Case Report

Management of child after traumatic epidural hematoma with pulmonary edema and arrhythmia

ABSTRACT

We present a case of severe neurogenic pulmonary edema and arrhythmia complicating management of a 7-year-old child with acute epidural hematoma and impending cerebral herniation. The underlying mechanisms for this are discussed as well as management of severe neurogenic pulmonary edema. We emphasize the need to recognize this rare complication early and institute prompt aggressive management.

Key words: Epidural hematoma; long QT; neurogenic pulmonary edema; pediatric closed head injuries

Introduction

Neurogenic pulmonary edema (NPE) is a potentially life-threatening condition characterized by accumulation of fluid in lung spaces and adventitia in association with intracranial pathology, typically intracranial hypertension. Described in association with cases of subarachnoid hemorrhage, supratentorial subdural hematoma, and posterior fossa hematoma,[1] the suddenness and apparent randomness of NPE presentation means that a high index of suspicion is required in order to diagnose it and institute prompt treatment. Most cases of NPE have been described in adults, some of whom may have had preexisting cardiopulmonary disease. We present a case of NPE in a previously healthy 7-year-old boy following a closed head injury with a large epidural hematoma and impending cerebral herniation.

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Case Presentation

A 7-year-old boy with no prior medical history presented 5 hours after a fall down five stairs at home. He complained of a headache immediately after falling but was lucid. When he was difficult to arouse from sleep 2 hours later, his mother drove him to the hospital. On admission, Glasgow Coma Scale was 3 with pupils initially fixed and dilated on the left, progressing to 6 mm fixed and dilated bilaterally with agonal breathing and copious frothy sputum. The patient was intubated on arrival with pink frothy fluid bubbling from the trachea. Administration of 0.9 normal saline 70 ml/kg and dopamine for significant hypotension (nadir systolic blood pressure 43 mmHg and diastolic blood pressure 34 mmHg) was instituted. Hypertonic saline was started for suspected intracranial hypertension. Portable chest X-ray

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showed severe diffuse hazy opacities throughout both lung fields, raising suspicion for NPE [Figure 1]. Computerized tomography showed a nondisplaced skull fracture of left parietal and squamous temporal bones, an underlying large left-sided epidural hematoma with 7 mm left-to-right midline shift, and early transtentorial and cerebellar tonsillar herniation [Figure 2a and b]. He was taken to the operating room (OR) less than one hour after arrival to the hospital, where he underwent left frontotemporoparietal craniotomy and evacuation of epidural hematoma.

Intraoperative course was complicated by copious amounts of pink, frothy fluid that occluded the endotracheal tube and required frequent suctioning. The patient was ventilated using Pressure Regulated Volume Control mode with tidal volume = 6 ml/kg, RR = 26 breaths/min, peak pressure = 36 cm H_20, PEEP = 8 cmH_20, FiO_2 = 1. Fentanyl-based anesthesia was supplemented with mannitol and furosemide. Fresh frozen plasma (total volume 300 ml) was administered for prothrombin time 17.7 sec (normal: 11.9–14.5 sec) and partial thromboplastin time 38.4 sec (normal: 22.5–38 sec). Case totals were: 700 ml 0.9 normal saline, urine output 660 ml, and estimated blood loss 100 ml. Intraoperative fluid balance was positive 240 ml over 3.5 hours. At the end of surgery, an attempt to transport ventilating with Ambu bag proved impossible because of fulminant NPE. Furthermore, the initial attempt to leave OR for PICU was delayed by a brief episode of unstable ventricular tachycardia that resolved after 2 minutes with chest compression, epinephrine, and resumption of ventilation via the ventilator at intraoperative settings. Dopamine and epinephrine infusions supported his blood pressure, and his heart rate was 110 beats per minute during this period. Arterial blood gas reflected difficulty with ventilation: pH 6.98, pCO_2 83.8 mm Hg, pO_2 481.4 mm Hg, bicarbonate 20 mmol/L, and $Sa0_2$ 99.9%. Serum sodium, potassium and calcium levels were within normal limits. In PICU, 12-lead EKG showed sinus tachycardia with prolonged QTc of 514 msec, which peaked after 24 hours at 573 msec (99th percentile QT_c is 460 msec for prepubescent children). Echocardiogram on postoperative day one showed normal cardiac function. QT_c interval normalized daily thereafter and was normal (433 msec) two weeks later. The patient did not have a family history of long QT syndrome or cardiac arrhythmias. After 12 days, the child was discharged to a rehabilitation center where he spent 2.5 weeks before going home. He regained his pre-injury neurologic function and returned to school with good grades but did have occasional migraine-type headaches.

Discussion

NPE is acute pulmonary edema occurring after a central neurologic insult. The pathophysiology is complex and still poorly understood. One theory involves a hemodynamic, or "blast injury" mechanism, in which the adrenergic response to sudden injury results in severe pulmonary vasoconstriction, resulting in increased pulmonary hydrostatic pressure and vascular permeability. A second involves an inflammatory mechanism resulting in increased pulmonary capillary permeability.[2] Chaari *et al*.[3] reported a case of severe head injury with NPE in whom transpulmonary thermodilution showed decreased stroke volume index and cardiac function index, with impairment of left ventricular function confirmed by echocardiogram. Meanwhile, extravascular lung water and pulmonary vascular permeability index were elevated. They concluded that NPE includes both cardiac dysfunction and lung injury.

In general, outcomes following surgical intervention for pediatric patients with epidural hematomas are good. One single center study of traumatic epidural hematomas in 39 children showed excellent outcome after surgical intervention with zero mortality, regardless of clinical status, cause of injury, or pupillary abnormalities.[4] However,

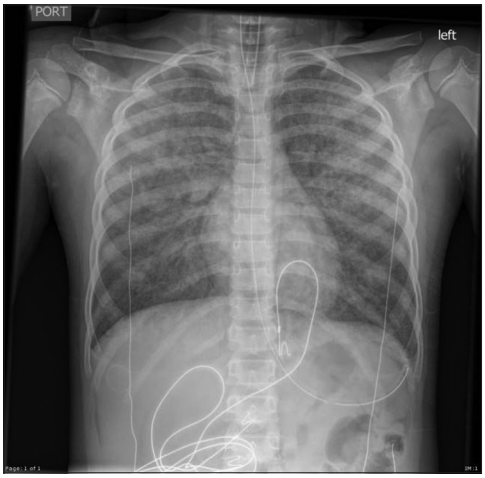

Figure 1: XR Chest single view: Diffuse fluffy infiltrates, immediately after intubation

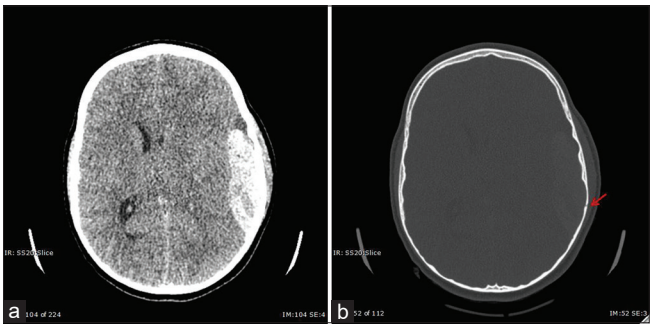

Figure 2: (a) Axial CT head, no IV contrast: Large left epidural hematoma with left to right shift. (b) Axial CT head: Parietal fracture

the association of NPE with epidural hematoma presents management challenges and significantly complicates the prospect of a good outcome. There are reports of pulmonary edema in children associated with subdural hematoma after abusive traumatic head injury[5] or in conjunction with Takotsubo cardiomyopathy after postoperative epidural hemorrhage.[6] QTc prolongation has been reported after acute cerebrovascular events in adults. Our case is a unique presentation of a large epidural hematoma after skull fracture complicated by NPE and ventricular arrhythmia associated with long QTc interval in a previously healthy child that normalized within weeks.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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