PSYCHOSIS IN SYSTEMIC LUPUS ERYTHEMATOSUS—A BRIEF REVIEW AND CASE REPORT

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Although considerable knowledge regarding the clinical and serological abnormalities associated with Systemic Lupus Erythematosus (SLE) is available, the etiology of this disorder is not yet clear. However, there is ample evidence to suggest that immunological mechanisms of tissue injury are important in its Pathogenesis. The prevalence of SLE is 2-3 per 100,000, and the disorder is 9 times more frequent in women than in men, occurring usually between the second and fifth decades of life. The clinical presentation and course of SLE are variable but amongst the commonest abnormalities reported are arthritis and arthralgia (92%), fever (84%), skin eruptions (72%), lymphadenopathy (59%), renal-involvement (53%), anorexia, nausea, vomiting (53%) myalgia (48%), pleuritis (45%) and central nervous system abnormalities (26%). The arthritis and arthralgia are fleeting and involve the hand and feet but deformities are rare. Classical cutaneous manifestations include a malar rash with a butterfly distribution, patchy alopecia, lupus hairs and ulceration of the mucous membranes of mouth and nose. Involvement of the CNS and kidneys is usually associated with a poorer prognosis. Biochemical abnormalities include anemia, leukopenia, thrombocytopenia, positive direct Coomb’s test, presence of antinuclear antibodies (ANA), L. E. Cell, hypo-complementaemia, hyper gamaglobulinaemia, positive rheumatoid factors and biologic false positive Wasserman Reaction.

The neurological manifestations of SLE include seizures, cranial nerve involvement, hemiparesis, para-paresis, peripheral neuropathies, movement disorders, autonomic disorders and disorders of mental function. Psychotic behaviour often accompanies SLE. Kaposi (1872) and Osler (1895) described delirium and delusions in their anecdotal descriptions of SLE. Later Fessel and Solomon (1960) presented a review of psychoses induced by SLE in 227 cases. Various incidences of psychosis have been reported e.g. 52% (Brody, 1953; O’Connor, 1959), 15% (Harvey et al., 1954), 49% (Stern and Robins, 1960), and 12% (Dubois and Tuffanelli, 1964). Confusional states are frequent as are acute deliria with disorientation, disturbances of attention, delusions, hallucinations, excessive motor activity and paranoia. Less frequently, dementia and a general deterioration of intellectual functions occur. “Functional psychoses” of affective and schizophrenic reaction types are the commonest. Shearn and Pirofsky (1953) found depression the commonest manifestation but may be not all of them were of a psychotic intensity. In 40 unselected cases with SLE, O’Connor (1959), found psychosis in 21; 11 were classified as “acute brain syndrome” and 10 as “functional” including 7 schizophrenic and 3 depressive reactions. In their subsequent series of 75 patients followed up to death “brain syndrome” was found in 39 and “Psychosis” in 45 (O’Connor and Musher, 1966). Although these authors felt that an element of “organicity” differentiated the
schizophrenic reaction in SLE, most observers have not found them distinct from other schizophrenias. Stem and Robins (1960) in a similar series reported that 26 out of 53 patients had psychosis: 7 were organic, 8 mixed, 6 schizophrenics and 2 depressives and of the 27 non-psychotic patients 8 had depressive reactions. Typical paranoid (Cares and Weinberg, 1958; Dietze and Voegtle, 1966; Malamud and Saver, 1954), catatonic (Dubois, 1966; Gold and Yahr, 1960; Honda, 1966; Malamud and Saver, 1954; Tseitin, 1963) and hebephrenic (Harrahan, 1954) forms have been described. Johnson and Richardson (1968) reported 8 cases with psychiatric signs and symptoms in their series of 24 cases. These included 3 with affective symptoms i.e. euphoria in terminal phase and a transient flattened affect associated with exacerbation of SLE and 5 schizophrenic Psychosis (3 till death and 1 transient). Others had transient psychotic episode, paranoid delusions, severe persistent psychosis and delirium with grandiosity. Estes and Christian (1971) found psychosis in 59% of their cases. Commonest were disorders of mental function and grand mal seizures i.e. 42% and 26% patients respectively. Psychoses included organic syndromes (disorientation, hallucinations or deterioration of mental functions) in 21% and functional psychoses (schizophrenic and affective reactions) in 16%. 8 patients had anxiety/depressive psychoses which in 2 was severe enough to require ECT's. Postulated mechanisms of development of psychiatric manifestations include a pre-psychotic personality disposition, a variety of cerebral lesions, toxic affects of SLE itself and the effect of steroid therapy. It is interesting to note however, that adrenocortico-steroid administration is of therapeutic value in these states. Various combinations of these factors are undoubtedly operative.

In this report we illustrate one case of SLE who manifested psychiatric signs and symptoms.

Case Report:

A. S. aged 19 years was hospitalized with a 2 year history of a multisystem involvement in the form of arthritis and arthralgia involving both wrists, recurrent pyrexia, myalgia, malaise, red-coloured cutaneous rash over cheeks and legs, alopecia, cervical lymphadenitis, ulcers in oral mucosa and bleeding gums. The illness followed a waxing and waning course. 6 months after the onset of symptoms, cortisone was started in a dose of 40 mg/day along with antipyretics, to which she responded after 3 months and therefore cortisone was gradually discontinued. There was a recurrence of symptoms in December, 1980 i.e. after 3 months and cortisone was restarted. This time a diagnosis of SLE was confirmed by positive LE cell and Antinuclear Antibodies. On a dose of 15 mg/day of cortisone in April, 1981, the patient developed irrelevant speech, and had experiences of being commanded by God to preach and do social service. Irritability, grandiose behaviour, suspecting her father of having an extramarital affair, incontinent affect, pressure of speech and occasional flight of ideas with audio-visual hallucinations were observed. During this period which lasted 6 weeks she was fully oriented but has a faint recollection of her experiences. An "organic psychotic" condition was considered a possibility; cortisone was stopped and she was successfully treated with haloperidol, chlorpromazine and 9 ECT's. Cortisone was restarted in low doses (10 mg/day) and the patient went back to college in July 1981 but experienced lack of concentration and irritability. She remained aloof and felt a lack of confidence.

In September 1981, the patient suffered from florid symptoms once again, became irritable and destructive, made suicidal threats, had occasional sleep disturbance and an exacerbation of oral ulcers which were in temporal correlation with reduction in dose of cortisone to 7.5 mg/day. Her dose of cortisone was increased to 20 mg/day which produced a good response. Depressive
features which persisted were subsequently treated with trimipramine and anxiolytics. She was subsequently maintained on 10 mg. cortisone per day and at the time of this report was well maintained.

**Investigations:**

Hb-between 9-12G% at different times, TLC-7,500/cmm. D.L.C.-P 46, L 45, E 3, M 6; E.S.R. 50-120 mm in 1st hour, C. Reactive Proteins+ve, Rh factor negative, L.E. cell positive, A.N.A. present in 1 : 10 dilution, ASO Titre 333 units, B.U.N. 10mg %, albuminuria ++++ and pus cells present in urine; E.K.G., X-rays of chest and K.U.B. were normal while that wrist joints revealed a rarefaction of the joint. Physical examination revealed oral ulcers (See photograph), alopecia, 'lupus hairs' and lymphadenitis.

Psychological tests revealed a normal I.Q. and average performance on Bender Gestalt Test and Weschler Memory Scale. Rorschach test did not reveal any abnormality.

**COMMENTS**

This patient manifested with classical physical signs and symptoms of SLE, the diagnosis being further confirmed by haematological and serological tests. Psychotic episodes which developed during the course of illness were treated with cortisone, neuroleptics and ECT's. It is important to distinguish these episodes from 'cortisone induced psychosis' by instituting treatment with cortisone. The 'psychotic episodes' were not accompanied by demonstrable organic deficits in intellectual or higher brain functions. A higher 5 year survival rate for patients with functional psychosis as compared to those with organic syndromes has been reported (Johnson and Richardson, 1968). Various other factors that may affect the prognosis are : age of onset (McComb and Patterson, 1959 ; Meislin and Rothfield, 1968), sex (Rupe and Nickel, 1959), presence or absence of Rheumatoid factors (Zvaiffer and Block, 1962 ; Davis and Bollat, 1964), multiple system involvement and time of instituting therapy.

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