Horner’s syndrome secondary to tube thoracostomy

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Tube thoracostomy is a common therapeutic approach applied in medical practice. Certain complications of this procedure have been described in the literature. Oculosympathetic paresis, or Horner’s syndrome, occurs from the interruption of second order preganglionic neurons and manifests as miosis, ptosis, hemifacial anhidrosis and enophthalmos. Iatrogenic Horner’s syndrome, on the other hand, very rarely couples with tube thoracostomy. Only seven cases have been described in the literature, two of whom were in the pediatric age group. Herein we present a three-year-old girl operated for diaphragmatic hernia who later developed Horner’s syndrome at the same side of the thorax tube. Upon the development of the pathology, the tube was repositioned and after one month only a slight ptosis persisted. Our patient seems to be the third case described in the literature. The clinical significance of this pathology is assessed in this report.

Key words: tube thoracostomy, Horner’s syndrome.

Case Report

A three-year-old girl admitted to our hospital with the diagnosis of right-sided diaphragmatic hernia. She had presented to her pediatrician with the complaints of cough and fever. On chest X-ray, the right contour of the diaphragma could not be followed. Together with the lateral and contrast X-ray studies, a lateral right diaphragmatic hernia with colonic loops inside the defect could easily be seen. Right thoracostomy and hernia repair were planned. During the operation the colonic loops were reduced to the abdomen and the diaphragmatic defect was primarily repaired with nonabsorbable sutures. A 24 F thorax tube was introduced before the closure of the wound. The preoperative and early postoperative course was uneventful. Three days postoperatively, miosis, ptosis and enophthalmos at her right eye were recognized (Fig. 1). The anteroposterior chest X-ray showed that the tip of the tube was localized and slightly curved just at the apex of the right hemithorax (Fig. 2). The position of the tube was suspected as the cause, and was repositioned. The chest tube was later removed, and the patient was discharged from the hospital on the sixth postoperative day. Two weeks later, the clinical appearance of the right eye showed progression but the syndromic look persisted. However, one month after the
operation, only a slight ptosis remained and almost total improvement was achieved. No further resolution of the pathology was observed. She had no postoperative complication regarding her diaphragmatic repair.

Discussion

Horner’s syndrome is the paresis of the oculosympathetic pathway. This pathway starts from the hypothalamus and continues to the eye. Nerve fibers stemming from hypothalamus reach the ciliospinal center in the spinal cord. Secondary preganglionic neurons located in this center leave the spinal cord in the ventral roots of C_8 and T_1 vertebra. These fibers pass through the first thoracic and inferior cervical ganglia and reach the superior cervical ganglia. Only the endoathoracic fascia separates parietal pleura from the ganglia. Ninety percent of Horner’s syndrome cases originate from compression mostly due to a malignant tumor in this region. Tube thoracostomy is one of the other rare entities that can cause this pathology.

Improper insertion of a chest tube can cause unwanted complications. Among these complications, Horner’s syndrome is a relatively uncommon entity. The close proximity of the superior cervical ganglia and thoracic cavity, especially at the apex of the thorax, can easily lead to external compression when the tip of the chest tube is located in this area. In our case the apical localization of the chest tube was evident (Fig. 2). The ipsilateral involvement of the eye and gradual improvement after the removal of the tube exclude any other possible cause. Even though tube thoracostomy is routinely applied in clinical practice, exact recognition of thoracic anatomy and sufficient consideration of the possible complications are mandatory for successful applications. From this point of view, the tip of the chest tube should not be placed close to the apex and, if so, should be repositioned immediately. The second thoracic vertebra is a suitable place for the tip localization. Another subject of discussion is whether catheter removal, after the development of Horner’s syndrome, leads to reversal of the pathology. According to the clinical experience, there have been cases of full resolution, no resolution at all, and of only partial resolution, such as in our case. A possible explanation can be that the scope of the pathology is dependent on the degree of the injury to the superior cervical ganglia. Thus, complete pressure ischemia or severe trauma may result in irreversible changes and local edema or hematoma formation may cause partial or reversible injury to the ganglia. Thus, repositioning or total removal of the chest tube should be evaluated when such a complication is experienced in clinical practice.

REFERENCES