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Acute Confusional States during Treatment with Risperidone

Dear Editor

Risperidone is an atypical antipsychotic, which has been reported as being particularly suitable for older patients, because of a lack of the usual adverse affects caused by typical antipsychotics, including extrapyramidal symptoms, sedation and a postural drop in blood pressure (Zayas and Grossberg, 1998; Thorpe, 1997). Cognition has been reported to improve as compared with no treatment or treatment with typical antipsychotics (Gallhofer *et al.*, 1996). It has been recommended for use in delirium in older people, who are more prone to these adverse affects (Sipahinalani and Masand, 1997).

More recently, however, there have been case reports suggesting that risperidone may be associated with cognitive impairment in older people. For example, Ravona-Springer *et al.* (1998) reported three cases of delirium associated with risperidone, although all had other possible reasons for becoming acutely confused. In addition, all were also taking other psychotropic medication. Furthermore, one was recovering from electroconvulsive therapy and one was hyponatraemic. A further case report (Tavcar and Dernovsek, 1998) described a rapidly developing delirium in an 85-year-old-woman, who was taking medications including risperidone, and had concurrent physical problems.

There have also been two case reports of confusion associated with concurrent risperidone and lithium treatment (Swanson *et al.*, 1995; Chen and Cardiasis, 1996). In the first, a 25-year-old man had a high fever and raised creatinine phosphokinase, and was diagnosed as having neuroleptic malignant syndrome. In the second, a 69-year-old woman was confused in the context of lithium toxicity and risperidone. Other factors, including the use of amatidine, and chronic obstructive airways disease, may have been important.

We would like to report a case of acute confusion associated with risperidone treatment. Demographic details have been altered slightly to prevent identification.

A 68-year-old gentleman with a history of schizophrenia, left ventricular failure (LVF) and atrial fibrillation (AF), was treated with digoxin. His first presentation with schizophrenia was in 1976. At this time he had a complex delusional system which he had acted upon, causing injury, but was cognitively intact.

One year before this admission, he had had persistent delusions regarding the water being infected which interfered with his fluid intake and at this time his medication was reassessed. Due to the lack of efficacy and a poor tolerance to increasing dosage, the sulpiride 300 mg b.d. was changed to risperidone 6 mg. His MMSE at this time was 27/30. His psychotic symptoms began to resolve, although he remained reluctant to drink. Over the following months he became gradually confused, scoring 19/30 on MMSE. A physical examination noted controlled AF, though no evidence of LVF, FBC, ESR, LFT AND TFT were unremarkable. U&Es revealed mild dehydration. MSU was negative. This deterioration in his cognition was accompanied by a decline in his self-care. He was admitted to hospital, after 'toas-

ting' his arm on an electric heater in order to 'cure' his arthritis, which led to superficial burns, dehydration, a fall and subsequent upper respiratory tract infection. This occurred on a background of persistent psychosis and several recent admissions.

On admission he scored 10/30 on mini-mental state. Repeat FBC, U&E, LFT, TFT and MSU were normal. ECG and CXR revealed only a chest infection, which was subsequently treated. Although medically fit, with no evidence of further infective focus, he remained confused. Over the course of five weeks, his confusion remained, MMSE remaining at 10/30. Repeat bloods, MSU and CXR were unremarkable. Digoxin levels were in the normal range. CT was unremarkable and the ECG revealed controlled AF. The EEG showed non-specific generalised cerebral dysfunction with low amplitude spike components seen in the left occipital region that did not amount to epileptiform activity. This was compatible with toxic-metabolic disturbance, renal failure or a dementing process.

As no other cause of prolonged confusional state was found, it was decided to stop his anti-psychotic medication. Within one week his MMSE had risen to 21/30 and two weeks later it had returned to 27/30. He remained psychotic and olanzapine 10 mg was commenced. After a further two weeks, his psychosis resolved, he was cognitively intact, communicative and socially reactive.

Risperidone has been recommended for older people in particular because of its pharmacological profile and lack of reported side effects. A retrospective pharmacoepidemiological study of 122 inpatients of over 65-years-old has been reported, in which 1.6% of patients with pre-existing cognitive impairment had a delirium whilst taking risperidone (Zarate *et al*., 1997). However, as neither the diagnosis nor investigations relating to these patients is reported, it is not clear what part, if any, risperidone played in its development.

Risperidone acts by blocking 5HT2 and partially blocking D2 receptors, which result in an antipsychotic effect and improvement of negative symptoms (Schotte *et al*., 1996). It is unclear how this would cause cognitive impairment. We considered the possibility that impairment was caused by a lowering of the seizure threshold and our patient was suffering from ictal or post-ictal confusion. There is little EEG evidence to support this hypothesis.

We wish to draw clinicians' attention to the possibility that despite the relative safety of risperidone in this age group, patients taking it may become acutely confused and that the differential diagnosis should include risperidone-induced confusion.

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