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Extreme suicidality following serotonin syndrome

SIR: Dursun *et al* (1995) suggest that toxic serotonin syndrome (TSS) remains underreported and misdiagnosed. We wish to report extreme suicidality in a 17-year-old schoolgirl following the serotonin syndrome attributed to phenelzine, dothiepin and lithium which she had been receiving for a month for a treatment-resistant depression. During treatment, the patient developed the cardinal features of TSS: pyrexia, opisthonus, rigidity, restlessness and tremor (Sternbach, 1991) in the absence of recent treatment with neuroleptics or other aetiologies (Dursun *et al*, 1995). She recovered from TSS within two weeks of discontinuing all drugs and reverted back to her previous depressive stupor – requiring feeding via a nasogastric tube. She was subsequently transferred to our care and six weeks after TSS, clomipramine 25 increasing to 50 mg was introduced. Within seven days of this, the patient developed an active suicidal drive. She made continuous efforts to lacerate herself and tried to ingest various objects, stating "I feel so awful I must die". Nursing staff had to remain hypervigilant as she would attempt to harm herself with any objects available such as light bulbs, pencils and glassware. The patient required continuous physical restraint from two nursing staff. Senior consultants with special expertise in mood disorders commented that the degree of suicidal drive, which lasted six weeks, was unprecedented in their experience. At six weeks the clomipramine was discontinued and the patient was treated with a repeat course of twelve ECT with a full recovery.

Although serotonergic drugs such as fluoxetine do not precipitate suicide (Beasley *et al*, 1991), it is suggested that paradoxical increases in suicidality may occur in some patients treated with drugs affecting serotonergic transmission (Mann & Kapur, 1991). This case suggests that, following serotonin syndrome, increased sensitivity to even cautious reinstatement of a drug with serotonergic properties (clomipramine), may occur. Although the serotonin syndrome is rare, careful study of patients in the aftermath of TSS is indicated.

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Brain damage and tardive dyskinesia

SIR: I note with interest Pourcher *et al* (1995) suggest "occult acquired brain damage" as an aetiological factor in the development of neuroleptic-induced tardive dyskinesia (TD). The following two cases suggest a similar risk factor for SSRI-induced extrapyramidal side effects (EPS) and TD.

Case 1: A 32-year-old man presented with a depressive illness and was treated with paroxetine 20 mg/day. At the age of 21 he had suffered a pontine haemorrhage due to undiagnosed hypertension that resulted in a left hemiplegia. He subsequently made a full neurological recovery. Within a few days of commencing paroxetine he developed left-sided dystonia of such severity that he was unable to walk. The paroxetine was discontinued and within a week his mobility had returned to baseline. He was then treated uneventfully with dothiepin.

Case 2: A 62-year old woman with Parkinson's disease and mild dementia presented with a depressive illness and was treated with paroxetine 20 mg/day. Her anti-parkinsonian medications were unchanged. Over a three week period her mobility declined, due to increased rigidity and bradykinesia, from walking with the aid of one person to requiring three people to mobilise her. She also developed buccolingual masticatory TD. The paroxetine was discontinued and over the course of a month she regained her mobility. The TD however remains unchanged at four months. Her depression has been treated with trazadone to some effect.