

# Case Report

## Claude Bernard–Horner syndrome resulting from pleural empyema\*

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### ABSTRACT

Claude Bernard–Horner syndrome presents various etiologies and occurs as the direct result of interrupted nerve signaling at any point along the nerve trajectory, be it intrathoracic or extrathoracic. Herein, we report a case of Claude Bernard–Horner syndrome caused by loculated pleural empyema located in the paravertebral region of the upper third of the right hemithorax. The patient was submitted to thoracotomy in order to drain the infected fluids. The end result was satisfactory, including resolution of the infection, pulmonary expansion, and remission of the syndrome.

**Keywords:** Pleural empyema/complications; Horner syndrome/etiology; Blepharoptosis; Thoracotomy; Case reports [publication type]

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### INTRODUCTION

Claude Bernard–Horner syndrome (CBHS) results from a blockage of the sympathetic innervation to the eye and face at any point along its trajectory. It is clinically associated with a discrete to moderate blepharoptosis due to paresis of the superior tarsal muscle, also known as Müller's muscle. The pupil presents variable myosis that depends on the location, degree and chronicity of the deficit. The syndrome is considered complete

when these symptoms are accompanied by anhidrosis of the ipsilateral hemiface, an increase in temperature, and facial hyperemia.<sup>(1-2)</sup>

The etiologic factors of CBHS are multiple and can be iatrogenic, traumatic or associated with systemic diseases. Pleural empyema is rarely the etiologic agent of this syndrome. The objective of this report is to present a case presenting this association.

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## CASE REPORT

A 32-year-old male patient presented with sudden onset of pain in the right shoulder upon physical exertion. He sought medical attention and was treated with anti-inflammatory, but there was no improvement in his symptoms. After two days, he presented fever and increased intensity of the pain. The X-ray of the right shoulder was normal, and the treatment was therefore continued. On the fifth day, the patient presented considerable worsening of the symptoms, with cellulitis in the entire right hemithorax, acute dyspnea, signs of septicemia, decreased arterial pressure, and tachypnea. He was referred to the intensive care unit for ventilatory support and diagnostic investigation.

The chest X-ray upon admission revealed a hypotransparency in the upper third of the right hemithorax (Figure 1). The computed tomography of the chest revealed an image suggestive of loculated pleural empyema located in the paravertebral region of the upper right hemithorax (Figure 2). The patient was submitted to an emergency exploratory posterolateral thoracotomy, with drainage of the empyema and pulmonary decortication. He remained on mechanical ventilation for six days and was discharged from the intensive care unit on postoperative day 10, with satisfactory evolution and resolution of the



Figure 1- Chest X-ray revealing a paramediastinal hypotransparency in the upper right hemithorax

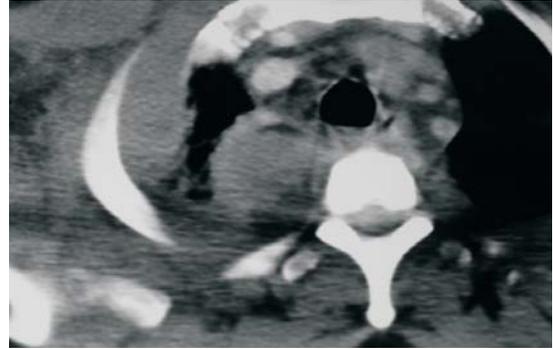


Figure 2 - Computed tomography of the chest revealing loculated pleural empyema located in the paravertebral upper right hemithorax



Figure 3 - Patient with right blepharoptosis

infection. After the ventilatory weaning procedure and extubation of the patient, while he was conscious, we observed blepharoptosis of the right eye. The patient perceived this ptosis on the fifth day after the onset of the clinical profile and before hospitalization, a fact that was confirmed by his family (Figure 3). The patient was discharged on postoperative day 14 and presented good general health status. After two months of postoperative outpatient follow-up evaluation, the patient presented total regression of the blepharoptosis.

## DISCUSSION

The sympathetic nerve responsible for innervating the eye and face follows a descending trajectory from the hypothalamus via the first-order motor neuron down to the brainstem and then follows to the stellate ganglion via the second-order motor neuron. Subsequently, it reaches the face via the third-order motor neuron and divides into two branches: one along the trajectory of the internal carotid artery (toward the smooth muscle of the eyelid and pupil); and

another along the external carotid artery (toward the sweat glands of the face).<sup>(1-4)</sup>

Due to the complex trajectory of the sympathetic pathway, interruptions in nerve signaling, resulting in CBHS, can occur at distinct levels. Therefore, CBHS cases are grouped by the location of lesions: brain; brainstem; spinal cord; thoracic vertebrae from C8 to T1; cervical sympathetic chain; and orbit.<sup>(1-2)</sup>

The hemispheric lesions that can cause the syndrome are extensive brain infarction and thalamic hemorrhage. The brainstem lesions are hypothalamic infarction, Wallenberg's syndrome, multiple sclerosis, tumors and brainstem encephalitis. Among spinal cord lesions are syringomyelia, vertebral tumors, spinal cord tumors and traumas.<sup>(1,5)</sup>

The lesions from C8 to T1 that can result in the symptomatology are Pancoast tumor, thoracic outlet syndrome and mechanical compressions. The lesions of the cervical sympathetic chain can be caused by carotid pathology, neoplastic infiltration, adenopathies or abscesses.<sup>(1,6-8)</sup>

Regarding iatrogenic causes, the incidence of CBHS resulting from video-assisted thoracoscopic sympathectomy has been shown to be 2.6%.<sup>(9)</sup> In a study conducted in Brazil in 2003,<sup>(10)</sup> a sample of 743 sympathectomy patients was analyzed, and the incidence of CBHS was reported to be 0.54%. Vascular procedures performed in open upper thoracic surgery can result in lesions of the stellate ganglion, as was described in a case described in 2000.<sup>(11)</sup> Procedures such as epidural anesthesia can also result in CBHS.<sup>(12)</sup>

Another complication related to the surgical intervention is the inappropriate positioning of the chest tube, which has been implicated as a factor in the genesis of CBHS. However, the risk can be eliminated by early correction of the tube position. Mechanical compression of the sympathetic chain probably leads to ischemia and postoperative neuropraxia. It is likely that such compression is directly related to the duration of the contact with the etiological agent.<sup>(13)</sup>

The clinical signs, together with the pharmacological evidence, facilitate the topographic diagnosis of the lesion and make it possible to clarify its etiology. The oculosympathetic postganglionic lesions do not include anhidrosis and are accompanied by only blepharoptosis and myosis. The use of hydroxyamphetamine hydrobromide 1% allows us to clearly distinguish between

preganglionic and postganglionic blockage since its administration causes pupil dilation in patients with preganglionic lesions but not in those with postganglionic lesions.<sup>(1,14-15)</sup>

There are multiple complications associated with pleural empyema, including the septicemia, lung entrapment and spontaneous drainage of the empyema into the skin or contiguous tissues.<sup>(16)</sup> Lesions of the sympathetic cervical thoracic chain caused by intrathoracic abscess are presented in the literature as etiological agents of CBHS. Nevertheless, in our review of the literature, we found no reference to this syndrome associated with loculated pleural empyema.

We have presented herein the case of a patient with loculated pleural empyema located in the upper right paravertebral region, in the topography of the sympathetic thoracic chain, leading to postoperative CBHS. The surgical intervention, which consisted of the removal of the empyema cavity and drainage of the chest, was successful, with resolution of the infection and remission of the syndrome.

In conclusion, although it is rarely the cause of CBHS, the loculated pleural empyema can be considered during the diagnostic study since it can result in mechanical compression of the sympathetic chain, which is reversible upon the removal of the causal factor.

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