-reactive protein (CRP) of 332 mg/L. He also had hyponatraemia (Na^+ 126 mmol/L) and elevated creatinine kinase (CK = 662 iu/L). Urine drug screen was negative to amphetamine, opiates, cannabinoids and cocaine.

Initially the patient was admitted to a medical ward and commenced on normal saline with intravenous antibiotics (co-amoxiclav) because of the raised inflammatory markers. The body temperature, CRP, and WBC count fell gradually; the CK level dropped as well. The blood culture came back negative. However, the patient remained agitated, was running around the ward, and experiencing visual and auditory hallucinations. He required PRN lorazepam and regular diazepam.

On day five of admission the patient was deemed medically fit and discharged from the medical ward. However, he was still agitated and confused about what had happened. Concerns were raised regarding his mental state, given his past psychiatric history and current problems. A Mental Health Act assessment was performed and the patient was admitted to a psychiatric unit under Section 2 of Mental Health Act 1983 on the same day.

On admission to the unit the persecutory delusions and hallucinations were still present but to a mild degree. The involuntary movements appeared more like muscle twitches, which occurred less frequently. He was observed on the ward and only PRN lorazepam was prescribed together with his regular medication. He then settled on the ward and did not require any further PRN medication.

After a few days the persecutory delusions and hallucinations wore off. The involuntary movements had stopped but left patches of numbness on his right arm, mainly on his fingers. The area was poorly defined and was not localized to a specific dermatome. The numbness disappeared after a few days without any complications.

The patient remained in the psychiatric unit for two weeks before discharge to the care of the Community Mental Health Team (CMHT).

Discussion

The exact components of Ivory Wave are unclear and thought to be variable.[4, 7] Studies have shown that the main ingredients include MDPV (3,4-methylenedioxypyrovalerone); desoxypipradrol, also known as 2-diphenylmethylpiperidine (2-DPMP); and lidocaine.[7, 8, 9] MDPV and desoxypipradrol are both synthetic stimulants. MDPV was first synthesized in 1969[10] and is found as a white or light tan powder.[4] Desoxypipradrol was initially developed by a pharmaceutical company in the 1950s as a treatment for Attention Deficit Hyperactivity Disorder (ADHD) and narcolepsy, but it was replaced by other related substances.[8] They both act as noradrenaline and dopamine reuptake inhibitors and their effects are thought to be similar to those of amphetamine and cocaine in high doses.[9, 11, 12]

Ivory Wave is known to bring on several desired effects, including increased energy and sociability, increased concentration, and sexual stimulation. There are also many physical and psychological unwanted effects reported including insomnia, severe agitation/anxiety, panic attacks, kidney pain, stomach cramps, tachycardia, hypertension, dilated pupils, headache, tinnitus, skin prickles and numbness, dizziness, and dyspnoea.[4, 13] These effects appear highly dose-dependent[4] and have been based on self-reports made by users on online forums.

In the UK there have been several media reports of hospital admissions related to Ivory Wave. The majority of patients were described to have acute psychotic symptoms, namely paranoid delusions, and auditory and/or visual hallucinations. A few of them had physical complications, requiring cardiac monitoring in ICU.[1, 2, 5] However, no detailed description of their clinical features were available.

In this case report, we have described chronologically the clinical features of a patient, who presented to A&E after taking Ivory Wave. The patient had a similar presentation to what it was described by Paolo Deluca and his colleagues.[4]The patient also experienced involuntary movements in his limbs which has not been reported before in the literature. We have also reported the blood test results: raised inflammatory markers (WCC and CRP) and CK.

The findings from this case, in combination with the limited literature, suggest that the use of 'Ivory Wave' can lead to serious complications including over-stimulation of the cardiovascular and nervous system, hyperthermia, and acute psychosis which can potentially result in severe illnesses or even death. The risk of these effects would be greater if the drug was combined with other recreational drugs or alcohol. In addition, the exact composition and strength of the substance may vary and users may not be completely aware of what chemicals they are consuming. This implies that users of Ivory Wave may to taking potentially dangerous substances with unknown effects.

In April 2010, MDPV was made a Class B drug in the UK together with other cathinone derivatives. In addition the UK Home Office has recently announced a ban on the *import* of desoxypipradrol and any products containing the chemical.[15] The use and availability of Ivory Wave in the UK is being closely monitored and may result in further legislative review. Changes in legislation, more research studies, and health education on Ivory Wave could help the public to realize that, irrespective of the legal status of a drug, recreational use of substances may pose a significant risk to their health.

Competing Interests
None declared
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Education & Training

Management and medical leadership – evaluation of training needs and pathways

Ovais Wadoo, Aadil Jan Shah, Aamer Sajjad and Dave Fearnley

ABSTRACT

Management as a component of training for doctors is well recognised. However, management training is not always a planned component of training and both the access to and content of training are variable throughout the National Health Service (NHS). In this article we emphasise the importance of management and leadership skills for trainees. We discuss alternative routes to attain the relevant competencies and share our experience on the value of formal qualifications versus 'on the job training.' Two authors (OW and AS) are pursuing a Master's degree in management. One author (AJS) took the route of 'on the job training' to attain competencies in management. Another author (DF), who has an MBA, is the Medical Director and Deputy Chief Executive of a mental health trust and organises management training for trainee psychiatrists in collaboration with the Director of Medical Education. The article is written from the perspective of psychiatric trainees although the content is largely generic and is of relevance to all specialties

Introduction

There has been a concerted attempt by government to engage doctors in management and the importance of medical management in psychiatry has never been greater. This commenced with the Griffiths Report on management within the National Health Service[1] (NHS) but had renewed emphasis 25 years later in Lord Darzi's report.[2] The NHS Next Stage Review Final Report '*High Quality Care for All*' sets out a vision for an NHS with quality at its heart. It places a new emphasis on enabling NHS staff to lead and manage the organisations in which they work. It pledges to incorporate leadership and management training into postgraduate medical curriculum. The proposal that management training should be integral to the training of all doctors, including psychiatrists, is not new.[3, 4]

Although management as a component of training for doctors is generally accepted, new consultants are often poorly prepared to deal with the complex organisational issues involved in taking on managerial responsibility.[5, 6] This is partly to do with prior training and partly because learning in this area needs to be based on experience. It is essential that they be adequately prepared to fulfil the responsibilities. Recent psychiatric literature has pointed to the need for psychiatrists to have skills to develop their management and leadership roles and has called for more than 'on the job training.'[7]

Management training for trainees – why?

It is important to recognise that all doctors will have some management responsibilities and it is a requirement of all doctors to fulfil these duties effectively as part of appraisal and revalidation. Medical training has traditionally focused on the clinical skills necessary to be a safe and competent clinician. It is increasingly important that doctors are not only competent clinicians but also have the skills to enable them to function efficiently and effectively within a complex healthcare system.

The aim for the doctor in training is to develop management skills in readiness to take on the responsibilities of a consultant. The management role of consultants is becoming more widely accepted and continually increasing, e.g. this may involve responsibility for teams, people, and the resources they use.[8] Furthermore, the changing role of consultant psychiatrists calls for consultants to have skills to fulfil management and leadership roles.[9] However, while not always recognised, all doctors including trainees are required to achieve some managerial functions from an early stage in their careers. Acquisition and application of leadership and management skills will enable them to contribute to the effective delivery of healthcare for patients.

The fast pace of change within healthcare provision means that it is important that current trainees have the appropriate skills for effective delivery of healthcare.[10] It is clearly no longer acceptable that development of management and leadership competencies is left as optional.

What are the competencies that we need to acquire?

Leadership and management are a key part of a doctor's professional work and the development of appropriate competencies needs to be an integral part of a doctor's training and development. The objectives of the skills of all psychiatrists in training has relied on a number of documents which include Good Medical Practice[11] produced by the General Medical

Council (GMC), Good Psychiatric Practice[12] produced by the Royal College of Psychiatrists, and the Medical Leadership Competency Framework (MLCF).[13] The Royal College of Psychiatrists recognise that psychiatrists will need to acquire a basic level of management skill, and this is reflected in the curriculum which outlines the knowledge and experience to be gained during specialty training.

The intended learning outcomes for trainees are to demonstrate the ability to work effectively with colleagues including team-working, developing appropriate leadership skills, and demonstrating the knowledge, skills and behaviours to manage time and problems effectively.[14] Furthermore the MLCF describes the leadership competencies that doctors need to acquire (Box 1). The MLCF was introduced in response to the recognised need to enhance medical engagement in leadership and was jointly developed by the Academy of Medical Royal Colleges, GMC and the NHS Institute for Innovation and Improvement.[15]

Box 1: Leadership competencies to be gained during speciality training

1. Demonstrating personal qualities
   - Developing self awareness
   - Managing yourself
   - Continuing personal development
   - Acting with integrity
2. Working with others
   - Developing networks
   - Building and maintaining relationships
   - Encouraging contribution
   - Working within teams
3. Managing services
   - Planning
   - Managing resources
   - Managing people
   - Managing performance
4. Improving services
   - Ensuring patient safety
   - Critically evaluating
   - Encouraging improvement and innovation
   - Facilitating transformation
5. Setting direction
   - Identifying the contexts for change
   - Applying knowledge and evidence
   - Making decisions
   - Evaluating impact

How to attain competencies in management and leadership - formal qualifications Versus 'On the job training'

It is important to realise that the acquisition of management competencies is an ongoing experience which starts early in one's career. Any trainee embarking on management training should consider very carefully the alternatives, assess their needs, and determine their own aims and objectives. It is often necessary to choose and tailor an individual training package. We share our experiences of two routes that can lead the trainee to acquire the relevant skills. For the convenience of the reader we will discuss these under the headings of 'formal qualifications' and 'on the job training.'

Formal qualifications (MSc in Health and Social Care Management)

There are many advanced courses on offer, leading to a management qualification, usually lasting several years. Some of these courses are MBA (Health Executive), MSc in Health and Social Care Management, MSc in Health and Public Leadership, Masters degree in Medical Leadership, and Masters in Medical Management.

We (OW and AS) are pursuing an MSc in Health and Social Care Management, through the Faculty of Health and Applied Social Sciences in Liverpool John Moores University, on a part-time basis using our dedicated special interest time (six sessions per month). This degree has been specifically designed to provide all health and social care professionals the opportunity to develop their knowledge and skills to facilitate their role as managers. The programme is structured in such a way as to facilitate the part-time student and enhance their learning experience.

The MSc is modular in structure. In the first year the student will undertake three core management modules. In the second year the student will undertake a research methods module, management module and an individual work-based project. The final year culminates in a dissertation involving a significant piece of research. The student can choose to register for CPDs and there is an option to exit after one year (60 credits) with a Postgraduate Certificate or after two years (120 credits) with a Diploma. University regulations allow students to gain credit for demonstration of relevant prior learning, whether certificated or not. The course format is shown in Box 2.

The ratio of coursework, in-house teaching and self-directed learning varies between modules. Each module usually requires half to one-day attendance of in-house teaching per week. The programme uses a variety of assessment procedures that include a written assignment of 2000–5000 words, video role-play, seminar presentations and work-based projects. Completion of the assignments represents the greatest challenges to time and requires commitment and motivation.

Box 2: Format of the MSc in Healthcare Managementat the Liverpool John Moores University

- Improving service delivery through human resource management (20 credits)
- The economics of World Class commissioning (20 credits)
- Advancing leadership for quality (20 credits)
- Research methods and data analysis (30 credits)
- Strategic management and entrepreneurship (20 credits)
- Individual study or work based learning (10 credits)
- Dissertation (60 credits)

Strengths and weaknesses of an MSc in Health and Social Care Management

Whilst on the course we were able to learn a variety of concepts that were completely new to us, but the main challenge was to put them into practice. As part of the course we had to work on management related projects in our workplaces, so that we could apply the learnt concepts in real time.

We believe that the MSc course has undoubtedly improved our understanding of team working and leadership whilst working on a work-based project. The projects were specific supervised experiences linked to key developmental objectives and enhanced our problem-solving and decision-making, the ability to analyse and reflect on situations, as well as the expected understanding of resource management and change management.

We have been able to analyse personal development needs to enhance personal effectiveness and leadership skills. It helped us to critically evaluate the impact of action learning for organisational development. We have gained an insight into the concepts of commissioning and the role of economic evaluation. We were able to critically appraise the impact of government policies on the commissioning process. Our skills and knowledge of human resource management within a framework of contemporary policy context has increased. We really do feel that it has improved our insight into change management.

We hope that completing a significant research project within an academic setting will further develop our research skills. So far it has been a valuable and stimulating experience that has provided us with both skills and knowledge in management. The teaching and learning approaches for all modules draw into the experiences of the workplace. All core module assessment tasks are linked to the workplace, which is particularly useful.

However the process of developing a dissertation proposal, finding a supervisor, gaining ethical approval and proceeding with the research is time consuming and at times frustrating. The financial cost is a significant consideration but can be partially funded through the study leave budget. Furthermore, there is funding available for some modules through the Strategic Health Authority. As we were using most of our special interest sessions to pursue the degree, we had to put an extra effort to develop additional clinical interests.

'On the job training'- what does that mean?

On the job management training may entail clinical managerial experience (e.g. organising outpatient clinics, developing systems for prioritising clinical work, managing teams, and drawing up on-call rotas), specific skills (e.g. chairing meetings, organising training days, and representation on committees), specific management experience (e.g. participation in service development) and resource management (non-clinical aspects of management such as human resources and finance).

The clinical setting provides many opportunities to gain knowledge, skills, attitudes and behaviours that are identified in the management and leadership curriculum. The diversity of daily clinical practice will enable the acquisition of appropriate skills and trainees need to take advantage of all the formal and informal learning opportunities.

These range from workplace-based 'learning sets'[16] and project based learning. It is the responsibility of the trainers to ensure adequate and appropriate educational opportunities are made available to the trainee. In turn the trainee should be enthusiastic and proactive in identifying their own gaps in knowledge, skills, attitudes and behaviour.

It is important to bear in mind that such training should be supplemented by selected formal courses. Some training schemes offer no organised management training, whilst some provide training as a short and often intense course.[17] A variety of courses have been developed for trainees, both at regional and national level. Trusts, Deaneries, independent organisations, universities and the Royal Colleges run such courses. These courses are normally short, lasting a week or less. The components of 'on the job training' in Merseycare NHS Trust and generic management courses offered by Mersey Deanery are listed in Boxes 3 and 4 respectively.

Box 3: Components of 'on the job training' in Merseycare NHS Trust

- Appropriate involvement of trainees in clinical teams
- Appropriate involvement of trainees in service development
- Shadowing arrangements in placements
- Undertake a management project
- Senior managers in the trust as mentors to trainees
- Action learning sets for trainees
- Trainees developing teaching and supervisory skills with junior colleagues
- Management seminars
- Representation on committees (e.g. school board, local negotiating committee, local education board etc)
- Two-day and three-day residential management training for higher trainees
- Generic management courses run by the deanery
- Personal development and management courses hosted by the College

Box 4: Generic management courses offered by Mersey Deanery

- Management and leadership
- Mentoring, appraisal, interview skills
- Effective team-working
- Managing change
- Time management
- Preventing and managing stress
- Negotiating skills
- Managing meetings

Strengths and weaknesses of 'on the job training'

'On the job training' may vary from one placement to another depending on the availability of resources and mentors. Achieving 'on the job' management experience depends on the enthusiasm of the senior trainee. It is more personalised and individually driven. Higher training posts do provide exposure to management issues, but do not necessarily provide in-depth management experience.

It is easier to gain experience in clinical management skills but it can be difficult to achieve specific management experience including resource management. Trainers with formal management roles do not routinely engage trainees in this aspect of their work, and similar experiences have been expressed in other training schemes.[18] Even if there are opportunities available to get involved in service development and other operational issues, one may struggle to commit any time.

Furthermore the loss of protected training (reduction of special interest to only two sessions for specialist trainees) to service provision has impacted on training.[19] The formal courses are confined to development of skills such as leadership, teamwork and management of conflict. Residential management courses are available, providing one week or less of intensive training. The amount of management theory and techniques that can be learned on such courses is limited. The limited theoretical training in management means that trainees are unlikely to be adequately prepared for the extensive management role.

Which one is for you?

Managing services and leading organisations is not for everyone. Nevertheless, the medical role has inherent elements of leading and managing patient care and therefore doctors are often involved in service improvement and development. Perhaps the key issue is whether qualifications alone are sufficient to equip a doctor to be an effective manager, or is experience simply enough? It is important to remember that management qualifications tend to involve real-time application of concepts (which may be the same as on the job training) but at the same time gives a solid knowledge base. Furthermore, limited experience (involvement in local management) is unlikely to be sufficient and therefore experience should ideally be supplemented by selected formal courses.

However, even with the most impressive portfolio of formal training, trainees will nevertheless have to demonstrate competence in leadership and management in their work. All trainees are adult learners who ought to take responsibility for their own education. Which route the trainee wants to take depends not only on what the trainee intends to do in his future role but also on where he trains and what resources are available. Training needs will differ depending on past experience, competence, and capabilities. It is important for the trainees to recognise that the training needs will differ depending on their interests and the type of consultant post to which they aspire.

Formal qualifications would suit those with a well-developed interest in management and a desire to make this a significant part of their ongoing career. If the trainee intends to take a lead management role it may be necessary and useful to complete a Master's degree. It will provide the trainee with both skills and knowledge in management and a well-recognised and formal degree in management. Having established that, it is worthwhile appraising the variety of courses available, as they vary significantly. It is helpful to determine the course's content, assess its relevance, and establish how much in-house teaching and self-directed learning is expected. For those who want to acquire management skills for better day-to-day functioning in their job, it is useful to analyse their personal development needs and complete relevant modules according to these needs. This could be attained through 'on the job training' if resources can be identified and secured. A final point to bear in mind is the Royal Colleges' direct contribution to developing management and leadership in trainees. For example the Royal College of Psychiatrists promotes engagement of doctors in management and has a dedicated Special Interest Group for management.

Competing Interests
None declared
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Vertigo-diagnosis and management in the primary care

Daljit Singh Sura and Stephen Newell

General Information

1. Vertigo is the hallucination of movement of the environment around the patient, or of the patient with respect to the environment [1]. It is not a fear of heights.
2. Vertigo is not necessarily the same as dizziness
3. Dizziness is a non-specific term which can be categorised into four different subtypes according to symptoms described by the patients:
   a. Vertigo
   b. Presyncope: the sense of impending faint, caused by a reduced total cerebral perfusion
   c. Light-headedness: often described as giddiness or wooziness [2]
   d. Disequilibrium: a feeling of unsteadiness or imbalance when standing [2]

Classification

Vertigo may be classified as:

1. Central - due to a brainstem or cerebellar disorder
2. Peripheral - due to disorders of the inner ear or the Vestibulocochlear (VIIIth) cranial nerve

Incidence/Prevalence:

Most patients who complain about dizziness do not have true vertigo:

1. 5 community based studies into dizziness indicated that around 30% of patients were found to have vertigo, rising to 56.4% in an older population [3]
2. A postal questionnaire study which examined 2064 patients, aged 18-65, 7% described true vertigo in the previous year [3]
3. A full time GP can therefore expect between 10-20 patients with vertigo in one year [3]
4. 93% of primary care patients with vertigo have either benign paroxysmal positional vertigo (BPPV), acute vestibular neuronitis, or Ménière's disease [4]. These conditions are highlighted in Table 2

Causes

A wide range of conditions can cause vertigo, and identifying whether deafness or CNS signs are present, can help narrow the differential diagnosis, as shown in *Table 1*.

Symptoms

1. Vertigo may be due to central lesions or peripheral lesions. Vertigo may also be psychogenic or occur in conditions which limit neck movement, such as vertigo caused by cervical spondylosis, or following a "whiplash" flexion-extension injury.
2. It is essential to determine whether the patient has a peripheral or central cause of vertigo [1].

Table 1 Causes of vertigo

Vertigo with deafness	Vertigo without deafness	Vertigo with intracranial signs
Ménière's disease	Vestibular neuronitis	Cerebellopontine angle tumour
Labyrinthitis	Benign positional vertigo	Cerebrovascular disease : TIA / CVA
Labyrinthine trauma	Acute vestibular dysfunction	Vertebro-basilar insufficiency and thromboembolism: - lateral medullary syndrome - subclavian steal syndrome - basilar migraine
Acoustic neuroma	Medication induced vertigo e.g. aminoglycosides	Brain tumour: - e.g. ependymoma or metastasis in the fourth ventricle
Acute cochleo-vestibular dysfunction	Cervical spondylosis	Migraine
Syphilis (rare)	Following flexion-extension injury	Multiple sclerosis
		Aura of epileptic attack – especially temporal lobe epilepsy
		Drugs – e.g. phenytoin, barbiturates
		Syringobulbia

3. Information obtained from the history that can be used to make this distinction includes [1]:
   a. The timing and duration of the vertigo
   b. Provoking or exacerbating factors
   c. Associated symptoms such as
      i. Pain
      ii. Nausea
      iii. Neurological symptoms
      iv. Hearing loss
4. Central vertigo:
   a. The vertigo usually develops gradually
   b. Except in: an acute central vertigo is probably vascular in origin, e.g. CVA
   c. Central lesions usually cause neurological signs in addition to the vertigo
   d. Auditory features tend to be uncommon.
   e. Causes severe imbalance
   f. Nystagmus is purely vertical, horizontal, or torsional and is not inhibited by fixation of eyes onto an object
5. The duration of vertigo episodes and associated auditory symptoms will help to narrow the differential diagnosis [5]. This is illustrated for various pathologies that cause vertigo, in Table 2

Table 2 Timing of symptoms			
Pathology	**Duration Of Episode**	**Associated Auditory Symptoms**	**Peripheral or Central Origin**
Benign Paroxysmal Positional Vertigo	Seconds	No	Peripheral
Vestibular Neuronitis	Days	No	Peripheral
Ménière's Disease	Hours	Yes	Peripheral
Perilymphatic Fistula	Seconds	Yes	Peripheral
Transient Ischemic Attack	Seconds / Hours	No	Central
Vertiginous Migraine	Hours	No	Central
Labyrinthitis	Days	Yes	Peripheral
Stroke	Days	No	Central
Acoustic Neuroma	Months	Yes	Peripheral
Cerebellar Tumour	Months	No	Central
Multiple Sclerosis	Months	No	Central

6. It is important to differentiate vertigo from non-rotatory dizziness (presyncope, disequilibrium, light-headedness). Patients can be asked whether they "felt light headed or felt as if the world was spinning around" during a dizzy spell [3].
7. Important points in the history:
   a. Onset - specific provoking events such as flying or trauma
   b. Duration:
      i. Seconds - Benign positional vertigo
      ii. Hours - Ménière's Disease
      iii. Weeks - Labyrinthitis, Post-head trauma, Vestibular neuronitis
      iv. Years - may be psychogenic
   c. Associated auditory symptoms - rare in primary CNS lesion
   d. Other associated symptoms
      i. Nausea and vomiting in a vestibular cause
      ii. Neurological symptoms such as visual disturbance, dysarthria in a central lesion

Physical/signs

1. Examination of ear drums (Otoscopy/ Pneumatic otoscopy) for:
   a. Vesicles (Ramsay Hunt syndrome)
   b. Cholesteatoma
2. Tuning fork tests for hearing loss – Rinne/Weber tests
3. Cranial nerve examination. Cranial nerves should be examined for signs of :
   a. Nerve palsies
   b. Sensorineural hearing loss
   c. Nystagmus [3]
4. Hennebert's sign [1]
   a. Vertigo or nystagmus caused by pushing on the tragus and external auditory meatus of the affected side
   b. Indicates the presence of a perilymphatic fistula.
5. Gait tests:
   a. Romberg's sign (not particularly useful in the diagnosis of vertigo [1])
   b. Heel-to- toe walking test
   c. Unterberger's stepping test [1] (The patient is asked to walk on the spot with their eyes closed – if the patient rotates to one side they have labyrinth lesion on that side
6. Dix-Hallpike manoeuvre [1]
   a. The most helpful test to perform on patients with vertigo[1]
   b. If rotational nystagmus occurs then the test is considered positive for BPPV. During a positive test, the fast phase of the rotatory nystagmus is toward the affected ear, which is the ear closest to the ground.
7. Head impulse test/head thrust test
   a. Useful in recognizing acute vestibulopathy [6]
8. Caloric tests
   a. Cold or warm water or air is irrigated into the external auditory canal
   b. Not commonly used

Investigations/Testing to consider:

1. Special auditory tests
   a. Audiometry helps establish the diagnosis of Ménière's disease
2. The history is most important and may give a quite good indication of the cause of vertigo. General medical causes such as anaemia, hypotension and hypoglycaemia may present with dizziness, and therefore should be investigated.
3. If features of CNS causes is suspected from the history or examination:
   a. CT/MRI Brain imaging as appropriate

Treatment

1. Treatment should ideally aim at the cause of the vertigo [7]:
   a. Medical management – as described below.
   b. Vestibular rehabilitation exercises – e.g. Cawthorne-Cooksey exercises [5].
      i. These exercises aim to help the patient return to normal activity more quickly.
      ii. Moving the eyes from side to side and up and down while in bed or sitting down - then moving the head, first with your eyes open and then closed
      iii. Other forms use gaze and gait stabilising exercises. Most exercises involve head movement
2. For most patients the main priority is effective control of the symptoms.
   a. For acute attacks, treatments include [5,8]: -
      i. Betahistine hydrochloride 8-16mg upto TDS
      ii. Cinnarizine, 15-30 mg TDS or
      iii. Prochlorperazine should be reserved for rapid relieve of acute symptoms only [8,12] - tablets 5-10 mg or buccal 3mg TDS or injection 12.5 mg IM or 25mg PR suppository - if vomiting
   b. Preventive measures for recurrent attacks include:
      i. Restrict salt and fluid intake - stop smoking and restrict excess coffee or alcohol [9,10]
      ii. Betahistine hydrochloride 16mg regularly TDS seems most effective in Ménière's
      iii. Cinnarizine 15-30 mg TDS
3. Points to consider
      i. Warn patients when drugs may sedate [10].
      ii. Prochlorperazine is less sedating than some other recommended antihistamines, but may cause a dystonic reaction (particularly in children and young women) [11].
      iii. Benzodiazepines are not recommended [9].
4. Recurrent vertigo
      i. The most important first step in the management of recurrent vertigo is to distinguish vertigo from 'dizziness'.
      ii. In attacks of vertigo there is a sense of mobile disequilibrium ("the room spinning") which, if severe, results in uncontrolled staggering in one direction which may be only prevented by grabbing a solid object[10].
5. Epley's manoeuvre
   a. Aims to remove debris from the semicircular canals and deposit it in the utricle where hair cells are not stimulated [1]
   b. Contraindications include [10]:
      i. Severe carotid stenosis
      ii. Unstable heart disease
      iii. Severe neck disease (cervical spondylosis with myelopathy)

iv. Advanced rheumatoid arthritis

Consultation and referral:

1. Refer to secondary care if [10]:
    i. Recurrent separate episodes
    ii. Neurological symptoms e.g. dysphasia, paraesthesiae or weakness
    iii. Associated sensorineural deafness
    iv. If there is an inadequate visualisation of the entire tympanic membrane or an abnormality (e.g. cholesteatoma)
    v. Atypical nystagmus e.g. non-horizontal, persisting for weeks, changing in direction or differing in each eye
    vi. Positive fistula sign: pressure on the tragus reproducing symptoms (suggests endolymphatic fistula
2. If the patient has hearing problems in addition to vertigo then referral should be made to an ENT specialist. Other cases should be referred to a neurologist [10].
3. While awaiting referral:
    i. Consider symptomatic drug treatment for no longer than 1 week because prolonged use may delay vestibular compensation
    ii. It is important that the person stops symptomatic treatment 48 hours before seeing a specialist, as it will interfere with diagnostic tests such as the Dix-Hallpike manoeuvre.
    iii. If the person's symptoms deteriorate, seek specialist advice.

When to consider hospitalization

1. Admit the patient to hospital if they have severe nausea and vomiting, and are unable to tolerate oral fluids [9].
2. Admit or urgently refer the person to a neurologist if they have:
    a. Very sudden onset of vertigo (within seconds) that persists.
    b. Acute vertigo associated with neurological symptoms or signs (e.g. new type of headache - especially occipital, gait disturbance, truncal ataxia, numbness, dysarthria, weakness) which may suggest CVA, TIA, or multiple sclerosis [9].
3. Admit or refer the person as an emergency to an ENT specialist if they have acute deafness without other typical features of Ménière's disease (tinnitus and a sensation of fullness in the ear). Sudden onset unilateral deafness would suggest acute ischaemia of the labyrinth or brainstem, but can also occur with infection or inflammation.
    a. Emergency treatment may restore hearing. The person should be seen within 12 hours of the onset of symptoms [9]
4. The urgency of referral depends on the severity of symptoms (e.g. requirement for intravenous fluids because of excessive vomiting) and the suspected diagnosis [9].

Patient Information

The Ménière's Society www.menieres.org.uk
www.patient.co.uk/doctor/Vertigo.htm

Competing Interests
None declared
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Viewpoint

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Psychiatry in the doldrums: what price happiness?

Francis J Dunne

A lifetime of happiness! No man alive could bear it: it would be hell on ear. *(George Bernard Shaw 1856-1950)*

Guess what? Antidepressants do not work for mild or moderate depression! This amazing 'revelation' seems to surface periodically as a popular item in the media and platform for the experts in living, especially since talking therapies are now considered the panacea for all ills. Despite methodological flaws in the research (as with all studies)[1] and noticeably, with less scrutiny of talking therapy research, this 'fact' is preferentially brought to our attention. That antidepressants have unpleasant side effects and are not always effective we have known all along. When one thinks about it, all drugs have adverse effects. Strange how antidepressants work - they seem to cause unpleasant adverse effects but not beneficial ones! No one doubts that neurotransmitters are involved in pain transmission or are responsible for muscle movement, yet biological pathways are dismissed when 'emotional' or 'psychological factors' are promoted as causing distress. By contrast, talking therapies cure the problem and are considered safe, it seems.

Am I alone in not being surprised? I have never understood how mild depression (whatever that is) becomes moderate, or how normal becomes mild, even with the International Classification of Diseases (ICD 10) and Diagnostic and Statistical Manual of Mental Disorders (DSM IV) to hand. And who has decided there be a minimum duration of two weeks for mild depression? Does it not count if one is suicidal for a week? The corollary of this is seen in another nugget of perceived wisdom masquerading as 'research' which informs us that 14 units and 21 units of alcohol per week are considered the upper limits of safe drinking for women and men, respectively. What if intake exceeds these magical figures? A rigid adherence to the dictum would castigate a woman or man as alcohol-dependent imbibing 15 and 22 units per week. This type of anecdotal research has no scientific meaning because one cannot equate units with a way of life, one's metabolism, stature, weight, and so forth. In the laboratory I can detect mild anaemia from severe anaemia because haemoglobin can be measured, and when to treat is usually quite clear-cut. In mental health studies, as with the alcohol example quoted, the theory is also vague. The usual response from 'researchers' in this field is that rating scales are capable of detecting differences in mood, say, which then determines the 'therapy' one receives. This is a fallacy. For example, in medicine, small variations in haemoglobin do not make the slightest difference to how a patient feels, though such fluctuations are important.

How do you measure tiredness? One can feel tired and have a normal haemoglobin level. In the elderly, for example, abnormal blood indices are often present despite an outwardly well appearance. The anaemia still needs to be treated. Laboratory tests are therefore used to confirm the severity of an illness and are objective, regardless of outward appearances. Treatment is given and the haemoglobin (in this example) returns to normal, without the patient even being aware in many cases. The difference between the above example and a mental health 'condition' is that there is no realistic cut-off point between feeling well and being unwell. Therefore, when to intervene is arbitrary. Am I tired because I'm depressed, or is it the other way round? Do two or more weeks of mild happiness mean I am ill? 'Is there such a thing as moderate happiness?' 'Should we be using mood stabilizing medication or talking therapies if we are mildly or moderately happy?' Absurd. No one speaks of another individual as being mildly or moderately happy. So why should it make sense to talk of someone as mildly or moderately depressed? What next – mildly or moderately normal? Severe conditions require treatment; mild upsets can be managed by simple alterations in lifestyle, and one does not need a medical doctor or an expert in living to tell you so. There is little point in expecting a favourable drug treatment outcome for say, hypertension, if the patient continues to smoke or is grossly overweight. Take the metabolic syndrome of dyslipidaemia, central obesity, hypertension, and insulin resistance: treatment involves removing the causative factors, not prescribing drugs to reduce weight.

I am a kind of paranoiac in reverse. I suspect people of plotting to make me happy.(*J.D. Salinger 1919-2010)*

The norm for most people is to get on with matters in hand and tolerate life's daily grind. Some good days, some bad. A lot depends on your financial status too. Nothing new there. It does not make sense to assume antidepressants will make the slightest difference to an individual's 'ups and downs,' as there is no clinical syndrome to address. Living is not a genetic condition, though alterations in genes affect living. There is the

risk of medicalising every difficulty one faces. Behaviour is often personality-driven and not a symptom of illness, and though personalities vary, one does not speak of a personality illness or personality condition. Even the term personality 'disorder' has come in for much criticism because of the difficulty in defining what is meant by personality.[2] One individual may be overtly aggressive, another too passive, and to embrace all eventualities, there is the term passive-aggressive. No point in being perfectionist because nowadays you may fit the obsessive character description. On reflection though, I would rather the cardiologist, surgeon, airline pilot, concert musician and so forth, err on the side of perfectionism! It does not require much imagination to realize that the real test of a 'condition' is when an individual begins to feel he/she is not functioning at a healthy level because of a pervasive sense of inertia, lassitude, lack of motivation, persistent gloominess and despair, for reasons apparent or not. Most people feel despondent at times, say after bereavement, or losing one's job, and likewise many individuals are more motivated, innovative, and ambitious than others. Some conditions, which seem to have a genetic basis, have stood the test of time, such as bipolar disorder, eating disorders, schizophrenia, borderline personality disorder and obsessive compulsive disorder; all other 'disorders' less so.

That antidepressants often fail to work is nothing new, even for severe depression because there are often too many factors at work. Patients who suffer from severe depression and suicidal ideation would be unlikely to be entering a clinical trial in any event. Furthermore, the theory of a chemical neurotransmitter imbalance is outmoded. It could be that an alteration in receptor sensitivity, either at the presynaptic or postsynaptic site, is the critical factor. Furthermore, it is conceivable that more neurotransmitters are involved than the handful we know of at present. What does the physician do then? Tell patients there is only a 70% chance of getting better with antidepressants and let them get on with it! Anyway, why should antidepressants be any different to other drugs used throughout the entire field of medicine? No drug has a 100% cure rate (save perhaps antibiotics or vaccines for specific infections). When one is well it is easy to be critical, cynical and dismissive. When a patient develops Hodgkin's Lymphoma or any other serious nonsurgical illness and is told there is a 70% chance of survival with medication it is highly likely he/she would optimistically choose the latter. Why should severe depression be any different?

Rating scales cannot be robustly be relied on, at least in psychiatry, as most information is descriptive and there are few instances when a scale can be regarded as having proven validity.3 The Hamilton Rating Scale, a commonly used measure of depression, contains a large number of items relating to sleep and anxiety, and hence sedative antidepressants may seem to be appropriate. It could therefore be argued that the patient is benefiting from a good night's sleep rather than any inherent antidepressant effect of the drug in question. Thus the side effect of the drug now has a therapeutic effect! This is akin to saying antihistamines work only through their sedative effect! Many scale items are poor contributors to the measurement of depression severity and others have poor interrater and retest reliability. Besides, mental and emotional diagnoses are so often ephemeral, and therefore defy 'rateability'. Another example is the Beck Depression Inventory (BDI), often used as a screening tool: because it is a self-report questionnaire, it poses problems in that the person completing it may distort responses. The question therefore is: how does one prove that antidepressants are effective on the basis of flawed clinical trials even when the evidence in clinical practice is obvious?

Meta-analysis is often used as 'proof' that research shows or does not show evidence to support a particular theory. However, meta-analysis itself is not foolproof. The methodology is complex and fraught with difficulty.[4] The sheer volume of material can impress the naïve and the search for negative outcomes, if it suits the preconceived, intended purpose, will be celebrated in the media as 'scientists discover' and so forth. A diligent search of the literature will uncover the sort of results one is looking for, because remember, there are lots of bad trials, no trials are identical, and there is heterogeneity among trial results.

It is clearly very difficult to devise a perfect rating scale, particularly in psychiatry where one is dealing in 'mind matters' and the pathoplasticity of mental disorders. Besides, leaving 'research' aside, the terminology in psychiatry as a whole is vague and interchanges between lay descriptions and 'psychiatric'. Does 'mad' mean psychotic? What is madness anyway? Is neurotic the same as being a worrier or chronically anxious? Can one be neurotic about one thing and not another? Is a teenager worried about exams (assuming he/she is fully prepared of course) normal, anxious, neurotic or unduly concerned? In medicine, matters are clearer by and large: blood pressure is high, low or normal. We are not comparing like with like, is the usual retort.

To be stupid, selfish, and have good health are three requirements for happiness, though if stupidity is lacking, all is lost*. (Gustave Flaubert 1821 - 1880)*

Although it is easy to accept that antidepressants are ineffective for mild or moderate depression, one has to consider that in even in major depression the effects of spontaneous remission (75% in 12 weeks in some instances)[5, 6] and natural fluctuations need to be taken into account. Even patients with chronic symptoms, who normally seek help when their symptoms are at their worst, sometimes improve anyway. Take a simple known fact: the prevalence of pain in patients with depression is high, around 65%, and the average prevalence of depression in pain clinics is nearly of a similar order. Pain symptoms in depression are not adequately treated by Selective Serotonin Reuptake Inhibitors (SSRIs) or indeed by amitriptyline (commonly used for pain relief) and hence depression is prolonged.[7] On

reflection it should not be too difficult to comprehend why drugs do not always work given that some three billion base pairs of deoxyribonucleic acid (DNA) make up the human genome. To add to the complexity, copy number variation refers to differences in the number of copies of a particular region in the genome, which is associated with susceptibility or resistance to disease.

Patients who are depressed and helped by medication are now being told by irresponsible 'counsellors' and sometimes by their own family doctors, that they are really only taking sugar pills, because of selective information taken from flawed antidepressant drug trials. By the same token should patients also give up their counselling sessions and take a sugar pill? It should not be forgotten that a true placebo control is impossible in psychotherapy unlike physical methods of treatment (though still difficult), whatever the flaws inherent in the latter. It seems odd that psychological data in 'therapy studies' carried out by non-clinicians and clinicians gets to be called 'science' whereas drug research carried out by scientists becomes 'flawed science?' Even so, countless dubious articles of 'human interest' manage to appear in prestigious medical journals under the apparent authorship of 'leading figures in the field' (being a pop celebrity physician or psychologist helps) where the psychobabble is fed to the reading classes who in turn regurgitate it to their naïve, well-intentioned adherents, and to the media. I don't blame the latter: all the media want is a good story; 'human interest' items will sell newspapers regardless of their quality or accurateness. People who run the media have little understanding of science and wear their ignorance as a badge of honour.[8] Therefore it comes as no surprise when it is discovered that antidepressants do not work for mild or moderate depression that the slogan becomes 'antidepressants do not work at all,' which is what the critical psychiatry faction wanted in the first place.

If you can't explain it simply you don't understand it well enough *(Albert Einstein 1879-1955)*

Ironically, as in neuropharmacology, it is through progress in molecular biology that advances in psychotherapy research will be made as molecular genetic findings unfold over the next few years; it is likely that biological vulnerability will become increasingly detectable; although single genes and polymorphisms will probably never account for a large proportion of variability, combinations of genes may increasingly identify specific types of environmental vulnerability.[9, 10] No mental health condition is 'all genetic or environmental.' However, it is through neuropharmacological research that the mechanisms of action of various drugs used in neurology and psychiatry have been identified and helped to develop an understanding of biological substrates underlying the aetiology of psychiatric disorders. Genetic studies help us understand why some individuals are more prone to becoming ill given the same environmental stress factors.

The overriding clinical impression by doctors in clinical practice and in hospital settings is that patients tolerate minor side effects in the hope that benefits will accrue in the long term, as they do with the very unpleasant adverse effects from other drugs used in medicine (chemotherapy, for example). It is incumbent for doctors to stress that antidepressants do work for severe depression (though not in all cases) and mood stabilizers are helpful in bipolar disorder, and advise about untoward effects.[11] Doctors should also emphasize that the therapeutic effect is not that of a placebo, much in the same way that methylphenidate helps many children with Attention Deficit Hyperactivity Disorder (ADHD) when it is properly diagnosed and not attributed to poor parenting skills. The beneficial effects of antidepressants when they do occur are noticed objectively, usually within four to eight weeks of taking the medication, sometimes sooner. Patients are not coerced into feeling better by the charismatic charm of the physician, who may be sceptical to begin with, in any event. Besides, 'charisma' wears a bit thin when one continues to feel miserable and unresponsive to treatment of whatever sort. Cognitive therapy is effective for those who are motivated and not too disabled with lethargic indifference to engage. Behavioural methods do work, because specific techniques are employed which allow accurate objective evidence (cessation of smoking, desensitisation for phobias, amelioration of obsessive rituals) to be gathered.

There is a vast grey area between what constitutes 'normal' and 'mild or moderate' depression. In most cases, even if one concedes that a patient is 'mildly or moderately' depressed there is usually no need to interfere, because everyday issues are usually the triggering factors. Most 'psychiatric' conditions are not psychiatric, and life's ills and worries are best left to the General Practitioner (GP) to offer advice, perhaps a close friend, or even a next-door neighbour. Best to throw away all the psychobabble bibles and 'treatment packages' by the experts in living who earn a good income exploiting patients' weaknesses. Instead, patients should be taught to rely more on their natural intuition and cultivate inner strengths and talents. When depression, mood swings, phobias, obsessive rituals, and inner turmoil (for example, derogatory hallucinations, tormenting thoughts) become overwhelming, that is the time to seek medical advice. Most people (unless delusional) know which category they fit into, and should be able to receive help or intervention to deal with mental anguish before it becomes too disabling.

Many patients get better with or without talking therapies or medication, through sheer determination. At least with pharmacotherapy the medication can be thrown out after a reasonable period of adequate dosage. Either the drug works or it does not. Psychotherapy theoretically, particularly psychoanalysis, has no end, and can prove very costly. The top-up sessions are not free either! Even the National Health Service (NHS) will only offer a certain number of sessions and then you are on your own. Of course, there is the homework and perhaps

a few more top-up sessions, if you make enough fuss! By all means investigate the alleged fraudulent business practices of Big Pharma and eliminate the biased positive results of drug trials. Author bias should also be scrutinized to eliminate personal prejudice. There is no need for patients to be duped by the empty rhetoric perpetuated by the experts in living if we are simultaneously led to believe that life's ills will be resolved through the use of a sugar pill.

To be conscious that you are ignorant is a great step to knowledge. *(Benjamin Disraeli 1804 – 1881)*

Within the field of psychiatry (and psychology) there are those who do not believe in drug treatments, ADHD, eating disorders, to mention a few. Everything is environmentally induced or caused by bad parenting, we are told by some self-appointed 'life experts'. And there are those who thrive on being deliberately controversial in an effort to raise their media profile and income. What a pity. Cardiology does not compete with cardiothoracic surgery nor does gynaecology compete with obstetrics, for example. Debate - yes. Antagonism - no. It is time for psychiatry to re-examine and distance itself from the popular psychobabble of the agony aunts and uncles before it completely loses its sense of professionalism. Because there are so many overlapping clinical scenarios within psychiatry and neurology, the former needs to align itself with the latter specialty and by doing so will gain respectability. The ever-widening chasm between psychiatry and other medical disciplines has been gathering momentum over the years, leaving psychiatry more alienated than ever. Perhaps there is also a case for subsuming some psychiatry specialties back into general psychiatry, for example, a Consultant General Psychiatrist with a 'special interest' in the conditions a Child Psychiatrist might be expected to deal with, such as Tourette's syndrome, ADHD, and psychoses. Fewer graduates are now interested in pursuing psychiatry because they do not want to study medicine for years only to end up being marginalized as an on-looker in some multidisciplinary setting, devoid of any responsibility or decision-making. It is not that Cinderella will not be going to the Ball; there will be no Ball to go to.

Competing Interests
None declared
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Medicine in Pictures

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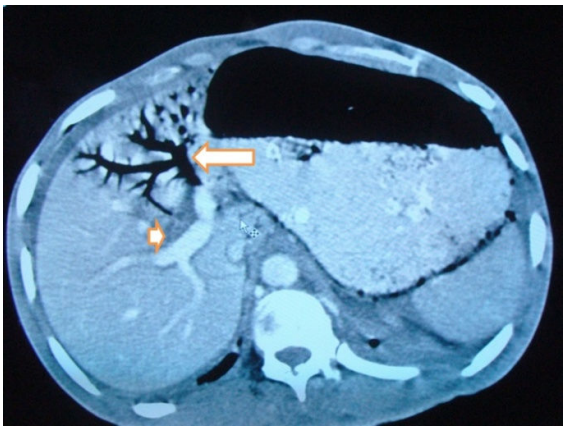
Portal vein air embolism

Suhail Y Hakim , Gursimran Singh Kundan and Sofia Gani Rah

A 45- year -old man hit by a speeding vehicle presented with chest and abdominal pains, along with dyspnea and vomiting. On examination, vitals signs were pulse rate of 122 per minute, blood pressure of 100/60 mm of Hg and respiratory rate of 28 per minute. Chest examination showed tenderness on the lower ribs. The patient had ecchymotic patches and abrasions on his lower chest and abdomen. Abdominal examination showed diffuse tenderness in all quadrants. The patient also had an open fracture of right femur. Arterial blood gas analysis showed hypoxia. Liver enzymes were normal. Chest X-ray and FAST (Focused Assessment with Sonography for Trauma) did not reveal any obvious pathology. CT scan of the chest showed small contused areas of lungs on both sides. Contrast CT abdomen showed the following picture.

Medicine in pictures



CECT (Contrast Enhanced Computed Tomography) of Abdomen.

Question: What is this radiological finding?

Answers:

1. Pneumobilia
2. Liver Laceration
3. Portal vein air embolism
4. Oriental cholangiohepatitis.

Correct answer and description – at the end of the article

Differential diagnosis:

Air in the portal vein has many causes including Necrotizing entero-colitis, Inflammatory bowel disease, Pneumatosis intestinalis, Mesenteric ischemia, Perforated peptic ulcer, Trauma etc.

Explanation:

Pneumobilia means air in the biliary tree. This condition refers to central location of the air which does not extend to within 2 cm of the liver capsule. It is most commonly seen in patients following surgery in which a biliary-enteric anastomosis has been created or a sphincterotomy (sphincter of Oddi) has been performed.

Liver laceration is seen as a non-enhancing region, linear or branching, hypo-dense wedge lesion extending to liver surface.

The CT findings in oriental cholangiohepatitis can present as intra- or extra hepatic duct stones, dilatation of the extra hepatic duct with relatively mild or no dilatation of the intrahepatic ducts or as localized dilatation of the lobar or segmental bile ducts.

Discussion:

Gas in the portal vein is a rare and usually fatal condition, and its presence in trauma is a rare occurrence. Various terms are used to describe the condition like hepatic portal vein gas (HPVG), pneumoportogram, gas embolism of the portal vein etc. It has to be differentiated from air in the biliary radicals. Portal venous gas manifests on CT as small, tubular air densities in the peripheral regions of the liver, predominantly in the left hepatic lobe in the left portal vein as it is more anterior. Due to the centrifugal flow of blood in the portal venous system, air bubbles appear to extend within 2 cm of the liver capsule [1, 2] . Susman and Senturia state that air in the biliary radicals is more centrally placed and extends up to the hilum and into the common hepatic duct due to the centripetal flow of bile [3] . In the pediatric age group the commonest cause attributable is

necrotizing entero-colitis, and along with pneumatosis intestinalis it is in fact pathognomic of the condition[4] . The pathophysiology behind air entering portal vein is intestinal mucosal damage leading to air entering the venules that connect into the portal vein. This was demonstrated in an experiment where hydrogen enema was given to a dog and subsequent mesenteric venous gas was noted[5]. Outcomes are poor in non-trauma cases. In the absence of CT findings associated with bowel ischemia, portal venous gas due to trauma or iatrogenic causes may be treated conservatively [6] . However, blunt trauma can be varying in severity and intensity, and the likelihood of polytrauma should be considered by the treating physician.

Correct answer is option 3 - Portal vein air embolism.
Portal Vein Air embolization also called HPVG (hepatic portal vein Gas).Long arrow showing air in the left portal vein and the arrow head showing the contrast filled right portal vein.

Competing Interests
None declared
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E-Interview

Interview with Professor Richard D Griffiths

Richard D Griffiths BSc, MD, FRCP, FHEA

Prof Griffiths is a Professor of Medicine (Intensive Care), Dept of Musculoskeletal Biology, Institute of Ageing & Chronic Disease, Faculty of Health & Life Sciences University of Liverpool, and Honorary Consultant Physician in Intensive Care Medicine, Whiston Hospital, UK.

He obtained a BSc in Physiology during undergraduate training in medicine (MBBS) at University College London during the '70s. During the early '80s in London obtained a research MD studying muscle energetics in the early days of human Magnetic Resonance Spectroscopy. Became a consultant in adult Intensive Care Medicine in 1985 following a move to Liverpool in 1984 and continued research interests in muscle and expanded these into nutrition (glutamine) and the critically ill. Since then has been a pioneer of the rehabilitation of the post-ICU patient. He extensively involved over the last two decades in undergraduate curriculum reform and as the Director of the Final Year has pioneered a fully portfolio based professional learning programme.

How long have you been working in your speciality?
I have been a consultant intensive care physician for more than 25 years.

Which aspect of your work do you find most satisfying?
To be able to improve patient care through clinical research and the training of medical students.

What achievements are you most proud of in your medical career?
Raising the awareness of the physical, psychological and cognitive challenges ICU patients and relatives face during recovery and contributing to the evidence base guiding rehabilitation.Clinical nutrition research on glutamine and identifying the need to use six month mortality outcomes in the critically ill. Creating a final year of undergraduate medical training that fosters professionalism and critical self awareness based upon a clinical portfolio and appraisal process that produces graduates fit for practice.

Which part of your job do you enjoy the least?
Very little, but perhaps the ever increasing bureaucracy of regulation in practice and research.

What are your views about the current status of medical training in your country and what do you think needs to change?
In the UK most medical schools have radically reformed their curriculum to meet the needs of modern medicine and life-long learning. In Liverpool our students are recognized to be well prepared with the skills to ensure patient safety and start foundation training following a course commended by clinicians, hospitals, examiners and GMC alike. Post-graduate changes have paralleled these developments and while the training structures and closer observations are to be commended the restrictions on working time remains a concern for the acquisition of real "shop floor" experience. Our trainees simply don't get enough "flying hours" as in the past.

How would you encourage more medical students into entering your speciality?
Intensive care medicine is popular. The problem for students is to understand how to get there. The new Faculty of Intensive Care medicine, that has just starte, brings an independent speciality out from under the umbrella of its various parent specialities and hopefully will provide the focus to make the career pathway clearer in the future.

What qualities do you think a good trainee should possess?
All those attributes that the GMC expect of a practitioner! In particular I like to see enthusiasm, self awareness and measured confidence, an enquiring and questioning mind and a degree of professional flexibility mixed with the ability to ask for help and advice. I need to trust them just as their patients need to as well.

What is the most important advice you could offer to a new trainee?
Stay calm, be professional and follow the basic principles of good medical practice doing the simple things well, and don't be afraid to ask for help.

What qualities do you think a good trainer should possess?
Maintain professionalism and be a role model at all times with the ability to listen, support and recognize the strengths as well as being firm with those things that need developing.

Do you think doctors are over-regulated compared with other professions?
No, while regulation does not itself prevent bad medicine it does prevent it being ignored.

Is there any aspect of current health policies in your country that are de-professionalising doctors? If yes what should be done to counter this trend?
De-professionalising only occurs when doctors avoid taking leadership roles. I think this was a fear in the recent past but in the last 10 years in the UK there has been a strong drive to redefine professionalism and the role of the doctor for the 21st century and it is central now to modern undergraduate and post graduate training with the importance of Consultants and GPs taking leadership roles in planning health care delivery.

Which scientific paper/publication has influenced you the most?
Huxley AF 1957 A theory of muscular contraction" Prog. In Biophys. And Biophys. Chem; 7:255.
Professor Sir Andrew Huxley was awarded the Nobel prize in medicine in 1963 with AL Hodgkin for nerve conduction but my personal memory is in muscle physiology (as one of my tutors) for his work on the theory of muscle contraction and the role of cross bridges. His clarity of thought was demonstrated in his ability to always ask the question everyone else wished they had asked! He was a kind and gentle teacher that gave time even for a simple medical student.

What single area of medical research in your speciality should be given priority?
The brain is the forgotten organ in multiple organ failure. We now recognize that acute brain dysfunction is a serious problem but we know little about its pathology, how to prevent it or recover from it.

What is the most challenging area in your speciality that needs further development?
There has been a rush towards ill conceived large scale pragmatic clinical effectiveness studies of various therapies few of which have shown much to change practice. Rather there is a need for more detailed scientific research to better define efficacy of therapies by exploring the pathological processes and the genetic and environmental influences of common disorders that result in multiple organ failure.

Which changes would substantially improve the quality of healthcare in your country?
Addressing the challenge of an ageing population and in particular the community medical and non-medical support of the aged infirm so that modern medicine does not grind to a halt.

Do you think doctors can make a valuable contribution to healthcare management? If so how?
By showing leadership and making the changes happen and not leaving it to others perhaps less informed to direct change.

How has the political environment affected your work?
I have tried to ignore it as much as possible. Politics is a business best left to politicians while the rest of the world gets on with life.

What are your interests outside of work?
I treasure my family, a marriage of 28 years, with two undergraduates in medicine and one in architecture and doing all the jobs they ask of a father. When not escaping to the south of France or walking I become a generalist handyman so it can be a gardener, electrician, plumber, decorator, carpenter, car mechanic……and the Sunday Roast!

If you were not a doctor, what would you do?
With the exception of playing a musical instrument anything that combines academia, teaching and its practical application, but with preference in the natural world.