or to the effects of the lesion interfering with central nervous system activity more directly. There is widely held agreement that in some cases, the psychiatric distress is directly due to extensions of tumours beyond the sella turcica (Lishman, 1978). Similarly, her psychiatric disturbance was not due to obstruction of the circulation of cerebral spinal fluid, since her tumour was very small and there was no evidence of increased intracranial pressure.

This individual did not report any social stressors which could account for her low mood or anxiety. Her negative family history, while not accounting for all genetic input, would suggest that she was not a strong risk for familial endogenous depression. The lack of precipitants combined with menstrual irregularity, weight gain and increased intrascapular fat caused clinical suspicion that her psychiatric complaints were related to an endocrinological problem. Although it would be premature to indicate a causality between her tumour and her psychiatric symptoms, the patient's history and the large body of literature concerning endocrinological disturbances in mental illness do provide modest convergent evidence in favour of an organic contribution to her illness.

The availability of routine screening of serum electrolytes and urea was important to this situation. Since the patient had few physical complaints and was seen by her regular physician four months before her psychiatric contact, one could have easily omitted blood chemistry from her evaluation. This omission could have led to tragic consequences. Although this patient had no primary physical complaints, her medical condition was quite serious. Pituitary tumours are merely a small part of the medical conditions which sometimes present as psychopathology. This case would not have come to proper treatment so quickly had it not been for the distressing nature of her psychiatric symptoms. This situation illustrates the importance of routine medical screening for psychiatric patients. It is unfortunate that cost-containment measures seek to limit the use of routine testing in patients without manifest physical distress. These circumstances emphasise the need for psychiatrists to practise good medicine and remain aware of the clinical features of medical syndromes. Further evaluation of the association between pituitary function and psychiatric disease is recommended.

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Systemic Lupus Erythematosus in a Woman with Mental Handicap

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A woman with a mental handicap developed SLE. This magnified the level of her handicap, and because of her difficulties in giving a history, mildly abnormal results of tests in any one system were dismissed over long periods of time. This resulted in very late diagnosis of a potentially treatable disease.

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Systemic lupus erythematosus (SLE) is a member of the collagen or connective-tissue diseases, as are rheumatoid arthritis, dermatomyositis, scleroderma and polyarteritis nodosa. A change in our conception of the course of SLE has come about as a result of more sensitive diagnostic methods. The disorder, which is more common than was previously thought, has an incidence of about 4.2 per 100 000 population (Ridley *et al*, 1988). It does not necessarily have a rapid and fulminant course as was previously believed (Hughes, 1979),

and it is markedly more common in females than males (ratio 9 : 1).

The onset is usually insidious, with the development of malaise, fatigue and low-grade fever. Migratory arthralgia develops in the majority of cases. Diffuse muscle aching is common. Skin changes are frequent, although the classic butterfly rash over the nose is by no means always seen. Purpura, alopecia, photosensitivity and Raynaud's phenomenon are common. Lymphadenopathy, oedema and anaemia are often disclosed. The kidney is the most frequent organ to be involved. Anorexia, nausea and vomiting are common. Hypertension is often marked.

Cerebral involvement in SLE is now reported in up to 60% of cases (Lishman, 1987). Mental disorders are the commonest neuropsychiatric manifestation, with acute and chronic reactions, functional psychoses, changes of personality and a variety of neurotic reactions. The majority of mental disturbances are transient, usually clearing within six weeks and rarely outlasting six months although episodes are often recurrent (Bresnihan, 1982; McCune *et al*, 1988). Seizures are also common.

Case report

CM was born of Irish parents in 1949. The pregnancy was full term, with a normal delivery. She was the youngest of six children and appeared to develop normally until the age of two when her mother felt that she was slightly slower developmentally than her siblings. She started at her local primary school but at the age of seven transferred to a special school because of poor academic progress. There were no behaviour problems at this stage. She progressed well at this school and left at the age of 16, when she got a job in a knitting factory. She stayed there for about three years and at the age of 20 she suddenly left home to live with a boyfriend. Her family did not know of her whereabouts and she was not seen during that year. When she arrived back home she appeared very distressed, disturbed and confused. She was thought to have visual hallucinations, was not sleeping well and was extremely aggressive to her family. She was seen by a neurologist, an electroencephalogram (EEG) was performed and a diagnosis of "brain damage and epilepsy" was made. She was treated with phenobarbitone and later with phenytoin.

She was admitted to a psychiatric hospital on a number of occasions during the next 12 months. No definite psychiatric diagnosis was recorded in the notes. Comments such as "a subnormal epileptic girl with a possible degenerative condition" and "possible drug side-effects" were recorded. Later that year (1973) she was transferred to a mental handicap hospital where she remained for the next 15 years.

On admission she was described as looking pale, ill and agitated, with poor concentration, and probably visual and auditory hallucinations. Although she used the spoken word she was unable to maintain a rational conversation with others. She regularly attacked other residents. At that time she had low levels of haemoglobin, folate and serum proteins and an erythrocyte sedimentation rate (ESR) of 50 mm/min. She was referred to a neurologist again. She had another EEG and a brain scan but no change in management was suggested. Her full-scale IQ on the Wechsler Adult Intelligence Scale was 55.

Her condition fluctuated over the next 12 years. She appeared to have recurrent periods of poor concentration and aggressive behaviour which required seclusion. During these disturbed periods she was often described as looking unwell. Her clinical notes, reviewed in retrospect, described fluctuating skin rashes and intermittent infections of skin and chest. She was referred to general physicians on two occasions but by the time that she was seen, the abnormal blood test which had led to referral had reverted to normal.

In April 1988 she moved to a National Health Service residential unit from the large hospital where she had been resident for 15 years as the hospital was scheduled for closure. At the first multidisciplinary review her history was felt to be atypical by the consultant psychiatrist and a tentative diagnosis of SLE was made. On examination she had bilateral central cataracts, skin pigmentation of the face, and chilblains on the toes. Her mental state revealed distractability, talkativeness and repeated incomprehensible utterances. She continually demanded cigarettes and became angry if not given them. Her full blood count and ESR were normal. However her antinuclear antibody and lupus anticoagulant (an anticardiolipin antibody) were both positive. She was referred to a department of immunology. Her complement-3 degradation products (C3d) were raised, computerised tomography was normal, and a skin biopsy was unrevealing.

At this time she was on two neuroleptics, an antidepressant, a benzodiazepine, and two anticonvulsants. She was started on prednisolone to treat the SLE and we were then able to stop her neuroleptics and anticonvulsants. After two months she became very agitated, with rapid and pressured speech, the content of which was impossible to assess. However, it was unclear whether this was due to prednisolone or to withdrawal of the anticonvulsants and neuroleptics. Haloperidol was then commenced and in June 1988 she started on azothiaprine.

To date, her mental state has improved although her concentration is still very poor. Much of her behaviour is bizarre and attention-seeking but it fluctuates. Although she has spoken language it is impossible to hold a rational conversation with her because of her poor concentration. However, aggressive behaviour is no longer present and the violent swings of mood do not appear to occur. Her only medication now is azothiaprine and haloperidol. Apart from an episode of nodular vasculitis about four weeks after starting azothiaprine she has remained in good physical health over the last 18 months.

Discussion

This case illustrates the difficulties that can arise when someone with a mental handicap develops a multi-system disorder. Since such a person may not

have the ability to describe the often transient and vague symptoms of a multisystem disorder, these are often dismissed by their carers and doctors. It may well be that the attitude of the doctor changes because the person has a mental handicap, and consequently vigorous investigation is not carried out. The transient nature of abnormalities is another factor which mitigates against early diagnosis.

It is likely that this woman had her first attack of SLE at the age of 19 when she left home. The fact that she had been labelled mentally handicapped, coupled with her inability to communicate vague and transient symptoms, led to the failure of diagnosis.

Ridley *et al* (1988) suggest that the prognosis for cerebral lupus is not as bad as was previously thought. This case would confirm that, as the patient has probably had the disease for 20 years. In this woman the task of rehabilitation is an enormous one which is hindered by her poor concentration. It is impossible to predict whether she will recover from the effects of spending 15 years of her life living in a large mental handicap hospital with people who were profoundly mentally handicapped.

Staff working in psychiatric and mental handicap services should be aware of the possibility of cerebral involvement by SLE in people with atypical symptoms. They should also be aware of the modern and more sensitive tests for diagnosis of the disorder (Bossingham, 1988).

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