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Letters to the editor

Depression and multiple sclerosis

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A 35-year-old professional man was hospitalised for depression in November 1995. During the past 8 years he experienced four attacks of a relapsing remitting multiple sclerosis (MS), clinically definite according to Poser's criteria (Poser et al, 1983). He also suffered from a bipolar affective disorder as does his sister, who receives prophylactic lithium treatment.

In June 1995, the patient had a spontaneous manic episode treated with lithium and neuroleptics. Since then lithium prophylaxis was maintained and he remained euthymic for a further 3 months. In October 1995, depressive symptoms progressively appeared, associated with a constricting pain of the lower limbs. The diagnosis of MS relapse was initially excluded as neurological examination was normal. The pain complaint was attributed to depression and the patient was referred to the psychiatric unit. He displayed the International Classification Diseases (10th revision) criteria for severe bipolar depression. He received clomipramine 150 mg/d for 2 weeks and 200 mg/d for 4 weeks without any improvement of his pain or depression. In fact, his pain had worsened and his exercise tolerance had decreased. A bilateral pyramidal syndrome had appeared together with a decrease in pin prick sensation below D6. The patient was maintained on clomipramine and lithium and intravenous methylprednisolone pulse therapy (IMPT) was started (500 mg/d during 5 days). Blood lithium levels were maintained between 0.5–0.9 mEq/L. A brief and moderate dietary salt restriction was prescribed and there were no clinical sign of lithium toxicity.

After IMPT, pain had decreased and his depression had dramatically improved. The Hamilton Depression Rating Scale (17 items) (Hamilton, 1967) had decreased from 17 to 3. The patient displayed no sign of hypomanic reaction and was discharged 4 days after the end of corticosteroid therapy. The neurological symptoms and signs disappeared within 3 weeks after discharge and the patient was still euthymic 6 months later.

It is understood that corticosteroids frequently induce affective disorders (Lewis and Smith, 1983). Nevertheless, we prescribed IMPT to our patient without reluctance for three reasons. Firstly, psychiatric side-effects are seldom observed in short course steroid treatments (Chrousos et al, 1993). Secondly, there is no evidence that a previous history of affective disorder predisposes to steroid-induced mental disorders. Thirdly, co-treatment with lithium has been shown to decrease the incidence of steroid-induced mental disorders (Falk et al, 1979).

Consequently, in cases of concomitant depression and MS relapse the use of IMPT under lithium coverage is feasible and possibly helpful. Our patient's depression recovered when he received corticosteroids. It may have been purely coincidental or could reflect a delayed response to clomipramine. The classic issue of the organic or psychogenic origins of depressive states during MS is beyond our compass, but it directly underlies a practical therapeutic question that is conspicuously absent in the literature: if considered as organic, should depression in MS be treated with corticosteroids? This issue remains cautiously evaded.

We await with interest other reports of concomitant bipolar depression and MS relapse in order to state recommendations for the clinical management of this condition.

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