perplexed and not fully oriented to time and place. I wish to report an adolescent girl who exhibited oneroid state with no basis for organicity, and who responded well to clozapine after failure of trials with haloperidol and risperidone.

**Case Report**

"Ms. A" is a 19-year-old unmarried woman from a socioeconomically lower class urban family. In the past, she developed an illness suggestive of paranoid schizophrenia (ICD 10 Criteria), characterized by withdrawn, abusive and violent behavior, delusions of persecution, and inappropriate affect of 5-month duration. She recovered spontaneously and was symptom-free for the next 2 months. Subsequently, she presented to us with confusional behavior without any apparent psychological or physical precipitating factor. Her confusional behavior lasted for 15 days and was almost continuous. During this time, she misidentified her family members and had urinary incontinence. She muttered and talked incoherently. Mental status examination revealed increased psychomotor activity, verbal stereotypy, and perseveration with loosening of association and perplexed mood. She exhibited double orientation and double book-keeping phenomena.[3,4] At times, she would misidentify the doctor as her brother, but on other occasions, she behaved like a patient with the same doctor. She would misidentify the hospital as her home and name a few patients as her relatives. She labeled half of the hospital as her house and half as the hospital. While in the hospital, she would follow regular hospital routine. Her look was perplexed. She had fleeting delusions of persecution, rapidly shifting visual hallucinations, and illusionary distortion of perceptual processes on several occasions.

A detailed, meaningful cognitive assessment was not possible in view of her persistent perplexity. Medical and neurological referrals were taken and no deficit was detected upon a detailed physical examination. Her computed tomography (CT) head scan and electroencephalogram (EEG) were normal. A routine hematological and biochemical investigation did not reveal any abnormal findings. She was put on a regimen of haloperidol, 20mg/day for 3 weeks, and, subsequently, risperidone, 6mg/day, with no improvement. Ultimately, I started her on a regimen of clozapine, and gradually increased the dosage to 200mg over a period of 3 weeks, with proper monitoring of leukocyte count. Ms. A showed marked improvement with clozapine not only in thinking but in her confusional behavior. Hence, she was stabilized on the same dose for 3 months without any relapse.

**Comment**

Oneroid state is a neglected condition probably because it deals more with clinical descriptions than with a phenomenological approach. This case did not fit into any category of the current psychiatric classificatory system. In contrast to our report where clozapine has been used successfully, there have been few reports of clozapine-related delirium or confusion.[3] In such cases, clozapine's anticholinergic properties or concomitant anticholinergic or CNS depressant medications may have been responsible for the delirium.

In conclusion, clozapine can be used safely in patients with functional confusional psychoses, such as oneroid state; hence, it may be considered as the first line of treatment. Further systematic collection of data in this regard may add to the confidence in prescribing clozapine in this state.

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**References**

1. Mayer-Gross: Schizophrenia, in Clinical Psychiatry, 3rd ed. Edited by Slater E, Roth M. London, Bailliere Tindall, 1965, pp 237–341
2. Kaptsan A, Miodownick C, Lerner V: Oneroid syndrome: a concept of use of for Western psychiatry. Isr J Psychiatry Relat Sci 2000; 37:278–265
3. Sims A: The disordered self, in Symptoms in the Mind, 3rd ed. Edited by Sims A. London, Saunders, 2003, pp 209–229
4. Gancro R, Lehmann H: Schizophrenia: clinical features, in Comprehensive Textbook of Psychiatry, 7th ed. Edited by Sadock BJ, Sadock VA. New York, Lippincott Williams & Wilkins, pp 1169–1199
5. Iqbal MM, Rahman A, Husain Z, et al: A clinical review of adverse effects and management. Ann Clin Psychiatry 2003; 15:33–48

## Clozapine-Induced Tardive Dystonia (Blepharospasm)

*SIR:* Tardive dystonia is a type of "tardive" movement disorder induced by antipsychotics and is characterized by involuntary muscle contraction, which may be tonic, spasmodic, patterned, or repetitive.[1] Blepharospasm is a type of focal tardive dystonia characterized by chronic intermittent or persistent closure of the eyelids. Although there are rare reports of tardive dystonia induced by atypical antipsychotics, such as olanzapine,[2] clozapine, in contrast, has been reported to ameliorate tardive dystonia.[3] Contrary to existing literature, we report a case of a patient developing blepharospasm with clozapine.

**Case Report**

"Mrs. F," a 46-year-old woman, presented with one month of suspiciousness, mumbling to herself,

and disturbed biological functions, and she was diagnosed with schizophreniform disorder. Past and family history revealed no psychiatric or neurological illness. Initially, she was treated with trifluoperazine (15mg/day) for 4 months, which was replaced with clozapine due to severe akathisia. The dose of clozapine was increased from 25mg to 100mg/day over a period of 2 weeks after performing hematological investigations, and she was maintained on the same dose for 4 months.

While being treated with clozapine, Mrs. F developed frequent and forceful blinking of her eyelids. She would blink her eyes about 40 times in a minute when exacerbated by bright light. An ophthalmological consultation was unremarkable for any eye pathology contributing to this presentation and the neurological exam did not reveal any abnormal movements, except for the blepharospasm. The patient had no prior personal or family history of blepharospasm or other movement disorders.

Five months later, clozapine was tapered and stopped, but Mrs. F continued to have blepharospasm even 4 weeks after discontinuing clozapine. Subsequently, clonazepam (1mg/day) was started and the blepharospasm disappeared a month after its initiation. Two years later, the patient had a second episode of psychotic illness and was again treated with clozapine, titrated up to 75mg/day. Five weeks later, she exhibited a similar form of blepharospasm, though it was not as severe. Again, clozapine was discontinued and Mrs. F had a complete resolution of the blepharospasm with clonazepam.

**Comment**

The reappearance of blepharospasm with the administration of clozapine strongly implicates this drug in this dystonic reaction. Blepharospasm is classified as a type of focal dystonia and has been reported to occur with atypical antipsychotics. In contrast to other atypicals, such as olanzapine, which have been anecdotally reported to cause tardive dystonia,[2] clozapine has been used in treating tardive dystonia, including blepharospasm.[3,4] However, in rare instances, clozapine has been reported to induce tardive dystonia as well.[5] In the above case, the patient developed cervical dystonia 2 years after clozapine was started. Given the rarity of reports of clozapine-induced tardive dystonia, our patient may have developed an idiosyncratic reaction to this drug. This is further supported by the observations that this patient did not develop tardive dystonia with trifluoperazine, which is a more potent $D_2$ receptor antagonist than clozapine, and that the proposed mechanism of drug-induced tardive dystonia is the sensitization of dopamine receptors following their blockade.[6] Another issue highlighted by this report and the existing literature is that clonazepam is an effective treatment of tardive dystonia,[3] and may be considered before more invasive procedures, such as botulinum toxin, are tried.

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**References**

1. Sienaert P, Peuskens J: Remission of tardive dystonia (blepharospasm) after ECT in a patient with treatment-refractory schizophrenia. J ECT 2005; 21:132–134
2. Charfi F, Cohen D, Houeto JL, et al: Tardive dystonia induced by atypical neuroleptics: a case report with olanzapine. J Child Adolesc Psychopharmacol 2004; 14:149–152
3. Shapleske J, Mickay AP, Mckenna PJ: Successful treatment of tardive dystonia with clozapine and clonazepam. Br J Psychiatry 1996; 168:516–518
4. Levin H, Reddy R: Clozapine in the treatment of neuroleptic-induced blepharospasm: a report of 4 cases. J Clin Psychiatry 2000; 61:140–143
5. Molho ES, Factor SA: Possible tardive dystonia resulting from clozapine therapy. Mov Disord 1999; 14:873–874
6. Trugman JM, Leadbetter R, Zalis ME, et al: Treatment of severe axial tardive dystonia with clozapine: case report and hypothesis. Mov Disord 1994; 9:441–446

## Clozapine-Induced Cataplexy

*SIR:* Cataplexy is an abnormal state characterized by atonia and believed to represent dissociated REM (rapid eye movement) phenomena that intrude into wakefulness. Cataplexy-like phenomenon as a rare side effect of clozapine treatment was initially reported by Chiles et al.[1] in a series of four people with schizophrenia who were on variable doses of clozapine. Blum[2] also noted cataplexy during clozapine treatment along with hyperthermia and increased REM sleep. We report another patient presenting with this rare side effect of clozapine and attempt to explain the phenomenon with possible neurobiological mechanisms.

**Case Report**

A 29-year-old unmarried woman with unremarkable past, family and personal history presented with an episodic illness of 4 years duration suggestive of schizoaffective disorder, manic type. As she remained symptomatic even after adequate trials with various antipsychotics along with mood stabilizers, clozapine was introduced. She was on a regimen of sodium valproate, 1000mg/day, when clozapine was initiated and gradually hiked to 150mg/day. Though her symptoms