HORNER'S SYNDROME AFTER TONSILLECTOMY
SÍNDROME DE HORNER TRAS AMIGDALECTOMÍA
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ABSTRACT

Case report: A 5-year-old girl underwent a tonsillectomy and developed a right Horner’s syndrome after the procedure: we present the clinical findings and the investigations performed.

Discussion: Horner’s syndrome is caused by a decrease in sympathetic innervation to the eye, and the defining signs are ptosis and miosis. The reported incidence of Horner’s syndrome following tonsillectomy is low, although it is one of the commonest operative procedures performed (1,2) (Arch Soc Esp Oftalmol 2008; 83: 129-132).

Key words: Pupil, anisocoria, Horner’s syndrome, ptosis, oculosympathetic paralysis.

INTRODUCTION

Horner’s syndrome was described by Johann Friedrich Horner in 1869. It is also called Bernard Horner syndrome or oculosympathetic paralysis. It is due to an injury in one of the cervical sympathetic chains, affecting the same side of the injury. It is characterized by the pathognomonic triad of ptosis, miosis and enophthalmos, although it can also include hemifacial dishydrosis and loss of the cilium-spinal reflex. This paper presents a Horner syndrome case after tonsil extraction of a 5 year-old girl.

CASE REPORT

A five year-old girl with a history of multiple episodes of amigdallitis and mid-otitis. In another hospital she had an amigdallectomy and adenoidectomy under general anesthesia as per usual practice. The operation was free of complications and without infiltration of post-op local anesthesia.

The immediate postop developed normally but 3 days later the patient exhibited fever and general deterioration. The parents observed that the left eye pupil was larger than the right one. The patient was admitted in the pediatric service of our hospital and
referred to the ophthalmology service where the Horner syndrome and mediastinitis diagnostic was reached.

The ophthalmological exploration revealed (fig. 1):
— Right eye miosis under natural light, with increased anisochoria in darkness. The dilatation range and speed of right pupil was lower than that of the left eye.
— Slight ptosis in right eye with discrete elevation of the lower eyelid.
— The rest of the ophthalmological examination was normal: no iris heterochromy or enophthalmos, normal ocular movements and the patient remained in orthotropy.

Cocaine eye drops at 10% were instilled in both eyes. After 15 minutes the right pupil failed to react while the unaffected left one dilated. This confirmed the Horner syndrome diagnostic (fig. 2).

After 6 months of evolution, ptosis receded and only a small anisochoria remained (fig. 3).

**DISCUSSION**

The oculosympathetic chain comprises 3 neurons (3):
— First order neuron axons which, starting at the hypothalamus, go through the brainstem to make synapse in Budge’s ciliium-spinal centre, at the level of C8-T2.
— Second order neuron axons which ascend to the apex pulmonis, go round the subclavian artery and make synapsis in the superior cervical ganglion at the level of the jaw angle, 1.5 cm behind the palatine amigdalla.

— From the third order neuron (postganglionic) the sudomotor fibres of the face follow the external carotide and the pupillary fibres go intracranial again to follow the internal carotide to the cavernous sinus, where they incorporate into the ophthalmic branch of the trigeminus up to the pupil dilating muscles, upper eyelid Müller’s muscle and lower eyelid retractor muscles.
— Horner syndrome is a consequence of an oculosympathetic paralysis and is characterized by:
— Slight ptosis, with elevation of the lower eyelid and apparent enophthalmos.
— Miosis (with normal pupil reactions) which produces a slight anisochoria (1 mm or smaller) and a characteristic delay in dilatation in darkness. Ani-
Sohoría is larger in darkness and for 5 seconds, becoming reduced after 15-20 seconds.
— Anhydrosis according to the location of the injury (barely evident in post-ganglinary forms).
— Iris heterochromia in congenital cases, with the involved iris being lighter.

Pharmacological tests:
Cocaine 2-10% test: pupils dilated normally, inhibiting the reabsorption of noradrenaline in the neuromuscular interface. However, the paralyzed pupil does not dilate. This test is more useful in doubtful cases because it only confirms the oculosympathetic injury but does not allow for its localization.

Hydroxyamphetamine test (Paredrina): releases noradrenaline in the neuromuscular interface, causing dilatation if the third neuron is intact, thus allowing to discriminate between pre- or post-ganglionic injury.

The Hydroxyamphetamine test is not available in Spain. In addition, it has an error margin of 10% (3) which increases in children due to trans-synaptic degeneration (4). It is recommendable to carry out a chest CAT scan and an ear and neck NMR in all Horner syndrome patients without clinical signs pointing to a diagnostic, although it is known that the ganglionic syndrome is frequently benign (3).

In children with an acute Horner syndrome of non-traumatic origin, neuroblastoma (cervical or in mediastinum) should be discarded as well as other space-occupying masses, taking into account the high frequency of false negative results in the catecholamine levels in urine. Vascular structures are frequently involved, which calls for an angio-NMR (4).

In our case, the tests (chest CAT and vanililmandelic acid levels in urine) were negative, and medias- tinis evolved favorably with medical treatment.

Horner’s syndrome has been described after tonsils surgery (2,5). Shissias maintained that the damage occurs during the operation due to anatomic variability and the closeness of the amigdalla fossa to the superior cervical ganglion (2). However, in a recently published article Hobson maintains that the cause of Horner’s syndrome could lie in the anesthetic infiltration carried out at the end of the surgery due to diffusion of the infiltrated drug (5). In our patient, this was discarded because no infiltration was made after the surgery.

Therefore, we conclude that in our case the Horner syndrome was due to the damage sustained by the superior cervical ganglion during the palatine amigdalla surgery. Said ganglion is only 1.5 cm behind the amigdalla (2,5). On the other hand, the damage was reversible and evolved up to a near complete disappearance of the condition.

REFERENCES