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Transient Phrenic Nerve Paralysis Associated With Status Asthmaticus

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Summary. Phrenic nerve paralysis is a condition typically occurring after invasive procedures in the chest and neck. Here we describe a case of transient unilateral diaphragmatic paralysis in a child with status asthmaticus complicated by complete right lung atelectasis. Common causes of this disorder and possible implications for our case are discussed. **Pediatr Pulmonol. 2004; 38:269–271.** © 2004 Wiley-Liss, Inc.

Key words: atelectasis; phrenic nerve paralysis; status asthmaticus.

INTRODUCTION

Status asthmaticus (SA) is a life-threatening condition with significant morbidity and mortality.[1] Common complications of SA include atelectasis, air leak, infection, and respiratory failure. Phrenic nerve paralysis (PNP) is occasionally reported as a complication of SA requiring mechanical ventilation and chest drainage.[2] We present a case of transient unilateral PNP in a child with SA complicated by unilateral lung atelectasis.

CASE REPORT

A 9-year-old boy with wheezing and atelectasis was referred to our pediatric intensive care unit (PICU) for impending respiratory failure. His medical history, following an uncomplicated perinatal course, included frequent episodes of wheezing and recent prick tests positive for house-dust mite and grass pollens. Of note, at 4 years of age, the child suffered from a blunt chest trauma due to a motor vehicle accident, when he hit the car seat in front of him with his trunk. No loss of consciousness or invasive procedures were reported. The chest X-ray obtained on that occasion was normal, and the child recovered without known sequelae. At the time of the current episode, his only medication was occasional use of asthma inhalers for wheezing relief.

The day before his referral to our unit, the child was admitted to a peripheral hospital because of coughing, wheezing, and right-sided chest pain. There was no fever or history suggestive of inhaled foreign body. Physical examination of the chest revealed bronchial breath sounds on the right lower field with a few scattered wheezes. A chest radiograph showed hazy infiltrates in the middle and lower left lobes, with normal position of both hemidiaphragms. In spite of appropriate treatment for acute asthma, overnight he experienced increasing respiratory distress and a need for supplemental oxygen (O_2). A repeated chest X-ray and a subsequent CT scan showed a complete atelectatic right lung with a compensatory hyperinflated left lung. The child was then referred to our hospital for ongoing treatment.

On admission to our PICU, the child had tachypnea (50 bpm) with chest retractions, reduced air entry with bronchial breath sounds on the right side, and diffuse wheezing on the left. Asymmetric breathing was also present, with reduced excursion of the right rib cage. Transcutaneous O_2 saturation was 84% in room air. The remainder of his physical examination was unremarkable. A chest X-ray confirmed complete atelectasis of the right lung with a mediastinal right shift (Fig. 1). The laboratory workup was not significant except for total IgE 3,080 kU/l. After starting a treatment with inhaled albuterol, i.v. methylprednisolone, and supplemental oxygen, the child underwent an urgent flexible fiberoptic bronchoscopy that revealed thick mucous secretions on the right bronchial tree. Repeated lavages with saline were performed, followed by chest physiotherapy.

On day 2, the chest X-ray showed a striking reduction of atelectasis but revealed an anomalous elevation of the right hemidiaphragm (Fig. 2). In fact, in addition to the breathing asymmetry of the chest, a paradoxical inward movement of the right upper abdominal quadrant was appreciable during inspiration. Fluoroscopy confirmed the

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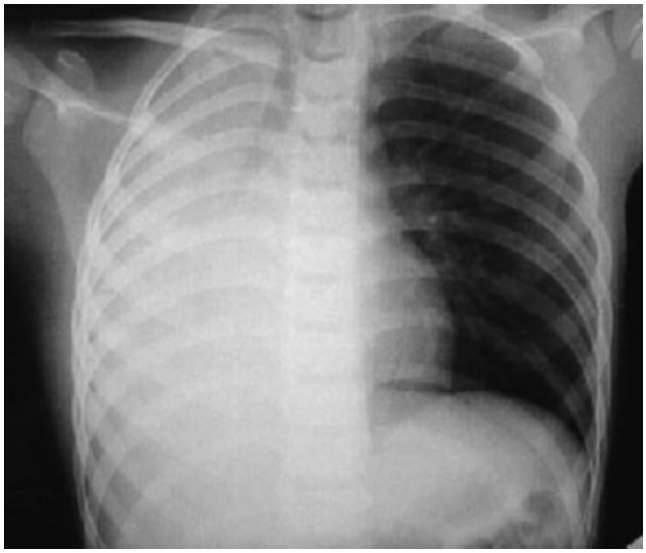

Fig. 1. Chest radiograph at admission, showing atelectatic right lung.

clinical suspicion of PNP, displaying the inspiratory paradoxical movement of the right hemidiaphragm. Over the next few days, a gradual recovery from respiratory symptoms occurred, and repeat chest X-rays (days 3 and 8) showed a progressive reduction in the elevation of the hemidiaphragm. The second fluoroscopy (day 8) also documented a striking reduction in his paradoxical movement, and the patient was transferred back to the referring hospital for ongoing treatment. No other neuromuscular abnormalities were detected over the period of his hospital stay.

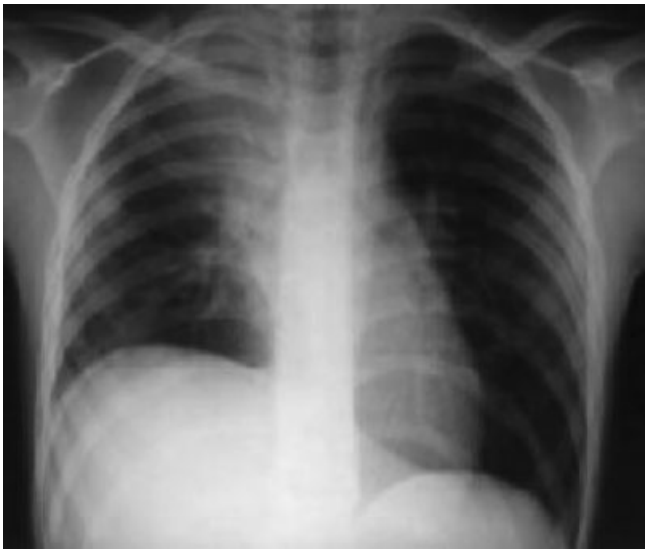

Fig. 2. Chest radiograph following fiberoptic bronchoscopic lavage, demonstrating elevation of right hemidiaphragm due to phrenic nerve paralysis.

One week after transfer (day 17), an electrophysiological study was performed to rule out a misdiagnosed neuropathy or myopathy. Determination of his peripheral (i.e., right arm) nerve conduction velocity and electromyography pattern yielded normal results. Three weeks later (day 38), a chest radiograph showed a clear right lung, and the ipsilateral hemidiaphragm returned to its normal position. At fluoroscopy, no abnormal displacement of the right hemidiaphragm was detected. Pulmonary function tests were normal as well. The child had symmetric and quiet breathing, while taking regular bronchodilator therapy.

DISCUSSION

Status asthmaticus (SA) is a condition that may have several complications.[1] Phrenic nerve paralysis (PNP) was anecdotally reported in a mechanically ventilated asthmatic needing a chest drainage.[2] The most common causes of pediatric unilateral PNP are birth injury, local injury (both iatrogenic and accidental), and neck and mediastinal tumors.[3–5] A careful medical history is the mainstay to rule out most causes of PNP.[2–5] In our patient, the perinatal history was unremarkable. A chest X-ray obtained at 4 years of age immediately after a motor vehicle accident was normal. Since then, there was no report of cervical or chest injury. PNP as a result of iatrogenic injury from local surgical procedure, central vessel cannulation, or chest tube insertion was also excluded based on clinical history.

At the time of this episode, the flexible bronchoscopy carried out in our unit could have been a traumatic procedure, but it was accomplished with no difficulty under appropriate sedation. On the other hand, PNP has never been described among its complications. No radiologic signs of neck or mediastinal masses were found. Lastly, we tried to rule out a possible neuropathy or myopathy through the electrophysiological study of peripheral nerve and muscle. Unfortunately this was feasible only during follow-up, and thus the findings of a normal nerve conduction velocity and normal electromyography pattern cannot definitely rule out an acute and reversible form of neuromuscular disorder at the time of symptoms.[6]

To our knowledge, this is the first report of a PNP during SA without a recognizable cause. It is noteworthy that we can only speculate as to whether PNP and atelectasis were associated in this case. PNP per se, through the rib cage hypomotility and inefficient cough, can entail an atelectasis.[3] This seems unlikely in our patient, since neither PNP was apparent nor was a known cause of PNP detectable prior to when atelectasis had ensued. In fact, in a case of preexisting PNP, asthma exacerbation resulted in hyperinflation of the affected chest side rather than atelectatsis.[7] Thus, for our patient, one may speculate that

the atelectatic retraction of the lung may have caused a ''stretching injury'' of the phrenic nerve, resulting in a transient hemidiaphragm paralysis.

In conclusion, in our asthmatic child, none of the recognized causes of phrenic nerve paralysis could be implicated with certainty. Further data are needed to clarify whether transient phrenic nerve paralysis may be considered a potential complication of status asthmaticus. In the meantime, the precise nature of such an association remains a matter of discussion.

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