Jaundice Associated with the Use of Risperidone in a Case of Presenile Dementia

Dear Editor

A 54-year-old Caucasian man with a 2-year history of progressive memory loss was admitted for management of his worsening aggression. A year previously, a full dementia work-up revealed no abnormality apart from ventricular dilation for which he had received a ventriculo-peritoneal shunt. The shunt achieved no beneficial effects and he presented on admission with an unsteady gait, urinary incontinence, aphasia and emaciation. With no addition to the picture on repeat dementia/delirium screening, he was started on diazepam and haloperidol, then thioridazine during the first week. Due to the emergence of extrapyramidal side-effects, both neuroleptics were stopped and diazepam increased to 8 mg thrice daily. Risperidone 1 mg bd was introduced allowing for a reduction in diazepam. A dramatic improvement was noted; by day 10 he sat alone and spoke a few logical words. Such was the transformation that he was discharged home in the care of his wife on day 20, taking risperidone 1 mg bd and diazepam 2 mg tds. Five days later he developed clinical jaundice. Serum analyses showed a raised total bilirubin (188 u/l) with no other abnormality in liver function tests or hepatitis screen. Risperidone was discontinued. Within 48 hours the jaundice resolved with a level of 19 u/l of bilirubin. Subsequently, the patient was transferred to residential care as he never regained the level of improvement seen whilst on risperidone even after a trial of other neuroleptics.

Risperidone is a new anti-psychotic which has its effects mainly on the $5HT_2$ receptors and less so on D_2 receptors (Seeman, 1992), the latter giving rise to the claim that it causes fewer extrapyramidal side-effects than older anti-psychotics. Risperidone is associated with sedation (Seeman, 1992), headache and nausea (Chouinard *et al.*, 1993), but *no development of jaundice has been reported.* No rechallenge was undertaken and the reaction cannot therefore be categorically attributed to risperidone. If, however, risperidone is responsible for the reaction in this case, are we to presume that it is an idiosyncratic reaction like that associated with chlorpromazine?

Neuroleptics have been associated with increased sensitivity in patients with dementia (Devanand, 1989); however, they remain the most effective treatment for controlling severe and prolonged aggression (Stotsky, 1972). Whether risperidone has specific cognitive advantages will become clearer with the conclusion of currently ongoing studies of the elderly population.

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