Case Report

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Pericardial effusion unmasked SLE in a young schizophrenic male : A case report

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Abstract

Systemic Lupus Erythematosus (SLE) is characterised by its multi-systemic involvement and has a chronic remitting and relapsing course. It can involve the nervous system on its central or peripheral components. While the prevalence of SLE is highest among females aged 14 to 64 years, males are not immune. The risk of SLE development in men is similar to that in the pre-pubertal or postmenopausal women. The median duration of clinical presentation to diagnosis ranges from 6 months to 12years. Keeping in view its female preponderance there is invariably a low clinical suspicion of SLE among males.

The clinical scenario of a young man is presented in this report who presented with acute mania and remained on psychiatric follow up for schizophrenia for one year after an initial electro convulsive therapy. The patient presented with fever and shortness of breath to King Abdul Aziz specialist hospital Taif ,Saudi Arabia .The index case was found to have pericardial effusion and on further evaluation proved to have SLE.Following treatment with steroids patient improved clinically, resumed his job and he is off all his anti-psychiatric medications now.

Keywords: SLE, Schizophrenia, Pericardial effusion, Electroconvulsive therapy

Introduction:

The spectrum of psychiatric illness in Systemic lupus erthyromatosus (SLE) include psychotic, depressive, subtle cognitive and personality disorders of histrionic type. The occurrence of psychiatric manifestations in SLE varies widely from 5 to 83%. It is postulated that there is a direct action of the disease on the central nervous system by autoantibodies namely anti phospholipid and anti-ribosome P auto antibodies or cytokines like interleukin 2, interleukin 6, alpha interferon [1]. During the course of the disease side-effects of glucocorticosteroids and hydroxychloroquine or anxious reaction to chronic and potentially lethal illness is postulated to be another mechanism of psychiatric manifestation of SLE . SLE patients are prone to develop myriad of psychological distress in addition to neuropsychitric manifestations which require a social and psychological support. While some of these manifestations are treated by corticosteroids and psychotropic drugs[1]medications with anticholinergic side-effects, like phenothiazines, tricyclic antidepressants and hydroxyzine which enhance the oral dryness should be avoided in SLE.

Clinical scenario:

A 27-year-old male suddenly developed aggressive behaviour for the first time in his life ,while on his work place. The patient had no insight into his illness and was brought to the local psychiatric hospital by his colleagues where he was admitted as a case of acute mania. He was managed with electroconvulsive therapy (EST) in addition to antipsychotic medication as neuro imaging including CT scan and MRI brain were normal. A few days later , the patient was discharged on anti-psychiatric medicines. However, after six months while on antipsychotic medication, he developed a low grade fever .He was admitted to a local hospital where in addition to base line investigations a lymph node biopsy was done which revealed follicular hyperplasia, without any abnormal cell. Patient's HBV, HCV, HIV were negative. The patient developed anorexia , significant weight loss and progressive difficulty in getting up from a sitting position .He also developed shortness of breath and presented to King Abdul Aziz specialist hospital in Taif, Saudi Arabia virtually in a bed bound state . He was admitted in the intensive care unit of the hospital .The examination revealed pallor, generalised lymph-adenopathy, palmer rash, alopecia and mouth ulcers. The patient had mild pericardial effusion and Mitral regurgitation (MR)++ on echocardiography. Further evaluation showed significant proteinuria. Serum ANA, dsDNA were positive .Lupus anticoagulant was negative. Keeping in view above symptoms and signs the patient was diagnosed as a case of SLE[2] (Mouth ulcers, Pericardial effusion ,ANA positive , dsDNA positive) The patient was managed with pulse dose of methylprednisolone 1g intravenously (IV) daily for 5days, followed by oral prednisone 60 mg once daily, which was tapered on follow up . Patient tolerated the treatment well and improved progressively . He became ambulatory and rejoined his job. The psychiatric medications were stopped.

However, on follow up the patient continued to have proteinuria 1.8 gm/24 hr. He was readmitted and the kidney biopsy revealed class IV lupus nephritis. He was given pulse cyclophosphamide 1gm/m^2 intravenously and later started on tablet Mycophenolate 1.5gm once daily. The proteinuria

improved and he is following our clinic for the last two years now .Patient's follow up investigations are shown in table 1.

Table: 1 Patients' hospital investigations and results

Test	Result Pre-treatment (On presentation)	Result Post-treatment (After 6 weeks)	Normal range
Haemoglobin	6.2	12.3	12. 2-15.3 gm/dl
White blood cell	3.2	6.7	6-16 × 109/l
Platelet	41,000	197	150-450 × 109/l
ESR	82mm first hour	56mm	
Total bilirubin	1.2	1.0	.0.8 to 1mg/dl
Direct bilirubin	1.0	0.8	0.-0.6µmol/L
AST	335	30	5-30U/L
ALT	257	29	5-30U/L
ALP	182	100	50-100U/L
GTT	497	65	7-30 IU/l
Albumin	39	39	38-54 g/l
Total protein	5.2	4.5	
INR	1.1	1.1	0.8-1.2
Urea	62	40	
Creatinine	1.2	1.0	
Na/K	131/3.8	142/3.6	
Serum glucose	100	102	65-110mg/dl
ANA	Positive		
Anti DsDNA	Positive		
Lupus anticoagulant	Negative		
24 hr urinary protein	2.3gm/L	500mg/L	<150mg/L

Discussion:

The correct diagnosis of central or even peripheral nervous system manifestations in patients with SLE can be challenging because of many SLE-related and non-SLE-related processes present in a patient. The index case proved to have acute mania as the first manifestation of SLE which remained under oblivion till he developed serositis another complication of SLE. While this patient came to clinical attention after one year a case of SLE masquerading schizophrenia for 14 years was reported by Funaunchi et al[3]. In another report, a 14-year-old boy with a two-year history of cognitive dysfunction and behavioural problems SLE was diagnosed after two years[4] . It appears that the psychiatric symptoms may occur as the first manifestation of juvenile SLE. It will not be out of place to mention that the psychiatric manifestation can be at times dire which could even result in harm to others in a given society. The case of Folie a trios syndrome, characterized by the transfer of delusional ideas from one person to two other persons culminating in murder has been reported in a patient with SLE[5] . In a significant retrospective data from China (a cohort of 518) neuropsychiatric manifestations in SLE were observed in 96(19%) of the above study cohort . The seizure disorder accounted for the most prevalent disorder of neuropsychiatric manifestations (NP) of SLE followed by cerebrovascular disease and acute confusional states. In the above study, 96 patients with psychiatric symptoms, acute psychosis was observed in 10(11%)patients. Authors in this study were of the opinion that this percentage could have been higher if subtle cognitive dysfunction were included as well. Authors of the same study further concluded that the antiphospholipid antibodies were significantly associated with NP manifestations, especially cerebrovascular disorders[6].

The autoantibodies have been found to be biomarkers for future neuropsychiatric events in SLE. A prospective study throughout ten years conducted among 1047 SLE patients demonstrated that individuals who had evidence of lupus anticoagulant (LA) were at an increased future risk of intracranial thrombosis. Further, those with anti-ribosomal P antibodies were at an increased future risk of lupus psychosis[7]. The Lupus anticoagulant in the index case was negative, and anti-ribosomal P antibodies were not available . A study by Sanna et al[8] have shown that an association exists between anti-NR2 antibodies and depressed mood in addition to decreased short-time memory and learning. Authors in this study concluded that antibodies to NMDA receptors thus might represent as one of the several mechanisms for cerebral dysfunction in patients with SLE.

The CT scan brain of the index case was normal. However, massive bilateral calcification of sub-cortical structures in a patient with SLE with the psychotic disorder has been reported[9]. The psychiatric diseases are related to vasculitis and non-inflammatory vasculopathy of the small cerebral blood vessels. Further, a study has shown that ninety per cent of the patients with psychosis, organic brain syndrome or generalised seizures had increased IgG antineuronal activity as compared with only 25 per cent of the patients who presented with hemiparesis or with chorea/hemiballismus. The authors in the above study concluded that the diffuse central nervous system manifestations of SLE are a direct result of the interaction of the antibody with neuronal cell membranes[10].

The management neuropsychiatric manifestation in SLE should include treatment of the disease itself and specific psychotropic treatment. The index case had rapid improvement following Glucocorticosteroid therapy. Intravenous infusions of immunosuppressive agents, such as cyclophosphamide, have been found to be effective in such conditions [1] . Psychotropic drugs may be used, but it is prudent to mention that SLE-inducing drugs, like chlorpromazine, carbamazepine and lithium carbonate must be avoided. Following treatment with steroids, the index case improved and all his antipsychiatric medications were finally stopped and he resumed his job.

To conclude the index case highlights that even though SLE is more frequent among females of childbearing age but males are no way immune to SLE . While evaluating patients with multiple unexplained somatic complaints and psychiatric symptoms SLE ought to be ruled out. The existence of neuropsychiatric manifestations in SLE constitutes an

indisputable clinical reality that every practitioner must be able to recognise and treat.

Competing Interests
None declared

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