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Editorial

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Depression and pain: is there a common pathway?

Francis J Dunne

Chronic pain, arbitrarily defined as that lasting longer than six months, is a clinical, social, and economic problem. It is often accompanied by feelings of low mood and despondency.

Whether chronic pain induces clinical depression or depression initiates psychosomatic pain (through physiological mechanisms) is difficult to prove. The burden of illness increases when patients suffer from both. Financial hardship and medical costs affect the quality of life which leads to difficulties in coping and further decreased functioning, making the treatment of both conditions more complicated. Therefore, better recognition, assessment, and treatment of comorbid pain and depression should, at least in theory, lead to better outcomes.

Pain is broadly categorized into three groups: nociceptive (any painful stimulus), neuropathic (for example, diabetes), and psychogenic. Nociceptive pain occurs with direct noxious stimuli. Neuropathic pain is a result of disease or injury to the nervous system or spinal cord. Psychogenic pain has no discernible physical origin. Although the precise physiological mechanisms are not entirely understood, there are two basic categories of sensory neurones. The first type is myelinated and fast conducting; the second is unmyelinated and slow conducting.

Acute pain which follows damage to tissue (an ankle sprain, for example) is usually correlated with hyperalgesia (an increase in the pain elicited by a noxious stimulus and felt as a sharp, burning sensation) and allodynia ('other' pain evoked by a normally innocuous stimulus) and serves to protect the injury from further trauma while allowing the damage to be repaired.

Depression is often a chronic disorder and though its symptoms may be alleviated by appropriate medication and other therapies, physical complaints tend to be more intractable. For example, fibromyalgia (FM), a syndrome characterized by widespread muscle pain and generalized tender points, is often associated with major depressive disorder.[1] However, although the vast majority of patients with fibromyalgia do not meet criteria for a psychiatric disorder, psychological symptoms are common. In a randomized controlled trial of primary care patients with musculoskeletal problems and depression, antidepressant medication followed by a self-management pain programme led to improvement in both.[2]The tricyclic antidepressant amitriptyline was traditionally used usually in small doses, to treat pain, with moderate success. In addition to its own intrinsic analgesic effect amitriptyline appears to enhance the effects of opioid analgesia. Other antidepressants are now in vogue; for example, duloxetine, a serotonin (5-HT)/noradrenaline (NA) reuptake inhibitor, is sometimes used for diabetic neuropathic pain.

Of the numerous neurotransmitters at least two, namely 5-HT and NA, may prove to be one common link between depression and pain. Both serotoninergic and noradrenergic pathways ascend from subcortical areas (brainstem, hypothalamus and thalamus) to the whole neocortex and mediate emotional and physiological responses.[3] Their pathways descend the spinal cord and suppress nociceptive inputs. Serotoninergic cell bodies located in the raphe nucleus in the brainstem, and noradrenergic neurones located in the locus coeruleus (also in the brainstem) send projections to various parts of the brain involved in the control of mood, appetite, sexual activity, attention and concentration. Theoretically at least, a dysfunction at the level of the serotoninergic and noradrenergic neurons could affect both ascending and descending pathways resulting in the psychological and physically painful symptoms of depression. Neurotransmitters may open or close the 'gate' on perception of painful stimuli. Therefore adrenergic and serotoninergic pathways from the brainstem to the spinal cord will inhibit incoming painful stimuli. This is perhaps an oversimplification as some sensory fibres enter via the ventral spinal roots.

The hypothalamic pituitary axis (HPA) is probably also involved. The hypothalamus, which synthesises and secretes neurohormones, has a wide range of physiological functions including regulation of thirst and hunger, sexual behaviour, defence reactions such as fear and rage, and circadian rhythm: disturbances of all these functions are frequently seen in depressed or anxious patients. The HPA is also affected in patients with physical stress as well as major depression, as shown by increased levels of adrenocorticotropic hormone and

cortisol in the plasma. Stimulation of the lateral areas of the hypothalamus produces a diffuse sympathetic discharge possibly because some areas of the hypothalamus control adrenaline and NA secretion. Prolonged stress associated with pain leads to depletion of central 5-HT and malfunction of other associated receptors.[4]

The hypothalamus and limbic system (whose boundaries are difficult to define) with its associated structures – the amygdala, hippocampus and septal nuclei, are involved in the mental and affective aspects of emotions. The amygdala, a cluster of nuclei in the medial temporal lobe, may have a role in the reciprocal relationship between pain and depression. The amygdala controls not only emotional behaviour but also memory. However, mixed results have been reported regarding the level of activity of the amygdala in response to pain.

Nociceptor afferents terminate within distinct regions of the dorsal horn and within the spinal cord, synapses are sites of considerable modification, hence the term 'gate' for the dorsal horn cells. The neurotransmitter for slow pain is believed to be substance P, and glutamate is the putative transmitter secreted by primary afferent fibres subserving fast pain.[5]

5-HT and NA neurotransmitter systems influence neuroplasticity in the brain. Most currently available antidepressants act through reuptake inhibition of either or both. Therefore, it would seem feasible to prescribe dual-action antidepressants when pain symptoms are associated with depression. However depressed patients with pain comorbidity are less likely to take antidepressant medications compared to those with depression alone. Also, individuals who develop pain or depression are at risk for developing the other, thus escalating the clinical management. Furthermore, when pain is refractory to treatment, it is associated with more depressive symptoms and worse depression outcomes, and vice versa. Depressive symptoms are very common in physically ill patients. Unfortunately, depression is often overlooked in pain patients because pain symptoms take priority or worse still, comorbid depression is not considered.

It is difficult to state with certainty whether or not unexplained pain is 'psychological'. Such an assumption might be perceived as demeaning and patronizing to patients and the suggestion of providing cognitive therapy misinterpreted as him/her overplaying their reaction to pain or that the pain is 'psychological'. Others do not like being labelled 'psychiatric', and are therefore reluctant to take antidepressants even when a physiological explanation is given. Pain perception involves physical and emotional factors and its primary function is to protect the organism from harm. It follows therefore, that pain thresholds and pain tolerance vary from individual to individual, and especially among patients with depression.

Antidepressants are frequently used in the treatment of depression and generalized anxiety disorders. Their use extends beyond these areas, however, and it is now accepted that antidepressants are efficacious in treating chronic pain syndromes in addition to their effects on psychological features such as low mood, inordinate guilt, or feelings of worthlessness. Because physical symptoms are often the main complaint in many depressed patients and pain is common as a presenting symptom, clinicians need to know about the dual use of antidepressants for both. Future antidepressants may involve neurotransmitters, other than 5-HT and NA, which could include dopaminergic pathways, opioid (antagonists of morphine-type drugs) receptors and the pentapeptides (enkephalins) which bind to these receptors.

Competing Interests
None declared
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Research Article

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Community-acquired urinary tract infection in the elderly

Mahesh E, Medha Y, Indumathi V A, Prithvi S Kumar, Mohammed Wasim Khan and Punith K

ABSTRACT

Background: Urinary tract infection (UTI) in the elderly poses a very serious problem. The knowledge of microbiology and antibiotic susceptibility of micro-organisms causing the disease is vital for defining the empirical treatment. There are no large-scale studies on the same from India.

Aim: To find out the common presenting symptomatology and risk factors associated with UTI and distribution of isolated uropathogens and their resistance pattern.

Settings and design: Prospective study done in a tertiary care centre in Bangalore.

Methods and material: The study included elderly patients aged 65 years and above who were admitted, or visited the outpatient departments in the hospital, and had confirmed UTI.

Results and conclusions: Fever (57/194 - 29.4%) and dysuria (52/194 - 26.8%) were the most common symptoms of UTI. Diabetes Mellitus (DM) was the most common risk factor associated with UTI. Extended Spectrum Beta-Lactamase (ESBL) producing Escherichia coli (E.coli) (93/194 - 47.94%) was the most commonly isolated pathogen. Of the total, 56.2% of the uropathogens showed ESBL positivity. Overall, the highest antibiotic resistance was recorded for Fluoroquinolones (79.9%).

Keywords: Uropathogen, Elderly, Antibiotic Resistance, ESBL

Introduction

Urinary tract infection (UTI) is the second most common infectious complaint in geriatric clinics overall, and the most common outpatient complaint caused by bacteria.[1] The diagnosis and treatment of UTI in the elderly is not the same as treating UTI in adults. In frail elderly patients with age-associated multiple severe underlying disorders and cognitive impairment, early recognition of bacteraemic UTI and prompt, appropriate treatment are critical in reducing the mortality.[2]Also, the extensive and inappropriate use of antimicrobial agents has invariably resulted in the development of antibiotic resistance which, in recent years, has become a major problem worldwide.[3] The diagnosis and empirical treatment of UTI in the elderly is challenging and a sound knowledge of the prevalent epidemiology of bacteria and their resistance pattern is necessary for the same. However, there is not much information on the aetiology and resistance pattern of UTI in the elderly in India. This study was done to find out the present uropathogen profile causing UTI in our centre and their antibiotic resistance patterns.

Subjects and methods

This prospective study was done at our tertiary care centre from January to December 2008. The study included all patients who were admitted or visited the outpatient departments in the hospital with symptoms of UTI during the study period and had UTI confirmed by positive urine culture reports. Only one sample from each subject was considered. Subjects with clinical symptoms of UTI but no growth on culture were excluded from final analysis. Subjects who were treated with another antimicrobial within the previous 48 hours, or within 24 hours if only a single dose and in the presence of an appropriate positive culture and ileal loops or vesicoureteral reflux were also excluded from the study. Complete data regarding demography, sex preponderance, associated symptoms, pathogenic organisms causing UTI and their antibiotic resistance were collected.

Overall, 194 subjects were included in the study (male: 116, female: 78). The mean age of the study population was 73.54±7.19 years, ranging between the ages of 65 and 96. The distribution of patients according to gender across various age groups is given in table 1. A general trend of more male subject enrolment was seen across all the age groups.

Table 1. Age and gender distribution of complicated and uncomplicated urinary tract infection.

Age group	Male	Percent	Female	Percent	Total	Percent
65-74	66	56.9	48	61.5	114	58.8
75-84	40	34.5	24	30.8	64	33.0
85-94	10	8.6	5	6.4	15	7.7
≥95	0	0	1	1.3	1	.5
Total	116	100.0	78	100.0	194	100.0

Isolation and identification of uropathogens

A clean catch midstream specimen, or suprapubic aspirate in subjects who were unable to give the former, was collected in a sterile, wide-mouth, leak-proof container to hold approximately 50ml from these subjects. Using a calibrated loop method of loop diameter 4 mm, 10 µL of the uncentrifuged specimen was transferred onto the agar plate and streak, using the modified Mayo's technique without flaming the loop for isolation, and

incubated at 35- 37^{0}C for 24 hours. A specimen was considered positive for UTI if a single organism was cultured at a concentration of >10^{5}Colony Forming Units/ml. The Gram-positive and Gram-negative organisms culture isolates were further identified by using various biochemical reactions up to genus/species level wherever applicable.

Antibiotic sensitivity testing

In the presence of any potential growth, antibiotic sensitivity testing was done by the Modified Kirby-Bauer disc diffusion method according to the Clinical and Laboratory Standards Institute (CLSI) guidelines.[4] The antibiotics tested were Imepenem, Meropenem, Ciprofloxacin, Ofloxacin, Norfloxacin, Amikacin, Gentamicin, Nitrofurantoin and Cotrimoxazole (Pathoteq Labs, India).

Extended Spectrum Beta-Lactamase (ESBL) detection

The screening for ESBL was done using Cefpodoxime (<17mm), Ceftazidime (<22mm), Aztreonam (<27mm), Cefotaxime (<27mm) and Ceftriaxone (<25mm). If the organisms showed the zone of inhibition lower than the minimum for any antibiotic disc, ESBL positivity was suspected. The phenotypic confirmation was done by testing the strain against Ceftazidime (Ca) and Ceftazidime/Clavulanic Acid. A > 5mm diameter of the zone of inhibition for Ceftazidime/Clavulanic Acid in comparison to Ceftazidime was considered indicative of ESBL production. Escherichia coli (E. coli) ATCC 25922 was used as ESBL negative and Klebsiella pneumoniae (K. pneumoniae) 700603 was used as ESBL positive reference strain.[4]

Statistical analysis

Descriptive statistics (totals, means, percentages, and standard deviations) were conducted using the statistical software package - SPSS Version 16.0 (SPSS Inc., Chicago, USA). Age, gender, organisms causing UTI, their antibiotic sensitivity and resistance, symptomatology of these subjects, and risk factors for UTI were included in the analysis and the results presented in tables and figures.

Results

Fever (57/194 - 29.4%) and dysuria (52/194 - 26.8%) were common symptoms of most UTI patients (Fig. 1). Diabetes mellitus (DM) and recent uro-genital instrumentation were the most common risk factors associated with UTI in the present study (Table 2). The organism profile and their antibiotic resistance profile were similar in patients with or without DM.

E. coli (138/194 - 71.1%) was the most commonly isolated pathogen responsible for UTI in the present study (Figure 2). 56.2% of the total infection was caused by ESBL positive organisms.

Figure 1. Various symptomatologies seen in patients with urinary tract infection during the initial presentation

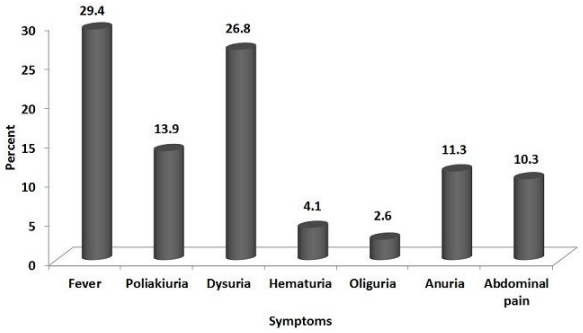
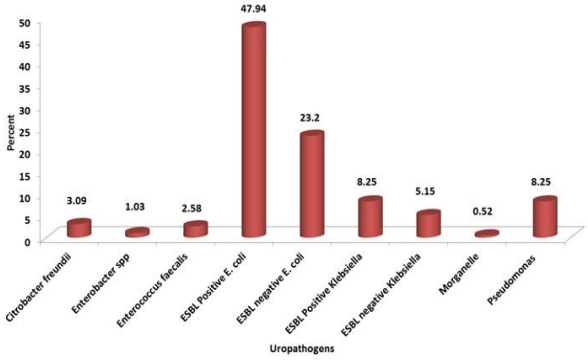


Table 2. Frequency of various risk factors in subjects with urinary tract infection.

Risk Factor	Frequency	Percent
Catheterization	29	14.9
Diabetes Mellitus	97	50.0
Immunosuppression	2	1.0
Recent history of uro-genital Instrumentation	43	22.2
Recurrent urinary tract infection	14	7.2
Renal stones	5	2.6

The antimicrobial potency and spectrum for nine selected antimicrobial agents (Imepenem, Meropenem, Ciprofloxacin, Norfloxacin, Ofloxacin, Gentamicin, Amikacin, Nitrofurantoin and Cotrimoxazole) against the uropathogens were studied. The highest and least antibiotic resistance was noted against fluoroquinolones (79.9%) and carbapenems (3.61%) respectively (Fig. 3).

Figure 2. Frequency and distribution pattern of urinary tract infection pathogens and percentage Extended Spectrum Beta-Lactamase (ESBL) production.

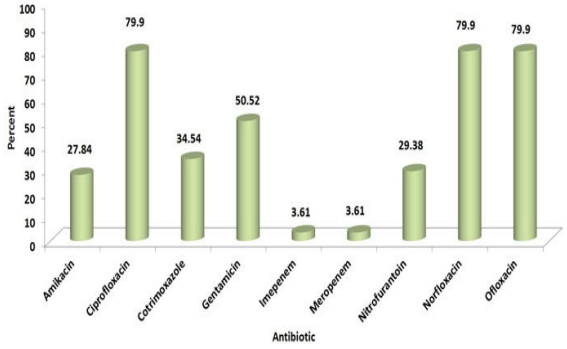


Discussion

While increased frequency and dysuria are usual symptoms of UTI, uncertainty looms around the same as these symptoms can be masked by catheterisation, or be common and chronic in the elderly even in the absence of UTI.[5-10]Fever was the most common symptom of UTI in the present study as with similar studies worldwide.[11-13] Studies have found that the elderly do

not lack a febrile response; that an elevated temperature was the most common initial symptom, a marker for a serious infection, and the most important clinical indicator for antibiotic treatment.[14-16]Whitelaw et al[17] reported that a delay in interpreting fever as a symptom of UTI led to a high mortality rate in the elderly within 24 hours of admission.

Figure 3. Resistance pattern to various antibiotics of the uropathogens



Diabetes isconsidered as an important risk factor for UTI with manyauthors having defined UTI in patients with DM as complicated when the UTI is symptomatic.[18-19] However, the authors did not find that DM influenced the organism profile and their antibiotic resistance in the present study. Bonadio et al[20] studied the influence of DM on the spectrum of uropathogens and antimicrobial resistance in elderly adult patients with asymptomatic UTI (mostly hospital-acquired). They found that DM *per se* did not seem to influence the isolation rate of different uropathogens and their susceptibility patterns to antimicrobials. These findings indicate that, although DM is a known immunomodulator, the role played by the same in altering the antibiotic resistance is minimal compared to recent invasive procedures.

Although the uropathogen profile of the present study resembles similar studies worldwide, the antibiotic resistance of these organisms was unusually high.[2, 21] Cotrimoxazole is the recommended drug for treating UTI. However, more than one third of the study subjects were resistant to the first-line drug. 79.9% of the uropathogens were resistant to fluoroquinolones, which are considered as the second-line drug. As prior fluoroquinolone use is a known risk factor for fluoroquinolone-resistantE. coli infection, it is plausible that frequent fluoroquinolone prescriptions may be contributing to the observed resistance.[22-23] Aypak et al [24] found that treatment durations were statistically longer than the recommended three-day course when patients were empirically treated with fluoroquinolones due to increased resistance rates, and suggested to discourage the empirical use of fluoroquinolones in UTI.

The most troublesome finding of the present study is that ESBL-positive organisms accounted for 56.2% of the total infection. Not much information on ESBL-producing organisms causing UTI is available from India and most of these reports are from the younger population. The prevalence of ESBL-positive UTI in these studies varied between 26.6% and 48.3%.[25-26] To the best of our knowledge, this is the highest ever reported prevalence of ESBL-positive UTI in the elderly worldwide. ESBL-producing organisms are frequently resistant to many of the antimicrobial agents usually recommended for the treatment. As lesser new antibiotics are available for their management, we need to be concerned of this issue in years to come especially in tertiary care centres. A unified antibiotic protocol is necessary to limit the morbidity and mortality associated with inappropriate and under-treatment of UTI.

The limitations of the present study were that altered mental status was not considered as one of the clinical manifestations of UTI in the elderly, which could have mitigated the total number of study subjects included in the study. In addition, the phenotypic confirmation of ESBL-positive organisms was done using only Ceftazidime/Clavulanic Acid and not Cefotaxime/Clavulanic Acid as per the latest CLSI guidelines. As a result, there may be under-reporting of the incidence of ESBL organisms in the present study.

In conclusion, we report a significantly high resistance to common antibiotics among the uropathogens in the present study. Furthermore, the very high rate of ESBL-positive UTI is of concern, and monitoring for the same is necessary to prevent treatment failure and increased morbidity and mortality with UTI.

Competing Interests

None declared

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Review Article

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Eating Disorders in Children and Adolescents

Fayyaz Khan and Uttom Chowdhury

Eating disorders are defined as those disorders in which there is excessive concern with the control of body weight and shape, accompanied by grossly inadequate, irregular or chaotic food intake. It is widely accepted that eating disorders occur in young adults and adolescents, however, a number of reports have described series of young patients with eating disorders aged from eight years upwards.[1,2]The range of disorders in children includes selective eating, food avoidance emotional disorder, functional dysphagia and pervasive refusal syndrome.

ANOREXIA NERVOSA.

The DSM IV diagnostic criteria for anorexia nervosa are as follows:

1. Refusal to maintain body weight at or above a minimally normal weight for age and height (e.g weight loss leading to maintenance of body weight less than 85% of expected; or failure to make weight gain during growth period).
2. Intense fear of gaining weight or becoming fat, even though underweight.
3. Disturbance in the way one's body weight and shape is experienced, undue influence of body weight or shape on self evaluation, or denial of the seriousness of current low body weight.
4. Absence of at least three consecutive menstrual cycles

Subtypes*:*

Restricting Type:

During the current episode of anorexia nervosa, the person has not regularly indulged in binge eating or purging behaviour.

Binge-Eating/Purging Type*:*

During the current episode of anorexia nervosa, the person has regularly indulged in binge eating or purging behaviour.[3]

The above criteria are intended primarily for use with older patients and do not adequately address the problems of anorexia nervosa in children. For example, criterion D in DSM IV applies only to post-menarcheal females and states that there should be an "absence of at least three consecutive menstrual cycles". This is clearly inapplicable in this age group, where the majority are premenarcheal. Equally unhelpful is the statement that weight should be maintained at less than 85% of that expected, for expected weight can only be calculated on the basis of height and age. Yet growth may also be impaired because of poor nutrition, so further adjustments have to be made. For these reasons, the Eating Disorders Team at Great Ormond Street Hospital for Sick Children in London, U.K, developed a more practical diagnostic criterion for early onset anorexia nervosa.[4] The current criteria is as follows:

Great Ormond Street diagnostic criteria for early-onset Anorexia Nervosa:

1. Determined weight loss (e.g. food avoidance, self-induced vomiting, excessive exercising and abuse of laxatives).
2. Abnormal cognitions regarding weight and/or shape.
3. Morbid preoccupation with weight and/or shape.[5]

Weight loss: Since children should be growing, static weight may be regarded as equivalent to weight loss in adults. Weight loss is a real cause for concern in children, since they have lower total body-fat deposits and therefore do not have much fat to lose. One measurement for weight loss uses the Tanner-Whitehouse Standards[6] where 100% represents the desired weight for a child's sex, age and height, and 80% or less is classed as 'wasting'.

Food avoidance: Children with anorexia nervosa give a variety of reasons for refusing food, the most common of which appears to be a fear of becoming obese. Other reasons include feelings of nausea or fullness, abdominal pain, appetite loss and difficulty swallowing. [7]

Self-induced vomiting: Fosson et al (1987) reported that at least 40% of the 48 children included in their study of early-onset anorexia nervosa were known to be vomiting at presentation.

Excessive exercising: This is not uncommon in children with anorexia nervosa. Daily exercise workouts may be a feature in these children's lives. Sometimes exercise may be carried out in secret in the privacy of the bedroom or bathroom.

Laxative abuse: This is not as common as in adult populations partly because children have less access or opportunity to obtain laxatives, but nevertheless, it still occurs.

Abnormal cognitions regarding weight and/or shape: The main beliefs are centred around body image and its distortion,

although it must be acknowledged that body image is difficult to assess reliably. Many children with anorexia nervosa will report that they consider themselves fat even when severely underweight, which is similar to the clinical observation seen in adult patients with the same condition.

Preoccupation with weight: Children with anorexia nervosa tend to be preoccupied by their own body weight and are often experts at calorie counting. This preoccupation is closely related to fear of fatness.

Physical Aspects

The majority of physical changes in anorexia nervosa are predominantly related to the effects of starvation and dehydration. This includes slow pulse rate, low blood pressure and poor circulation leading to cold hands and feet. Often there is excess fine hair especially on the back, known as 'lanugo'. Teeth may be pitted, eroded and decayed from gastric acid during vomiting.

A wide range of biochemical changes have been described in anorexia nervosa, although there is little information specifically relating to children. These include low haemoglobin and white cell count, low levels of potassium and chloride, raised liver enzymes such as alanine transaminase and alkaline phosphatase, and low levels of plasma zinc and serum iron.

A number of endocrine changes appear in anorexia nervosa and evidence suggests that this is due to the secondary effects of starvation. Changes include increased cortisol, growth hormone and cholecystokinin, and decreased luteinizing hormone, follicle stimulating hormone, oestrogen, triodothyronine and thyroid stimulating hormone.

BULIMIA NERVOSA

The DSM IV diagnostic criteria for bulimia nervosa is as follows [8]:

1. Recurrent episodes of binge eating e.g, eating large amounts of food in two hours, and a sense of lack of control during the episode.
2. Regular use of methods of weight control:
3. vomiting
4. laxatives
5. diuretics
6. fasting or strict diet
7. vigorous exercise.
8. Minimum average of two binges a week in three months.
9. Self-evaluation is influenced by body weight or shape.
10. The disturbance does not occur exclusively during episodes of anorexia nervosa.

Sub-types:

Purging Type: During the current episode of bulimia nervosa, the person regularly engages in self-induced vomiting or laxative misuse, diuretics or enemas.

Non-purging Type: During the current episode of bulimia nervosa, the person has used other inappropriate compensating behaviours such as fasting or excessive exercise, but not regularly used purging behaviour. [2]

Self-induced vomiting can lead to complications such as fluid and electrolyte disturbance and gastro-intestinal bleeding. Other physical complications include dental erosions, enlargement of the salivary glands, and muscle weakness.

OTHER EATING DISORDERS IN CHILDREN

Food Avoidance Emotional Disorder

This term was first introduced by Higgs et al (1989)[9], to describe a group of underweight children presenting with inadequate food intake and emotional disturbance who did not meet the criteria for anorexia nervosa.

The operational definition we use has evolved from Higgs and colleagues original description together with clinical experience and is as follows:

1. Food avoidance not accounted for by primary affective disorder.
2. Weight loss.
3. Mood disturbance not meeting criteria for primary affective disorder.
4. No abnormal cognitions regarding weight or shape.
5. No morbid preoccupation regarding weight or shape.
6. No organic brain disease or psychosis.

Selective Eating

Selective eaters are a group of children who present with very restricted eating habits in terms of the range of foods they will accept. Characteristics include:

1. Have eaten a narrow range of foods for at least 2 years.
2. Unwillingness to try new foods.
3. No abnormal cognitions regarding weight or shape.
4. No fear of choking or vomiting.
5. Weight may be low, normal or high.

Pervasive Refusal Syndrome

This term was first described by Lask et al (1991).[10] The main features are:

1. Profound refusal to eat, drink, walk, talk or self-care.
2. Determined resistance to efforts to help.

Initially these children present with features fairly typical of anorexia nervosa, but the food avoidance is gradually followed by a more generalised avoidance with a marked fear response.

Functional Dysphagia

Children with this condition generally present with complaints of difficulty or pain on swallowing. Features include:

1. Food avoidance.
2. Fear of swallowing, choking or vomiting.
3. No abnormal cognitions regarding weight or shape.
4. No morbid preoccupation regarding weight or shape.
5. No organic brain disease or psychosis.

For more information on the above eating disorders in children see Lask & Bryant-Waugh (1999).[5]

INCIDENCE AND PREVALENCE

For a number of reasons, the incidence and prevalence of childhood-onset anorexia are not known. There have been no epidemiological studies which have focussed specifically on this age group and the strict diagnostic criteria used in wider epidemiological studies may lead to a substantial underestimate of the true incidence.[2] However studies in adolescent populations estimate the prevalence to be in the order of 0.1-0.2%[11, 12] and it is likely to be even lower in children. Although debatable, an increase in actual referral rate of anorexia nervosa in children has been reported.[2]Gender distribution: Five to ten percent of cases of anorexia nervosa in the adolescent and young adult population occur in males.[13] However, in children, studies have reported that between 19 and 30% of children with anorexia nervosa have been boys.[7,9,14,15]

At present, there is little epidemiological information on the other eating disorders in children.

MANAGEMENT AND INTERVENTIONS[16]

Initial assessment

Comprehensive assessment should include physical, psychological and social components. Those with low to moderate risk should be managed as an outpatient. Those who are severely emaciated, with serious risk of self harm, with severe deterioration or with poor response to treatment are deemed high risk and should be considered for inpatient treatment or urgent referral to specialist services.

Anorexia nervosa – outpatient care

Psychological interventions

Psychological interventions are the key element in the management of anorexia.

The delivery of psychological interventions should be accompanied by regular monitoring of a patient's physical state including weight and specific indicators of increased medical risk.

• When delivering psychological treatment consider, in conjunction with the patient:
– Cognitive analytical therapy (CAT)
– Cognitive behaviour therapy (CBT)
– Interpersonal psychotherapy (IPT)
– Focal psychodynamic therapy
– Family interventions focused explicitly on eating disorders
• Focus of treatment should be on weight gain, healthy eating, and reducing other symptoms related to eating disorders.
• Dietary counselling should not be provided as the sole treatment for anorexia nervosa.

Medication

Pharmacological interventions have a very limited evidence base for the treatment of anorexia nervosa.

• Medication is not effective as sole or primary treatment; caution should be exercised in its use for comorbid conditions such as depression or obsessive–compulsive disorder, as these may resolve with weight gain alone
• Avoid using drugs that affect the heart such as antipsychotics, tricyclic antidepressants, some antibiotics and some antihistamines.

Anorexia nervosa – inpatient care

•Body Mass Index (BMI) is a measure of weight in relation to height. Normal BMI range is 18.5-24.9. BMI below 17 is a concern and GPs should consider referral to specialist services. However, BMI below 15 is serious and inpatient care should be considered.
• Consider inpatient treatment for patients with high or moderate physical risk, who have not improved with appropriate outpatient treatment or have significant risk of suicide or severe self-harm.
• Admit to setting that can provide the skilled implementation of refeeding with careful physical monitoring (particularly in the first few days of refeeding) and in combination with psychosocial interventions
• Consider increased risk of self-harm and suicide at times of transition for patients with anorexia nervosa, especially that of the binge–purging sub-type.

Psychological treatment

• Psychological treatment is a key element of an inpatient stay, but evidence for what kind of treatment or approach to treatment is effective, is limited.
• A structured symptom-focused treatment regimen with the expectation of weight gain should be provided, with careful monitoring of the physical status during refeeding.

• Provide psychological treatment with a focus on both eating behaviour and attitudes to weight and shape, and wider psychosocial issues with the expectation of weight gain
• Do not use rigid behaviour modification programmes.

Feeding against the will of the patient

• Feeding against the will of the patient should be an intervention of last resort in care and should only be done in the context of the Mental Health Act 1983 or Children Act 1989.

Post-hospitalisation treatment in adults

• Following discharge, extend the duration of psychological treatment over that normally provided to those who have not been hospitalised, typically for at least 12 months.
• Offer outpatient psychological treatment that focuses on both eating behaviour and attitudes to weight and shape, and on wider psychosocial issues, with regular monitoring of both physical and psychological risk.

Anorexia nervosa –physical management

Anorexia nervosa carries considerable risk of serious physical morbidity. Awareness of the risk, careful monitoring and, where appropriate, close liaison with an experienced physician, are important in the management of the physical complications of anorexia nervosa.

Managing weight gain

• Aim for an average weekly weight gain of 0.5–1 kg in inpatient settings and 0.5 kg in outpatient settings. This requires about 3500 to 7000 extra calories a week
• Provide regular physical monitoring and consider multivitamin/multimineral supplementation in oral form for both inpatients and outpatients.
• Total parenteral nutrition should not be used unless there is significant gastrointestinal dysfunction.

Managing risk

• Inform patients and their carers if the risk to their physical health is high
• Involve a physician or paediatrician with expertise in the treatment of medically at-risk patients for all individuals who are at risk medically.
• Consider more intensive prenatal care for pregnant women to ensure adequate prenatal nutrition and fetal development.
• Oestrogen administration should not be used to treat bone-density problems in children and adolescents as this may lead to premature fusion of the epiphyses.
• Healthcare professionals should advise people with eating disorders and osteoporosis or related bone disorders to refrain from physical activity that significantly increases the likelihood of falls.

Additional considerations for children and adolescents

The involvement of families and other carers is particularly important.

The right to confidentiality of children and adolescents with eating disorders should be respected.

Family members, including siblings, should normally be included in the treatment of children and adolescents with eating disorders. Interventions may include sharing of information, advice on behavioural management and facilitating communication.

Anorexia nervosa

• Family interventions that directly address the eating disorders should be offered to children and adolescents with anorexia nervosa.
• Offer children and adolescents individual appointments with a health professional separate from those with their family members or carers.
• For children and adolescents, once a healthy weight is reached, ensure increased energy and necessary nutrients are available in the diet to support growth and development.
• Involve carers of children and adolescents in any dietary education or meal planning.

Inpatient care of children and adolescents with anorexia nervosa

• Inpatient care of children and adolescents should be within age-appropriate facilities (with the potential for separate children and adolescent services), which have the capacity to provide appropriate educational and related activities. They should also balance the need for treatment and urgent weight restoration with the educational and social needs of the young person.
• Consider using the Mental Health Act 1983 or the right of those with parental responsibility to override the young person's refusal to receive treatment that is deemed essential.
• Seek legal advice and consider proceedings under the Children Act 1989 if the patient and those with parental responsibility refuse treatment where treatment is deemed essential.

Bulimia nervosa

Following the initial assessment consider:

• As a possible first step, an evidence-based self-help programme – direct encouragement and support to patients undertaking such a programme may improve outcomes. This may be sufficient treatment for a limited subset of patients.

Psychological treatment should form the key element of treatment, so consider:

• For adolescents: Cognitive behavioural therapy for bulimia nervosa (CBT-BN) adapted as needed to suit their age, circumstances and level of development.
• Where there has been no response to CBT or it has been declined: other psychological treatments, particularly interpersonal psychotherapy (IPT). (Note: patients should be informed that IPT takes 8–12 months to achieve results comparable with CBT-BN).

Medication may have a role

• Consider a trial of an antidepressant drug as an alternative or additional first step to using an evidence-based self-help programme.
• In terms of tolerability and reduction of symptoms, SSRIs (specifically fluoxetine) are the drug of first choice for the treatment of bulimia nervosa.
• The effective dose of fluoxetine may be higher than for depression.
• Beneficial effects will be rapidly apparent and are likely to reduce the frequency of binge eating and purging, but the long-term effects are unknown.
• No drugs, other than antidepressants, are recommended for the treatment of bulimia nervosa.

Physical management

• Assess fluid and electrolyte balance where vomiting is frequent or there is frequent use of laxatives.
• If electrolyte balance is disturbed, consider behavioural management as the first option
• If supplementation is required, use oral rather than intravenous preparations.

Bulimia nervosa – inpatient or day care

• Consider inpatient treatment for patients with risk of suicide or severe self-harm.
• Admit patients to a setting with experience of managing this disorder.

PROGNOSIS [17]

If untreated, anorexia nervosa carries high mortality rates of

10-15%. If treated, one third have full recovery, one third partial recovery and one third have chronic problems. Poor prognostic factors for anorexia nervosa include: chronic illness, late age of onset, bulimic features such as vomiting and purging, anxiety when eating with others, excessive weight loss, poor childhood social adjustment, poor parental relationships and male sex.

Prognosis for Bulimia nervosa is generally good, unless there are significant issues of low self esteem or evidence of severe personality disorder.

USEFUL RESOURCES

National Eating Disorder Association
Tel: 0800 931 2237
Website: www.nationaleatingdisorders.org

Royal College of Psychiatrists
17 Belgrave Square
London SW1X 8PG
Tel: 0171 235 2351
Website: www.rcpsych.ac.uk

Competing Interests
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Review Article

BJMP 2011;4(1):a402

Diffuse Alveolar Haemorrhage with ANCA associated vaculitis-review of Literature

Fadi Hammoudeh, Muhammad K. Perwaiz, Setu Patolia, Frances M. Schmidt, Narayan Neupane, Neerja Gulati, Danilo Enriquez, Joseph Quist, Mehjabeen Zahir, and Eneh Kennedy

ABSTRACT

Patients with Wegner's Granulomatosis often present with diffuse alveolar haemorrhage alongside the classical triad of haemoptysis, anaemia and progressive dyspnoea. The diagnosis is confirmed by bronchoalveolar lavage with serial aspirated aliquots of fluid revealing persistently bloody returns. Lung biopsy is very helpful if it shows granulomatous inflammation and vasculitis however it lacks sensitivity and specificity. Studies suggest that the detection of antineutrophilcytoplasmic antibodies (ANCA) along with Proteinase-3 can substitute for biopsy for the diagnosis of Wegner's Granulomatosis in patients who present with diffuse alveolar haemorrhage.

KEYWORDS

Diffuse Alveolar Haemorrhage (DAH), Wegener’s Granulomatosis (WG), Anti-neutrophil cytoplasmic antibodies (ANCA), classical antineutrophil cytoplasmic antibodies (C-ANCA), anti proteinas-3 (PR3)

Definition

Diffuse Alveolar Haemorrhage (DAH) is a rare but serious and frequently life-threatening complication of a variety of conditions. DAH refers to a clinical syndrome resulting from injury to the alveolar capillaries, arterioles, and venules leading to red blood cell accumulation in the distal air spaces because of leakage of alveolar capillaries. Most cases of DAH are caused by capillaritis associated with systemic autoimmune diseases such as ANCA-associated vasculitis, anti-GBM disease, and systemic lupus erythematosus.[1] Treatment is with immunosuppressants for patients with autoimmune causes and respiratory support if needed.

Diffuse alveolar haemorrhage syndrome is not a specific entity but is a syndrome that suggests a differential diagnosis and a specific sequence of testing.

Aetiology

Many disorders can cause alveolar haemorrhage; they include

- Autoimmune disorders (e.g., systemic vasculitides, Goodpasture's syndrome, antiphospholipid antibody syndrome)
- Pulmonary infections (e.g., invasive aspergillosis, hantavirus infection)
- Toxic exposures (e.g., trimellitic anhydride, isocyanates, crack cocaine, certain pesticides)
- Drug reactions (e.g., propylthiouracil, diphenylhydantoin, amiodarone, methotrexate, , nitrofurantoin, bleomycin, montelukast, infliximab
- Cardiac disorders (e.g., mitral stenosis)
- Coagulation disorders caused by diseases or anticoagulant drugs
- Isolated pauci-immune pulmonary capillaritis
- Idiopathic pulmonary haemosiderosis
- Bone marrow or solid organ transplantation.

Clinical Presentation

The clinical presentation of diffuse alveolar haemorrhage may reflect either alveolar bleeding alone or features of the underlying cause (e.g., haematuria in Wegener granulomatosis, arthritis in systemic lupus erythematosus). Hence, its recognition requires a high degree of suspicion. Some patients present with severe acute respiratory distress requiring mechanical ventilation. However, dyspnoea, cough, and fever are the common initial symptoms and are most often acute or subacute (i.e., present for less than a week). The fever is usually due to the underlying cause, such as lupus. Haemoptysis may be absent at the time of presentation in up to a third of patients because the total alveolar volume is large and can absorb large amounts of blood, without extending more proximally into the airways. Apparent haemoptysis, if present, must be differentiated from haematemesis or pseudohaemoptysis (alveolar flooding with fluid that resembles blood, as in *Serratia marcescens* pneumonia, in which the reddish hue of the infecting organism can create the impression of alveolar bleeding).

Chest X-ray and Chest CT scan typically shows bilateral infiltrates (figure 1 &2)

DAH & ANCA associated vasculitides

Wegener's Granulomatosis (WG) is an uncommon disease that affects about 1 in 20,000 to 1 in 30,000 people. WG is defined by the triad of granulomatous inflammation of the respiratory

tract, vasculitis of small to medium-size vessels and necrotizing glomerulonephritis. The onset of WG may be indolent with few symptoms, or it may have a rapid and severe onset. About 90% of patients have symptoms of a cold or runny nose or sinusitis that fail to respond to the usual therapeutic measures and last considerably longer than the usual upper respiratory tract infection. Other symptoms include nasal membrane ulcerations and crusting, saddle-nose deformity, inflammation of the ear with hearing problems, inflammation of the eye with sight problems, cough (with or without the presence of blood), pleuritis, (inflammation of the lining of the lung), rash and/or skin sores, fever, lethargy weakness, loss of appetite, weight loss, arthritic joint pain, night sweats, and haematuria which may or may not be indicated by a change in urine colour.Thediagnosis of WG depends on the combination of clinical presentation, serological markers, and histopathological findings. ANCA is a sensitive and specific marker for ANCA-associated systemic vasculitis. In a study done by U. Schönermarck et al,[9] 624 ANCA- positive patients were included, (C-ANCA: 333, P-ANCA: 291). C-ANCA were highly sensitive (81%) and specific (99.5%) for WG, resulting in high positive predictive value (PPV) (94%). Many studies showed that combining proteinase 3 (PR3) and C-ANCA results(C-ANCA/PR3) increases specificity and Positive Predictive Value close to 100%, but reduces sensitivity close to 70%.[10,11,13,14] In summary, the presence of C-ANCA & PR3 antibody is highly suggestive of WG. This led to reevaluation of the role of biopsy for diagnosis of WG in multiple studies.[4, 14, 15]

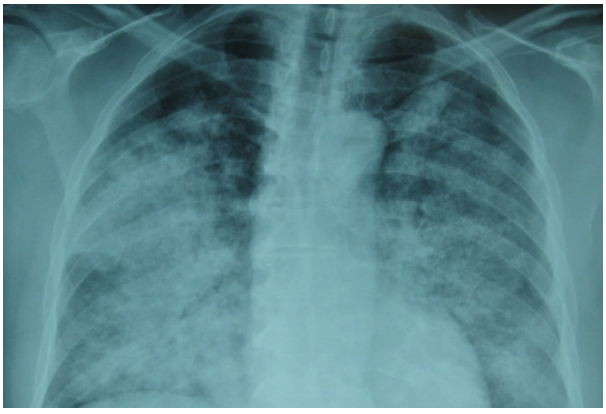

Figure 1

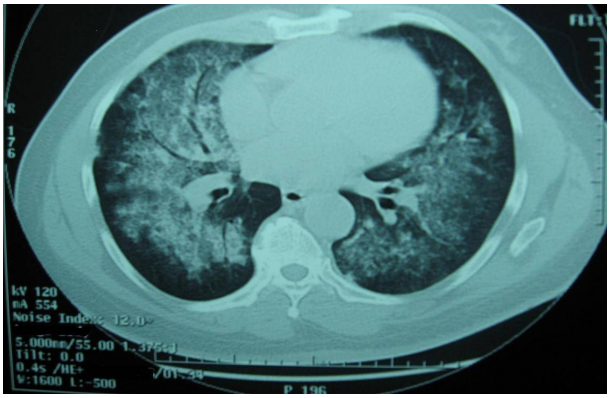

Figure 2

DAH & ANCA associated vasculitides

Wegener's Granulomatosis (WG) is an uncommon disease that affects about 1 in 20,000 to 1 in 30,000 people. WG is defined by the triad of granulomatous inflammation of the respiratory tract, vasculitis of small to medium-size vessels and necrotizing glomerulonephritis. The onset of WG may be indolent with few symptoms, or it may have a rapid and severe onset. About 90% of patients have symptoms of a cold or runny nose or sinusitis that fail to respond to the usual therapeutic measures and last considerably longer than the usual upper respiratory tract infection. Other symptoms include nasal membrane ulcerations and crusting, saddle-nose deformity, inflammation of the ear with hearing problems, inflammation of the eye with sight problems, cough (with or without the presence of blood), pleuritis, (inflammation of the lining of the lung), rash and/or skin sores, fever, lethargy weakness, loss of appetite, weight loss, arthritic joint pain, night sweats, and haematuria which may or may not be indicated by a change in urine colour.Thediagnosis of WG depends on the combination of clinical presentation, serological markers, and histopathological findings. ANCA is a sensitive and specific marker for ANCA-associated systemic vasculitis. In a study done by U. Schönermarck et al,[9] 624 ANCA- positive patients were included, (C-ANCA: 333, P-ANCA: 291). C-ANCA were highly sensitive (81%) and specific (99.5%) for WG, resulting in high positive predictive value (PPV) (94%). Many studies showed that combining proteinase 3 (PR3) and C-ANCA results(C-ANCA/PR3) increases specificity and Positive Predictive Value close to 100%, but reduces sensitivity close to 70%.[10,11,13,14] In summary, the presence of C-ANCA & PR3 antibody is highly suggestive of WG. This led to reevaluation of the role of biopsy for diagnosis of WG in multiple studies.[4, 14, 15]

The site of biopsy is dependent upon the clinical status. A nasal or sinus biopsy may be the least invasive way to diagnose WG. Renal biopsy is helpful if there is evidence of renal insufficiency or glomerulonephritis. A lung biopsy should only be considered if potentially diagnostic tissue cannot be obtained from any other site.[1] Hoffman et al performed a total of 82 open lung biopsies in patients with small vessel vasculitis of which 89% showed evidence of combined vasculitis and necrosis, granulomas and necrosis were found in 90%.[16] 59 transbronchial biopsies were performed in 48 patients and only four specimens had evidence of vasculitis and granulomas were identified in an additional three. Thus, the role of transbronchial biopsies in these patients is limited and open lung biopsies are more informative but carry a higher morbidity and mortality.

The incidence of DAH has beenreported as between 7-45% in Wegner's Granulomatosis (WG), and 10-30% in Microscopic Polyangitis (MPA).[3, 5, 6] The lungs are the most commonly affected organ in WG with evidence of involvement in over 90% of patients during the course of their disease; in 9% it is the only organ affected. [5,7] In MPA lung involvement is less

common than inWG, and occurs in up to 50% of cases during the course of the disease.[8]Pulmonary involvement ranges from subclinical changes on high resolution computed tomography to devastating haemoptysis. Approximately 5% of patients will have a fulminant presentation requiring assisted ventilation.

Treatment

Patients with DAH with or without glomerulonephritis, who are found to have ANCA positive can be generally assumed to have WG or MPA. The type of ANCA (PR3-ANCA or MPO-ANCA) found is irrelevant with respect to the initial management of this patients.[1] The backbone of therapy is the early identification of disease followed by the rapid induction of disease control with immunosuppression. Early recognition is crucial, because the prompt institution of supportive measures and immunosuppressive therapy is required for survival. The intensity of the initial treatment depends on the severity of the disease. Based on the European Vasculitis Study Group (EUVAS), which categorized the patients in groups according to the severity of their disease, the presence of DAH put the patient in the severe disease group.[17] The management of these patients is a combination of corticosteroid and cyclophospamide. S.L Hogan showed that cyclophosphamide reduces mortality and increase the likelihood of inducing remission in patients with ANCA-associated vasculitis. [18]

DAH is animportant cause of morbidity and mortality in ANCA- associated vasculitis, the mortality rate may reach 66%, which is six times greater than vasculitis without alveolar hemorrhage.[3,19,20,21] Based on the high mortality rate with DAH in ANCA-associated vasculitis, and reduction in mortality shown with cyclophosphamide, treatment with cyclophosphamide should be started as early as possible, based on the clinical presentation and the presence of ANCA, without waiting histological confirmation.

Key points

1. Patients with Wegner's Granulomatosis often present with diffuse alveolar haemorrhage. These patients must be treated promptly as delay in treatment results in high morbidity and mortality.
2. Lung biopsy is very helpful if it shows granulomatous inflammation and vasculitis however it lacks sensitivity and specificity.
3. Detection of C-ANCA with Proteinase-3 can substitute for biopsy in the diagnosis of WG in patients who present with diffuse alveolar haemorrhage.

Conclusion

DAH leading to acute respiratory distress syndrome is a rare and life threatening condition in adults with ANCA positive vasculitis. Patients with DAH with or without glomerulonephritis, who are found to have ANCA positive can be generally assumed to have WG or MPA, and diagnostic lung biopsy may be deferred. Early institution of treatment with prednisone and cyclophosphamide can significantly reduce morbidity and mortality.

Competing Interests
None declared

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Case Report

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An Unusual Cause of Chronic Dyspnoea

Fadi Seif and Lamia H. Ibrahim

Case presentation

A 73 year old lady presented for assessment of her recurrent right sided pleural effusion. She had a history of gallstones and underwent open cholecystectomy. One month after surgery the patient had recurrent pleural effusion requiring thoracocentesis on a monthly basis. On the chest x-ray, the pleural effusion was seen exclusively on the right side occupying the whole right hemithorax.

The pleural fluid was transudative on multiple occasions and there was no evidence of malignant cells. Her echocardiography revealed preserved cardiac function. An abdominal ultrasound showed findings of cirrhosis and splenomegaly consistent with portal hypertension.

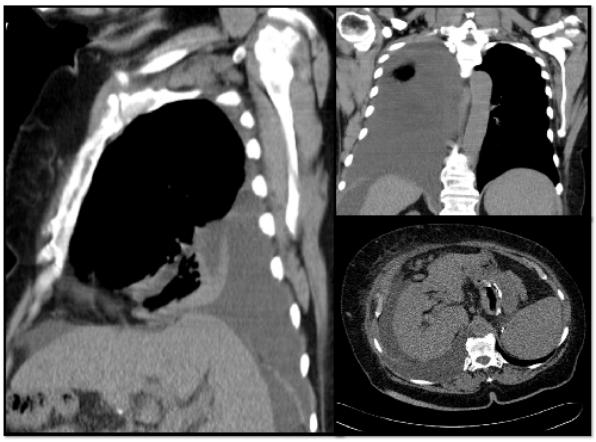

Image 1

Computerised tomography (CT) of the chest and abdomen revealed a large right-sided pleural effusion and minimal ascites (Image 1). An ultrasound guided paracentesis was performed with difficulty and only 17cc of fluid.was obtained. The abdominal fluid showed similar consistency as the pleural fluid. The blood workup at the same time was unremarkable.

Intra-peritoneal administration of 99mTc-sulphur colloid was attempted but failed in the absence of ascites. Computed tomography with three dimensional reconstruction at the diaphragmatic level revealed a defect in the posterior aspect of the right hemidiaphragm (Image 2 black arrow) and also revealed irregular contours of the liver, an indirect sign of diaphragmatic defect (Image 2 white arrow).

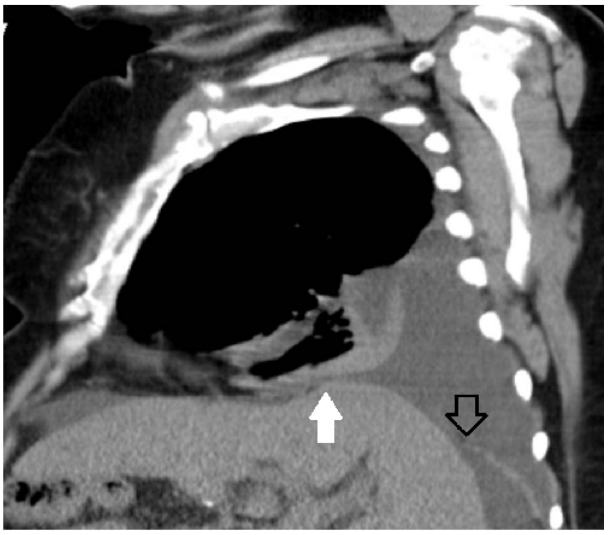

Image 2

The patient declined any surgical intervention at that point including the option of pleurodesis. She was started on diuretics and a low salt diet with significant improvement.

Discussion:

Pleural effusion due to hepatic cirrhosis and ascites is well known, but hepatic hydrothorax in the absence of ascites is a rare complication. We report a case of liver cirrhosis with a large and recurring right sided pleural effusion that had an apparent abdominal source in the absence of ascites. We review the characteristics and treatment for hepatic hydrothorax in the absence of ascites.

Hepatic hydrothorax is defined as the presence of significant pleural effusion in a cirrhotic patient without primary pulmonary or cardiac disease[1]. Postulated mechanisms for the development of pleural effusions in patients with hepatic cirrhosis include: hypoalbuminemia and decreased oncotic pressure leakage of the plasma from the hypertensive azygos vein, lymphatic leak from the thoracic duct, passage of ascitic

fluid to the pleural space by way of lymphatic channels in the diaphragm, and transfer of peritoneal fluid directly via diaphragmatic defects[2].

The usual unilaterality of hepatic hydrothorax could be attributed to a congenital factor, but not to physiologic mechanisms[3]. The most likely explanation appears to be that ascitic fluid passes through congenital or acquired fenestrations in the diaphragm directly into the pleural space[2]. The description of hepatic hydrothorax in the absence of ascites is very rare[1]. The flow of the ascitic fluid into the pleural space equaled the rate of ascites production in patients with this entity[3].

The composition of pleural fluid from hepatic hydrothorax is similar to that of ascitic fluid. Pleural effusions associated with portal hypertension are always transudative[1]. Nuclear scans can be performed to establish the diagnosis of hepatic hydrothorax with fairly high accuracy. Intra-peritoneal administration of 99mTc-human serum albumin or 99mTc-sulphur colloid can be used to demonstrate the communication between the peritoneal and pleural space. Recent advances in radiological imaging have enabled investigators to examine in detail the diaphragmatic defects responsible for the development of hepatic hydrothorax[1].

The management is challenging and frequently associated with poor outcomes in most cases. Dietary restriction of sodium intake and the addition of diuretics is the initial approach. Thoracocentesis can be performed in patients with dyspnoea due to hepatic hydrothorax for immediate relief of symptoms. When thoracocentesis is required too frequently in patients on maximal sodium restriction and optimal diuretics, alternative treatment options must be considered[1, 3].

Over the last few years, new insights into the pathogenesis of this entity have lead to improved treatment modalities such as portosystemic shunts (TIPS) and video-assisted thoracoscopy (VATS) for closure of diaphragmatic defects. Both, though temporary measures, are perhaps the best available bridging to liver transplantation in selected patients with refractory hepatic hydrothorax[2, 3].

Competing Interests
None declared
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Case Report

BJMP 2011;4(1):a407

COPD Exacerbation with Concurrent Stress Cardiomyopathy: A Case of Double Dyspnoea

Jennifer L. Pham , Steven R Bruhl and Mujeeb Sheikh

ABSTRACT
We present an interesting case of severe dyspnea due to chronic obstructive airway disease exacerbation and upon further evaluation a diagnosis of stress cardiomyopathy was entertained. We highlight a management of this particular case and provide a brief review of stress cardiomyopathy.

Case presentation

A 52 year-old Caucasian male with a history of chronic obstructive airway disease (COPD) presented to the emergency department complaining of progressive shortness of breath. Two days prior, the patient had presented to the ED with similar complaints that resolved with aerosol treatments and the patient was discharged on a metered dose inhaler (MDI). The patient had been prescribed MDI's (metered dose inhalers) previously for management of his COPD, but due to financial constraints he had been unable to fill his prescription for the past month. Emergency medical services (EMS) suspected COPD exacerbation and administered 40 mg prednisone IV and two albuterol-ipratropriumnebulisertreatments en route to the hospital, which improved the patient's breathing symptoms.

Upon arrival to the hospital, his blood pressure was 129/90, respirations 28, pulse 127, and he had an oxygen saturation of 100% on 7L/min. Physical examination revealed increased work of breathing, and wheezes in all lung fields with prolonged expiratory phase. The cardiovascular exam was normal except for tachycardia. A Routine electrocardiogram (ECG) revealed sinus tachycardia and T wave inversions in anterior leads. Chest x-ray showed old scarring in the left lower lobe. Routine cardiac enzymes showed mild elevation with a serum troponin level of 0.68ng/ml (normal range 0.0ng/ml-0.05ng/ml). The second set of troponin peaked at 1.66 ng/ml (normal 0.0ng/ml-0.05ng/ml). In view of the elevated cardiac enzymes atransthoracicechocardiogram was performed which demonstrated multiple wall motion abnormalities and reduced left ventricular ejection fraction of 25%. Coronary angiography demonstrated normal coronary arteries. Left ventriculography revealed hypokinetc mid-anterior and apical segment with a hypercontractile base with reduced ejection fraction (EF) of around 25% (normal range EF 55-65%) (Figure 1)

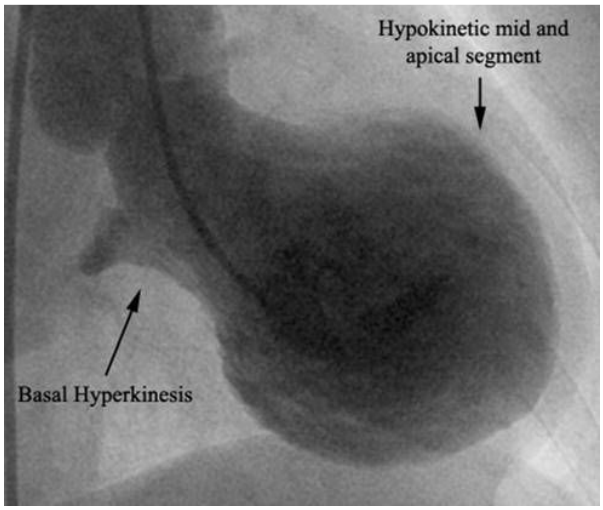

Figure 1. Left ventriculography demonstrating the classic appearance of Takotsubo cardiomyopathy

In light of the systolic dysfunction not in proportion with the degree of coronary artery stenosis and the multiple areas of wall motion abnormalities seen on echocardiogram, the diagnosis of Takotsubo cardiomyopathy (TCMP) was made. The diagnosis was further supported by the presence of ECG changes, troponin elevation, and the added social stresses of being unemployed. Over the course of his stay in hospital, the patient's breathing improved with oral prednisone, inhaled tiotropium, and fluticasone/salmeterol. The patient was also treated with an angiotension converting enzyme inhibitor (ACE inhibitor), aspirin, statin, and beta-blockers. There were no adverse coronary events during the course of his hospital stay and the patient was discharged after four days. A Follow up echocardiogram after 4 weeks showed normal left ventricular systolic function.

Discussion

Takotsubo cardiomyopathy (TCMP), also called stress-induced cardiomyopathy, apical ballooning syndrome, or broken heart syndrome, is a transient systolic dysfunction of the ventricles in the absence of significant coronary artery disease. Once thought to be a rare syndrome, TCMP is increasingly being identified in clinical practice, however, the prevalence and incidence are not known. It is estimated that 0.7-2.5% of patients who present with acute coronary syndrome are found to have TCMP[1] .The majority of these patients are postmenopausal females, with a mean age of 62-75 years. They may present with chest pain and have a recent history of an emotional stress or severe medical illness. [1]

The clinical manifestations of TCMP can mimic those of an acute myocardial infarction. Although, chest pain is a common presenting symptom, patients may also have complaints ofdyspnoeaand arrhythmias. In our casedyspnoeawas the predominant symptom and was easily confused with COPD exacerbation. Recently a few cases of concomitant stress cardiomyopathy with obstructive airway disease have been documented in literature. [2-4] While the pathophysiology of the coexistence of these two disorders is not fully understood, it is thought that both stress induced cardiac dysfunction due to exaggerated sympathetic activation and use of sympathomimetic bronchodilators instigates the myocardial stunning in such patients. Furthermore, an emotional stressor, such as death of a family member, or a physiological stressor, such as an acute medical illness, is thought to be a trigger for cardiomyopathy. [5] It is believed that the syndrome is not a result of anischemia, but there is some evidence to suggest thatoestrogenlevels may have a role in modulating the sympatho-adrenal outflow in TCMP. In mice models, chronic oestrogen supplementation seemed to have protective effects from exaggerated sympathetic outflow from the heart and brain[6] . Postmenopausal women with low levels ofoestrogenmay be more vulnerable to the exaggerated catecholamine release in responses to stressors.[7]

The characteristic finding in TCMP is a transient mid-ventricular or apical ballooning due to a hypokinetic portion seen on echocardiogram or on a left ventriculography. Systolic dysfunction is usually transient, inconsistent to the perfusion area of a single coronary artery, and usually resolves within 4-6 weeks. [8] Additional findings include ECG changes with ST segment deviations in precordial leads being the most common. Cardiac enzymes have been noted to have moderate elevations.[9.]

As data regarding the treatment of TCMP is limited, medical management mainly consists of symptomatic therapy with aspirin, ACE inhibitors, beta-blockers, and diuretics, also used in acute coronary syndrome.[10] Patients who present acutely are treated as acute coronary syndrome and often receive emergency coronary angiography. However, less invasive imaging techniques, such as echocardiograms, should first be examined carefully. Due to the transient nature of the syndrome, the duration of treatment is unknown with some studies suggesting that there is no benefit with chronic treatment. [11] The prognosis is fairly good, with in hospital mortality rates being reported to range from 0-8%, and recovery of left ventricular function in the majority of patients. [9, 12]

TCMP is difficult to distinguish from acute coronary syndrome on first presentation. Our patient had significant social stress. She presented with severedyspnoeaand was treated for COPD exacerbation. Elevation of cardiac enzymes and ECG changes lead to further evaluation and diagnosis of stress cardiomyopathy. This atypical presentation of TCMP showcases the importance ofutilisingthe routine noninvasive imaging and laboratory values to guide the diagnosis. Furthermore physicians need to maintain a high clinical suspicion for this syndrome.

Competing Interests
None Declared
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Case Report

Theophylline Toxicity – A Forgotten Entity

N Altaie , S Malik and S Robertson

Introduction

It is often forgotten that smoking inducescytochrome P450 (CYP) 1A2, resulting in altered concentrations and required doses of drugs metabolized by this route. Conversely, upon cessation of smoking, concentrations of these drugs can rise to toxic levels unless appropriate dose adjustments are made. Medical staff, and others involved with smoking cessation counselling, need to be aware of this if potential harm to patients is to be avoided. Here, we describe a patient who developed tonic clonic seizures due to Theophylline toxicity, having ceased smoking 2 weeks earlier.

Case Report

Our patient is a 76-year-old lady who presented to A&E feeling generally unwell, with a two day history of dizziness. Her medical history included atrial fibrillation, which was treated with Digoxin 62.5mcg, and Chronic Obstructive Pulmonary Disease, for which she took a combination inhaler of 25mcg Salmeterol Xinafoate and 250mcg Fluticasone Propionate, 2 puffs twice a day, plus Theophylline MR 175mg twice daily. She had successfully given up smoking 2 weeks prior to admission, having smoked 100 cigarettes per day over the previous 40 years. On admission, she was in atrial fibrillation, well controlled with a heart rate of 90 beats per minute, and normotensive, with no evidence of postural hypotension. Respiratory system examination revealed prolonged expiration, in keeping with COPD, but the rest of the examination was unremarkable. Routine blood investigations, including full blood count, urea, creatinine and electrolytes, liver function tests and C-reactive protein, were all normal apart from a serum potassium level of 3.0mmol/l (NR 3.5-5.0mmol/l). Theophylline concentrations were not tested at this point. A chest X-ray showed hyper-inflated lung fields in keeping with Chronic Obstructive Pulmonary Disease.

The patient was admitted for observation, and treated with Trimethoprim for a presumed urinary tract infection on the basis of urinalysis, which was positive for leukocytes and nitrites.

Two days following admission, the patient developed facial spasms and twitching of her muscles of her upper limbs. Over the next 24 hours, the patient had two witnessed tonic-clonic seizures, which were terminated with intravenous Lorazepam. An urgent CT (Computed Tomography) scan of the brain was performed. This showed changes in keeping with small vessel disease only, nothing to account for new onset of seizures. Following a post-ictal period, the patient recovered, but then the following day had a further two tonic-clonic seizures. It was at this point that a blood Theophylline concentration was requested.

The Theophylline concentration was reported as 41.6mcg/ml (NR 10-19.9mcg/ml), more than twice the upper safe therapeutic concentration. A search of the laboratory system revealed that this patient's Theophylline concentration had been within the therapeutic range when last checked 2 months prior to admission, while she was still smoking.

The Theophylline was immediately stopped and the patient closely monitored at the Medical High Dependency Unit for further fits, arrhythmias or electrolyte disturbances. Other causes of seizures had been investigated and excluded.

The patient's neurological symptoms improved dramatically following cessation of Theophylline, with no further twitching, muscle spasms or seizures. Within three days her Theophylline concentration returned to a safe level, but the drug was not recommenced. Unfortunately, however, the patient died from sepsis two weeks following her admission without having left hospital.

Discussion

Theophylline has largely fallen out of favour as a treatment for airways disease due to its very narrow therapeutic index. Even within the therapeutic range, side effects frequently occur. These side effects range from mild, including tremor and gastrointestinal disturbance, to serious and potentially life threatening, such as seizures and cardiac arrhythmias. Following acute overdose, hypokalemia, hyperglycemia, hypercalcemia, hypophosphatemia, and acidosis commonly occur.

Theophylline is mainly metabolised in the liver by demethylation or oxidation using the cytochrome P450 system.

The 8-hydroxylation of Theophylline to 1,3-dimethyluric acid (1,3-DMU) via cytochrome P450 1A2 is the major pathway.

The cytochrome P450 enzyme CYP1A2 mediates the rate-limiting step in the metabolism of Theophylline[1], andthe polycyclic aromatic hydrocarbons found in cigarette smoke are potent inducers of this enzyme[2]. For this reason, smokers may need up to double the dose of Theophylline to achieve therapeutic effect compared with non-smokers[3].

The relationship between smoking cessation and Theophylline has also been the subject of many studies. Lee et al demonstrated that stopping smoking for 1 week resulted in a 37.6% decrease in clearanceand a 35.8% increase in the half-life of Theophylline, and that the dose needs to be reduced by one fourth to one third after brief abstinence from tobacco to prevent potentially toxic concentrations[4]. For this reason, careful monitoring of plasma Theophylline concentration should be considered essentialfor optimal dosing in patients following smoking cessation. The study by Faber et al recommends that the dose of CYP1A2 substrates such as Theophylline should automatically be reduced by 10% on cessation of heavy smoking and thereafter be guided by plasma concentration monitoring[5].

We found one report in which Theophylline toxicity resulted in a patient's death following a similar presentation to the one described above[6]. This highlights the close attention that must be paid to drug concentration monitoring by physicians when advising patients to quit smoking.

Theophylline is not the only drug which needs to be considered when patients stop smoking. Other examples are Clozapine, Olanzapine, Haloperidol and Flecanide to name a few. These drugs are also substrates for CYP1A2[7].

Conclusion

When advising patients to stop smoking, it is essential that physicians routinely review the drugs that the patient is taking to look for those that may require dose adjustments. In the case of Theophylline, careful monitoring of Theophylline concentrations, for instance weekly in the first few weeks following smoking cessation, is essential to avoid potentially life-threatening complications.

Competing Interests
None Declared
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Case Report

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Painless aortic dissection presenting with congestive heart failure

Usman Ali , Wai Hang Cheung and Ashis Banerjee

ABSTRACT

A 44 year old man, previously in good health, presented with congestive heart failure, the onset of which was probably four weeks previously. A diagnostic label of community acquired pneumonia led to delay in the diagnosis of type A aortic dissection.

This required surgical management which resulted in a good outcome. The absence of chest pain may have contributed to the delay in diagnosis. Aortic dissection should form part of the differential diagnosis of unexplained acute congestive heart failure.

Case History

A 44 year old male presented to the emergency department complaining of shortness of breath. The symptoms had commenced suddenly four weeks ago. He had been breathless at rest, and subsequently developed a productive cough with white sputum. He denied chest pain. He was known to have the sickle cell trait but was otherwise in good health. He was a non-smoker.

Since the onset of symptoms, and prior to this admission, the patient presented to two different emergency departments. The working diagnosis was, and remained, community acquired pneumonia. On initial presentation empirical treatment for a community acquired pneumonia was commenced. Failure to improve resulted in additional cover for atypical organisms and the prescription of a short course of steroids on the subsequent admissions.

Initial observations revealed the patient was tachypnoeic and tachycardic, with a respiratory rate of 25 breaths per minute, heart rate of 114 beats per minute. He was apyrexial (temperature of 36.5°C). Pulse oximetry showed an oxygen saturation of 94% on room air. His blood pressure was recorded as 183/99 millimetres of mercury.

On examination large volume peripheral pulses, raised jugular venous pressure (5 cm), bi-basal crepitations, and bilateral ankle oedema were elicited/identified. Auscultation of the heart revealed a loud diastolic murmur audible throughout the praecordium.

A 12 lead ECG showed normal sinus rhythm, normal axis and left ventricular hypertrophy. Arterial blood gas analysis on room air showed a pH of 7.46, pa02 9.6 kPa, pCO2 4.3 kPa, HCO3 23.8 mmol/L, BE + 0.8 and lactate of 0.7 mmol/L. Routine venous blood tests did not identify any elevated markers of infection or inflammation. A chest radiograph (Figure 1) showed cardiomegaly and pulmonary oedema.

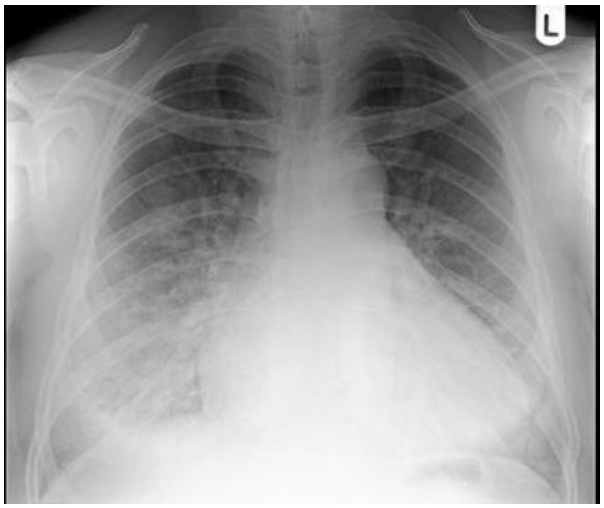

Figure 1

The patient was administered oxygen and given a diuretic to improve his ventilation.

The working diagnosis was congestive cardiac failure in the presence of what was presumed to be a new murmur. Urgent echocardiography revealed an aortic root of 6.2cm diameter at sinus level, with an evident dissection flap. There was no obvious haematoma. Severe free flowing aortic regurgitation, a dilated hyperdynamic left ventricle and a 0.7 cm diameter pericardial effusion anteriorly were also noted. It was concluded that the patient had a sealed 7cm type A aortic dissection. This was confirmed by a CT scan (Figure 2).

Large bore venous access was obtained and an intravenous beta blocker (Labetalol) administered. Urgent transfer to a tertiary cardio-thoracic surgical centre was made. He underwent aortic root and valve replacement, along with coronary artery bypass grafting to the right coronary artery using a reversed long saphenous vein graft. Postoperatively, he was anticoagulated on Warfarin, and was also placed on beta blockade therapy

(Bisoprolol), a diuretic (Frusemide), an ACE inhibitor (Ramipril), and a statin (Simvastatin).

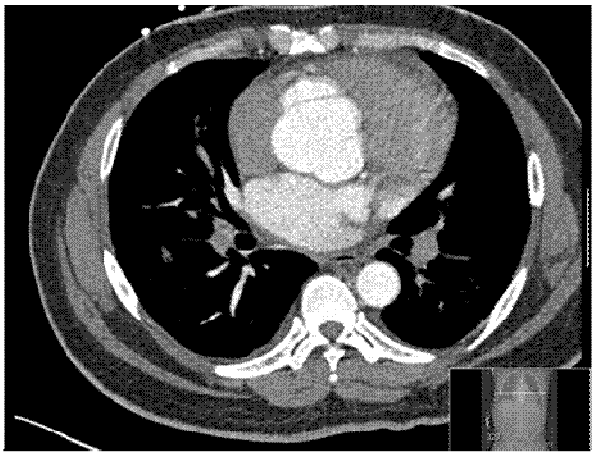

Figure 2

Discussion

Aortic dissection is a medical emergency. If left unrecognised or untreated mortality can be as high as 80% in two weeks, or 90% within three months[1,2]. 96 % of patients with aortic dissection present primarily with chest pain. The remaining 10% present with symptoms secondary to impairment of blood supply to other organ systems[3]. Dissections involving the ascending aorta present with retrosternal chest pain, while interscapular pain suggests involvement of the descending aorta. Pleuritic pain may indicate haemorrhage in the pericardial sac, with the potential for acute cardiac tamponade.

Only 6% of aortic dissections present with acute congestive cardiac failure. Patients presenting with aortic dissection and congestive cardiac failure are more likely to present without chest pain and have a valvular abnormality. When chest pain is present, the pain is more often mild and less likely to be abrupt in onset. Patients are less likely to be hypertensive on presentation and more likely to present in shock. These patients are more likely to have Stanford type A dissection . Congestive cardiac failure does lead to a delay in surgical intervention[4].

Congestive cardiac failure is usually due to aortic regurgitation from aortic valve disease, incomplete aortic leaflet closure, or aortic valve disruption. In the setting of unexplained cardiac failure aortic dissection should be considered, especially when an aortic regurgitant murmur has been detected clinically. Heart failure has been associated with supravalvular aortic stenosis in the presence of a painless type A dissection, in a patient presenting with persistent cough[5]. Rupture of aortic dissection into the right atrium, right ventricle, or main pulmonary artery may lead to a left to right shunt and congestive heart failure[6].

Painless aortic dissection has been recorded in other contexts, particularly with chronic dissection and in patients with Marfan's syndrome.The absence of chest pain should not exclude aortic dissection.

Competing Interests
None declared

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Clinical Practice

Incidental adnexal mass at Caesarean section - the value of implementing a comprehensive consenting process

Ingrid Paredes , Marlon Pastrana , Alasdair Gordon and Toh Lick Tan

ABSTRACT
Informed consent is an important part of good medical practice. Potentially, added, but not essential procedures may only become obvious during surgery. Therefore comprehensive consent to cover such a situation is advisable. In this report, we illustrate the value of a standardised consent form which addresses the issue.

Introduction

Examination of the ovaries at caesarean section is a normal practice as ovarian pathology may be found. The incidence of an adnexal mass found at caesarean section ranges from 1 in 123 [1] to 329 [2]. Ovarian cysts rarely develop de novo in late pregnancy, but rather persist from early pregnancy. About 4 in 5 ovarian cysts detected in the first trimester scan resolve spontaneously. Also, 4 in 5 of ovarian cysts persisting into the second trimester will also be present in the post-natal period as complex cysts such as serous cystadenomas, mature cystic teratomas, endometriomas and mucinous cystadenomas [3]. It therefore seems sensible to remove the ovarian cyst for histology at caesarean section rather than subject the woman to the anxiety of multiple investigations and/or another laparotomy, particularly when ovarian cystectomy during caesarean section does not appear to increase morbidity of the procedure [1].

We present a case of incidental ovarian cyst found at elective caesarean section to illustrate the value of a comprehensive consenting process.

Case Report

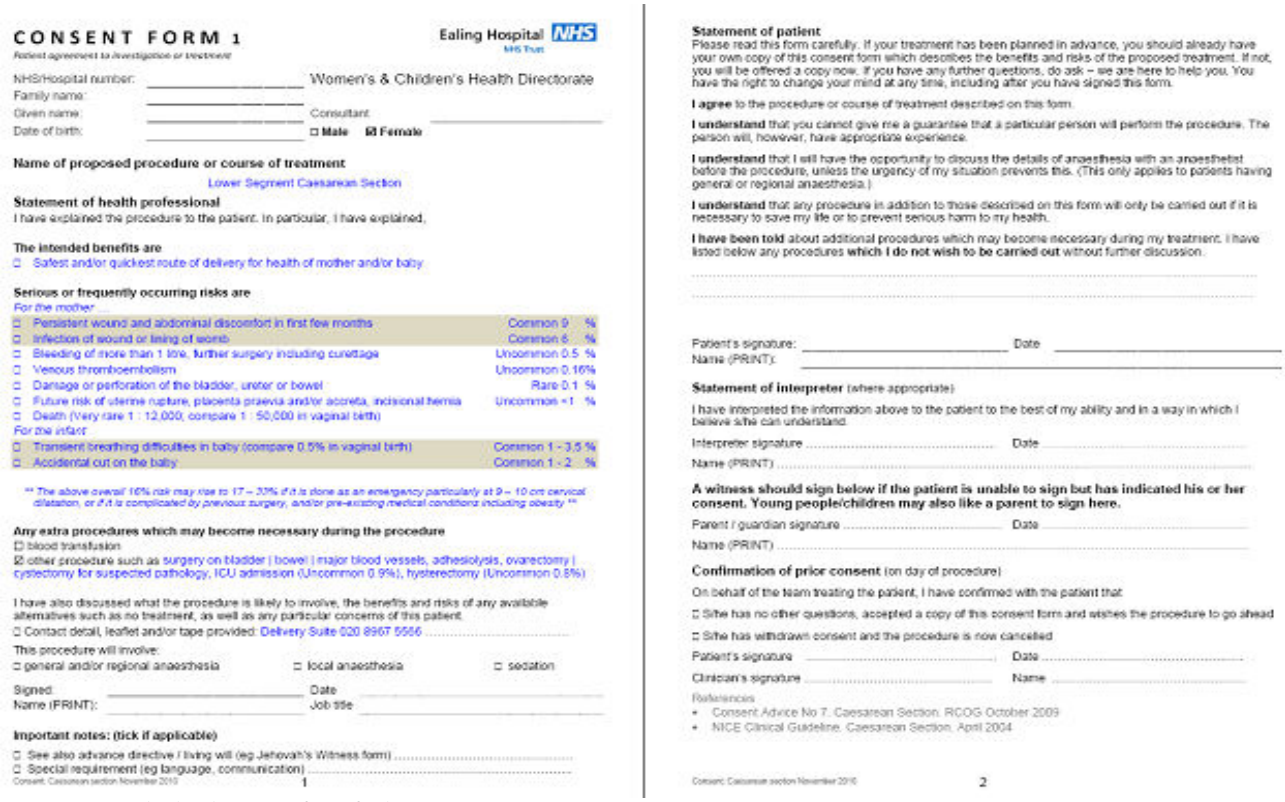
A 35 year-old para 1 + 0 healthy Polish woman was admitted for elective lower segment caesarean section (LSCS) at 39[+4] weeks gestation in view of a previous caesarean section 2 years ago for failure to progress in the first stage of labour.She was booked in a neighbouring hospital for her antenatal care where she was counselled and consented for the procedure by her consultant. Her pregnancy was uncomplicated and routine pregnancy scans were unremarkable. Apart from drainage of a breast abscess 2 years ago, she had no medical history of note.

Written consent for elective LSCS was obtained by the junior doctor on duty before the consultant pre-operative ward round. However, the directorate's standardised consent (figure 1) form was not used. The woman was therefore again counselled and written consent for elective LSCS obtained for the third time now including previously omitted additional procedures that might be performed during the course of the surgery.

At the uncomplicated LSCS under spinal anaesthetic, routine inspection of the uterus and adnexa revealed a 30 x 20 x 15 mm pedunculated firm pale mass attached to the left ovary suggestive of a fibroma. The findings were relayed to the woman, and confirmation of consent for the ovarian cystectomy was obtained. The abnormal ovarian mass was removed with conservation of the left ovary. Histology of the mass subsequently confirmed it to be an ovarian fibroma / fibrothecoma.

Discussion

The Royal College of Obstetricians and Gynaecologists (RCOG) recommend that clinicians should seek prior consent to treat any problem which might arise [4]. Indeed, in its Consent Advice for caesarean section, it states that discussion of appropriate but not essential procedures, such as ovarian cystectomy at caesarean section, should take place before undertaking the procedure [5]. This supports the position of the Department of Health which states that a procedure should not be performed merely because it is convenient, and that it is good practice where possible to seek the person's consent to the proposed procedure well in advance, when there is time to respond to the person's questions and provide adequate information [6].



CONSENT FORM 1

Ealing Hospital NHS
NHS Trust

Patient agreement to investigation or treatment

NHS/Hospital number:
Family name:
Given name:
Date of birth:

Women's & Children's Health Directorate

Consultant

☐ Male ☑ Female

Name of proposed procedure or course of treatment

Lower Segment Caesarean Section

Statement of health professional

I have explained the procedure to the patient. In particular, I have explained,

The intended benefits are

☐ Safest and/or quickest route of delivery for health of mother and/or baby

Serious or frequently occurring risks are

For the mother

☐ Persistent wound and abdominal discomfort in first few months	Common 9 %
☐ Infection of wound or lining of womb	Common 6 %
☐ Bleeding of more than 1 litre, further surgery including curettage	Uncommon 0.5 %
☐ Venous thromboembolism	Uncommon 0.16%
☐ Damage or perforation of the bladder, ureter or bowel	Rare 0.1 %
☐ Future risk of uterine rupture, placenta praevia and/or accreta, incisional hernia	Uncommon <1 %
☐ Death (Very rare 1 : 12,000; compare 1 : 50,000 in vaginal birth)	

For the infant

☐ Transient breathing difficulties in baby (compare 0.5% in vaginal birth)	Common 1 - 3.5 %
☐ Accidental cut on the baby	Common 1 - 2 %

*** The above overall 16% risk may rise to 17 – 33% if it is done as an emergency particularly at 9 – 10 cm cervical dilatation, or if it is complicated by previous surgery, and/or pre-existing medical conditions including obesity ***

Any extra procedures which may become necessary during the procedure

☐ blood transfusion
☑ other procedure such as surgery on bladder / bowel / major blood vessels, adhesiolysis, ovarectomy / cystectomy for suspected pathology, ICU admission (Uncommon 0.9%), hysterectomy (Uncommon 0.8%)

I have also discussed what the procedure is likely to involve, the benefits and risks of any available alternatives such as no treatment, as well as any particular concerns of this patient.

☐ Contact detail, leaflet and/or tape provided: Delivery Suite 020 8967 5566

This procedure will involve:

☐ general and/or regional anaesthesia ☐ local anaesthesia ☐ sedation

Signed: Date
Name (PRINT): Job title

Important notes: (tick if applicable)

☐ See also advance directive / living will (eg Jehovah's Witness form)
☐ Special requirement (eg language, communication)

Consent: Caesarean section November 2010 1

Statement of patient

Please read this form carefully. If your treatment has been planned in advance, you should already have your own copy of this consent form which describes the benefits and risks of the proposed treatment. If not, you will be offered a copy now. If you have any further questions, do ask – we are here to help you. You have the right to change your mind at any time, including after you have signed this form.

I agree to the procedure or course of treatment described on this form.

I understand that you cannot give me a guarantee that a particular person will perform the procedure. The person will, however, have appropriate experience.

I understand that I will have the opportunity to discuss the details of anaesthesia with an anaesthetist before the procedure, unless the urgency of my situation prevents this. (This only applies to patients having general or regional anaesthesia.)

I understand that any procedure in addition to those described on this form will only be carried out if it is necessary to save my life or to prevent serious harm to my health.

I have been told about additional procedures which may become necessary during my treatment. I have listed below any procedures **which I do not wish to be carried out** without further discussion.

Patient's signature: Date
Name (PRINT):

Statement of interpreter (where appropriate)

I have interpreted the information above to the patient to the best of my ability and in a way in which I believe s/he can understand.

Interpreter signature Date
Name (PRINT)

A witness should sign below if the patient is unable to sign but has indicated his or her consent. Young people/children may also like a parent to sign here.

Parent / guardian signature Date
Name (PRINT)

Confirmation of prior consent (on day of procedure)

On behalf of the team treating the patient, I have confirmed with the patient that:

☐ S/he has no other questions, accepted a copy of this consent form and wishes the procedure to go ahead

☐ S/he has withdrawn consent and the procedure is now cancelled

Patient's signature Date
Clinician's signature Name

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- Consent Advice No 7: Caesarean Section. RCOG October 2009
- NICE Clinical Guideline: Caesarean Section. April 2004

Consent: Caesarean section November 2010 2

Figure 1. Standardised consent form for lower segment caesarean section

In spite of the publication of the above guidelines well over a year ago, our case supports the belief that most obstetricians omit discussion and/or documentation of ovarian cystectomy at LSCS, and indeed other risks or additional procedures that may be relevant as showed in figure 1. This may be because the clinician is unaware of the recommendations, not familiar with the potential risks or findings at surgery, or that there is simply insufficient time to document comprehensively.

Our directorate has adopted the use of standardised consent forms for common procedures. These forms are available on our intranet which can be edited allowing clinicians to amend the risks and additional procedures as appropriate in each individual case. We believe the verified printed consent form offers legible and comprehensive documentation of the counselling process, as well as prompting clinicians to discuss key issues such as those recommended by the RCOG Consent Advice. We advocate the use of such standardised consent forms in improving the care of patients and supporting clinicians to deliver optimal services.

Competing Interests
None declared

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Education and Training

An analysis of the learning needs of undergraduate medical students in a developing country: the learning needs are similar to students in the West, but resources differ

Mahmood Tariq and Memon Abdul Razak

ABSTRACT

Objective: To explore the perception of undergraduate medical students in developing world regarding teaching and learning of various basic clinical skills, identifying their learning needs and directing resources of the university accordingly.

Design / Setting: A questionnaire study implemented at the time of completion of voluntary clinical skills course for four weeks at the attached Liaquat University of Medical and Health Sciences, Hyderabad, Pakistan.

Participants: All students who went through the course during consecutive sixteen months commencing from October 2008 and ending in January 2010.

Results: History taking and laboratory investigations were deemed least important by students, perhaps because these are taught to them during normal attachments in all wards. The Friedmans mean rank was highest for passing of a naso-gastric tube (12.24) and catheterisation of urinary bladder (11.66). The students felt the greatest need to learn these two invasive procedures because they are not taught elsewhere, followed by equipments and drugs used in anaesthesia, sterilisation and inserting an intravenous cannulation. The students ranked the learning of the following skills in the middle: giving intra-venous and intra-muscular injections, taking blood, providing pre or post-operative care (mainly pre-operative fitness assessment and monitoring heamo-dynamic stability post operatively), and being able to identify surgical instruments. Asymptotic as well as Monte-Carlo significance was very high. ($p<0.000$)

Conclusions: Student's views should form a key part of design when considering development of a skills course and resources should be geared to meet these student learning needs.

KEYWORDS

Simulators, Medical Education, Skills course, skills training, learning needs

Background:

Controlled teaching environment can be provided to undergraduate medical students for learning certain basic clinical skills through using manikins or models before they have to perform them on real patients. Many medical schools in the United Kingdom now have clinical skills laboratories which are equipped with a large host of such learning resources. Many medical schools promote their clinical laboratories and have dedicated lead consultants to teach, monitor and develop clinical skills. Furthermore, a wide variety of skills can be taught in these laboratories. For example, the University of Leeds Medical school offers teaching to second year undergraduate medical students in basic life support, vital signs, injections, blood glucose monitoring, cannulation and venepuncture while third year medical students are taught fundoscopy, rectal examination, urinary bladder catheterisation, doing an ECG and examination of the breast. Similarly, simulators have also been used in postgraduate medical teaching, for example, Colonoscopy simulators have been used to calculate efficiency ratio of learners. [1]

However not all places in the world have clinical skills laboratories. Alternatively, some institutions teach the undergraduate medical students various basic skills in clinical supervised settings before they take up their first job as physicians. Some have assessed the level of training that their institutions have thus offered and tried to improve upon deficits. Mario Sicaja et al [2] from Zagreb University evaluated 252 students using a questionnaire asking the students to self assess their abilities on nine groups of skills and asked 129 faculty teachers to simultaneously assess the minimum necessary level of skills they expected from the graduating students. They concluded that the teachers expected higher level of clinical skills from students than that assessed by the students. Similarly, in postgraduate teaching, the students' learning needs have been assessed by determining the difference in expectation of trainers and the trainees.[3]

Effectiveness of basic clinical skills training programmes has been documented and it has been suggested that longitudinal skills training offers superior preparation for abilities during the clerkship [4]. It has also been suggested that whereas students from medical schools using traditional curriculum may not differ in their knowledge based performance, (demonstrated by Multiple Clinical Questions i.e. MCQ scores) from students at medical schools with clinical skills training, the later perform better on clinical examination (measured by Objective Structured Clinical Examination i.e. OSCE). [5]

However, in many other medical schools worldwide, which implement the traditional undergraduate medical curriculum, there are no clinical skills teaching sessions for undergraduate medical students. The students get to learn this first time when

they are doing internship and are in direct contact with the patients. It was the same situation in Liaquat University of Medical and Health Sciences (LUMHS) in Hyderabad, Pakistan. This is a large medical school which is based in the second largest city of Sindh province of Pakistan. The first author was invited from United Kingdom to review and advise on the undergraduate medical curriculum at LUMHS. The professor of plastic surgery (second author) had started to run a voluntary clinical skills course in his department covering some general basic clinical skills and provided his data to the first author for analysis and review.

This study was devised to analyse the views of participating students regarding the course and determine their learning needs. Based on the learning needs, one could identify which resources are needed and what the University should aim to provide. Furthermore, there are no such published studies from Pakistan. This study was aimed at providing scientific information on the learning needs of undergraduate medical students from the developing world, which may be deemed to be different from the medical students in the western world.

Methods:

All medical students from fourth and final year at LUMHS were invited to attend four weeks of clinical skills course at the plastic surgery department voluntarily. The students had to attend the department after their normal working hours. There were dedicated junior doctors in the department who were given the responsibility to teach the attendees hands on clinical skills in a structured manner. The skills included history taking, organising blood tests, vene-puncture, giving intravenous and intra-muscular injections, intravenous cannulation, urinary bladder catheterisation, passing naso-gastric tube, dressing of surgical wounds, basic pre-operative assessment, basic post-operative assessment including haemo-dynamic stability, surgical theatre mannerism, principles of sterilization, identification of common surgical instruments and equipment and identification of types of drugs used in anaesthesia. The students were provided a questionnaire asking for feedback on the course which they had to fill at the end. The study was approved by the University Research and Ethics Committee. The data was computerised and statistically analysed using a statistical package. It involved all students who went through the course during consecutive sixteen months commencing from October 2008 and ending in January 2010.

Results:

90 students were recruited to the study. Students were from both sexes and both fourth year and final year. There were 32 male (35.6%) and 58 female (64.4%) students. Of the total 90 students, 62 (68.9%) were from 4th year while 28 (31.1%) students were from final year. They were all volunteers who were willing to attend the course after their normal working hours and were allocated seats on a first come first serve basis. No student was refused entry to the course and all participants were provided questionnaire on feedback at the time of completion of the course. The response rate was 100% although this may be because students were actively encouraged by the teaching staff to ensure that feedback questionnaires were filled in.

History taking and laboratory investigations were deemed least important by students, perhaps because these are taught to them during normal attachments in all wards for clinical teaching.

Ranks

	Mean Rank
History taking	6.22
Lab investigations	6.22
Venesection	6.29
Giving injection	6.68
I/v cannulation	7.46
Catheterisation	11.66
Naso-gastric intubation	12.24
Dressing wound	6.29
Pre-operative assessment	6.71
Theatre environment	6.53
Post-operative assessment	6.62
Principles of sterilisation	7.73
Types of anaesthesia	7.81
Surgical instruments	6.54

Test Statistics [a]

N	90
Chi-Square	615.431
Df	13
Asymp. Sig.	0.000
Monte Carlo Sig.	0.000
99% Confidence Interval Lower Bound	0.000
99% Confidence Interval Upper Bound	0.000

a : Friedman Test

The Friedman mean rank was highest for catheterisation of urinary bladder (11.66) and passing of a naso-gastric tube (12.44). The students felt the greatest need to learn these two basic clinical skills perhaps because they are not taught elsewhere. This was followed by anaesthesia, sterilisation and passing an intravenous cannulation. The students ranked learning of the learning of the following skills in the middle: giving injections, taking blood, providing pre or post-operative care and being able to identify surgical instruments. Taking

history and arranging laboratory investigations were both ranked the lowest at 6.22. The Friedman asymptotic significance was high (p<0.000). Despite being a good sample size, Monte Carlo significance at confidence interval of 99% was very high (p<000.0). Tables above summarise the statistics

Discussion:

Our study has shown that undergraduate medical students from the developing world greatly value a basic clinical skills course, and are particularly keen on being taught naso-gastric intubation and urinary bladder catheterisation. They seem to get enough exposure in the wards on history taking and arranging laboratory tests, but identify learning needs in other clinical skills.

This study is limited to data collection from one large medical university, but the sample size has been large, observation has been over a period of one year, statistical significance has been very high and response rate has been extremely good. The teaching staff actively encouraged the students to fill the feedback questionnaires, and this could arguably lead to some response bias.

There are no such previous studies from Pakistan to compare our findings with. This study therefore can make a good baseline for local institutions to further develop and build upon. Roy Remmen's group compared four medical schools on clinical skills of students, and demonstrated positive effect of both longitudinal skill training as well as utilisation of problem based approach in these skill courses. Our study did not provide any longitudinal data and problem based learning approach was not utilised either. Our data is a cross sectional study.

There seem to be three levels of engagement in learning basic clinical skills. One side of the spectrum has structured teaching in clinical skills laboratories with simulation, models and manikins, while the other has no teaching of clinical skills at all, until the physician starts to work with real patients. In the middle is the model of teaching clinical skills on the wards, before graduating as doctors. The former model with clinical skills laboratories requires the most resource. Which model of teaching is adopted by any individual medical university may be dependant upon the local resources, as well as the demands of the local regulatory bodies. During this study, we were able to realise the pattern of clinical skills teaching at some other medical universities in Pakistan, India and Bangladesh as random examples of southeast Asia. We learned that most institutions in this part of the world do not undertake any formal clinical skills teaching, and certainly there are hardly any clinical skills laboratories. This voluntary attempt by the professor of plastic surgery at LUMHS is therefore commendable.

This study has also identified the keenness of students to learn some specific skills through such courses prior to graduation. With a move to more globalisation of medical protocols and guidelines, a greater uniformity should also emerge in the ways in which doctors in the east or the west hemisphere of the world learn medical knowledge, attitudes and skills. There may thus be need for researchers in medical education to encourage and push for adoption of clinical skills teaching courses prior to medical graduation in the developing world.

Furthermore this study has yet again reiterated that student's views should form a key part in the curriculum design when considering development of a clinical skills course, and resources should be geared to meet these learning needs of students.

Competing Interests
None declared
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E-Interview

Interview with Dr James Moon

Dr James Moon is a Senior Lecturer and Consultant Cardiologist at UCL and the Heart Hospital. He set up and runs the cardiac MRI department dividing his time between clinical practice and research.

He is part of a team of 5 research fellows in the new Heart Hospital Imaging department. He is interested in understanding the structure and function of the heart, particularly the heart muscle, and in detecting abnormalities of the heart to better target treatment.

How long have you been working in your speciality?

12 years (3 as consultant)

Which aspect of your work do you find most satisfying?

The creative aspects of research - joining the dots on information that does not fit and constructing a coherent body of work.

What achievements are you most proud of in your medical career?

Changing statin prescribing in England – as a registrar, I did not having access to cardiac MRI for 2 years and I worked relentlessly at all levels of the healthcare system –including up to Commons Health Select Committee - on this with the result that £1billion was saved or diverted to treat more individuals with statins – the UK now has the highest uptake of statins for primary prevention in the world.

Developing new ways of detecting different types of disease with MRI or CT scanners – in its latest iteration, we may be onto a technique that can measure a fundamental process common to most diseases and organs – not just the heart, and with CT as well as MRI: the volume of cells, fibrosis and their ratio.

Which part of your job do you enjoy the least?

New bureaucracy which we did without just a few years ago..

What are your views about the current status of medical training in your country and what do you think needs to change?

I worry about a tickbox 'learning portfolio' culture which dumbs down initiative and personal responsibility leaving a misplaced sense of entitlement.

How would you encourage more medical students into entering your speciality?

I've not seen the need to – cardiology is a fantastic, over-subscribed specialty with something for everyone so its pretty competitive.

What qualities do you think a good trainee should possess?

The same as those of a doctor. I have never seen this trainee:consultant divide; there is a continuum of learning and responsibility development.

What is the most important advice you could offer to a new trainee?

My advice is about learning rather than being a trainee. Medicine does not have that many raw facts to learn. What it does have is interconnected systems. Rarely consciously try and learn information – rather, try and link everything you have ever learned together, preventing isolated islands of knowledge. It takes longer to create the story, but you will never forget it and it's far more rewarding. If you encounter something new - a tricky JVP waveform, an ECG repolarisation abnormality or

some esoteric MRCP clinical sign, invoke your know of the physical world and apply it to explain the new phenomena – write the essay, deconvolute the phenomena and build it back up, perhaps with subtle changes to see where that gets you. You have spent decades learning about the Krebb's cycle, anatomy, electron transport, fractals, Newtonian dynamics, Brownian motion, fluid dynamics, conservation of energy, entropy, cell structure, evolutionary biology, statistics etc etc – use them.

What qualities do you think a good trainer should possess?

I am not sure I know, but generating enthusiasm in people, and then rewarding and promoting it - that's a good starting point.

Do you think doctors are over-regulated compared with other professions?

No.

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?

It's the effects on individuals that concern me. I fully understand the need for process, protocol teamwork and hierarchy, but these remove individual responsibility

Which scientific paper/publication has influenced you the most?

The non-medical maths/science/philosophy books and magazines I read at school and university. I particularly remember Martin Gardner recreational maths books. Recently I have used fractals and the concepts behind trapdoor ciphers in my understanding of cardiology.

What single area of medical research in your speciality should be given priority?

Prioritize individual researchers/teams rather than topics to create progress through their enthusiasm and own perceptions of priorities.

What is the most challenging area in your speciality that needs further development?

Managing our increasing technical capability which comes with ever reducing incremental benefit.

Which changes would substantially improve the quality of healthcare in your country?

I would overhaul the way society pays for and develops drugs. I would focus on increasing drug company reward for the risk associated with genuine innovation whilst reducing reward for expensive 'me too' drugs with no added value. My group estimated that about 10% of the NHS drug budget could immediately be reallocated improving societal value for money in prescribing, paying for all those much needed NICE decision cancer type drugs and concurrently turbocharging rather empty pharmaceutical drug development pipelines.

Do you think doctors can make a valuable contribution to healthcare management? If so how?

Absolutely. If you have transparently good and altruistic ideas, are selfless about who gets he credit for them, and sufficiently driven to achieve results, the NHS is a wonderful place - its like a demagnetized iron – apply a sufficiently persuasive external field, and the domains line up, generating far more force and direction than expected.

How has the political environment affected your work?

The UK has been great for my field –new techniques are adopted early and the international bane of my field - cardiology-radiology turf wars are less acrimonious here as socialized medicine does not reimburse on a pay per procedure basis..

What are your interests outside of work?

My young family, recreational science, cooking.

If you were not a doctor, what would you do?

Who knows. Perhaps an economist or maybe evolutionary biologist.