Short communication

Irreversible Horner's syndrome after bilateral thoracoscopic sympathectomy

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**ABSTRACT**

Case report: A 19-year-old boy who developed a right Horner's syndrome after a bilateral sympathectomy as a treatment for palmoplantar hyperhidrosis.

Discussion: Horner's syndrome is defined by the occurrence of miosis, ptosis and enophthalmos as a result of involvement of sympathetic innervation. This is quite rare, but identification is very important because it may also be an ominous sign secondary to a neoplasm, neurological diseases, or surgery of the sympathetic chain, as in our case.

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**SÍNDROME DE HORNER IRREVERSIBLE TRAS SIMPATECTOMÍA TORACOSCÓPICA BILATERAL**

**RESUMEN**

Caso clínico: Paciente varón de 19 años que presenta síndrome de Horner de lado derecho tras simpatectomía bilateral como tratamiento a una hiperhidrosis palmoplantar.

Discusión: El síndrome de Horner es definido por la aparición de miosis, ptosis y enoftalmos como consecuencia de la afectación de la inervación simpática. Es una afectación bastante infrecuente, sin embargo es muy importante su identificación, pues puede corresponder a un signo ominoso secundario a neoplasias, enfermedades neurológicas o a cirugía de la cadena simpática, como es nuestro caso.

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Introduction

Palmar and plantar hyperhidrosis consists in excessive sweating in said areas due to sympathetic nervous system hyperfunction.\textsuperscript{1,2} One option for treatment is the elimination of said hyper-function by means of sympathectomy or sympathectolysis through thoracoscopic pathway.\textsuperscript{3} The possible complications of this technique include pneumothorax, hemothorax and other less frequent complications such as Horner’s syndrome which is usually temporary due to nervous irritation, with permanent injury being exceptional.\textsuperscript{1,3}

Horner’s syndrome can be classified by the level at which the sympathetic pathway is interrupted: it could affect first order neurons, as in verteobasilar insufficiency or tumor. It could also affect second order neurons such as in pulmonary carcinoma or thyroid adenoma among others. In addition, it could give rise to a third order neuron disorder, internal carotid dissection, or Tolosa–Hunt syndrome, among others.\textsuperscript{3}

The case presented herein is that of a patient operated on for palmar and plantar hyperhidrosis who exhibited irreversible Horner’s syndrome as a sequel after 2 years’ follow-up without remission.

Clinical case

The case is of a male patient, aged 19 years, without relevant history, who underwent bilateral thoracoscopic sympathectomy for treating armpit and palm-sole hyperhidrosis. During surgery the sympathetic chain ganglions were ablated with electrocoagulation at the level of T2, T3 and T4.

The first day after surgery the patient was referred to the Ophthalmology Service after observing anisocoria.

Upon ophthalmological examination, the patient exhibited the following:

- Slight right eye palpebral ptosis which did not cover the pupil axis and slight elevation of the lower eyelid, or “reverse ptosis” (Figs. 1 and 2).
- Miosis in the right eye, increasing anisocoria under soft lighting, small pupil with poor dilatation (Figs. 1 and 2).

Discussion

The first description of the Claude Bernard Horner syndrome in humans was published in 1869 by Johann Friedrich Horner, a Swiss ophthalmologist. The syndrome is due to the interruption of the sympathetic innervation of the eye and ocular annexes. It is caused by a range of etiologies including cere-
brovascular accidents in the hypothalamus, in the encephalic trunk (Wallenberg syndrome), carotid artery lesions, tumors in the lung upper lobe or pleura and cluster cephalgia. It can express as a rare complication of thoracoscopic surgery with incidence ranging between 1% and 5% according to different series. In these cases, the lesion is generally produced in the pre-ganglonary sympathetic fibers close to the stellate ganglion.\(^2\)\(^4\) It exhibits a pathognomonic clinical triad with miosis and ptosis in the ipsilateral affected eye, and anhidrosis of said hemi-face as a consequence of oculosympathetic chain palsy.\(^5\) Other specific findings could be present such as delayed midriasis or enophthalmos in the affected eye, or iris heterochromia in congenital cases.

Pharmacological tests for diagnosis are as follows:

- 10% cocaine test to dilate pupils inducing sympathetic stimulation and inhibiting recapture of noradrenaline in the neuromuscular union. However, a pupil with sympathetic palsy does not dilate and produces a pupil size difference of 1 mm. One drop should be administered in each eye, repeating 1 min. later and examined after 15 min. This test confirms the oculosympathetic injury but does not facilitate its location.

- 1% hydroxyamphetamine test to release noradrenaline in the neuromuscular union, provoking dilatation if the third neuron is intact. This test enables discrimination between pre- and post-ganglionic damage. It must be carried out 48 h after the cocaine test to avoid the additive effect. One drop should be administered in each eye, repeating 1 min. later and examining the pupils 30 min. later.

In cases of post-surgery complications, this syndrome generally resolves spontaneously but in this case the damage is probably irreversible due to the persistence of the condition after 2 years of follow-up.\(^2\)\(^3\)\(^5\)

Management approach for these patients is usually conservative with expectant attitude, although in selected cases surgical treatment could be considered, for example to reinsert the aponeurosis of the elevator.

A diagnosis of Horner’s syndrome must always be considered in cases involving trauma, surgery, infections or neoplasia. The clinical history is the cornerstone for diagnosis.

**Conflict of interest**

No conflict of interests has been declared by the authors.

**References**