CASE REPORTS

Systemic lupus erythematosus presenting as morbid jealousy

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Summary

A patient fulfilling the diagnostic criteria for systemic lupus erythematosus and presenting with morbid jealousy is described. There was evidence of cerebral lupus. Her physical and mental symptoms responded to a combination of chlorpromazine and steroids. The morbid mental process was probably caused by her physical condition while the content of her disordered thought and behaviour was determined by her introverted premorbid personality, religiosity, unhappy childhood experiences and frustrated desire for children.

Introduction

Although various psychiatric symptoms have been associated with systemic Lupus erythematosus (SLE), (Editorial, 1975; Feinglass et al. 1976) morbid jealousy (Shepherd, 1961) as the presenting and predominant feature of this condition is unrecorded. Such a patient is now described and comments are made on problems of psychopathology and management.

Case report

A deeply religious, married woman aged 45-years, of Irish Catholic origin, was admitted to a psychiatric ward in August 1978, after she had exhibited systematized paranoid delusions involving her husband, and bizarre and unpredictable behaviour towards him. She claimed that he had been unfaithful to her for years, had fathered more than 100 children, was telling others that she had VD, and they had not had a valid church wedding. She intended to confront the priest to obtain written evidence concerning her marriage. An illegitimate child, she had never known her father. She married when 22 years old, and had 2 miscarriages but not the children she wanted. She had got on with her husband until becoming suspicious of him 10 months before. Always a quiet, introverted person, she had evidently not abused drugs or alcohol and was not taking drugs before admission.

Her medical history was somewhat unclear but apparently at 22 years she had a pulmonary embolism; and later deep venous thrombosis and a varicose ulcer treated with warfarin from January 1976 until February 1977, transitory episodes of visual impairment and facial spasm (July 1977) and short bouts of mental confusion and depression (June 1978) for which she was briefly prescribed amitriptyline in small doses.

On examination The patient was quite unconcerned, despite complaining bitterly about her husband. There was no significant mood or cognitive impairment. She had a photosensitive butterfly rash of the face and signs of alopecia. BP was 140/90 mmHg. There were bilateral leg ulcers and pitting ankle oedema.

 Investigations The significant abnormalities were as follows. ESR, 70 mm/hr; thrombocytopenia, 46 x 10^9–82 x 10^9/l; prolonged kaolin-cephalin time, 65 sec (control, 37 sec) with a circulating inhibitor of prothrombin activation; raised serum Ig, IgG 18-0 g/l (normal 6.5–15.0 g/l) IgA, 9.6 g/l (normal 0.8–3.4 g/l). Circulating immune complexes detected by Clq radio-immune binding assay. Thyroglobulin but not antinuclear antibodies detected by immunofluorescence. Precipitating antibodies to DNA present by radio-immunoassay (30–60%); upper limit of normal, 25%) and antibodies to extractable nuclear Sm antigens also present. Positive direct Coombs test using antiserum to IgG and to C3D. Serum C3 reduced at 53 mg% (normal, 80–150 mg%). Lymphocytotoxins not detected but 18O brain scanning showed focal reduction of oxygen utilization and to a lesser extent in blood flow in the frontal area (Pinching et al., 1978). Skin biopsy of clinically unaffected skin showed linear IgM deposition. Creatinine clearance impaired at 42 ml/min (normal 80–120 ml/min) but no other evidence of renal disease. Lung function tests normal.
Chloroquine 200 mg daily was started on 4 August but her delusions persisted, behaviour deteriorated (she threatened to burn their house down) and she began to exhibit disorientation in time and place, and memory impairment. Two weeks later she was also given chlorpromazine 125 mg daily with improvement in her behaviour and mental state (for example she spent a quiet weekend at home with her husband), and on 10 September she was discharged to day care. However, because her leg ulcer, hair loss and weight loss persisted, chloroquine was replaced by prednisolone daily on 20 October initially 150 mg/day decreasing to 20 mg/day over the next 2 weeks and maintained subsequently at this level with general physical and mental improvement and a rise in platelet count to \(120 \times 10^9\).1

**Discussion**

This patient was psychotic. Morbid jealousy was diagnosed on the basis of well systematized delusions concerning her husband’s fidelity, occurring in clear consciousness, the absence of typical features of manic-depression or schizophrenia, and her well preserved personality. SLE was diagnosed on the basis of the clinical features of hair loss with a light-sensitive rash and the laboratory abnormalities, namely auto-immune thrombocytopenia, a circulating anti-coagulant, antibodies to nuclear antigens, serum immune complexes, a positive Coombs’ test, reduced C3 and Ig deposition in the skin. These fulfilled the criteria for the diagnosis of SLE (Hughes, 1978).

The impairment of cognitive function and the abnormal brain scan suggested cerebral lupus. She responded physically and mentally to steroids and chlorpromazine, after chloroquine alone and in combination with chlorpromazine had proved ineffective. The improvement with steroids was consistent with what has been observed in cerebral lupus (Hughes, 1978). The psychopathology of morbid jealousy is controversial (for example Freud described projection of repressed homosexual desires on to others) but it is likely that the morbid mental process was caused by her physical condition while the content of her delusions and behaviour was determined by her pre-morbid personality, religiosity, illegitimacy, lack of paternal care and frustrated desire for children.

**Acknowledgment**

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**References**


