Horner Syndrome as a Rare Complication Following Parathyroidectomy: A Case Report

Caso Clínico: Síndrome de Horner como uma Complicação Rara após Paratiroidectomia

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ABSTRACT

Horner syndrome arises from disruption of the oculosympathetic pathway, presenting with ptosis, miosis, and hemifacial anhidrosis. It is a rare complication following parathyroid surgery.

A 64-year-old female developed right upper eyelid ptosis 24 hours after a right superior parathyroidectomy a year earlier. Histopathology confirmed a parathyroid adenoma. Ophthalmological examination revealed right-sided miosis, with delayed pupillary dilation under scotopic conditions. No hemifacial anhidrosis was noted. Diagnosis was confirmed through apraclonidine test. Surgery for reinsertion of the right upper eyelid retractors resolved the ptosis. The anisocoria persisted.

This case underscores the importance of recognizing Horner syndrome as a rare complication following parathyroid surgery. General surgeons should be aware of the oculosympathetic pathway's vulnerability. A comprehensive clinical history and ophthalmological examination are crucial for identifying potentially serious neurological conditions.

KEYWORDS: Horner Syndrome/etiology; Parathyroidectomy/adverse effects; Postoperative Complications.

RESUMO

A síndrome de Horner resulta da disrupção da via oculossimpática, manifestando-se com miose, ptose e anidrose. É uma complicação rara após cirurgia de paratiroide.

Uma mulher de 64 anos desenvolveu ptose da pálpebra superior direita 24 horas após uma paratiroidectomia superior direita (um ano antes). A histopatologia confirmou um adenoma da paratiroide. O exame oftalmológico revelou miose direita, com atraso da dilatação em condições escotópicas. Sem anidrose hemifacial. O teste da aproclonidina confirmou o diagnóstico. A cirurgia de reinserção dos retratores da pálpebra superior resolveu a ptose. A anisocoria persistiu.

É importante reconhecer a síndrome de Horner como uma complicação rara após cirurgia de paratiroide. Cirurgiões gerais devem estar cientes da vulnerabilidade da via oculossimpática. A história clínica e exame oftalmológicos completos são essenciais para identificar condições neurológicas potencialmente graves.

PALAVRAS-CHAVE: Complicações Pós-Operatórias; Paratiroidectomia/efeitos adversos; Síndrome de Horner/etiologia.

INTRODUCTION

Horner syndrome (HS) arises from disruption of the oculosympathetic pathway, classically manifesting with ipsilesional miosis, ptosis and facial anhidrosis. These can affect esthetics and quality of life. Miosis results from the disruption of the innervation to the pupillary dilator muscle with unantagonized action of the iris sphincter, resulting in anisocoria due to delayed pupillary dilation under scotopic conditions (dilation lag[1]). Ptosis occurs due to paresis of the Muller muscle in superior and inferior eyelids (reverse ptosis[2]), forming a narrower palpebral fissure (pseudo-enophtalmos). The distribution of the ipsilesional facial anhidrosis depends on the affected neuron. Other manifestations include iris heterochromia in congenital HS or long-standing cases.[3-6] The clinical signs may be subtle and often unnoticed.

Oculosympathetic pathway consists of three neurons, establishing important anatomical relationships during their course to the eye. The first neuron originates in the hypothalamus, descends through the brainstem and spinal cord, and emerges at the levels of C8 to T2. The second neuron loops near the pulmonary apex and ascends within the superior cervical chain to synapse at the superior cervical ganglion, where the third neuron emerges between the jugular vein and the common carotid artery. The fibers responsible for regulating ipsilateral hemifacial sweating accompany the external carotid artery, while the remaining fibers travel along the intern carotid artery to the cavernous sinus. At this level, they are in proximity to the ipsilateral abducens nerve, after which the oculosympathetic fibers follow the first division of the trigeminal nerve, without synapsing at the ciliary ganglion. Due to its long, circuitous route, various factors contribute to its damage and the subsequent development of HS.[4-7] Classically, etiologies are divided into congenital causes, such as congenital anomalies or birth trauma, and acquired causes, including iatrogenic injury after thoracic and neck surgeries, tumors, trauma, and vascular conditions. It may be the first manifestation of a life-threatening condition, such as internal carotid artery dissection (classically presenting with pain), pulmonary apex neoplasia, or neuroblastoma (pediatric patients).[8] In a patient presenting with ptosis, the ophthalmologist must perform a meticulous examination, including detailed evaluation of pupillary function and ocular motility.

The presented case refers to a HS after a parathyroidectomy for the treatment of a parathyroid adenoma. The relevance of this article stems from the limited number of reported cases of HS following parathyroid surgery, despite this being a common procedure. Awareness of rare complications is the first step towards their prevention and early detection.

CASE REPORT

We present the case of a 64-year-old female with no relevant medical history and a surgical history including a conventional total thyroidectomy 15 years earlier for papillary thyroid carcinoma and a right superior open parathyroidectomy 1 year earlier for parathyroid adenoma leading to primary hyperparathyroidism. Both surgical interventions were executed without immediate complications.

The patient was referred by her family physician for the evaluation of right upper eyelid ptosis. During the clinical history assessment, the patient reported that the symptomatology started during the first 24 hours after parathyroidectomy. Nonetheless, there was no related information on the medical records of the general surgery and endocrinology consultations.

Ophthalmological examination confirmed the right upper eyelid ptosis. Additionally, it showed right-sided miosis, with delayed pupillary dilation under scotopic conditions. No associated objective or subjective hemifacial anhidrosis was noted. The remainder of the ophthalmological examination was unremarkable: no ocular motility deficits, best-corrected visual acuity was 20/20, and slit-lamp anterior segment observation and fundoscopy showed no abnormalities. Neurological examination was unremarkable. The diagnosis was confirmed by pharmacological testing with apraclonidine. Before instillation, the right and left pupil diameters were 2.7 and 3.8 mm, respectively. After instillation, the diameters were 7.0 and 4.3 mm, respectively (Figs. 1a and 1b).

The patient underwent surgery for reinsertion of the right upper eyelid retractors (via posterior approach), which was completed uneventfully. This resolved the ptosis and led to substantial improvement in both aesthetic appearance and eyelid function (Figs. 2a and 2b). The anisocoria persisted throughout the follow-up.

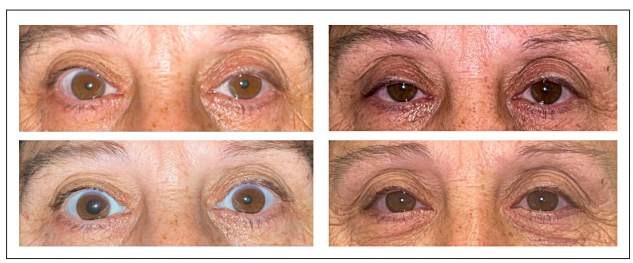

Figure 1. a) anisocoria before apraclonidine instillation b) reverse anisocoria after apraclonidine isntillation.

Figure 2. a) ptosis before and b) after surgery with reinsertion of superior eyelid retractors.

DISCUSSION

The disruption of the oculosympathetic pathway in HS typically presents with ipsilateral miosis, ptosis, and facial anhidrosis. In this case, although two of the three classic signs were present, the diagnosis was delayed by one year. Increased clinical vigilance and awareness of this complication could potentially have led to an earlier diagnosis.

This condition represents a rare post-thyroidectomy complication, ranging from 0.03% to 5% (mostly 0.2%) in a recent narrative review.[9] It has been observed following

open or endoscopic surgeries and other minimally invasive techniques, for treating both benign and malignant conditions.[7,9] The scientific evidence is limited regarding the risk factors for this complication. Additionally, to our knowledge, little is known about the prevalence of HS following isolated parathyroid surgery.[10-12] The retrospective study conducted at a center for endocrine surgery by Harding et al reported only one case among 410 patients who underwent isolated parathyroidectomy, which occurred following an uncomplicated minimally invasive procedure for parathyroid adenoma.[10] In a case series by Allen and Meyer, HS was described in six patients after neck surgery, including one with a history of parathyroid surgery and cervical catheter placement performed elsewhere; however, the precise timing of symptom onset was unclear, making causality difficult to determine.[11] McCrory *et al* described direct injury to the sympathetic chain during parathyroid exploration, where a cervical ganglioneuroma was mistakenly excised as a presumed parathyroid adenoma.[12]

Several mechanisms have been proposed to explain damage to the sympathetic chain during neck procedures,[7,10,13-15] including intra-operative cervical sympathetic chain complete or incomplete section, ischemic lesions, stretching by retractors, damage induced by energy instruments and post-operative compression by hematomas or seromas. The prognosis is directly related with the cause of the damage. In the reported case, neither the surgical report nor the postoperative consultation records contain sufficient detail to allow for a definitive inference regarding the mechanism of injury in the patient described.

Although the diagnosis was strongly suggested by the clinical presentation, pharmacological testing remains the gold standard for confirmation. Currently, the most commonly used test, which we performed in our patient, is the apraclonidine (0.5% or 1%) test, an α2-adrenergic agonist and weak α1-adrenergic agonist that has little effect on pupil size in normal eyes. In HS the denervation of the pupillary dilator muscle leads to supersensitivity of the postsynaptic α1-receptors. Therefore, instilling apraclonidine will reverse anisocoria, as the affected pupil will respond and dilate while the normal one will not.[16] The cocaine (4% or 10%) test is the classical test; however, it is now less used because of the difficulties in obtaining the substance. The hydroxyamphetamine test, which has many false negatives and is not widely available, is rarely used in clinical practice for localizing the damage.[5,6,17,18] Given the direct cause-effect relationship and the prolonged clinical course, no imaging studies were performed. Almog *et al*[19] showed that initial neuro-ophthalmological evaluation was sufficient to establish etiology in most HS cases. Nonetheless, it is imperative to always consider the use of targeted imaging studies to exclude serious etiologies whenever there is any clinical suspicion, or when the underlying cause remains unclear.[4,19,20]

Regarding treatment, some specific etiologies benefit from early surgical intervention, such as liquid collections drainage. However, literature shows that there is a great potential for at least partial recovery[7,9] during the first months to 1 year after surgery with conservative measures. The reported patient presented with ptosis 1 year after surgery, making spontaneous resolution less likely. Therefore, we proposed a surgical treatment with reinsertion of the right upper eyelid retractors (via posterior approach). The procedure was uneventful, resolving the ptosis and resulting in a good esthetic and functional outcome. The patient was satisfied with the surgical outcome. Miosis persisted during the follow-up.

CONCLUSION

This case underscores the importance of recognizing HS as a rare but noteworthy complication that requires a high index of suspicion after parathyroid surgery. It can have a major impact in esthetics and quality of life.

General surgeons should be aware of the vulnerability of the oculosympathetic pathway due to its close and highly variable anatomical relationship with these structures, with standardized and appropriate surgical guidelines that aim its preservation. Close post-operative follow-up is also essential.

Ophthalmologists must be proficient in identifying HS as a potential consequence of cervical surgeries, highlighting the importance of obtaining a detailed clinical history for accurate diagnosis and treatment.

When assessing a patient with ptosis, the ophthalmologist should conduct a thorough examination, including evaluation of pupillary function and extraocular motility. Without this, the diagnosis of serious and life-threatening neurological conditions, such as HS, may be missed.

CONTRIBUTORSHIP STATEMENT / DECLARAÇÃO DE CONTRIBUIÇÃO

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MVP and CA: Writing of the manuscript and final revision.

All the authors approved the final version to be published.

FM: Revisão bibliográfica, redação do manuscrito e revisão final.

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Todos os autores aprovaram a versão final a ser publicada.

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