



Clinical Diagnosis and Management of Oculogyric Crisis: A Narrative Review

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ABSTRACT

Oculogyric crisis (OGC) is an acute dystonic reaction characterized by sustained, involuntary upward deviation of the eyes caused by repetitive spasms of the extraocular muscles. Episodes may last from several seconds to several hours. OGC is often misdiagnosed, particularly outside neurology and psychiatry, partly because of its variable presentation and its resemblance to other neurological or psychogenic movement disorders. Its pathophysiological mechanisms are not yet fully understood, although disruption of dopaminergic neurotransmission is a consistent finding. OGC is most frequently associated with the use of medications that affect dopaminergic pathways, including antipsychotics and certain antiemetics; it may also co-occur with underlying neurological or psychiatric conditions. Effective management typically involves withdrawing the causative agent and administering anticholinergic medications. This narrative review summarizes the etiology, pathophysiology, clinical manifestations, differential diagnosis, and management of OGC, providing a practical clinical overview for general practitioners and relevant specialists. Greater awareness of OGC may facilitate timely recognition and appropriate management while reducing diagnostic delays and unnecessary investigations.

Keywords: Dystonic reaction, extraocular muscles, neurology, oculogyric crisis, psychiatry.



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INTRODUCTION

Oculogyric crisis (OGC) is an acute neurological event characterized by sustained and involuntary upward deviation of the eyes, resulting from recurrent tonic spasms of the extraocular muscles. Episodes may vary in duration, ranging from several seconds to several hours, and are often paroxysmal.¹ OGC is classified as a form of dystonia, a movement disorder characterized by sustained muscle contractions that cause abnormal movements or postures.² As a form of episodic dystonia, the diagnosis of OGC requires a comprehensive clinical understanding of its various causes, symptom patterns, and underlying pathophysiological mechanisms.¹

Initially, OGC was commonly observed in patients with post-encephalitic parkinsonism. However, subsequent research has shown that this condition may occur across a wide range of clinical contexts, including drug reactions (particularly to antipsychotics and antiemetics), genetic or sporadic movement disorders, and structural brain lesions.^{3,4}

While the precise prevalence of OGC in Indonesia remains unknown, a brief review from China reported that the prevalence of OGC is approximately 5.3%.³ The incidence of antipsychotic-induced OGC has been reported to range from 0.9% to 3.4%.⁵ The clinical presentation of OGC can mimic seizures, encephalitis, and psychogenic disorders, which may lead to inappropriate diagnostic investigations and unnecessary use of anticonvulsant therapy. Given the limited number of studies on this topic, this review summarizes the etiology, pathophysiology, clinical manifestations, differential diagnoses, and management approaches for OGC to provide clinicians with practical guidance.

MAIN REVIEW

SEARCH STRATEGY

We conducted a literature search for relevant articles on OGC using Google Scholar and PubMed, combining the following keywords: "oculogyric crisis", "krisis okulogirik", "dystonia", and "movement disorder". Although we did not apply strict review protocols, we evaluated articles based on their relevance

to the etiology, risk factors, pathophysiology, clinical manifestations, differential diagnosis, and management of OGC. This review included open-access full texts published between 2016 and the present, as well as several earlier publications. Articles written in English or Indonesian were considered.

ETIOLOGY

The underlying causes of *oculogyric crisis* (OGC) are diverse and can broadly be categorized into three major groups: medication use, neurological disorders, and hereditary or genetic factors. Drug-induced OGC is the most frequently encountered form. Drug-induced OGC is generally associated with pharmacological agents that block dopamine D2 receptors, including antipsychotic (neuroleptic) and antiemetic agents.⁶ Ondansetron is one antiemetic that has been reported to cause extrapyramidal adverse effects in the form of OGC; however, the mechanism remains unclear, as ondansetron has no direct effect on dopaminergic, histaminergic, adrenergic, or cholinergic receptor activity. Nevertheless,

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ondansetron is believed to have the potential to inhibit increased dopaminergic activity.⁷

These medications disrupt the balance of neurotransmitter systems, particularly within the basal ganglia, which play an important role in regulating motor function. This imbalance may trigger dystonic reactions, one of which is OGC. The reaction is characterized by a sudden upward deviation of the eyes that persists for a period due to extraocular muscle spasm. A six-year prevalence study of OGC episodes found that the incidence of OGC among patients receiving second-generation antipsychotics reached 1.8%, most commonly due to risperidone or olanzapine (as monotherapy or in combination with other antipsychotics); the episodes resolved after switching the antipsychotic medication.⁸

Table 1 summarizes medications reported to induce OGC reactions.^{4,6,7}

Table 1. Medications reported to induce dystonic reactions.^{4,6,7}

Classification	Drug Name
First-generation antipsychotics	Haloperidol
	Fluphenazine
	Flupentixol
	Perphenazine
	Chlorpromazine
	Zuclopenthixol
Second-generation antipsychotics	Risperidone
	Amisulpride
	Aripiprazole
	Olanzapine
	Quetiapine
	Clozapine
	Ziprasidone
	Lurasidone
Antidepressants	Imipramine
	Escitalopram
	Fluvoxamine
Anticonvulsants	Carbamazepine
	Lamotrigine
	Gabapentin
Antiemetics	Metoclopramide
	Phenothiazines
	Ondansetron

Neurological disorders associated with OGC include conditions affecting brain structures such as the basal ganglia, brainstem, and cerebellum.⁹ Structural abnormalities,

including brain tumors, cerebral infarction, and demyelinating plaques, may disrupt neural pathways that regulate eye movements and therefore have the potential to cause OGC through impaired coordination among brain regions involved in ocular motor control. In addition, several studies have suggested that the cerebellum may contribute to the pathophysiology of dystonia, including OGC, because of its involvement in motor and sensory signal processing.^{10–12}

Neurodegenerative disorders are also known to affect control of eye movements, particularly saccadic movements; these alterations not only reflect motor impairment but are also related to broader cognitive functions.¹³ One relevant example is neuronal intranuclear hyaline inclusion disease (NIID), which is characterized by eosinophilic intranuclear inclusions in neurons of the central, peripheral, and autonomic nervous systems. This degenerative multisystem disorder is characterized by widespread muscle spasms, speech and swallowing disturbances, tremors, impaired motor coordination, progressive muscle weakness, muscle atrophy, and OGC.¹

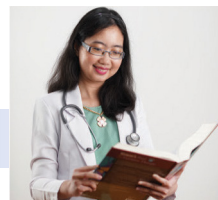
A review by Mahal, et al., found that OGC commonly occurs in disorders involving dysfunction of dopamine production and reuptake, collectively referred to as monoamine neurotransmitter disorders.¹ Direct disruption of dopaminergic pathways also affects dopamine and serotonin levels. Parkinsonism may also occur together with dystonia due to dopamine deficiency. Brain MRI findings in these neurometabolic disorders are generally normal; diagnosis is established only through neurotransmitter studies of cerebrospinal fluid samples or genetic testing.¹⁴

Another condition that may be present with OGC is Rett syndrome, a neurodegenerative disorder that primarily affects girls. Common manifestations include gait disturbance, bruxism, OGC, parkinsonism, and dystonia. In addition, tyrosine hydroxylase (TOH) deficiency, a genetic disorder of the catecholamine production pathway, may cause dystonia, tremor, hypersensitivity to levodopa therapy, OGC, akinesia, and muscle rigidity.¹

Several rarer autosomal recessive genetic disorders have also been reported to manifest as OGC, including Kufor-Rakeb disease, hemophagocytic lymphohistiocytosis, sepiapterin reductase deficiency, and GTP cyclohydrolase I deficiency. In addition, hereditary dopamine transporter deficiency syndrome, caused by homozygous mutations in the SLC6A3 gene, is characterized by orolingual dyskinesia and OGC.¹⁵ Aromatic L-amino acid decarboxylase (AADC) deficiency, a rare neurometabolic disorder, causes impaired serotonin and dopamine production, leading to developmental delay, hypotonia, autonomic dysfunction, and movement disorders such as OGC, dystonia, or hypokinesia.^{16,17}

Another disorder that may cause OGC is GLUT1 deficiency, which is caused by mutations in the SLC2A1 gene on chromosome 1p35–31.3. Symptoms include intellectual disability, epilepsy, and exercise-induced dyskinesia. OGC may be an early indicator in most patients.¹⁸ Vesicular dopamine-serotonin transporter disease occurs because of a de novo mutation in the monoamine transporter gene SLC18A2 (p.Pro237His), causing hypotonia, bradykinesia, epilepsy, autonomic dysfunction, and OGC.¹⁹ 6-Pyruvoyl-tetrahydropterin synthase deficiency causes hyperphenylalaninemia and reduces dopamine and serotonin levels in the central nervous system, resulting in hypotonia, seizures, OGC, and developmental impairment.²⁰ Although rare, OGC may also present as a symptom of homozygous PLA2G6 mutation.²¹

OGC has been reported in association with several etiologies, including structural lesions such as striatal capsular infarction and brainstem lesions, Wilson disease, acute herpes virus-related brainstem encephalitis (accompanied by stupor), Japanese encephalitis (JE), vascular parkinsonism, paraneoplastic syndrome, and parkinsonism associated with PLA2G6 gene mutation after L-dopa treatment.¹⁴ In addition, OGC has been reported in a two-year-old patient with trisomy 21 who developed posterior reversible encephalopathy syndrome, which subsequently progressed to directionless eye movements.¹⁴



Other reports include single cases of Hallervorden-Spatz syndrome²² and West syndrome.²³ In many of these case reports, the descriptions of OGC were either not detailed or showed atypical features, such as forward eye fixation in vascular parkinsonism or improvement of symptoms after antiepileptic drug administration in cases of brainstem lesions. Other eye movement disorders reported as single cases include a “dystonic oculogyric state” in patients with parkinsonism and basal ganglia calcification²⁴ and “extraocular muscle dystonia” in patients with hepatocerebral degeneration.¹⁴

RISK FACTORS

Risk factors for OGC require further investigation, although OGC has frequently been associated with medication use, particularly antipsychotic agents. OGC is commonly observed in young adult men receiving high-potency antipsychotics, particularly higher neuroleptic doses and parenteral administration such as intramuscular injection. It has been associated with an increased risk of acute dystonic reactions, including OGC, suggesting that both the intensity of dopamine receptor blockade and the route of administration may influence susceptibility.²⁵ Although OGC is more commonly associated with high-potency first-generation antipsychotics, but several case reports have noted that OGC may also occur with second-generation antipsychotics, including olanzapine, quetiapine, amisulpride, aripiprazole, and lurasidone.^{26,27} Several cases have developed OGC following dose escalation, with improvement after dose reduction or discontinuation of the suspected agent, suggesting a possible dose-related susceptibility in some patients.²⁷ Patients with neurological disorders (i.e. a history of dystonia or other extrapyramidal reactions), particularly hereditary or genetic conditions, and previous exposure to antipsychotic medications are also more vulnerable to OGC.¹⁷ Although OGC is neither fatal nor life-threatening, as an adverse drug effect, it has been reported to reduce adherence to regular medication use, worsen sleep quality, and increase chronic fatigue. In addition, OGC symptoms may become a source of social stigma, causing patients to feel embarrassed or alienated and thereby avoid social activities. Ultimately, this condition

may result in significant psychological and physical functional impairment and affect overall quality of life.²⁷

PATHOPHYSIOLOGY

Although the fundamental pathophysiology of OGC is not fully understood, most cases show reduced dopamine levels.¹⁴ OGC is frequently observed in Parkinson disease,²⁸ and pathological findings indicate involvement of the substantia nigra.²⁹ The use of anticholinergics such as trihexyphenidyl, benztropine, benhexol, biperiden, procyclidine, or other centrally acting anticholinergics, which are known to increase dopamine release, has been shown to provide therapeutic benefit in OGC.¹ These findings support the hypothesis that reduced dopaminergic activity contributes to the pathogenesis of OGC. However, the effectiveness of L-dopa in patients with OGC is inconsistent; some patients show initial symptomatic improvement, followed by relapse.¹⁴

The strong association between OGC and dopamine receptor-blocking pharmacological agents further supports a hypodopaminergic state in OGC. Inhibition of several dopamine receptor subtypes appears sufficient to trigger OGC, given that both typical and atypical dopamine receptor antagonists may be causative agents. OGC is often associated with acute dystonic reactions; however, no direct association has been found with drug-induced parkinsonism following long-term exposure. This suggests that OGC is more likely triggered by direct dopamine receptor blockade or acute dopamine deficiency.¹⁴

Several other hypotheses regarding the pathophysiology of OGC have also been proposed, including hyperactivity of striatal cholinergic pathways,³⁰ imbalances between dopaminergic and cholinergic neurotransmitters,¹ and, less commonly, dopaminergic hyperactivity in the striatum.³¹ Nevertheless, the most frequently reported mechanism remains related to dopaminergic pathway dysfunction.¹

Although the precise pathophysiology of OGC remains uncertain, the available evidence most strongly supports a hypodopaminergic state as a central mechanism. Slow and Lang

highlighted the consistent association of OGC with dopaminergic dysfunction, particularly in patients exposed to dopamine-receptor blocking agents and in inherited disorders affecting dopamine synthesis or metabolism, with several neurometabolic conditions demonstrating reduced cerebrospinal fluid homovanillic acid levels. The association between dopamine deficiency severity and OGC occurrence, along with improvement after dopaminergic treatment in selected disorders, further supports this hypothesis.¹⁴

The involvement of striatal cholinergic activity has been proposed as a potential downstream consequence of dopaminergic dysfunction rather than as an independent primary mechanism. Reduced dopaminergic signaling may produce relative cholinergic overactivity within the striatum, disrupting the dopaminergic-cholinergic balance and leading to abnormal motor output. The clinical effectiveness of anticholinergic agents in drug-induced OGC provides supportive evidence for this mechanism.¹⁴

However, the evidence for a primary hypercholinergic mechanism is less direct than that supporting dopaminergic dysfunction. Similarly, Barow, et al., emphasized that although dopaminergic dysfunction provides a common mechanistic link across drug-induced OGC, hereditary and sporadic movement disorders, and focal brain lesions, the dopaminergic-cholinergic imbalance does not fully explain all forms of OGC. Tardive OGC and OGC triggered by agents that do not act mainly on dopaminergic pathways may depend on additional mechanisms such as altered GABAergic and serotonergic signaling and maladaptive synaptic plasticity.⁴ Accordingly, a hypodopaminergic state remains the best-supported mechanism, with cholinergic activity more plausibly acting as a downstream contributor.

CLINICAL MANIFESTATIONS

OGC is an acute dystonic reaction characterized by brief muscle spasms that cause upward deviation of the eyes. The onset of OGC is generally sudden, promptly after the administration of drugs, or less often after a long-term exposure to neuroleptics. Typical episodes last minutes to hours and usually resolve within 24 to 48 hours after



discontinuing exposure to the causative agent.^{4,14} These spasms may be preceded or followed by psychiatric symptoms, including anxiety, restlessness, obsessive urges, increased sensitivity to light, or visual distortion.¹ OGC may also be accompanied by various other clinical manifestations, including an abnormally increased blink rate, uncontrolled tongue protrusion, eyelid spasm (blepharospasm), cervical dystonia, and signs of autonomic dysfunction. Autonomic symptoms may include excessive sweating, increased blood pressure, tachycardia, pupillary dilation (mydriasis), facial flushing, hypersalivation, and difficulty urinating.¹

Slow, *et al.*, proposed diagnostic criteria for OGC, divided into primary and supportive criteria (**Table 2**). First, OGC requires paroxysmal, tonic, conjugate

ocular deviation. Second, the duration of each episode should range from several minutes to hours. Preserved consciousness is a highly important diagnostic criterion because it helps distinguish OGC from differential diagnoses that resemble OGC, such as seizures, which are usually followed by impaired consciousness, and ocular bobbing. Additional criteria may include the presence of other dystonic disorders and an association with low dopamine levels (which should be supported by clinical symptoms and cerebrospinal fluid studies). In addition, patients remain conscious during ocular deviation, or the episode may be preceded by discomfort or anxiety. Slow, *et al.*, proposed that all three primary criteria, along with at least three additional/supportive criteria, should be fulfilled to establish a diagnosis of OGC.¹⁴

For diagnosing OGC, history-taking is essential, as the most common etiology is medication-related adverse effects (**Figure 1**).¹⁴ All medications taken by the patient should be documented, including additional recreational substances (which may include neuroleptics). If a medication-related etiology has been excluded, consider other genetic or hereditary etiologies. Clinicians may inquire about family history; however, the absence of a family history of genetic disease does not completely exclude a genetic disorder, given the possibility of autosomal recessive inheritance.

Brain MRI may be performed even though findings are generally normal, because it is important to exclude specific neurological conditions such as hypomyelination with atrophy of the basal ganglia and cerebellum (H-ABC).¹⁴

Age at onset may also help identify the cause of OGC. Childhood-onset OGC is usually caused by neurometabolic disorders involving dopamine metabolic pathways, resulting in disturbances in dopamine levels or in overall serotonin and dopamine levels. Childhood-onset OGC is often misdiagnosed as cerebral palsy because of findings suggestive of autosomal recessive genetic abnormalities. Therefore, genetic testing and cerebrospinal fluid studies are required to diagnose OGC. Adult-onset OGC is usually neurodegenerative and requires genetic investigations or other specific tests, such as rectal biopsy, to evaluate for neuronal intranuclear hyaline inclusion disease (NIID), one of the neurodegenerative conditions that may manifest as OGC.¹⁴

In resource-variable settings, the diagnosis of OGC remains primarily clinical, and specialized diagnostic testing (i.e. cerebrospinal fluid neurotransmitter analysis and genetic testing) is often unavailable, particularly in emergency care. Therefore, careful characterization of the ocular episodes, medication history, age at onset, associated neurological features, and family history remains essential for initial clinical assessment.¹⁴

DIFFERENTIAL DIAGNOSIS

OGC is an acute dystonic reaction involving the extraocular muscles and may resemble

Table 2. Primary and supportive criteria for the diagnosis of OGC.¹⁴

Primary Criteria	Supportive Criteria
Tonic and conjugate deviation of the eyes	Preceded by anxiety/discomfort
Duration: several minutes to hours	The patient is conscious and feels distressed or experiences functional impairment due to the condition
Preserved consciousness	Dystonia
	Associated with low dopamine levels and improves with anticholinergic or dopaminergic medication

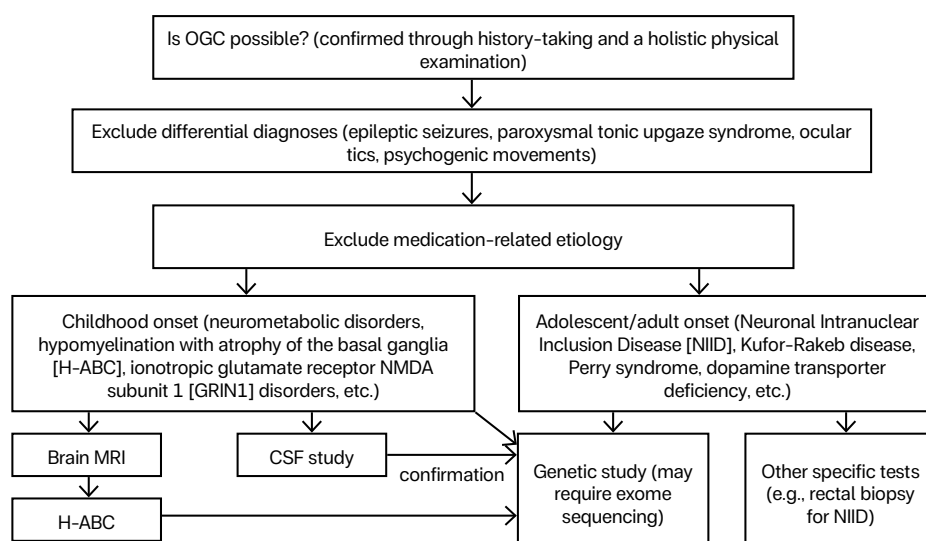


Figure 1. Diagnostic framework for OGC.¹⁴

Abbreviations: OGC: Oculogyric crisis; MRI: Magnetic resonance imaging; H-ABC: Hypomyelination with atrophy of the basal ganglia and cerebellum; CSF: Cerebrospinal fluid.



other dystonic or dyskinetic movements. This condition has a strong association with the use of neuroleptic agents, as well as various other neurological disorders that may clinically mimic a range of movement disorders. However, routine neurological or ophthalmological examinations are often normal, which may complicate the diagnosis.³²

A thorough medical history and careful clinical examination are required to distinguish OGC from other differential diagnoses. One differential diagnosis most closely resembling OGC is anti-NMDAR encephalitis. In patients with anti-NMDAR encephalitis, OGC often occurs together with seizures, making it difficult to determine whether eye deviation is caused purely by OGC or by a seizure phenomenon. Seizures due to anti-NMDAR encephalitis are characterized by altered consciousness.¹⁴ A clinical comparison with other conditions is shown in **Table 3**.¹

MANAGEMENT

If a medication is identified as the cause of OGC, reduce the dose of the causative medication. When dose reduction does not improve symptoms, complete discontinuation

is recommended.^{26,32} Dose adjustment or drug discontinuation generally produces favorable outcomes in antiemetic- or antipsychotic-induced OGC, because both types of medication are known to interfere with dopaminergic transmission. Individual susceptibility to specific medications has also been found to influence the occurrence of OGC and is therefore an important factor in determining management strategies.³

In clinical practice, attention should be given to dopamine-receptor blocking medications such as haloperidol and metoclopramide, which may be encountered in psychiatric and acute-care settings.^{14,35} A focused medication history, including recent initiation or dose escalation, is essential for identifying the potential offending agent and facilitating prompt treatment.^{1,14}

For patients presenting to the emergency department with suspected OGC, the initial approach should include assessment of airway, breathing, circulation, and level of consciousness, followed by confirmation of the characteristic ocular deviation and review of recent medication exposure.

Once the adjustment of the causative medication is insufficiently effective, several pharmacological options may be used as alternative therapies. In acute cases, intravenous or intramuscular antihistamines (diphenhydramine, chlorpheniramine, or promethazine) or benzodiazepines (diazepam) are strongly recommended. These medications generally relieve OGC symptoms within minutes. If symptoms persist, the dose may be repeated after 15 to 30 minutes. To prevent recurrence, recommend oral anticholinergics such as trihexyphenidyl for at least 4 to 7 days. In non-acute cases, oral anticholinergics may also be first-line, with oral benzodiazepines such as clonazepam added if the expected effect is not achieved.^{1,3,32}

Some antipsychotics, such as clozapine or quetiapine, have a lower risk of dystonia; persistent OGC symptoms have improved in long-term antipsychotic users who switched their treatment to quetiapine or clozapine.^{3,28} It should be noted that the therapeutic effect in tardive OGC may be limited, because clozapine itself can also cause OGC.³

Levodopa may be considered as a treatment option for OGC and has produced improvement in patients with OGC accompanied by parkinsonism.¹⁴ However, levodopa may also induce dyskinesia (levodopa-induced ocular dyskinesia), characterized by blepharospasm occurring 10–15 minutes after administration, accompanied by peribuccal or buccolingual dyskinesia.³³ Based on these findings, the relationship between levodopa use and OGC requires further investigation.^{14,33}

CONCLUSION

OGC is an eye movement disorder that is strongly associated with various neurological disorders, particularly dystonia. OGC is most commonly caused by medication use, especially dopamine receptor-blocking agents that are frequently found among antiemetic and antipsychotic medications. In addition, certain neurological conditions or genetic factors may trigger OGC. Disruption of dopaminergic pathways is presumed to be the main mechanism underlying OGC, although the exact pathophysiology remains incompletely understood. From a clinical perspective, early recognition of OGC is

Table 3. Differential diagnosis of OGC.¹

Diagnosis	Description
Seizures or epilepsy	- Focal seizures are characterized by involuntary and repetitive tonic/clonic movements of the head accompanied by eye deviation - Primarily caused by cortical disturbances - Associated with impaired consciousness - EEG should be used as a diagnostic modality
Ocular tics	Eye deviation is associated with tic disorders, usually in children aged 6 to 12 years.
Ocular dyskinesia	- Repetitive, transient upward eye movements of which the patient is unaware - Associated with other dyskinesias (e.g., tardive dyskinesia or L-dopa-induced dyskinesia), rather than a mental disorder
Paroxysmal tonic upgaze	- Upward eye deviation lasting from several seconds to hours, generally with childhood onset (age 2 weeks to 90 months) - In 80% of cases, onset occurs before the age of 2 years - Episodes resolve spontaneously 1 to 48 months after onset - May also occur in children with cerebellar ataxia or developmental disorders due to GRID2 mutations
Psychogenic	Distressing ocular deviation associated with other functional movements



essential, particularly for general practitioners and clinicians working in emergency or primary care settings. In patients presenting with acute or recurrent ocular deviation with preserved consciousness, clinicians should obtain a thorough medication history, particularly regarding recent exposure to dopamine receptor-blocking agents. Misdiagnosis may lead to unnecessary investigations or inappropriate treatments. Dose adjustment or discontinuation is strongly recommended in cases of drug-induced OGC, while intravenous or intramuscular therapy may be used in severe or acute cases.

Despite increasing recognition of OGC, several important knowledge gaps remain. Further studies are required to clarify its definitive pathophysiological mechanisms. In addition, epidemiological studies, especially

in Indonesia, are needed, as local prevalence and incidence data are currently unavailable. Finally, evidence regarding the optimal management of refractory or recurrent OGC remains limited, particularly concerning treatment strategies when withdrawal of the causative agent and conventional pharmacological therapy are ineffective. Future prospective and comparative studies are therefore needed to establish more standardized diagnostic and therapeutic approaches.

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Author Contributions

HS and CJT contributed to the conception

and design, data collection, and drafting of the manuscript. All authors have reviewed and approved the manuscript.

Conflict of Interest

The authors have no conflict of interest to declare.

Declaration on the Use of Artificial Intelligence (AI)

The authors declare that no artificial intelligence tools were used in the conduct of this study or preparation of this manuscript. All authors affirm that the entire content of the manuscript has been reviewed, verified, and approved by the authors, and that full responsibility for the content remains with the authors.

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