Case Report

Horner Syndrome Following Thyroid Surgery: The Clinical and Pharmacological Presentations

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Abstract

Purpose: To report the clinical and pharmacological findings of a patient with iatrogenic Horner syndrome (HS) which occurred after thyroid surgery.

Case Report: A 29-year-old man was referred to our emergency ward due to anisocoria and unilateral eyelid ptosis reported by the patient immediately after a recent thyroidectomy for a papillary carcinoma. Ophthalmologic examination revealed 3 mm ptosis of the right eyelid. In dim illumination, the right and left pupil size was measured 3 and 6 mm, respectively. In bright illumination, the amount of anisocoria decreased; the near pupillary reaction was intact. Brain and neck magnetic resonance imaging and chest radiography were normal. Pharmacological tests with 10% cocaine, 1% hydroxyamphetamine and 1% phenylephrine localized the interruption of the oculosympathetic pathway with postganglionic third-order neuron involvement. After 6 months of follow-up, no sign of recovery was recorded.

Conclusion: Despite HS could appear to be a rare complication of thyroid surgery, it is of importance for the neck surgeons to be aware that oculosympathetic pathway (OSP) is a potentially vulnerable structure with close anatomical relationship with the thyroid gland, and for the ophthalmologists that HS may occur secondary to neck surgery and taking an accurate history is mandatory.

Keywords: Horner syndrome; Miosis; Ptosis; Thyroid Surgery

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INTRODUCTION

Johann Friedrich Horner first described the clinical presentation of ptosis, miosis, and anhydrosis caused by interruption of the oculosympathetic pathway (OSP).[1] OSP supplies innervation to the ipsilateral face sweat glands, dilator muscles of the pupil and retractor muscles of the eyelids. It consists of three neurons and two relay centers, beginning in the central nervous system and concluding at the eye. OSP lesion can be diagnosed and localized clinically through the combination of history, clinical examination, and pharmacological testing. In the cases of malignancy, imaging of the entire sympathetic pathway is warranted.

Horner syndrome (HS) is a rare complication of thyroid surgery with only few cases reported; clinical characteristics and OSP lesion localization have not been described in detail.

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CASE REPORT

A 29-year-old male was referred to our emergency ward due to new-onset anisocoria and unilateral ptosis. One week before, he had undergone a total thyroidectomy under general anesthesia for a thyroid nodule (papillary carcinoma TIR 5; based on five-tiered system of classification of thyroid lesions) detected in the right lobe of his thyroid gland. The patient reported the onset of ocular signs and symptoms immediately after waking up from anesthesia. Ophthalmologic examination revealed a 3 mm right upper eyelid ptosis without anhydrosis and any evidences of vascular dilatation of the face. In dim illumination, the right and left pupils measured 3 and 6 mm, respectively [Figure 1]. In bright illumination, the amount of anisocoria decreased [Figure 2]. The near pupillary reaction was intact. A complete neurological examination confirmed otherwise normal cranial nerves. Brain and neck magnetic resonance imaging and chest radiography were unremarkable. In order to confirm our hypothesis of iatrogenic HS and to anatomically localize the OSP lesion, we performed all pharmacological tests on separate days. Pathologic 10% cocaine test with no pupil dilation of the affected eye after instillation confirmed the HS diagnosis. Persistent right eye miosis after 1% hydroxyamphetamine solution instillation revealed a postganglionic third-order neuron (TON) involvement. TON lesion localization was also confirmed by pupil dilation and temporary regression of ptosis in the affected eye when 1% phenylephrine was placed in the conjunctival sac [Figure 3]. Currently, the patient has been followed-up for 6 months without any sign of recovery.

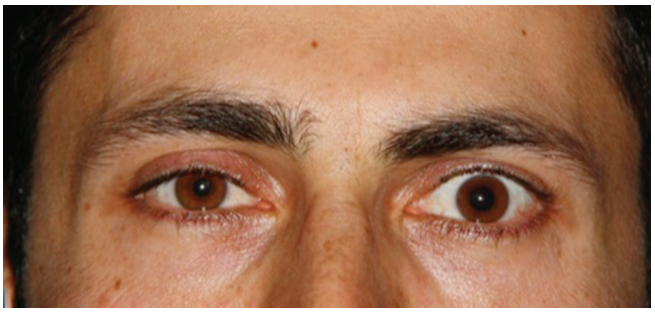

Figure 1. Right eye miosis, blepharoptosis and lower eyelid margin "inverse ptosis".

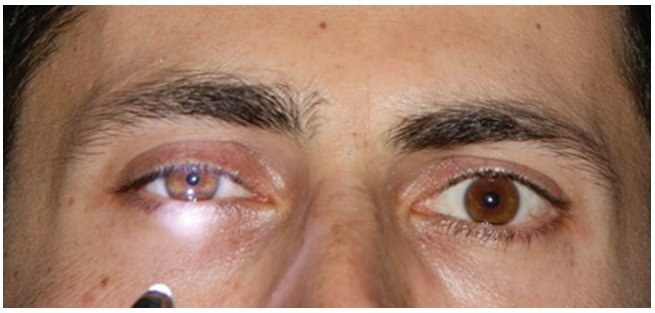

Figure 2. Decreased anisocoria in bright illumination.

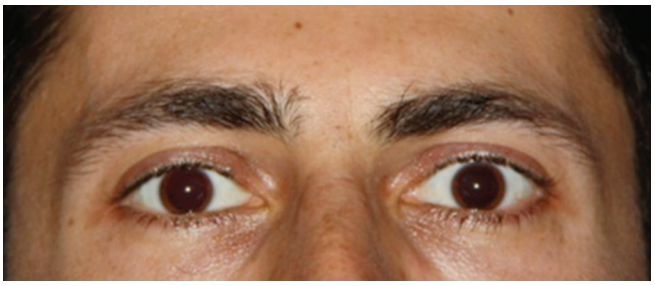

Figure 3. Pupil dilatation and temporary regression of the ptosis following instillation of 1% phenylephrine eye drop.

DISCUSSION

Horner syndrome can be classified as preganglionic or postganglionic based on the location of the OSP lesion with reference to the superior cervical ganglion; the preganglionic segments include both the first-order and second-order neurons while the TON is postganglionic. Pharmacological localization is challenging because of poor availability of the reagents. Instillation of one drop of 10% cocaine solution is used for pharmacologic diagnosis: cocaine blocks the re-uptake of norepinephrine released at the neuromuscular junctions of the iris dilator muscle, thereby increases the amount of norepinephrine available to stimulate the muscle. Following instillation in a normal eye, the pupil will dilate but in HS, the pupil will dilate poorly because a little or no norepinephrine will be released into the synaptic cleft. Instillation of 1% hydroxyamphetamine solution can be used for differential diagnosis of central and preganglionic from postganglionic lesions; it will release norepinephrine from the sympathetic synaptic terminal, which will dilate pupil in HS only if the postganglionic neuron is intact. An alternative drug for localizing the site of the lesion is 1% phenylephrine. Due to the principle of denervation supersensitivity, in the HS produced by a lesion interrupting the postganglionic fibers, the pupil dilates and the ptosis temporarily regresses when 1% phenylephrine is placed in the conjunctival sac of the affected eye. The pupil of a patient with central (first order) HS should not dilate while in a preganglionic HS (second order) the pupil may dilate minimally.[2,3]

HS following conventional thyroidectomy is a very rare complication (0.2–0.27% of cases).[4] Recent reports described this complication both after the video-assisted minimally invasive and the robotic thyroid surgery.[5,6] It is possible to postulate several mechanisms whereby the sympathetic chain may be injured during the course of surgical intervention including postoperative hematoma, ischemia-induced neural damage, stretching of the cervical sympathetic chain, damage to the communication between the cervical sympathetic chain and the recurrent

laryngeal nerve during its identification and the potential of anatomic variation in which the recurrent laryngeal nerve give off a communicating branch to the cervical sympathetic chain.[7] Approximately 70% of patients who suffer from HS following thyroidectomy, experience a permanent damage or an incomplete recovery; while, the remaining recover completely after a variable period ranging from 5 days to 15 months after surgery. Prognosis depends on the mechanism of injury; there is often a spontaneous recovery in the cases of indirect injury of the sympathetic plexus. In other subjects, the symptoms persist over time.[8]

We found only 29 cases of HS after thyroidectomy reported in the literature.[8-13] Although the clinical characteristics and OSP lesion localization were not described in detail in the reported cases, there are two interesting features in almost all cases. The first is the lack of anhydrosis and vascular symptoms of the face on the side of the lesion as observed in our case, and the second is the recovery of the eyelid ptosis coupled with a persistent miosis. To the best of our knowledge, our case report is the first to describe in detail not only the clinical characteristics but also pharmacologic OSP lesion localization of the iatrogenic HS.

Although OSP surgical damage could appear as a rare complication, it is quite important for thyroid surgeons to be aware that OSP is a potentially at-risk structure due to its close anatomical relationship with the thyroid gland, and for the ophthalmologists that HS can occur secondary to thyroid surgery and taking the accurate patient history is mandatory. The current patient was referred to our emergency ward by another ophthalmologist for a radiological evaluation of anisocoria and ptosis with no suspicion of iatrogenic HS. Imaging is not mandatory in all cases of HS. In our case, patient's young age, history of malignancy and neurological consultation suggested us to perform imaging of the entire sympathetic chain in order to exclude infarction of the cerebellar or vertebral artery, brain metastasis or compressing hematoma. Our report shows that a careful surgical technique can reduce the risk of HS, and a combination of history, clinical examination, and pharmacological testing would be beneficial for early diagnosis.

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Nil.

Conflicts of Interest

There are no conflicts of interest.

REFERENCES

1. Horner JF. On a form of ptosis. *Klin Monbl Augenheilkd* 1869;7:193-198.
2. Reede DL, Garcon E, Smoker WRK, Kardon R. Horner's Syndrome: Clinical and Radiographic evaluation. *Neuroimag Clin N Am* 2008;18:369-385.
3. Walton KA and Buono LM. Horner syndrome. *Curr Opin Ophthalmol* 2003;14:357-363.
4. Cozzaglio L, Coladonato M, Doci R, Travaglini P, Vizzotto L, Osio M, et al. Horner's syndrome as a complication of thyroidectomy: Report of a case. *Surg Today* 2008;38:1114-1116.
5. Harding JL, Sywak MS, Sidhu S, Delbridge LW. Horner's syndrome in association with thyroid and parathyroid disease. *ANZ J Surg* 2004;74:442-445.
6. Kang SW, Park JH, Jeong JS, Lee CR, Park S, Lee SH, et al. Prospects of robotic thyroidectomy using a gasless, transaxillary approach for the management of thyroid carcinoma. *Surg Laparosc Endosc Percutan Tech* 2011;21:223-229.
7. Harding JL, Sywak MS, Sidhu S, Delbridge LW. Horner's syndrome in association with thyroid and parathyroid disease. *ANZ J Surg* 2004;74:442-445.
8. González-Aguado R, Morales-Angulo C, Obeso-Agüera S, Longarela-Herrero Y, García-Zornoza R, Acle Cervera L. Horner's syndrome after neck surgery. *Acta Otorrinolaringol Esp* 2012;63:299-302.
9. Lange MJ. Morbidity and mortality in thyroidectomy. *Proc R Soc Med* 1961;54:871-873.
10. Beahrs OH, Vandertoll DJ. Complications of secondary thyroidectomy. *Surg Gynecol Obstet* 1963;117:535-539.
11. Smith I, Murley RS. Damage to the cervical sympathetic system during operations on the thyroid gland. *Br J Surg* 1965;52:673-675.
12. Solomon P, Irish J, Gullane P. Horner's syndrome following a thyroidectomy. *J Otolaryngol* 993;22:454-456.
13. Italiano D, Cammaroto S, Cedro C, Bramanti P, Ferlazzo E. Horner syndrome following thyroidectomy. *Neurol Sci* 2011;32:531.