

# Horner's syndrome: an extremely rare complication of tube thoracostomy

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## SUMMARY

Chest tube insertion is the most commonly performed procedure in the thoracic surgery practice. This surgical procedure has various complications, however, Horner's syndrome resulting from compression of the stellat ganglia due to apical insertion of chest tube is extremely rare. In this article a 21-year-old male patient who was treated with chest tube insertion for spontaneous pneumothorax at the left side is presented. The chest tube was removed after four days, however, classical triad of Horner's syndrome including unilateral ptosis, miosis and anhidrosis developed. On the control examination performed after three months symptoms improved except for slight ptosis. It should be kept in mind that Horner's syndrome may develop as a complication of apically placed chest tube.

**Key words:** Horner' syndrome, pneumothorax, thoracostomy

## ÖZET

**Horner sendromu: tüp torakostominin oldukça nadir görülen bir komplikasyonu**

Göğüs tüpü uygulaması toraks cerrahisi pratiğinde en sık uygulanan prosedürdür. Bu cerrahi prosedür birçok değişik komplikasyon içerir, ancak göğüs tüpünün apikal yerleştirilmesine bağlı olarak stellat gangliyonun basısı sonucu oluşan Horner sendromu gelişimi oldukça nadir görülür. Bu yazıda 21 yaşında sol taraflı spontan pnömotoraks nedeniyle göğüs tüpü yerleştirilerek tedavi edilen bir erkek hasta sunulmuştur. Hastanın göğüs tüpü dört gün sonra çekildi, ancak tek taraflı pitozis, miyozis ve anhidrozis triadından oluşan Horner sendromu gelişti. Üç ay sonra yapılan kontrol muayenesinde hafif pitozis dışında semptomların düzeldiği görüldü. Apikal olarak yerleştirilen göğüs tüpünün bir komplikasyonu olarak Horner sendromu gelişebileceği akıldaki bulundurulmalıdır.

**Anahtar kelimeler:** Horner sendromu, pnömotoraks, torakostomi

## Introduction

Tube thoracostomy (TT) is a life-saving and safe surgical procedure with low morbidity rate. The early and late complications of TT may be life-threatening. The probable complications of TT should be considered by the thoracic surgeons in order to avoid morbidity and mortality related with this procedure (1). The incidence of Horner's syndrome (HS) is approximately 1.3% in thoracic surgery practice (2). The reversibility of this clinical condition is related with the etiologic factor. The compression of stellat ganglia with the tip of chest tube can be accepted as a reversible etiologic factor. However there are some cases in the medical literature in whom symptoms have not completely recovered after chest tube insertion (3). We think that the reversibility of this clinical condition is closely related with the duration and force of the compression of stellat ganglia, and these patients should be closely followed up.

## Case Report

A 21-year-old male patient was admitted to our emergency department with the complaint of chest pain and dyspnea. There were decreased breath sounds at the left side and his chest X-ray revealed partial spontaneous pneumothorax. He was referred to our clinic for chest tube insertion. A 28-gauge chest tube was inserted through the fifth intercostal space on the anterior axillary line and connected to underwater seal. His lung expanded and no air leak was determined after the chest tube insertion (Figure 1). At presentation and during the treatment we did not observe any ptosis or miosis. The thorax computerized tomography (CT) was performed for the evaluation of bullae. It revealed a few bullae with a diameter of less than one cm and the apical placement of the tip of chest tube (Figure 2). We removed his

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**Figure 1.** Chest X-ray of the patient showing apical placement of the chest tube

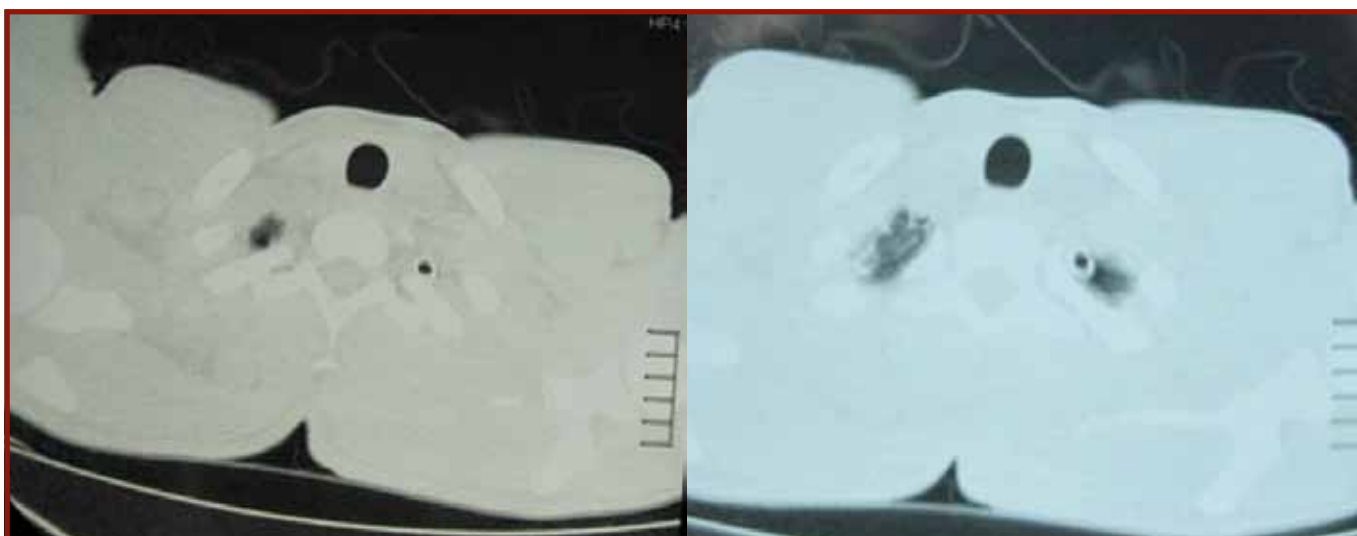
chest tube on the 4th day and got aware of the ptosis and miosis on the left side after removal of the chest tube (Figure 3). The patient declared that this condition had occurred with the placement of tube thoracostomy. The compression time of stellat ganglia via tip of chest tube was estimated to be four days. After three weeks he admitted to another thoracic surgery department with the same symptoms and recurrent spontaneous pneumothorax was detected. Then he underwent left axillary thoracotomy for the resection of bullae. The symptoms recovered but a slight ptosis

was present after a follow-up of three months. In our case the compression time of stellat ganglia is longer in comparison to the literature findings. Although this clinical condition is usually reversible, the compression time of stellat ganglia is the primary factor determining the duration of reversibility.

### Discussion

HS may result from various etiologies such as central, preganglionic and postganglionic reasons. The preganglionic etiologies include Pancoast tumour, apical pulmonary tuberculosis, thoracic surgery including chest tube insertion, carotid dissection or aneurysm, cervical tumors, and trauma to the neck or chest. The latter etiology is the most common cause. The anatomic destruction of stellat ganglia by tumor invasion or trauma is an irreversible condition. However the apical placement of chest tube is an iatrogenic and somewhat reversible reason. Therefore the chest tube placement and its re-positioning as soon as possible is very important when HS occurred.

Cocaine is a sympathomimetic agent that produces pupillary dilation after topical administration by blocking re-uptake of norepinephrine and enhancing its concentration at the iris dilator. Cocaine-induced mydriasis requires an intact oculosympathetic pathway; pharmacologic confirmation of HS is demonstrated if topical application of this agent produces less dilation, or not at all, compared with the fellow pupil. The “cocaine test” has become the standard diagnostic method for confirming clinically suspected HS. Typical protocols consist of applying one to two drops of cocaine 4% to 10% to both eyes and com-



**Figure 2.** Thorax computed tomographic scan of the patient showing apical placement of the chest tube

paring the amount of dilation of the abnormal pupil with the normal pupil (4). This confirmatory test is useful in patients with multiple trauma in order to determine the level of cause. In our case the etiologic factor was obvious and we did not perform any confirmatory test like cocaine administration.

The first case of HS occurring due to placement of chest tube has first been reported by Rosseggar and Fritsch in 1980 (5). The thin endothoracic fascia between the apical parietal pleura and stellate ganglion is a predisposing factor for the development of HS. The movement of tip of chest tube with the expansion of lung can injury the stellate ganglia. The outcome of these patients is variable and the resolution of the HS cannot be guaranteed (5,6). Neuropraxia defined as reversible impairment of impulse transmit is responsible for the pathophysiology of this iatrogenic etiologic factor. However, in our case and in one of the cases in the study of Kaya et al. the symptoms did not improve completely when the compression time was more than three days (2). Therefore we can not explain the pathophysiology just due to neuropraxia because of the limited reversibility.

HS can be the presenting symptom of pneumothorax. In these cases air collection in the hemithorax is responsible for the compression of stellate ganglia. This clinical condition is usually related with the tension pneumothorax. There has been only one reported case in the literature, however, our case did not present with HS (7).

Althought chest tube placement undergoing thoracotomy is a safe procedure as it is achieved under direct observation, Kaya at al. have reported that two of five patients have had HS after thoracotomy procedure (2). This complication should be kept in mind when placing an apical chest tube. The recovery of HS caused by chest tube insertion is closely related with compression time of stellate ganglia, and such patients should be closely followed up particularly in apical placement of chest tube.

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