PHOTO ESSAY

Oculogyric Crisis with Downward Deviation – A Photo Essay

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ABSTRACT

Oculogyric crisis (OGC) describes the clinical phenomenon of sustained dystonic, conjugate and typically upward deviation of the eyes. A few cases with downward or lateral deviations have been described.^{1,2}

ARTICLE HISTORY

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KEYWORDS

Oculogyric crisis; downward; neuroleptics

Case presentation

A 52-year old man was referred to our department by the cardiovascular surgery team complaining of involuntary downward ocular deviation. He had had sternal haematoma due to traumatic chest injury and underwent surgery three days previously. Two days after the operation he developed delirium for which haloperidol 1 mg oral three times a day was initiated. On the third day of the therapy, he was seen because of the downward deviation of the eyes. On examination he was found to be oriented and to cooperate fully. Sustained conjugate downward deviation of eyes was present (Figure 1). The patient could bring his eyes back near to the primary position with combination of forceful effort and eye blinking, but he could not sustain it, and the eyes resumed their downward position within seconds. The rest of the neurological examination was unremarkable. His blood tests and brain MRI findings were unremarkable. He was given Biperiden iv 2 mg after that he was treated with oral Biperiden Hcl 2 mg two times a day and alprozolam 0.5 mg oral two times a day. When he was seen a month after treatment, his eyes were normally positioned (Figure 2). He was able to look upwards (Figure 3).

Discussion

Oculogyric crisis (OGC) describes the clinical phenomenon of sustained dystonic, conjugate and typically upward deviation of the eyes, although a few cases with downward or lateral deviations were

described.^{1,2} OGC was first described in patients with encephalitis lethargica, but is most commonly seen in the present day in patients exposed to neuroleptics.³ Although the pathophysiology of acute dystonic reactions is still unclear, it is believed to be due to a deficit in central dopamine transmission resulting in overactive striatal acetylcholine release.⁴ The spectrum of conditions associated with OGCs is wide and encompasses three main categories of disorders: (1) drug-induced disorders, (2) hereditary and sporadic movement disorders and (3) disorders related to focal brain lesions.¹ Sixty-eight per cent of all reported patients with drug-induced OGCs we could identify were due to neuroleptics.¹ Both typical and atypical neuroleptics induced OGCs; however, the use of typical neuroleptics and OGCs was more commonly reported.¹ Patients who have experienced a single episode of acute dystonic reaction are at higher risk for future dystonic reactions when exposed to other dopamine receptor blocking agents.⁵ It is commonly reported as an acute disorder.¹ Clinical presentation may vary from very brief and subtle eye deviation as an isolated symptom to more severe and even painful forms accompanied by neck flexion, jaw opening, blepharospasm, tongue protrusion and autonomic signs, such as perspiration, pupillary dilation, increases in blood pressure and heart rate.¹ The common basis of these disorders is a metabolic, anatomical or functional disruption of the nigrostriatal pathway, mainly of dopamine metabolism.

Treatment strategies are variable and depend on the aetiology of OGCs.¹ In drug-induced

Figure 1. Sustained conjugate downward deviation of the eyes.

Figure 2. After treatment eyes at primal position.

OGCs, the first step of management should include removing or, if not possible, reducing the dose of the offending agent.¹ In acute cases, administration of anticholinergics, such as benztropine (e.g. 2-mg intravenous) and biperiden (e.g. 5-mg intramuscular) can be

used.¹ Oral administration of anticholinergics may be the most feasible approach for cases seen outside the emergency setting.¹ In cases of persistent lack of response, oral treatment with benzodiazepines such as clonazepam (e.g. 0.5–4 mg) might provide symptom relief.¹

Figure 3. After treatment patient was able to look upward.

However, it should be noted that treatment effects might be limited in tardive OGCs.¹ A few patients have been reported to exhibit drug-induced oculogyric crises that recurred spontaneously months to years after discontinuation of the offending dopamine receptor blocking agent. Episodes reoccurred spontaneously, but remained responsive to anticholinergics.⁶

Disclosure statement

The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the article.

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