

LETTERS TO THE EDITOR

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Letters to the Editor

IS MONITORING OF PLATELETS NECESSARY DURING CLOZAPINE THERAPY ?

Sir,

The availability of clozapine in India at prices relatively lower than those in the west has made this a feasible treatment option for many patients with schizophrenia or schizoaffective disorder whose illness had not responded to conventional treatment. However, it is still an expensive treatment, added to which are the costs of haematological monitoring. In order to minimise treatment costs, it is imperative to review the schedule for monitoring in the Indian context, to reflect the actual risk of agranulocytosis (Tharyan, 1998).

Current recommendations do not require the routine monitoring of platelet counts but a report by Eranti & Chaturvedi (1998) documenting asymptomatic marked thrombocyte fluctuations, without similar leucocyte variations or agranulocytosis, suggested the need for routine platelet monitoring. There is little evidence at present to support such a recommendation. In addition, spurious results may be obtained with routine thrombocyte counts that may result in deserving patients being potentially denied effective therapy, as is illustrated by the following example.

A 22-year-old single male was started on haloperidol in 1991 for a psychotic illness, which on follow up was diagnosed as schizoaffective disorder - bipolar type, fulfilling diagnostic criteria in DSM - IV (American Psychiatric Association, 1995). Lithium was added in 1996 after the emergence of manic symptoms and subsequently he has had a fluctuating course requiring continued haloperidol for persistent delusional beliefs and intermittent treatment with fluoxetine for depressive episodes. Haloperidol was replaced by risperidone 8 mg/day in mid 1997 and in early 1998 carbamazepine was added to achieve better mood control. By mid 1998, he started

evidencing buccolingual involuntary movements and in view of his distressing mood fluctuations and persistent delusional beliefs, a decision was made to try mono-therapy with clozapine.

Baseline white cell count was normal but platelet count was 83,000/cu mm. Since he evidenced no clinical features of thrombocytopenia, a repeat platelet count was ordered. Simultaneously all psychotropic medication was tapered off and clozapine was initiated at 25 mg/day on 19/10/98. Platelet count on 24/10/98 was 1,10,000/cu mm on a clozapine dose of 75 mg/day. Clozapine was continued but as a matter of caution, platelet counts were ordered on 28/10/98.

On 31/10/98 the patient was seen by another consultant who, on noting a platelet count of 82,000/cu mm with a total WBC count of 8,500/cu mm, stopped clozapine. A platelet count repeated on the same day was 86,000/cu mm, again with a normal total and differential white cell count and no symptoms of thrombocytopenia.

At this point a consultation was sought with haematologists who advised that a manual count of the slide be performed on the last sample of blood given for platelet evaluation. This was reported to show 1,20,000 platelets/cu mm. According to the haematologists, the reason for the low platelet counts on previous occasions was probably erroneous readings produced by automated counts with the Coulter counter, which tends to read platelet aggregates as a single large platelet, thereby under-reporting the total number of platelets. A manual count revealed this error.

Clozapine was restarted on the next day and subsequent platelet counts done manually have all been normal, albeit with marked variations in absolute number. The patient is currently fully functional on a clozapine dose of 150 mg/day, employed, euthymic and free of delusional beliefs. Monitoring of platelet counts has been long discontinued.

Unless systematic evaluation reveals symptomatic thrombocytopenia or thrombocytosis, independent of agranulocytosis induced by clozapine, routine monitoring of

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platelets appears unwarranted at present.

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