LETTERS TO THE EDITOR

Claude Bernard–Horner Syndrome as a Rare Complication of Postoperative Drainage

To the Editor: The Claude Bernard–Horner syndrome was first described in humans in 1869 associated with palpebral ptosis, enophthalmos, and myosis. It is the clinical manifestation of cervical sympathetic chain involvement, most commonly in the stellate ganglion, which is located at the cervicothoracic junction. Multiple causes have been described for this syndrome, including neoplastic, neurologic, iatrogenic, traumatic, and vascular processes. Among iatrogenic causes mentioned most often in the literature are those resulting from jugular catheterization, from thoracic sympathectomy as a treatment for hyperhidrosis, from epidural anesthesia, and from resection of
mediastinal tumors. We report the case of a 28-year-old woman who developed iatrogenic Claude Bernard–Horner syndrome related to postoperative pleural drainage.

The patient reported no relevant medical history, and accordingly a thoracoscopic examination was made after the second episode of spontaneous pneumothorax in the left lung. The apex of lung was resected, revealing evident bullous dystrophy. No adverse events occurred during surgery, and there was no concurrent thoracic involvement. Pleurodesis and pleurectomy were not performed. No epidural catheters or central airway tubes were inserted during preparation for anesthesia. The pleural cavity was drained through a 24F silicone tube which was positioned in the left side of the chest towards the pleural apex and over the anterior part of the lung. The tube was connected to suction of −20 cm H₂O. Immediate postoperative radiographs revealed almost complete re-expansion of the lung and proximity of the drainage tube to the area surrounding the stellate ganglion. Claude Bernard–Horner syndrome in the left lung was the only postoperative complication, appearing at 36 hours, with severe palpebral ptosis, enophthalmos, conjunctival injection, and myosis. The pleural drain was immediately removed, once pulmonary re-expansion and the absence of air leaks had been confirmed. Resolution of the syndrome gradually occurred over 6 months until complete remission was achieved.

The literature makes little mention of closed or postoperative pleural drainage, in any disease context, as a rare cause of Claude Bernard–Horner syndrome. However, frequency may be higher than that reflected in publications. Some authors report different pathophysiologic causes of iatrogenic damage to the stellate ganglion: injury, section or direct burn; compression by a local hematoma; heat transfer from the electric scalpel; drainage tube contact (pressure necrosis); or direct aspiration over the ganglion. We believe that the last mechanism accounts for the unusual complication in our case. In some cases, signs have been short-lived, lasting from a few days to up to 6 months. However, most cases described involve permanent damage and have serious cosmetic, medical and legal implications.

Although Claude Bernard–Horner syndrome is a rare complication, like other authors, we consider that the tip of the pleural tube should not be located near the cervicothoracic junction, and that the drain should be repositioned or removed before the onset of the syndrome.

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