

Correspondence

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Co-occurrence of polydactyly and psychosis

Sir: We wish to comment on the preliminary report listing five cases of co-occurrence of polydactyly and psychosis (Cardno *et al*, 1998), which concluded that there was preliminary evidence that polydactyly was over-represented in individuals with familial schizophrenia and related psychotic illnesses.

We were interested in the report as we had a 36-year-old Asian male who suffered from chronic relapsing schizophrenia and also had pre-axial polydactyly with an extra thumb on the left hand. There was no family history of polydactyly but his eldest brother suffered from schizophrenia. Our case was similar to the fifth case described by the authors (a Caucasian young male, 41 years old) except the ethnicity.

O'Callaghan *et al* (1991) concluded that minor physical abnormalities indicated early dysmorphogenesis in schizophrenia, particularly in males (all the cases described by the authors were male), which appeared to be associated more reliably with genetic than obstetric factors and with cognitive impairment. They also found that a family history of schizophrenia was particularly associated with abnormalities of the mouth.

Post-axial polydactyly (little finger side) occurs as an isolated lesion in Black people, inherited as an autosomal dominant trait, but in White people may be associated with other anomalies and syndromes. We note that three out of the four cases of post-axial polydactyly described by the authors also had a family history of polydactyly. Pre-axial polydactyly with extra thumbs is common in White people; it is usually sporadic and unilateral (Nelson *et al*, 1992). (Unfortunately this vital information has been omitted from the recent edition of the same text, which Cardno *et al* cited (Nelson *et al*, 1996).) In our opinion, as isolated pre-axial polydactyly of digits is likely to be sporadic, such cases

should not be included in the same group as familial post-axial polydactyly in future research.

We also note that the first case described by Cardno *et al* (an Indian male, 64 years old) did not have any family history of schizophrenia or related psychotic illnesses. We feel that further research should recognise these differences and focus on patients with familial polydactyly and familial schizophrenia.

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O'Callaghan, E., Larkin, C., Kinsella, A., et al (1991) Familial, obstetric and other clinical correlates of minor physical anomalies in schizophrenia. *American Journal of Psychiatry*, **148**, 479-483.

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Olanzapine in the treatment of psychotic depression

Sir: In connection with the review by Tollefson & Kuntz (1999), examining the clinical studies involving olanzapine, we would like to report its effective use in the treatment of psychotic depression.

Two patients with severe psychotic depression, responsive solely to electroconvulsive therapy (ECT), refused further treatment necessitating pharmacotherapeutic change. Both patients were receiving maximal doses of venlafaxine (375 mg daily), with lithium augmentation, in combination with an antipsychotic (thioridazine or sulpiride). Severity of illness

precluded the use of placebo and so, over a period of three months, we prospectively conducted an open study, involving antipsychotic substitution with olanzapine (10 mg). This was the only change implemented. Objective and subjective mood and psychotic symptoms were rated weekly, blind to treatment status (Hamilton, 1960; Beck *et al*, 1961; Cliffe *et al*, 1995). Prior to olanzapine substitution, ECT had not been administered for at least two months and the maximum tolerable doses of traditional antipsychotics had been prescribed without success.

In both subjects it was found that within three weeks of starting olanzapine the psychotic symptoms diminished and mood began to improve. With ongoing treatment clinical improvement continued and both patients eventually recovered sufficiently to enable discharge from hospital.

The atypical antipsychotic olanzapine may, therefore, have a place in the management of treatment-resistant psychotic depression. This would be in keeping with its effects on comorbid mood symptoms in schizophrenia (Tollefson & Kuntz, 1999) and its suggested adjunctive role in the treatment of bipolar disorder (Carlos *et al*, 1998).

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Hamilton, M. (1960) A rating scale for depression. *Journal of Neurology, Neurosurgery and Psychiatry*, **23**, 56-62.

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Use of long-acting benzodiazepines in older people

Sir: Taylor *et al* (1998) produced data from Liverpool showing no reduction in overall benzodiazepine use among the elderly during a 10-year period, and that there was a high rate of inappropriate use of the drugs.