

## Unusual Complications of Empyema Thoracis: Diaphragmatic Palsy and Horner's Syndrome

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### ABSTRACT

We report here a 3 month old child with empyema thoracis, who developed complications of diaphragmatic palsy and Horner's syndrome. These complications of empyema thoracis have not been reported earlier. We discuss the possible mechanisms for these complications. [Indian J Pediatr 2006; 73 (10) : 941-943] E-mail: rakesh\_lodha@hotmail.com

**Key words :** *Empyema thoracis; Diaphragmatic Palsy; Horner's syndrome*

Empyema thoracis may occur in 5- 10% of children with bacterial pneumonias.<sup>1</sup> Various complications of empyema thoracis have been described: bronchopleural fistula, pyopneumothorax, pericarditis, peritonitis, systemic sepsis, etc. We describe here two unusual complications of empyema thoracis: diaphragmatic palsy and Horner's syndrome. To best of our knowledge, these complications have not been reported earlier.

### CASE REPORT

A 3-months-old girl, previously thriving well, presented to emergency services with history of high grade fever for 5 days, swelling over left axillary region for 3 days, excessive crying for 2 days, rapid breathing and decreased oral acceptance for 2 days, and decreased sensorium for 12 hours. There was no history of rash, cyanosis, vomiting, neck retraction, or seizures. On examination, child was irritable and stuporous. The child was febrile. The pulse rate was 180 per minute, pulses were feeble, and peripheries were cold. The capillary filling time was 4 seconds. The respiratory rate was 58 per minute with chest indrawing. A diffuse, erythematous, warm, tender, non-fluctuant and indurated swelling 5cm X 5cm was seen in the left axillary area with surrounding edema; a pus point was seen on its centre. There were decreased chest movements and air entry on left side suggesting a pleural fluid collection. Crepitations were heard on both

the sides of chest, more on the right. Liver was enlarged 5 cm below costal margin. Cardiovascular examination was normal except for tachycardia. Central nervous system examination revealed an irritable and distressed child with normal cranial nerve examination; motor and sensory systems were normal. There were no signs of meningitis or raised intracranial pressure.

A diagnosis of very severe pneumonia with chest wall abscess with pleural effusion with septic shock was made. Patient was intubated and mechanically ventilated for increasing respiratory distress. The child was resuscitated with normal saline boluses and infusion of dopamine at 10 mcg/Kg/min. The child was administered cefotaxime, amikacin, and vancomycin. The child was shifted to the Pediatric intensive care unit. Arterial blood gas report revealed compensated metabolic acidosis. Chest X-ray showed left sided pleural effusion with infiltrates in the right lung (Fig. 1). Hemoglobin was 7.8 gm/dL, total

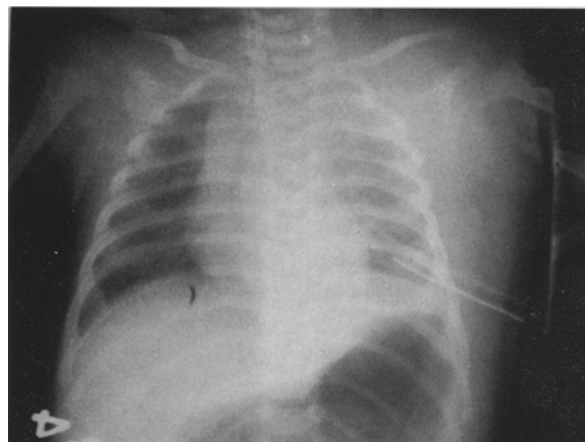


Fig. 1. Chest radiograph at admission.

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leukocyte count of  $7900/\text{mm}^3$  with 46% polymorphs, 52% lymphocytes, and 2% eosinophils), platelet count was  $80000/\text{mm}^3$ . C-Reactive Protein was negative. Contrast enhanced CT scan of the chest revealed left pleural effusion with enhancing walls suggestive of empyema extending in to the anterior mediastinum and left chest wall (Fig. 2); consolidation was seen in left upper and left lower lobes, and right upper lobe. There was mediastinal shift to right side.

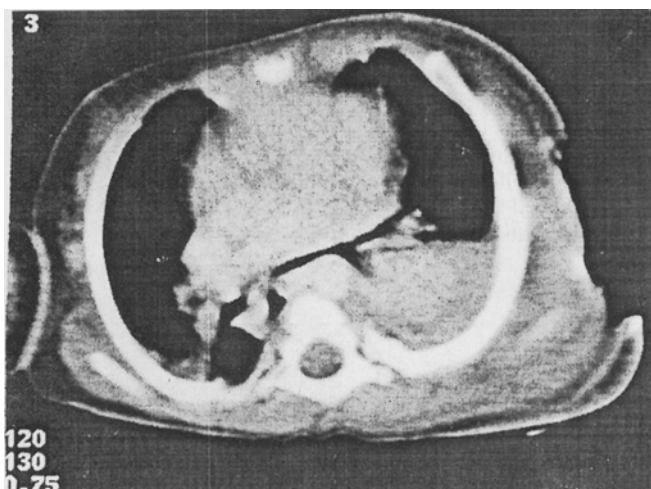


Fig. 2. CT chest at admission

A chest tube was inserted for left empyema and this revealed purulent material. Aspiration of the swelling in left axilla did not yield any material. An ulcer appeared over the swelling in the left axilla. Pus from chest tube revealed gram-positive cocci and culture was positive for methicillin sensitive *Staphylococcus aureus*.

Over next few days, the ulcer in left axilla gradually increased in size and child developed surrounding edema involving scalp, neck, anterior and posterior chest wall including lumbar region. Wound debridement was done and daily dressing was done using EUSOL and diluted povidone iodine. By day 16 of hospital stay wound extended up to pectoralis major. Thereafter, granulation tissue started appearing on the base of wound and general condition of the child improved. The chest radiographs showed decrease in the size of empyema and resolution of infiltrates. The chest tube was removed on 20<sup>th</sup> day of hospital stay.

On day 27 of admission child was noticed to have ptosis of left eye with miosis. There was no other neurological abnormality. The chest X-ray this time showed raised left diaphragm; the lung fields were clear (Fig. 3). Left diaphragm palsy was suspected and confirmed by fluoroscopy examination, which showed no movement of diaphragm on left side. Child was gradually weaned from ventilator and extubated on day 30 of hospital stay. The child was subsequently discharged from hospital on day 40 with no change in the findings of Horner's syndrome and diaphragmatic palsy.

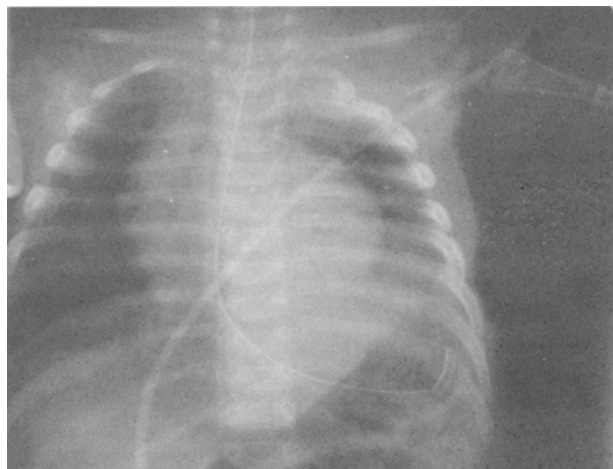


Fig. 3. Chest radiograph showing elevated left dome of diaphragm.

## DISCUSSION

We have described here a child with left sided empyema thoracis who subsequently developed left diaphragmatic palsy and left Horner's syndrome.

Both unilateral and bilateral diaphragmatic palsy are very rare in children. The combination of Horner syndrome, phrenic nerve palsy, and palsy of the ipsilateral recurrent laryngeal nerve has been described in adults with tumors – Rowland Payne syndrome.<sup>2</sup> The same has been described in a child with cervical neuroblastoma.<sup>3</sup> Various causes for diaphragmatic palsy have been described in children including birth trauma, surgical procedures involving neck and chest, mediastinal and pulmonary tumors and surgical manipulation of diaphragm during abdominal surgery.<sup>4</sup> Recently, it is also been reported to complicate central venous catheterization.<sup>5</sup>

Phrenic nerve has got a long course making it vulnerable to damage. It arises from  $C_3$ ,  $C_4$  and  $C_5$  roots and passes over scalene muscles and between the subclavian vein and artery under the visceral pleura, and then it runs along the pleura lateral to the pericardium to reach the diaphragm. Horner's syndrome occurs secondary to injury to sympathetic ganglion- stellate ganglion situated at  $C_8$ ,  $T_1$ . Horner's syndrome could result from neck surgeries, cardiac surgeries and thoracotomy. It is also been described secondary to tube thoracostomy and chest trauma with clavicle and first rib fracture.<sup>6</sup> Our case had undergone debridement in the left lateral chest wall. It is unlikely that this could have injured the phrenic nerve and sympathetic ganglion.

Our patient was noticed to have simultaneous onset of left Horner's syndrome (miosis and ptosis) and left diaphragm palsy with out any associated brachial plexus injury. There was no laryngeal nerve palsy. These complications have not been reported earlier in children. It is possible that there was injury to the left phrenic nerve

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due to inflammation and infection in left pleural space. The CT scan of the chest had revealed left empyema thoracis extending in to the anterior mediastinum and left chest wall. The other possibility is that the inflammation and infection in the apex of left lung could lead to irritation and injury to phrenic nerve as well as the stellate ganglion. Absence of any motor abnormality in the left upper limb rules out any cervical cord lesion. The other possibility is of osteomyelitis of the left first rib. While Horner's syndrome has been described in cases of fracture of first rib,<sup>6</sup> the same has not been the case with osteomyelitis. Also, there was no evidence of osteomyelitis on the chest radiographs or the CT scan of the chest in our case. It is also possible that the child had necrotizing fasciitis and the extension of infection led to involvement of stellate ganglion leading to Horner's syndrome.

In conclusion, we have presented a case of left empyema thoracis with unusual complications of ipsilateral diaphragmatic palsy and Horner's syndrome.

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