**Letter to editor / *Editöre mektup***

# Olanzapine-induced delayed-onset oculogyric crisis
## *(Olanzapine bağlı geç başlangıçlı okülojirik kriz)*

**Mustafa İSPİR, Osman BAKKAL, İbrahim GÜNDOĞMUŞ, Ayhan ALGÜL**

**To the Editor,**

Olanzapine is an atypical antipsychotic drug that is very effective and has weak potential for extrapyramidal adverse effects. Oculogyric crisis (OGC) is an acute dystonic reaction that usually occurs after initiation of antipsychotic treatment, characterized by spasmodic deviations of the eyes, lasting for a few minutes to several hours.[1,2] Among the atypical antipsychotics, cases of olanzapine, clozapine and ziprasidone induced acute OGC have been reported.[3] As far as we know a few cases have been reported about delayed onset or tardive OGC with olanzapine. We reported a case of olanzapine-induced OGC that started after 1 month of initration of the treatment and improved addition of oral biperiden.

We reported a case of oculogyric crisis in a 21 year-old man due to olanzapine monotherapy after his first psychotic episode. He exhibited psychotic symptoms in the form of social withdrawal, restricted affect and paranoid delusions. In his personel history there were no brain damage and drug or alcohol issues. His family history was noncontributory. He was hospitalized and given 10 mg of olanzapine at night. Symptomatic improvement was noticed, but he developed a distressing OGC after 1 months. It would occur every 2 to 3 days lasting up to a fixed upward gaze. These episodes lasted for 15 to 20 minutes and spontanously resolved without any additional medications. There were no other associated involuntary movements and no worsening of the psychiatric symptoms. A Biperiden (4 mg/d) was added in divided doses and there was gradual reduction of duration and frequency of OGC that eventually subsided.

In the case of OGC, there was a great response to low-dose anticholinergic at one week.

In the literature, there is medical case of dystonia due to the long-term usage of olanzapine and case of tardive dystonia which was not responding to anticholinergics, and the cases of OGC appeared in the first one or two days. The distinctive feature of our case is the appearance of the situation after a month, and responded in a week after supplementation of anticholergics. The certain mechanism of neuroleptic-induced OGC is just unclear and possibly attributable to higher striatal inhibition of dopamine function or higher dopamine-acetylcholine antagonism.[4] Considering recovery of OGC with anticholinergics, a dopamine deficiency and acetylcholine excess may be implicated, for OGCs. However, why some cases develop acute and, others, tardive OGCs is not known.[5] It is hypothesized that sensitizing effect of striatum from past antipsychotic exposures may contribute toward delayed of OGC. But our case was the first episode psychosis. In conclusion, although possibility of such a side effect is weak, clinicians should be known with this side effect and alert to early detection and rapid initiation of treatment.

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Departmant of Psychiatry, Sultan Abdulhamid Han Training and Research Hospital, İstanbul, Turkey
**E-mail:** mispirkadirli@gmail.com