REVISTA NEUROCIÊNCIAS

Oculogyric Crises: an underdiagnosed condition

Crises Oculógiras: uma condição subdiagnosticada

Crisis Oculógiras: una condición subdiagnosticada

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Resumo

Introdução. A distonia é um distúrbio do movimento hipercinético caracterizado por posturas anormais de natureza torcional ou movimentos repetitivos. A crise oculógira (COG) é um tipo específico de distonia que compromete músculos extraoculares. Embora rara, COG pode gerar grandes dificuldades diagnósticas. **Objetivo**. Realizar uma revisão narrativa sobre COG para chamar atenção dos neurologistas para essa condição debilitante. **Método.** Realizamos uma revisão não sistemática de artigos publicados em inglês ou português por meio do Google Scholar, de 2000 a 2025, utilizando os termos "oculogyric crises/oculogyric crisis" ou "crises oculógiras". **Resultados.** Obtivemos 239 manuscritos sobre COG. Trinta e dois artigos foram selecionados para esta revisão. A COG é uma distonia focal ou segmentar infrequente que afeta os músculos extraoculares. Classicamente atribuída ao uso agudo de antagonistas dos receptores de dopamina, também pode persistir com o uso crônico de fármacos antidopaminérgicos. Novos casos continuam sendo relatados, apesar do uso de novos agentes antipsicóticos atípicos. Além dos antipsicóticos, a COG pode surgir com uso de fármacos antieméticos e terapias medicamentosas para o parkinsonismo. Outras condições neurometabólicas, genéticas e estruturais são responsáveis por alguns casos. Representa um desafio diagnóstico devido à sua curta duração e à semelhança com outras condições graves, como crises epilépticas e encefalite. O tratamento baseia-se na administração de fármacos anticolinérgicos ou benzodiazepínicos. **Conclusões.** A COG é uma doença infrequente que pode ser causada por fármacos de uso rotineiro na Neurologia. Neurologistas devem estar atentos à sua ocorrência e etiologias para realizar um diagnóstico rápido e suprimir o agente causador.

Unitermos. Distonia; Agentes Antipsicóticos; Dopamina; Receptores

Abstract

Introduction. Dystonia is a hyperkinetic movement disorder characterized by abnormal postures of a twisting nature or repetitive movements. Oculogyric crises (OGC) are a specific type of dystonia compromising extraocular muscles. Although rare, OGC can cause great diagnostic difficulties. **Objective**. To perform a narrative review of OGC to draw the attention of neurologists to this debilitating condition. **Method.** We performed a non-systematic review of published papers in English or Portuguese through Google Scholar from 2000 until 2025 using the terms "oculogyric crises/oculogyric crisis" or "crises oculógiras". **Results.** We obtained a total of 239 manuscripts about OGC. Thirty two papers were selected for this review. OGC is an infrequent focal or segmental dystonia affecting extraocular muscles. Classically attributed to the acute use of dopamine receptor antagonists, it can also persist with chronic antidopaminergic use. Of relevance is the finding that new cases continue to be reported, despite the use of new atypical antipsychotic agents. Besides antipsychotics, OGC arises from the use of antiemetics drugs and drug therapies for parkinsonism. Other neurometabolic, genetic and structural conditions are responsible for some cases. It represents

a diagnostic challenge due to its short duration and its similarity to other serious conditions as epileptic seizures and encephalitis. Treatment is based on the administration of anticholinergic or benzodiazepine drugs. **Conclusions.** OGC is an infrequent disease that can be caused by drugs routinely used in Neurology. Neurologists should be aware of its occurrence and causes in order to make a rapid diagnosis and suppress the causative agent.
Keywords. Dystonia; Antipsychotic Agents; Dopamine; Receptors

Resumen

Introducción. La distonía es un trastorno del movimiento hipercinético caracterizado por posturas anormales de naturaleza torsional o movimientos repetitivos. La crisis oculógira (COG) es un tipo específico de distonía que compromete los músculos extraoculares. Aunque rara, la COG puede generar grandes dificultades diagnósticas. **Objetivo**. Realizar una revisión narrativa sobre la COG para llamar la atención de los neurólogos sobre esta condición debilitante. **Método.** Realizamos una revisión no sistemática de artículos publicados en inglés o portugués a través de Google Scholar desde 2000 hasta 2025 utilizando los términos "oculogyric crises/oculogyric crisis" o "crisis oculógiras". **Resultados.** Obtuvimos un total de 239 manuscritos sobre COG. Se seleccionaron treinta y dos artículos para esta revisión. La COG es una distonía focal o segmentaria infrecuente que afecta los músculos extraoculares. Clásicamente atribuida al uso agudo de antagonistas de los receptores de dopamina, también puede persistir con el uso crónico de fármacos antidopaminérgicos. Es relevante el hallazgo de que continúan reportándose nuevos casos a pesar del uso de nuevos agentes antipsicóticos atípicos. Además de los antipsicóticos, la COG puede surgir por el uso de fármacos antieméticos y terapias farmacológicas para el parkinsonismo. Otras condiciones neurometabólicas, genéticas y estructurales son responsables de algunos casos. Representa un desafío diagnóstico debido a su corta duración y su similitud con otras condiciones graves, como crisis epilépticas y encefalitis. El tratamiento se basa en la administración de fármacos anticolinérgicos o benzodiacepinas. **Conclusiones.** La COG es una enfermedad infrecuente que puede ser causada por fármacos de uso rutinario en Neurología. Los neurólogos deben estar atentos a su aparición y sus causas para realizar un diagnóstico rápido y suprimir el agente causal.
Palabras clave. Distonía; Agentes Antipsicóticos; Dopamina; Receptores

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INTRODUCTION

Dystonia is a hyperkinetic movement disorder in which there is an abnormal posture of a twisting nature or a repetitive patterned or tremulous movement, that is often initiated or worsened by voluntary action[1]; It arises from involuntary co-contraction of agonist and antagonist muscles[2] and can be classified as focal, segmental, multifocal, generalized, or hemidystonia based on the affected body region. Focal dystonia is confined to a single body region and cervical dystonia is the most common adult-

onset focal dystonia, followed by blepharospasm. Segmental dystonia affects two or more contiguous parts of the body[2,3].

Oculogyric crises (OGC) are an infrequent subtype of focal or segmental dystonia, affecting extraocular muscles and is mainly characterized by paroxysmal involuntary eye muscle spasms with fixed upward gaze. Rarely OGC presents with a downward fixed gaze[4,5]. When OGC is the only dystonic manifestation, it is poorly diagnosed. When associated with other dystonic movements more easily observable, mainly in the cranio-cervical region, the diagnosis is straightforward. OGCs are often accompanied by increased blinking of the eyes, ocular pain, backward and lateral flexions of the neck, blepharospasm, wide open mouth, and tongue protrusion[6,7].

An episode of OGC lasts a few seconds to several hours, more commonly minutes[8]. Short duration with no direct observation often delays the diagnosis. The most frequent cause of OGC is an acute dystonic reaction to dopamine receptor antagonists (DRAs). Other causes of OGC at a lower frequency are chronic use of DRAs, pharmacologic therapy with levodopa for parkinsonian syndromes, neurometabolic, genetic, and secondary to structural lesions[5].

The objective of this paper is to perform a narrative review of OGC, discussing its clinical diagnosis, pathophysiology, risk factors, differential diagnosis and treatment.

METHOD

We performed a non-systematic review using the terms/keywords "oculogyric crisis" or "oculogyric crises" or "crises oculógiras" present in the titles of articles in the English or Portuguese languages from January 2000 until January 2025. The database used was Google Scholar. We included experimental, review, case reports, and expert opinions papers with no specific criteria for exclusion. A few papers from the initial search were selected based on their relevance to the discussion, prioritizing studies that contributed to the clinical, pathophysiological, and therapeutic understanding of the topic. While no strict inclusion or exclusion criteria were predefined, the final selection was guided by the quality and significance of the content presented.

RESULTS AND DISCUSSION

We found a total of 239 articles about OGC. Finally, 32 papers were chosen for the review, 31 of them in English and one in Portuguese. The results were organized in the items of (1) Semiology of OGC; (2) Causative agents of OGC; (3) Pathophysiology of OGC; (4) Risk factors of OGC; (5) Differential diagnosis of OGC and (6) Treatment of OGC.

Semiology

OGC is a neurological disorder classically characterized by focal ocular muscle dystonia, which was first observed in patients with post-encephalitic parkinsonism in 1928[4,9].

Clinical symptoms and subtle eye deviation are sustained for variable periods, lasting intermittently from seconds to several hours, often minutes. It can be associated to painful symptoms in ocular muscles derived from dystonia or accompanied by jaw opening, neck flexion, tongue protrusion, blepharospasm and autonomic signs, such as pupillary dilation or perspiration[10]. In addition, psychiatric symptoms such as agitation, anxiety, auditory, tactile or visual hallucination have also been reported[7,11]. Fundamental for diagnosis, there is no loss of consciousness during an episode of OGC and the patient can fully report the symptoms or discomfort to the examinator. OGC is usually challenging because the episodes are self-limited and rarely witnessed by the physician[12].

Causative agents

Conditions associated with OGC are often subdivided into three categories: A) drug-induced, B) hereditary or sporadic disorders, and C) focal structural lesions[13].

The most frequent cause for an OGC is an acute dystonic reaction secondary to the administration of dopamine receptor antagonists (DRAs). Among these are neuroleptic medications, with first-generation antipsychotics such as haloperidol, pimozide, and others having the highest risk, followed by atypical second-generation antipsychotics (risperidone, olanzapine, quetiapine)[14,15]. Other new antipsychotic drugs such as aripiprazole present different mechanisms of action from DRAs. Aripiprazole is a third-

generation atypical antipsychotic, that functions as a partial agonist at dopamine receptors (D2 subtype) and serotonin receptors 5-HT1A, and as an antagonist at 5-HT2A receptors. Recently it became responsible for most cases reported of OGC in psychiatric disorders due to its better tolerability and crescent prescription[16,17]. Rarely, lithium treatment causes OGC as an acute dystonic reaction, mainly in doses above 900 mg/day[10].

In acute drug reactions, OGC develops after a few hours after intake, with 90% of cases occurring until 96 hours post-drug exposition. However, some cases develop after weeks or months from a precipitating drug[18,19].

It is fundamental to emphasize that, in addition to OGC as acute dystonic reactions, chronic use of antipsychotics can provoke OGC as a dystonic tardive syndrome[6,7]. Some authors renamed OGC as "tardive ocular deviation", since there is no true rotation nor does it always manifest as a crisis[19]. Tardive dystonia occurs after three to six months of exposition to DRAs and must persist for 1 month[20]. In this situation, the OGC may not respond to the withdrawal of DRA treatment.[6] On the contrary, rarely DRAs can also cause OGC when abruptly discontinued and can improve with reintroduction of the drug[13].

OGC is often a non-fatal condition, and individuals may show a lack of adherence to DRAs treatment due to social stigma from the emergence of OGC in public. Furthermore, OGC may be confused with symptoms of a conversive disorder associated to psychiatric conditions[11,15].

Excluding the use of antipsychotic medications (DRAs), other drugs are commonly involved in OGC. Antiemetics such as benzamides (metoclopramide, clebopride), anticonvulsants (carbamazepine, oxcarbazepine, lamotrigine, gabapentin), or antidepressants selective serotonin reuptake inhibitors (citalopram, escitalopram, fluoxetine, fluvoxamine) are frequently involved[9,12,21,22]. Rarely, antimalarial treatments more used in developing countries are responsible for OGC such as chloroquine and artemether/lumefantrine[23,24].

Furthermore, differences in frequency of OGC cases across continents may derive from misdiagnosis and underreporting. For example, in Brazil, bromopride, a benzamide not available in Europe and the USA, can cause acute dystonic reactions originated by its DRAs mechanism of action[22]. However, OGC from bromopride administration is not reported in Brazilian literature at the same frequency as metoclopramide, suggesting underreporting or misdiagnosis. The development of extrapyramidal symptoms and OGC at therapeutic antiemetics doses may be related to a homozygous genetic polymorphism, which induces the inactivity of a subclass of the Cytochrome P450 liver enzyme[22].

Antidepressants-induced OGC can be explained by the hyperstimulation of serotonin receptors subtype 5-HT2, inhibition of dopaminergic activity, and abnormalities of cholinergic and gabaergic activity[18].

Metabolic encephalopathies such as Wilson's disease,

mainly in young individuals <40 years old, must always be investigated[25]. Other metabolic encephalopathies caused by genetic mutations in enzymes related to the production of biogenic amines, such as dopamine, noradrenaline, and serotonin can produce OGC. These include deficiency of aromatic L-amino acid decarboxylase (AADC), sepiapterin reductase (SR), tyrosine hydroxylase (TH), and guanosine-triphosphate cyclohydrolase type I (GTPCH-I). Among these disorders, AADC is the most frequent. AADC is an autosomal recessive, neurometabolic disorder caused by disease-causing variants in the dihydroxyphenylalanine decarboxylase. OGC is a frequent event in these cases. AADC diagnosis is confirmed by cerebrospinal fluid analysis, neurotransmitter testing, plasma AADC activity testing and genetic testing[5,26].

Kufor-Rakeb (PARK9) is another rare cause of OGC caused by mutations in the ATP13A2 gene, which encodes a lysosomal protein that may play a role in the breakdown of alpha-synuclein leading to neurodegeneration of dopaminergic neurons. PARK9 typically presents with parkinsonism, cognitive decline, spasticity, and eye movement abnormalities including supranuclear gaze palsy and rarely OGC[27].

Other causal factors such as structural focal lesions in the midbrain and basal ganglia are conditions that cause OGC by anatomical disruption of the nigrostriatal pathway. In these cases, brain magnetic resonance images can help in differential diagnosis[18].

Pathophysiology

The exact mechanism responsible for OGC is not completely understood. Pathophysiology of OGC may involve an imbalance between dopaminergic and cholinergic neurotransmission in the nigrostriatal pathway. Hyper or hypodopaminergic states have been suggested[4,16,28]. In addition, chronic use of ADRs could lead to compensatory upregulation and/or hypersensitivity of postsynaptic dopamine (particularly D2) receptors. Furthermore, chronic use of ADRs could cause increased dopamine turnover and oxygen free radicals production with toxic effects in nigrostriatal pathways. This mechanism could explain why OGC could continue after stopping the drug[19,25].

Gamma-aminobutyric acid (GABA) interneuron abnormalities, leading to hypersensitivity of striatal dopamine receptors can be responsible for the condition[4]. Abnormalities of gabaergic activity in basal ganglia (medium spinal neurons) and mainly in nigrostriatal pathways can be related to the occurrence of OGC. This mechanism could explain the efficacy of benzodiazepines in OGC[25].

Risk Factors

Male sex has a higher incidence of OGC compared to women by probable hormonal influence. Estrogen may act as a protective factor[25]. Contradictorily to this theory, most cases of OGC from antiemetics with dopamine antagonism occurred in the female sex[13].

Young individuals have higher dopaminergic status compared to the elderly. ADRs decrease dopaminergic status in higher intensity in young age compared to old. This hypodopaminergic status of acute installation can increase the risks of OGC. Elderly individuals present a progressive degeneration of dopaminergic pathways, decreasing the chance of sudden abnormalities in dopaminergic status and occurrence of OGC[13].

Other risk factors are severe illness as dehydration, hypocalcemia, high antipsychotic dose, parenteral administration of antipsychotic, high potency of antipsychotic drugs with elevated D2 receptor antagonism, abrupt discontinuation of anticholinergic medication, and family or personal history of dystonia[18].

Differential Diagnosis

Differential Diagnosis from OGC involves several diseases. Among these, epileptic seizures and encephalitis must be timely investigated. In OGC, there is no loss of consciousness, in opposition to epileptic seizures or encephalitis, and often these conditions have abnormalities in electroencephalogram or brain magnetic resonance images. Considering epilepsy subtypes, Frontal lobe epilepsy is frequently confused with OGC. Frontal lobe epilepsy is characterized by versive seizures with upward movements and lateral forced head[12,21]. Encephalitis, mainly anti-NMDAR, can occur with upward eye movements or be associated with true OGC. Loss of consciousness and

electroencephalogram abnormalities are essential to differential diagnosis[27].

In terms of frequency, dyskinesias caused by levodopa therapy in patients with parkinsonism, mainly Parkinson's disease, tics, psychogenic, and paroxysmal tonic upgaze are differential of OGC[14]. Differential diagnosis between tics and OGC can be challenging. Eye movement tics in children are seen between the ages of 6 and 12 years and are characterized by a stereotyped conjugate deviation of the eyes upward and outward[21].

Antipsychotics are used for treatments of hyperkinetic movements such as chorea and tics. Treatment of tics with DRAs can cause OGC. However, individuals with tics can voluntarily suppress the movement temporarily with internal discomfort (premonitory sensation), and increase tics after suppression, unlike OGC. In OGC, the movement is more sustained and can be associated with pain. Tics have a short duration and there is usually no pain[29].

In parkinsonism, mainly Parkinson's disease, OGC occurs as a manifestation of motor complications of levodopa treatment. In this situation, there are related cases of OGC in dyskinesia of peak-dose or in the wearing-off phenomenon[28,30]. Furthermore, the ocular movement can represent a dyskinesia of the choreiform subtype, characterized by short-lasting horizontal or upward conjugate gaze deviation of extra-ocular muscles. In levodopa-induced ocular dyskinesia, there is no pain or autonomic symptoms and the movement may be suppressed

by visual fixation, in opposition to OGC[30].

Paroxysmal tonic upgaze is characterized by infantile or early childhood onset, with episodes of sustained conjugate upward deviation of the eyes. In addition, neck flexion and concomitant episodic ataxia are present. It is a benign movement ocular with a good prognosis and disappears at 1 to 48 months after onset[21,27].

Ocular movements in comatose patients such as bobbing or dipping can be confused with OGC. Although, also in these conditions the level of consciousness is not preserved. Psychogenic or functional disorders with ocular movements can be differentiated from OGC by distractible ocular movements and association with functional movements in other body regions[27].

Treatment

The mainstay of treatment in acute drug-induced OGC is anticholinergic medications. Parenteral anticholinergics are rapidly effecting agents that are frequently used in the treatment of OGC. Benzatropine or biperiden are the anticholinergics of choice. The relief occurs often in 15 to 30 minutes. A new dose can be administered in cases of lack of efficacy. Anticholinergics must be maintained orally for 5 to 7 days to prevent recurrence after the success of the initial treatment[4]. Intravenous antihistaminics such as diphenhydramine are alternatives. Benzodiazepines, including clonazepam and diazepam, may also be useful in OGC caused by antipsychotics[10]. However, benzodiazepines

increase de risk of breath disturbs in cases of segmental OGC associated with laryngeal dystonia. OGCs may be recurrent in decrease or re-exposure to the drug. Thus, complete withdrawal is recommended[31]. Oculogyric crises usually disappear within 24 to 48 hours upon withdrawal or reduction of the antipsychotic/antidopaminergic medication[8,31]. However, in psychiatric illness, when suspension of treatment is impossible, a decrease in the drug or substitution of antipsychotics must be tried. Clozapine is the drug most commonly used and also causes OGC in some individuals, although at low frequency[15]. Furthermore, as a tardive syndrome, OGC can also improve with an increase in DRAs doses[19]. We have not found the use of inhibitors of vesicular monoamine transporters (VMAT-2) tetrabenazine, deutetrabenazine, and valbenazine in OGC derived from tardive dystonia syndrome. VMAT-2 inhibitors cause a high rate of negative effects on mood, with an increase in depression and suicidality, with elevated risks of administration in a non-life-threatening condition such as OGC. In opposition, cases reported of OGC as a manifestation of acute dystonia from the use of VMAT-2 inhibitors to hyperkinetic movements are most commonly related[7,29].

Electro-convulsive therapy (ECT) is a non-pharmacologic treatment for OGC when it is a manifestation of tardive dystonic syndrome of chronic exposition to ADRs. ECT is an alternative therapy when the patient does not improve with anticholinergics, benzodiazepines, or changes

to ADRs with low risks of OGC and treatment cannot be stopped[32].

Treatment of OGC related to peak-dose or wearing-off periods in Parkinson's disease is an adjustment in dose administration of levodopa. Levodopa adjustment varies from reducing the daily dose of the drug to partitioning the dose into multiple administrations[28]. In opposition, levodopa does not improve or deteriorate ocular dyskinesia by levodopa[30]. The responsiveness to levodopa therapy is one of the criteria for differentiating some metabolic encephalopathies that cause OGC and are related to dopamine biosynthesis. For instance, sepiapterin deficiency and AADC deficiency. The OGC improves with levodopa in the former, whereas it will not be effective in the latter[5].

Ultimately, in cases of OGC as a manifestation of tardive dystonia, in opposition to other tardive focal/segmental dystonias, there are no reports of therapies with botulinum toxin or surgery, not only considering the risks and costs of these procedures but also the short duration of the episodes[20].

CONCLUSIONS

In summary, OGC is an underestimated diagnosis and continues to occur despite the development of new antipsychotic drugs. It can occur as a manifestation of an acute or tardive dystonic syndrome. Furthermore, OGC also occurs as a side effect of other classes of drugs common in generalist clinics, such as antiemetics, anticonvulsants, or

antidepressants. The diagnosis of this condition can be a challenge, as it involves the differentiation of life-threatening conditions such as epileptic seizures, encephalitis, or other conditions. OGC diagnosis can be difficult in patients with psychiatric illnesses. The correct diagnosis is necessary for timely and proper treatment with anticholinergics or benzodiazepines in acute cases or suspension or change of dopamine receptor antagonists in chronic cases.

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