Horner Syndrome After Anterior Revision Surgery for Cervical Spondylotic Myelopathy: A Very Rare Complication

A Case Report

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Abstract

Case: We report a case of postoperative left-sided Horner syndrome (blepharoptosis, miosis, and anhidrosis) after revision surgery for cervical spondylotic myelopathy. A C4 corpectomy and an anterior cervical fusion from C3 to C5 were performed through a left longitudinal approach in a patient with a surgical history of an anterior discectomy and a fusion from C5 to C6. One year after the revision surgery, the patient had recovered from the anhidrosis and the miosis, but the blepharoptosis was not fully resolved.

Conclusion: Horner syndrome resulting from surgical injury to the ipsilateral cervical sympathetic chain is a very rare complication of anterior spine surgery that has been reported when the lower cervical levels have been approached. Awareness of this important cervical structure may help to avoid this complication.

The anterior approach to the cervical spine was first described by Cloward[1] and Smith and Robinson in 1958[2], and has been used since then to address different cervical spine conditions. This effective approach allows treatment of several conditions, such as disc herniation, myelopathy, tumors, fractures, and infections. This procedure is not without risks, and there are some reported complications. Dysphagia, injury to the recurrent laryngeal nerve, as well as injury to the common carotid artery, the internal jugular vein, the trachea, and the esophagus are among the most frequently reported complications[3-7].

Horner syndrome (blepharoptosis as well as ipsilateral pupillary miosis and facial anhidrosis) can be caused by impairment to the sympathetic pathway. Impairment can occur anywhere along this 3-neuron pathway. The sympathetic supply to the eye and the ocular adnexa starts in the ipsilateral aspect of the hypothalamus, goes down the cervical spinal cord, and travels up the sympathetic chain to the ipsilateral eye[8,9]. It can be present as part of the clinical picture of other diseases and syndromes, including Pancoast tumors, intradural and/or epidural tumors, thoracic outlet syndrome, syringomyelia, brachial-plexus injury, and aortic dissection. Horner syndrome also can be a complication of spinal pathology and/or surgery. Recently, it has been reported as a presentation of a high thoracic disc herniation[10], and also may occur after anterior cervical spine surgery due to an intraoperative lesion of the cervical sympathetic trunk (CST). Although seen as a minor complication, it can cause relevant concern because blepharoptosis can be disfiguring and can cause visual-field impairment. The patient also can have an associated ipsilateral nasal congestion[11,12].

The reported rate of Horner syndrome after anterior cervical discectomy and fusion varies between 0.06%[13] and 3.8%[14], although most reports are based on case series by a single surgeon[3-7]. It is more frequently associated with an oblique corpectomy after an anterolateral approach to the cervical spine, with a reported rate as high as 57%[15]. The rate also is higher after revision surgery, possibly due to distortion of the anatomy and extensive exposure of the cervical structures. Full recovery occurs in 80% to 100% of patients within 3 to 6 months[2-4,13]. Given its rarity and subtle clinical presentation, it often may be underdiagnosed. The objective of this paper is to report a case of

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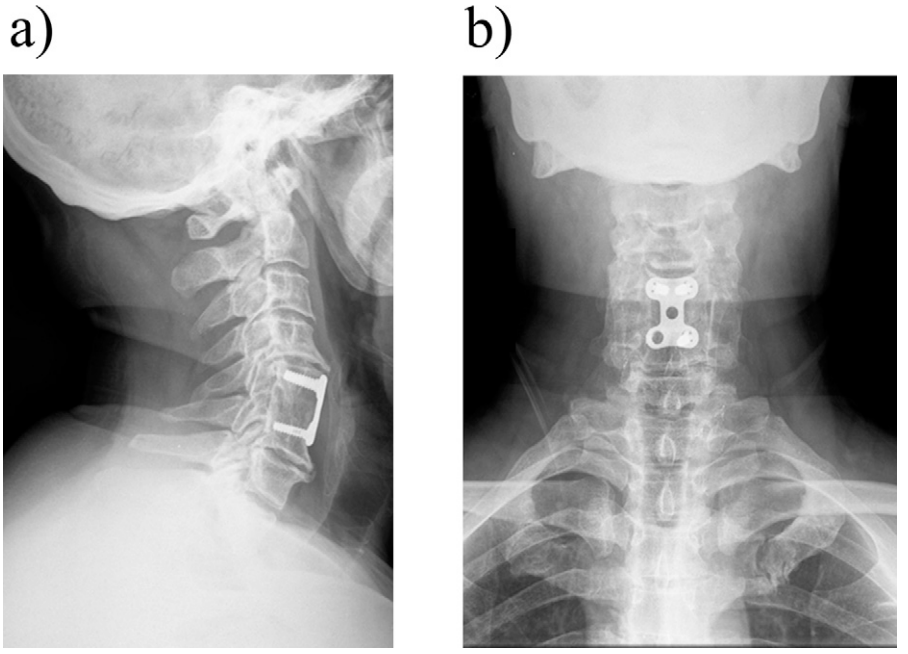

Fig. 1

Preoperative lateral (**Fig. 1-A**) and anteroposterior (**Fig. 1-B**) radiographs showing the fusion from C5 to C6 with an anterior plate.

Horner syndrome after revision anterior spine surgery and raise awareness about this rare and rarely reported complication.

The patient was informed that data concerning the case would be submitted for publication, and he provided consent.

Case Report

A 55-year-old man with a surgical history of an anterior discectomy and a fusion from C5 to C6 that had been performed 13 years earlier (Fig. 1) presented with a 6-month history of gradual-onset cervical pain and upper and lower-limb paresthesia.

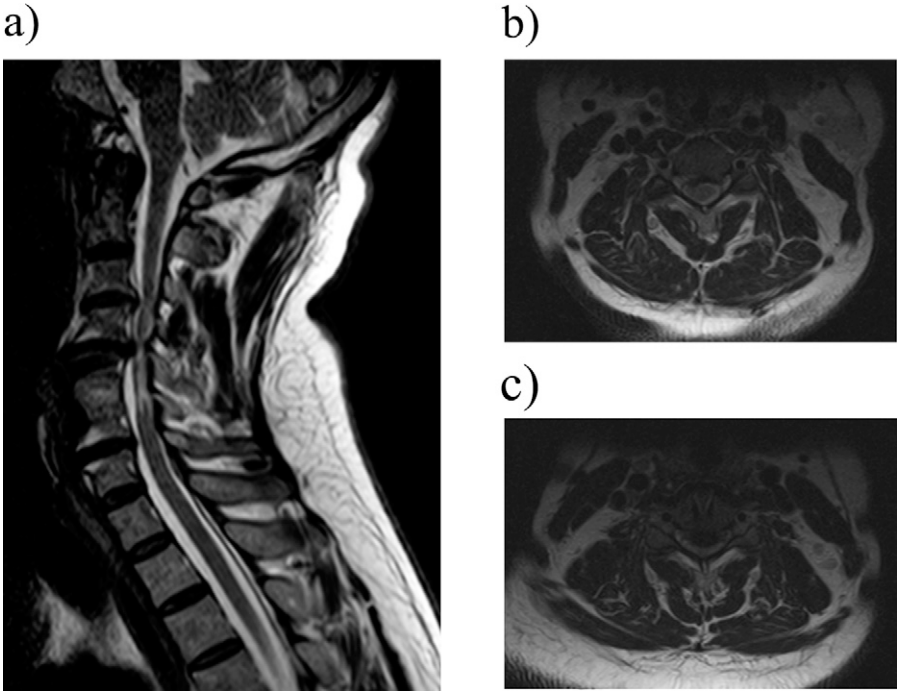

Fig. 2

Cervical spine T2-weighted MRI showing spondylosis from C3 to C5 and C4-C5 myelopathy. The views include sagittal (**Fig. 2-A**), axial at the C3 to C4 level (**Fig. 2-B**), and axial at the C4 to C5 level (**Fig. 2-C**).

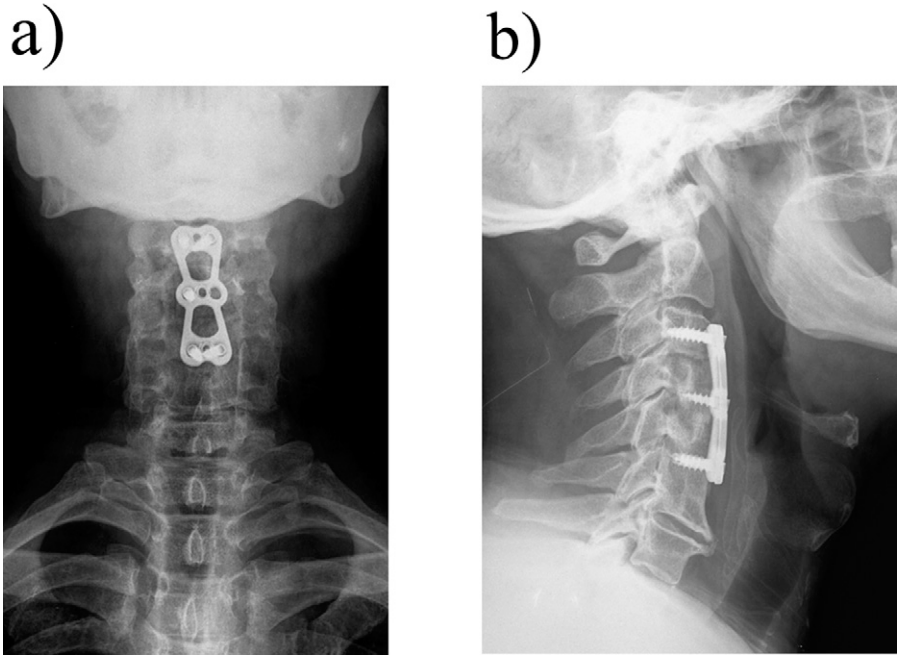

Fig. 3

Postoperative anteroposterior (**Fig. 3-A**) and lateral (**Fig. 3-B**) radiographs showing the prior C5-C6 fusion and the fusion from C3 to C5 with the plate and an autograft.

He noted loss of manual dexterity and a "shocklike" sensation down the spine that had progressed to an ataxic gait and bladder and bowel dysfunction over the previous 6 weeks.

Magnetic resonance imaging (MRI) revealed C3-C4 and C4-C5 spondylosis (Fig. 2) with associated myelopathy at C4-C5; the patient was scheduled for surgery 4 days later. The revision surgery was performed through a left longitudinal approach; the plate and screws from the previous surgery were removed, and a C4 corpectomy and an anterior cervical fusion from C3 to C5 were performed (Fig. 3).

Postoperatively, the examination revealed that the patient had blepharoptosis and miosis of the left eye and anhidrosis of the left side of the face, characteristic of a left-sided Horner syndrome (Fig. 4-A). The patient had progressive but incomplete recovery of the ataxic gait and the bladder and bowel dysfunction. Although the patient had recovered from the anhidrosis and miosis at 1 year postsurgery, the blepharoptosis had not fully resolved (Fig. 4-B).

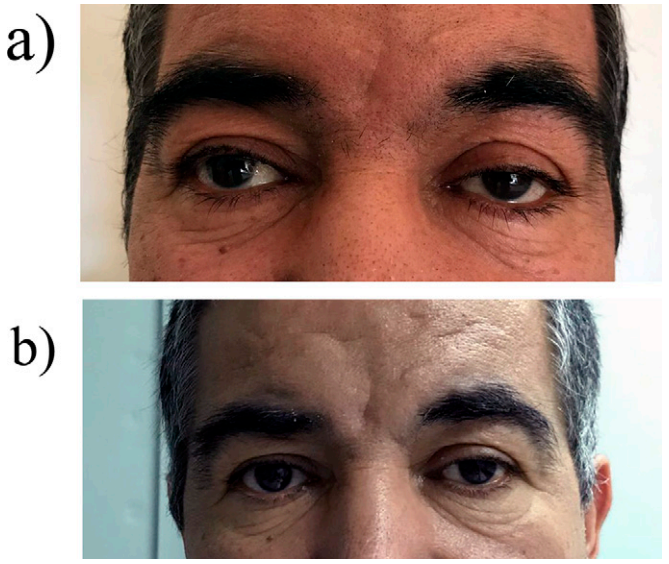

Fig. 4

Figs. 4-A and 4-B Postoperative photographs. **Fig. 4-A** Left-sided blepharoptosis and miosis are evident. **Fig. 4-B** Relative improvement was evident 1 year later.

Discussion

Johann Friedrich Horner first described the classic association of blepharoptosis and ipsilateral pupillary miosis and facial anhidrosis, known as Horner syndrome (oculosympathetic paresis), in 1869, following in the footsteps of Claude Bernard, who had studied the physiology of the sympathetic pathway to the eye in 1852[16]. The sympathetic pathway to the eye begins at the preganglionic neurons that are located at the level of the C8-to-T2 segments of the spinal cord, the ciliospinal center of Budge (the intermediolateral column of Clark). Axons from these neurons leave the cervical spine through the ventral spinal root to 1 or 2 white rami communicantes, and enter the CST at this level. They travel along the CST to the superior cervical ganglion located at the level of C3, where they synapse with postganglionic neurons. The superior cervical ganglion rests immediately anterior to the common carotid artery bifurcation, and the axons that arise from it form the carotid plexus. This plexus is located around the carotid arteries, and the axons continue along it, traveling to the ciliary ganglion. These fibers then pass through this ganglion without

TABLE I Anatomical Studies of the Cervical Sympathetic Trunk and Its Relationship to the Longus Colli Muscle*

Author, Year	Length of CST	Diameter of CST	Distance Between the Medial Borders of the Right and Left LCM	Distance Between the Medial Border of the LCM and the CST	No. of Cadavers
Civelek et al., 2008[20]		3.3 ± 0.6 mm at C6		11.6 ± 1.6 mm at C6	30
Ebraheim et al., 2000[12]	9.7 ± 2.1 mm	2.7 ± 0.6 mm at C6	7.9 ± 2.2 mm at C3; 10.1 ± 3.1 mm at C4; 12.3 ± 3.1 mm at C5; 13.8 ± 2.2 mm at C6	10.6 ± 2.6 mm at C6	28
Kiray et al., 2005[19]			17.2 mm at C3; 12.4 mm at C7		12

*CST = cervical sympathetic trunk, and LCM = longus colli muscle.

synapsing, and enter the eye via the short ciliary nerves, although some sympathetic fibers also can go through the long ciliary nerves and the optic canal[17].

Disruption of the sympathetic innervation of the eye and the ocular adnexa can occur at different levels. According to the location of the disruption, the causes of Horner syndrome can be central, preganglionic, or postganglionic. Preganglionic damage to the sympathetic pathway to the eye can result from a lesion to the CST that occurs during surgery with an anterior or an anterolateral approach to the cervical spine. This complication is rare but documented, especially after use of the anterolateral approach to the cervical spine. A lesion to the CST can be caused by extensive cervical dissection that is used to expose the lateral border of the cervical body, the uncovertebral joint, or the transverse foramen[18].

Anatomical studies have investigated the course, the location, and the anatomical variations of the CST and its relation to satellite structures (Table I); this knowledge allows a safer surgical technique and may help to prevent injury to this structure[12,19,20]. The CST travels posteromedially to the carotid sheath; it lies over the longus colli muscle, converging medially at C6, and the longus colli diverges laterally at this level. Extending from the anterior tubercle of the atlas to the bodies of the C3 to T3 vertebrae, this muscle is the longest and most medial of the prevertebral muscles. The CST is located an average of 11.6 mm lateral to the medial border of the longus colli muscle at the level

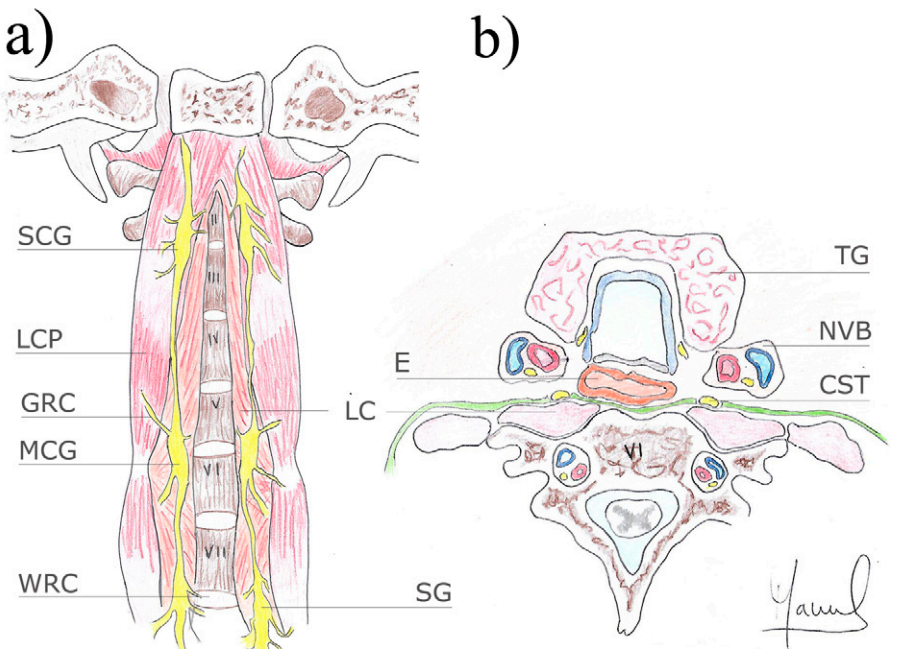

Fig. 5

Illustrations of the coronal (**Fig. 5-A**) and axial (**Fig. 5-B**) views showing the anatomy of the cervical sympathetic trunk. SCG = superior cervical ganglion, LCP = longus capitis muscle, GRC = gray rami communicantes, MCG = middle cervical ganglion, WRC = white rami communicantes, LC = longus colli muscle, SG = stellate ganglion, E = esophagus, TG = thyroid gland, NVB = neurovascular bundle, and CST = cervical sympathetic trunk.

of C6, and is closer to the medial border of the longus colli muscle at the C6 level than at the C3 level. This make the CST more vulnerable to injury with a surgical approach to the lower cervical levels[12,19,20]. Awareness of this anatomy may help to identify and preserve the sympathetic chain. To prevent lesions to the CST, when approaching the anterior cervical spine, the surgeon should stay in the midline when addressing the vertebral bodies, and avoid cutting the longus colli muscle transversely, dissection of the prevertebral fascia, or forceful retraction of the longus colli muscle and neurovascular bundle (Fig. 5).

With our patient, we were not able to identify the precise location of the lesion to the left side of the CST. It is possible that the fibrosis and scarring that were encountered during this revision procedure and the extensive dissection that was required may have led to an inadvertent injury to the CST, which contributed to the Horner syndrome.

The prognosis of Horner syndrome depends on the mechanism of the lesion. If the lesion is indirect (e.g., by retraction), there often will be a spontaneous recovery. However, in cases of complete section of the CST, symptoms will be permanent.

Patients with blepharoptosis may have a substantial cosmetic concern as well as an associated restriction of the upper field of view with a slight loss of vision[11]. The amount of light that reaches the retina is diminished in this situation, causing a reduction of visual acuity, especially in situations of low light. Anisocoria usually is benign in these patients. Palpebral ptosis can be corrected by surgical repair, with good aesthetic and functional results. As an alternative to surgery, use of the alpha-adrenergic agonist phenylephrine can raise the upper eyelid up to 2 mm by acting on the Müller muscle[21].

The reported recovery rate of Horner syndrome is between 80% and 100% after an anterior or an anterolateral approach in cervical spine surgery. In our patient, the blepharoptosis had not completely resolved at 1 year postsurgery. ■

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