in peliosis hepatis. Microscopic peliosis is often confused with extreme sinusoidal dilatation and hepatocellular dropout. In sinusoidal dilatation, the liver plate structure is intact, and lesions are perportal or midzonal, especially when associated with oral contraceptives and pregnancy. In hepatocellular dropout, there is collapse of liver cell plates without loss of reticulin fibers.

It is appropriate to classify the lesions according to the apparent etiology, as this correlates with distinctive histological and clinical features. In addition to anabolic, estrogenic, or adrenocortical steroids, macroscopic peliosis hepatis has been reported in malnutrition,[3] leukemia, tuberculosis, leprosy, vasculitis, lymphoma,[3] and AIDS. Bacillary peliosis in human immunodeficiency virus infection can manifest as massive hemoperitoneum.[4] In the present study, the patient gave a history of taking NSAIDS for vague abdominal pain, and the probability score by Naranjo algorithm[5] for NSAIDS use and adverse drug reaction[6] was 0.[doubtful adverse drug reaction]

Spontaneous regression can occur in peliosis hepatis with interruption of the inducing factor, or withdrawal of the specific drug in case of drug-induced peliosis. Recurrent spontaneous intrahepatic hemorrhage from peliosis has also been reported[6] and is rarely fatal; it is due to extensive parenchymal destruction. Hemorrhage complicating localized peliosis hepatis may require a partial hepatectomy. In the present study, the prolonged consumption of NSAIDS might have been responsible for progressive dilatation, rupture of subcapsular cysts, and the resultant hemoperitoneum. The patient was given only palliative treatment and advised sonography during the next visit.

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OLANZAPINE-INDUCED OCULOGYRIC CRISIS IN A PATIENT WITH SCHIZOPHRENIA

Sir,

Oculogyric crisis is an acute dystonic reaction characterized by sustained upward deviation of eyeballs and restlessness. Such reactions generally occur after administration of typical antipsychotics but are considered rare with atypical antipsychotics, particularly olanzapine. Until now, there are only two reports of olanzapine-induced oculogyric crisis from India, one in a patient of bipolar disorder[1] and another in a child with post-encephalitic syndrome.[2] Indeed, literature review on June 1, 2008, in ENTREZ PUBMED revealed only another German report in a patient with general anxiety disorder,[3] Interestingly, although the commonest indication of olanzapine is schizophrenia, olanzapine-induced oculogyric crisis was never reported in schizophrenia. Below, we describe such a case.

A 25-year-old man, suffering from schizophrenia for 6 months, was initially treated with risperidone 8 mg/day and fluoxetine 40 mg/day (for associated depressive symptoms). But in higher doses, olanzapine has been shown to have high D2 affinity, increasing the chance of oculogyric crisis. Probably for this, this patient did not have any problem with olanzapine at lower level except tremor. However, once his symptoms resolved, the patient stopped taking medicine and relapsed within 2 weeks. This time, a different consultant placed him on olanzapine 10 mg/day and imipramine 75 mg/day. After 1 month with this regimen, the patient reported partial improvement, along with tremor. As olanzapine is not known to cause tremor commonly, imipramine was thought to be the reason and was stopped. Indeed, olanzapine was hiked up to 15 mg/day as the patient had partial improvement. But within a few days, the patient started having repeated episodes of sustained upward deviations of eyeballs, along with anxiety; restlessness; and backward flexions of neck, which transiently resolved with injection promethazine 25 mg. However, regular addition of trihexyphenidyl 2 mg/day failed to stop recurrences of the crises, which ultimately required replacement of olanzapine with risperidone. Naranjo’s algorithm[4] indicated a probability score of 8 of olanzapine-induced oculogyric crisis. With risperidone 3 mg/day, the patient is now maintaining remission without having tremor or oculogyric crisis any further.

High dopamine-acetylcholine antagonism or high striatal dopamine inhibition has been suggested to underlie neuroleptic-induced oculogyric crisis.[1] So olanzapine with an intermediate level of D2-binding affinity[5] is not expected to cause oculogyric crisis. Probably for this, this patient did not have any problem with olanzapine at lower level except tremor. But in higher doses, olanzapine has been shown to have high D2 affinity, increasing the chance of oculogyric crisis.[6] Also, high anticholinergic property of imipramine may have prevented the occurrence of oculogyric crisis initially until it was withdrawn. However, this does not explain the failure of trihexyphenidyl to control this side effect. It may imply that probably trihexyphenidyl is not very effective in the treatment of repeated oculogyric crisis with olanzapine. Indeed, we may need to change the antipsychotic in such a case.

Recent Clinical Antipsychotic Trials of
Intervention Effectiveness (CATIE) study[6] has seriously doubted the prevalent belief of atypical antipsychotics including olanzapine having lesser neurological side effects. Our case report supports this doubt. Although possibility of such a side effect is low, we still need a cautious approach regarding this agent, particularly while prescribing a higher dose.

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