Case Report: Neonate With Stridor and Subcutaneous Emphysema as the Only Signs of Physical Abuse

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abstract

A stridulous, dysphonic cry with no external signs of trauma is a unique and unusual presenting sign for physical abuse. We report a previously healthy neonate with unremarkable birth history and medical history who presented with stridor and hypopharyngeal perforation due to physical abuse. This case highlights the need for further evaluation for traumatic injuries in the setting of unexplained new-onset stridor and consideration of physical abuse in the differential diagnosis.

Child physical abuse (PA) commonly occurs in the United States and is a diagnosis many providers find challenging. Because of the high risk of recurrence of abuse if the diagnosis is missed, diagnosing PA is crucial to prevent further physical (including death) and emotional harm.\(^1\)–\(^5\) The most recent reported incidence of PA is 6.5 per 1000 children (481 000 children) per year in the United States. The rates of children hospitalized with serious injuries due to PA with resultant death are highest in the first year of life.\(^6\)–\(^11\)

Hypopharyngeal perforation is extremely uncommon in children.\(^12\) A large case series of significant pediatric oropharyngeal trauma demonstrated no resultant pharyngeal perforation.\(^13\) A literature review of cases of hypopharyngeal perforation excluding known iatrogenic or external trauma causes demonstrated that 16 of 24 reported cases resulted from PA, and 3 were suspicious for PA.\(^12\)

We report a 28 day-old neonate with stridor due to right vocal cord paralysis, history of bloody oral secretions, subcutaneous emphysema, and pneumomediastinum but no external evidence of trauma or mouth/throat trauma. The injuries resulted from PA. This is the first reported case of this constellation of findings due to PA with a unique pathophysiologic mechanism.

CASE PRESENTATION

This previously healthy 28 day-old neonate presented to a local emergency department (ED) after her parents noted abrupt onset of noisy breathing and saw blood in her mouth when she awoke from a nap in her bassinet.

In the local ED, biphasic stridor and respiratory distress noted. Bloody oral secretions were present before suctioning by the outside ED, with no deep suctioning or other instrumentation of the airway performed. The patient was then transferred via Survival Flight to our PICU. Birth history was unremarkable with no history of a difficult extraction, stridor, or respiratory distress in the neonatal period. No respiratory distress reported before new-onset stridor. Formula intake and weight gain had been appropriate since birth.

Upon our physical examination, the patient demonstrated high-pitched inspiratory stridor, which was audible at rest and became louder during...
The patient was a neonate crying with heart rate of 190 beats per minute, respiratory rate of 45 per minute, and oxygen saturation of 98% on room air. The patient had a strong cry that was mildly dysphonic. There was no evidence of mouth or oropharyngeal trauma with no petechiae, bruising, or lacerations. Frenula were intact. There were no skin abrasions or bruising on the face, neck, or thoracic cavity. There was no palpable subcutaneous emphysema. Facial movements were symmetric and intact.

The pediatric otolaryngology team was consulted for further evaluation of stridor. Flexible laryngoscopy was performed at the bedside, which demonstrated right true vocal cord paralysis with the cord in the paramedian position. There was normal true vocal cord movement on the left. There were no mucosal abnormalities or bleeding visualized.

Plain film of neck and chest x-ray demonstrated air in the soft tissues of the right neck extending from the skull base into the mediastinum (Fig 1). Computed tomography (CT) of the neck and chest were performed to rule out a mediastinal mass as the source of vocal cord paralysis. The CT imaging demonstrated extensive gas dissecting through the soft tissue structures of the neck from the skull base to the level of the carina, and dissecting close to the esophagus and right mainstem bronchus (Fig 2). No clear source of dissecting gas was identified from the imaging.

Barium esophagram was performed and demonstrated evidence of a hypopharyngeal perforation with extravasation of contrast into the soft tissues in the area of the right hypopharyngeal-esophageal junction at the level of the thoracic inlet (Fig 3).

The patient was taken to the operating room for direct laryngoscopy and bronchoscopy with laryngeal electromyography (EMG) to further evaluate her right true vocal cord paralysis and hypopharyngeal perforation. Direct laryngoscopy findings were similar to flexible laryngoscopy findings, with no evidence of gross pharyngeal or hypopharyngeal trauma. Rigid bronchoscopy was unremarkable. Laryngeal EMG needle electrodes were placed into the right and left vocalis muscles, and simultaneous recordings were obtained. EMG demonstrated significantly decreased function on the right, and normal function on the left.

Head CT and brain magnetic resonance imaging showed no evidence of intracranial abnormality. Skeletal survey revealed no evidence of fractures or other osseous injury.

The Child Protection Team was consulted because of suspected PA, leading to the neonate’s unexplained hypopharyngeal perforation and unilateral vocal cord paralysis. Parents denied mouth or oropharyngeal trauma, and none was noted on examination. A report was then filed with Children’s Protective Services.

Subsequently, the patient’s father confessed to causing her injuries. During her naptime, just before the onset of stridor and noted bloody secretions, the father “gave her a solid jolt, causing her head to go back and forth forcefully.” Father was, subsequently, convicted of felony child abuse.

Our patient demonstrated full recovery of right vocal cord function at 2 months postonset. This was confirmed when she returned to clinic for flexible laryngoscopy with the findings of normal vocal cord mobility bilaterally and a normal cry without dysphonia.

**DISCUSSION**

Laryngotracheal (LT) injuries account for ~0.05% of trauma admissions in children. These injuries are, most commonly, related to blunt neck trauma. Although not prevalent, LT injuries are second only to intracranial injury as the most common cause of death among patients with head and neck trauma.14–18

The most common presenting sign for LT trauma is hoarseness.19,20 There are numerous other presenting symptoms/signs for head and neck trauma, including dysphagia, pain, dyspnea, hemoptysis, and signs of airway obstruction, such as stridor or tachypnea. Additional signs include drooling and cervical subcutaneous...
Barium swallow. This perforation was not visualized with flexible laryngoscopy because the perforation was likely small and, because the patient was awake, the muscles were contracting, further decreasing the likelihood of visualization. The perforation was also not detected on direct laryngoscopy, likely due to remucosalization over the days between presentation and evaluation in the operating room and the small size of the perforation. Remucosalization occurs over days to 1 to 2 weeks. This patient’s repeat esophagram 7 days after presentation did not reveal any hypopharyngeal leak.

The etiology of the vocal cord paralysis was presumably secondary to a neuropraxic injury from neck hyperextension/flexion. Neck hyperextension/flexion or blunt trauma (including thoracic trauma) are also possible etiologies for a hypopharyngeal perforation.\(^{21}\) The finding of unexplained hypopharyngeal perforation alone is strongly suggestive of PA. This case demonstrates that isolated, new-onset vocal cord paralysis in the setting of a previously healthy neonate may warrant further evaluation for PA.

The importance of a systematic diagnostic workup of new-onset stridor in an infant is illustrated in this case. New-onset stridor prompted plain film imaging of the neck, as well as an otolaryngology evaluation with bedside flexible laryngoscopy. Identification of the unilateral vocal cord paralysis as the cause of stridor then expedited further workup with direct laryngoscopy and bronchoscopy under anesthesia, as well as laryngeal EMG. Laryngeal EMG can provide useful data concerning muscle denervation and reinnervation, and serial EMG can be useful for monitoring recovery and establishing a reliable prognosis and treatment plan.\(^{22}\) Patients with a suspected neuropraxia as the etiology of their vocal cord paralysis, as was suspected in this case, have the highest rate of recovery. A recovery rate of 71\% at 2 years after onset has been demonstrated in this population.\(^{23}\)

Small pharyngeal or hypopharyngeal perforations resolve spontaneously with conservative measures, which include strict nothing-by-mouth status and initiation of nutrition via nasogastric tube passed under direct visualization or radiologic guidance. Perforations are reevaluated after a week with an esophagram to document resolution. Broad-spectrum intravenous antibiotics are also indicated in these patients. Rigid endoscopy performed in the operating room allows for accurate assessment of the perforation and survey for other mucosal injuries, as well as the removal of any foreign bodies.\(^{12}\) Surgical intervention may be warranted if the patient develops a retropharyngeal abscess, mediastinitis, or a mediastinal pseudocyst.

Early speech-language pathology involvement is important to rule out aspiration and provide modifications to optimize patient feeding because 23\% of infants with unilateral cord paralysis have feeding difficulty.\(^{23}\)

**CONCLUSIONS**

New-onset vocal cord paralysis in an infant with unclear etiology should prompt workup for related injuries and PA. Hypopharyngeal perforation or other upper aerodigestive tract perforation may coexist with traumatic vocal cord paralysis and is strongly suggestive of PA.

**ABBREVIATIONS**

CT: computed tomography
ED: emergency department
EMG: electromyography
LT: laryngotracheal
PA: physical abuse
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