Spotlight on Oculogyric Crisis: A Review

Pankaj Mahal[1], Navratan Suthar[1], Naresh Nebhinani[1]

ABSTRACT

Background: Oculogyric crisis (OGC) is a form of acute dystonia characterized by sustained dystonic, conjugate, and upward deviation of the eyes. It was initially reported in patients with postencephalitic parkinsonism. But later, other factors such as medications, movement disorders, metabolic disorders, and focal brain lesions were also found to be associated with OGC.

Methods: The literature regarding OGC was searched via PubMed, Google Scholar, and through citations in relevant articles till December 2019, with keywords including OGC, oculogyric eye movements, tonic eye movement, neuroleptics and OGC, antipsychotics and OGC, and all combinations of these. Only original articles (abstract or full text) that were published in the English language were reviewed.

Results: Hypodopaminergic state is implicated in the pathogenesis of OGC. Common risk factors are younger age, male sex, severe illness, high neuroleptic dose, parenteral administration of neuroleptics, high potency of neuroleptic drugs, abrupt discontinuation of anticholinergic medication, and family history of dystonia.

Conclusion: OGC is an acute dystonic reaction leading to tonic upward deviation of eyes. It is associated with various neurometabolic, neurodegenerative, and movement disorders and medications such as antipsychotics, antiemetics, antidepressants, antiepileptics, and antimalarials. OGC can adversely impact the compliance and prognosis of the primary illness. Hence, it needs to be managed at earlier stages with appropriate medication, primarily anticholinergics.

Keywords: OGC, oculogyric crisis, blepharospasm, acute dystonia

Key Message: Practitioners must know the common risk factors and etiopathogenesis of OGC. It would help them in choosing appropriate medication as well as timely identification and management.

Oculogyric Crisis (OGC) is a form of dystonic movement disorder characterized by paroxysmal, conjugate, and typically upward deviation of the eyeball, which occurs for seconds to hours.[1,2] Onset of OGC is generally acute, but sometimes it may develop after a few weeks or months of a precipitating event. Additionally, the patient may have increased blinking of the eyes, neck dystonia, tongue protrusion, blepharospasm, and autonomic signs such as perspiration, increased blood pressure, tachycardia, pupillary dilation, facial flushing, salivation, and difficulty in micturition.[1–3] Psychiatric symptoms such as anxiety, visual hallucinations or illusions, auditory hallucinations, catatonic phenomena, transitory delusions, or obsessive ideas may also accompany an OGC experience.[3]

Besides antipsychotics, other medications such as antiemetics, antidepressants, antiepileptics, and antimalarials are also associated with OGC.[4] It is also seen with various genetic or metabolic disorders that affect dopamine production, storage, or reuptake. The exact incidence of OGC is currently unknown, though one study reported it to be around 5.3%,[5,6] whereas the incidence of OGC with antipsychotics is in the range 0.9%–3.4%.[7] It is important to distinguish OGC from other similar presenting conditions such as epileptic seizures, paroxysmal tonic upgaze syndrome, or oculogyric tics.[8–10] In this review, we provide an overview of the various causes, risk factors, pathophysiology, diagnosis, differentials/mimickers, and management of OGC.

Materials and Methods

The literature regarding OGC was searched via PubMed and Google Scholar and through citations in relevant articles till December 2019, with keywords including OGC oculogyric eye movements, tonic eye movement, neuroleptics and OGC, antipsychotics and OGC, and various combinations of these. An

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additional search was performed for specific topics under this review. Only original articles (abstract or full text) that were published in the English language were reviewed. More than 200 articles related to the topic were found, out of which the articles relevant to psychiatry are primarily included here. Most of the publications related to OGC are either case reports or case series. In this review, we have focused on the adult population with OGC and its association with psychiatric symptoms/diagnosis.

Results

Etiology of OGC

Though the exact pathogenesis of OGC is not certain, various factors that contribute to its occurrence have been reported.

OGC is seen with different kinds of movement disorders such as tongue protrusion, lip-smacking, blepharospasm, choreoathetosis, anterocollis, and retrocollis.[11] Various neurological disorders are also found to be associated with OGC, such as Parkinson's disease, familial Parkinson's–dementia syndrome, dopa-responsive dystonia, parkinsonism with basal ganglia calcifications (Fahr's disease), neurosyphilis, multiple sclerosis, ataxia–telangiectasia, Wilson disease, and acute herpetic brain-stem encephalitis.[11]

Various genetic, neurometabolic disorders also play a part in the etiology of OGC. Hereditary dopamine transporter deficiency syndrome, in which, due to novel homozygous SLC6A3 gene mutations, there would be orolingual dyskinetic movements and OGC.[12] As a result of aromatic L-amino acid decarboxylase (AADC) deficiency, a rare autosomal recessive neurometabolic disorder characterized by a deficit of the AADC, which is involved in serotonin and dopamine biosynthesis,[13,14] the patient may have developmental delay, autonomic symptoms, hypotonia and movement disorder including OGC, dystonia or hypokinesia.[15] In GLUT1 deficiency syndrome, which is due to mutation in the SLC2A1 gene on chromosome 1p35-31.3 and often presents with mental retardation, epilepsy, paroxysmal exercise-induced dyskinesia, OGC can occur as an early sign of the disease in most patients.[16] Brain dopamine–serotonin vesicular transport disease, which occurs due to novel mutation in the monoamine transporter gene SLC18A2 (p.Pro237His), leads to hypotonia, bradykinesia, epilepsy, autonomic dysfunction, and OGC.[17] 6-pyruvoyl-tetrahydropterin synthase deficiency, which results in hyperphenylalaninemia along with dopamine and serotonin depletion in the central nervous system, usually presents with hypotonia, seizures, OGC, and developmental delay.[18,19] PLA2G6 associated neurodegeneration (PLAN), which occurs due to a homozygous mutation of PLA2G6, is a rare cause of OGC.[20]

Some neurodegenerative disorders are also found to be associated with the occurrence of OGC, such as mutations in the GRIN1 gene, which affects the function of both N-methyl-D-aspartate and dopamine D1 receptors, leading to oculomotor dystonic reactions;[21,22] Neuronal intranuclear hyaline inclusion disease, a multisystem degenerative disorder that involves both central and peripheral nervous systems, causing diffuse muscle spasms, dysarthria, dysphagia, tremors, ataxia, OGC, progressive muscle weakness, and atrophy[23]; Rett syndrome, progressive neurodegenerative disorder in females, leads to gait disturbance, bruxism, OGC, parkinsonism, and dystonia[24] and tyrosine hydroxylase (TOH) deficiency, an inborn error of catecholamine biosynthesis causing dystonia along with tremor, hypersensitivity to levodopa therapy, OGC, akinesia, and rigidity.[25,26] Other autosomal recessive disorders that rarely present with OGC are Kufor Rakeb,[27] haemophagocytic lymphohistiocytosis,[28] sepiapterin reductase deficiency,[29,30] and GTP cyclohydrolase I deficiency.[31]

Some brain lesions are also associated with OGC, such as lesions in periaqueductal and midbrain tegmentum,[32] brainstem,[33] or substantia nigra[34] and cystic glioma in the region of the posterior third ventricle.[35]

Finally, medications which would increase the risk of developing OGC include various groups such as antipsychotics, antiemetics, anticonvulsants, and antidepressants (**Table 1**). Acute dystonic reactions are commonly seen with the use of high-potency typical antipsychotics, relatively uncommon with atypical antipsychotics,[36] and rarely reported with clozapine.[37] A few cases of acute dystonic reaction, including OGC, have been associated with anesthetic agents like propofol, sevoflurane, nitrous oxide, and fentanyl administration.[38]

In recent times, most of the literature is associated with varied causes of OGC belonging to either neurometabolic disorders such as AADC deficiency, sepiapterin reductase deficiency, TUBB4A-related leukodystrophy[39] or medications (apart from what mentioned in **Table 1**) like anti-tubercular drugs,[40] or clebopride, a non-selective benzamide with antidopaminergic activity.[41]

Mechanism of OGC

The exact mechanism of OGC is not clear. Most of the brain lesions that lead to OGC are found in the nigrostriatal pathway.[2] Besides, several hypotheses have been given for drug-induced OGC such as striatal cholinergic hyperactivity,[42] striatal dopaminergic hypoactivity (commonly reported),[43] or the rarely reported striatal dopaminergic hyperactivity.[44] The majority of the conditions are related to dopaminergic dysfunction, including disorders of dopamine metabolism leading to low dopamine levels, such as TOH deficiency and AADC deficiency.[13,25]

TABLE 1.

Drugs Commonly Implicated in Oculogyric Crisis[2,43,57]

Drugs Classification	Drugs
First-generation antipsychotics (FGA)	Haloperidol Fluphenazine Flupentixol Perphenazine Chlorpromazine Zuclopenthixol
Second-generation antipsychotics (SGA)	Risperidone Amisulpride Aripiprazole Olanzapine Quetiapine Clozapine Ziprasidone Lurasidone
Antidepressants	Imipramine Escitalopram Fluvoxamine
Anticonvulsants	Carbamazepine Lamotrigine Gabapentin

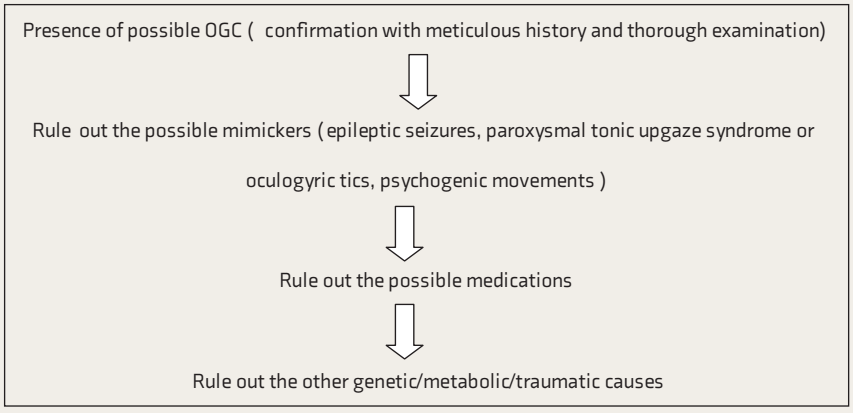
TABLE 2.

Proposed Criteria for the Diagnosis of Oculogyric Crisis[43]

Required Criteria	Supportive Criteria
• Tonic, conjugate deviation of eyes • Minutes to hours in duration • Consciousness preserved	• Preceded by anxiety, discomfort • The patient is aware of and bothered or disabled by the ocular deviations • Associated dystonia • Associated with a low dopamine state and improved by anticholinergics or dopaminergic medication

FIGURE 1.

Approach to a Patient with OGC-Like Phenomenon



OGC: Oculogyric crisis.

An imbalance between dopaminergic and cholinergic neurotransmission in the striatum has also been proposed for drug-induced OGC.[45]

Hypersensitivity of striatal dopamine receptors due to chronic dopamine receptor blockage, neurodegeneration of striatal interneurons, dysfunction of striatal gamma-aminobutyric acid (GABA)-ergic interneurons, or maladaptive synaptic plasticity are reported to be the causes of tardive involuntary movements.[46] Antidepressants-induced OGC is explained with hyperstimulation of 5-HT2 receptors, inhibition of dopaminergic activity, and alteration of cholinergic and GABAergic activity.[47]

Risk Factors of OGC

Certain characteristics of patients would make them more vulnerable for the emergence of OGC, such as younger age,[48,49] male sex,[48] greater severity of illness,[49] greater baseline psychopathology,[50] increasing neuroleptic dose,[48] parenteral administration of neuroleptics,[51] high potency of neuroleptic drugs,[52] abrupt discontinuation of anticholinergic medication within the first few weeks of initiating neuroleptics,[51] metabolic conditions (e.g., hypocalcemia, hyperthyroidism, hyperparathyroidism),[51] recent cocaine use,[51] and family history of dystonia.[44]

Diagnosis of OGC

Diagnosis of OGC is clinical, based on typical symptoms such as paroxysmal, conjugate, and typically upward deviation of the eyeball for seconds to hours. Slow et al.[43] has proposed the required and supportive criteria for diagnosing OGC (**Table 2**). Whenever clinicians encounter an OGC-like phenomenon, they should approach in a manner as suggested in **Figure 1**.

Differential Diagnosis/ Mimickers of OGC

Several clinical entities look similar to OGC. Therefore, meticulous history and thorough clinical examination should be done for differentiating other phenomena from OGC (**Table 3**).

Management of OGC

The mainstay of treatment of OGC is based on its etiology.[2] In the case of drug-induced OGC (e.g., antipsychotics), generally, the first approach is to taper down the causative drug. On the persistence of OGC on reducing the dose, discontinuation of the offending agent is advised, with shifting to other agents with a lesser propensity to cause dystonic reactions.[3,43]

For the management of cases where the mentioned strategy would not cause improvement, other pharmacological management options should be tried. In OGC, primarily anticholinergics such as trihexyphenidyl, benztropine, benzhexol, biperiden, procyclidine, or other centrally acting anticholinergic drugs or antihistaminergic agents (e.g., diphenhydramine, phenhydramine, chlorpheniramine, promethazine) are commonly used.[51] In most cases of acute dystonic reactions, the intravenous route is the preferred route of choice as it leads to improvement within 10 minutes. Alternatively, intramuscular (if you cannot access intravenous line) or oral (most unreliable, due to extensive gut first-pass metabolism) route can be used.[54] Sometimes benzodiazepines[2] and rarely dopamine agonists are also used.[3] To avoid recurrence, it is recommended to continue the management of OGC for at least a week but sometimes for longer periods in case of tardive OGC. If the above line of treatment fails, then clozapine remains a promising option[2] as it works via the stimulation of M4 muscarinic receptors, which results in inhibition of D1 receptors or the direct blockade of D1 receptors.[43] But rarely, in some cases, OGC has been reported with the use of clozapine.[37] Electro-convulsive therapy can also be given in tardive OGC cases with psychotic disorders.[55]

Prophylactic use of anticholinergic drugs for 1–2 weeks is indicated in patients with greater vulnerability for OGC with certain antipsychotics, though the available guidelines are inconsistent for the duration of use of such prophylactic agents.[49] Levodopa may be useful in Parkinson's disease with OGC. Anticholinergics are also found to be beneficial in OGC associated with focal brain lesions.

The dystonic reactions are associated with considerable physical and psychosocial consequences such as increased psychiatric comorbidity,[56] poor compli-

TABLE 3.

Common Differential Diagnosis for OGC[8–10]resulting in sustained unnatural positioning

Differentials	Description
Seizures	Repetitive, clonic, or tonic eye movements It is usually associated with loss of consciousness or impaired responsiveness, with other seizure phenomena such as tongue bite, incontinence, fall, confusion, and EEG changes Seizures are primarily cortical, while the OGC is primarily subcortical-striatal in etiology. Video-EEG monitoring can help in differentiating from OGC.[53]
Paroxysmal tonic upgaze	Upward eye deviation lasting seconds to hours Reported in children only
Ocular dyskinesias	Repetitive, upward gaze usually lasting only a few seconds Associated with L-dopa-induced dyskinesias and tardive dyskinesias. Generally, short-lasting, not associated with anxiety, and the patient often remains unaware of the eye movements.
Ocular tics	Brief ocular deviations It is associated with other tics and can be suppressed
Psychogenic	Distractible ocular movements and associated with other functional movements, and other functional findings

OGC: Oculogyric crisis.

ance,[57] increased fatigue, and sleep disturbances, leading to decrease quality of life.[58] OGC and blepharospasm can significantly affect daily living activities such as driving, shaving, combing, or dressing. These effects are more pronounced with tardive dystonia cases and can even lead to social avoidance, isolation, and stigmatization.[59] Hence, both non-motor and motor features associated with dystonia should be managed in order to improve the quality of life.

Conclusion

The timely diagnosis and early identification of the causative medication play an important role in the course and management of OGC. It can cause extreme agitation, which can mimic worsening of psychosis in psychiatric patients.[2] Treatment of OGC is mainly causal, as its etiology is multifactorial, involving both dopaminergic and cholinergic systems. Doctors must be aware of the varied presentation of OGC, as recurrent dystonic reactions have obvious implications on medication adherence and course and prognosis of primary disorders. OGC should be managed with meticulous history, examination, serial assessments, medications, and regular follow-up.

Declaration of Conflicting Interests

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