Rare disease

Non-cardiogenic pulmonary oedema following accidental electrocution in a toddler

Shweta Singh, Jhuma Sankar, Nandkishore Dubey

Department of Pediatrics, PGIMER, Dr RML Hospital, New Delhi, India

Correspondence to Dr Jhuma Sankar, jhumasankar@gmail.com

Summary

Non-cardiogenic pulmonary oedema due to electrocution is an underdiagnosed clinical entity. The authors report a toddler who presented with symptoms and signs of respiratory failure following accidental electrocution. His chest radiography showed bilateral diffuse infiltrates and cardiac involvement was ruled out by echocardiography. The child was managed symptomatically and discharged in a stable condition. The cause of pulmonary oedema in this child was thought to be neurogenic in origin as cardiac and pulmonary causes were ruled out. Non-cardiogenic pulmonary oedema is an uncommonly recognised entity which occurs with any neurological or non-neurological event stimulating the vasomotor centres. There are divergent theories to explain the cause for this rare phenomenon but none has been proved. The authors chose to report this case to highlight this uncommon complication of electrocution hitherto unreported in children.

BACKGROUND

Non-cardiogenic pulmonary oedema is a clinical condition characterised by hypoxemia, bilateral diffuse infiltrates on chest x-ray, no evidence of left ventricular dysfunction (LVD) (pulmonary capillary wedge pressure of ≤ 18 mm Hg) and a predisposing clinical condition.¹ The classical pathophysiological finding is the abnormal protein and fluid accumulation in the alveoli. Major causes of non-cardiogenic pulmonary oedema are acute respiratory distress syndrome, high-altitude pulmonary oedema and neurogenic pulmonary oedema.²

To the best of our knowledge, non-cardiogenic pulmonary oedema following electrical injury has not been previously reported in children. Here, we report a child with pulmonary oedema of probably neurogenic origin following accidental electrocution; to draw attention towards this rare form of complication.

CASE PRESENTATION

A 3-year-old boy presented to the emergency ward in an unconscious state following accidental electrocution at his home. At admission, he was unconscious and had severe respiratory distress. His heart rate was 154/min with feeble peripheral pulses, capillary refill time was 4 s with cool extremities and blood pressure (BP) was 78/54 mm Hg. His respiratory rate was 72/min; he had intercostal/subcostal retractions, nasal flaring and decreased air entry on both sides with coarse crepitations on auscultation. His SpO₂ was 61% on room air which increased to 74% on 100% oxygen. He was having tonic posturing with brisk deep tendon reflexes and bilateral extensor plantars. Cardiovascular and per abdominal examination was normal. Local examination of the site of injury revealed second degree burn injury involving the face and right shoulder.

INVESTIGATIONS

His chest x-ray revealed bilateral diffuse homogenous infiltrates with normal cardiac silhouette and blood gas estimation revealed severe hypoxemia (42 mm Hg). His ECG revealed non-specific ST changes. Qualitative Troponin-T assay with Troponin-T sensitive, Troponin-T Rapid Test kit (Cobas; Roche Diagnostics GmbH, Germany) was negative; creatine kinase-MB was normal (18 U/l) and echocardiography revealed normal cardiac function.

DIFFERENTIAL DIAGNOSIS

Pulmonary oedema of cardiac origin (ruled out by relevant investigations).

TREATMENT

He was intubated and ventilated (synchronised intermittent mandatory ventilation pressure mode peak inspiratory pressure=25, positive end-expiratory pressure=8, FiO₂ 100%, RR=25, Ti=0.5). Following ventilation his pinkish frothy endotracheal secretions decreased over the next 24 h and he was gradually weaned from ventilator and extubated after 56 h of admission. His local wound started healing with antiseptic dressing.

OUTCOME AND FOLLOW-UP

He was discharged after 6 days of hospital stay and on follow-up after 3 months of discharge is asymptomatic and doing well.

DISCUSSION

Electrical injury is a common and serious form of injury in children with an epidemiological data suggesting a prevalence of about 21–30% in developing and 3–5% in developed countries.^{3 4} The age group involved has a bimodal

peak: first peak in toddlers and second peak in adolescent age group with preponderance of males. The type of injury involved depends on various factors like the voltage of the current (high voltage arbitrarily taken as >1000 V and low voltage <1000 V), lightning injury and direct versus alternating current electric injury. Mortality is highest in the lightning strikes (17.6%) while it is 5.3% in high voltage and it is 2.8% in low voltage injuries and is least in electric arc injuries without passage of current through the patient (1.1%).⁵ Complications are most common in the high voltage group. The complications commonly studied are cardiac arrhythmias, neurologic sequelae and renal failure which occurs in up to 20% of cases but non-cardiogenic pulmonary oedema has not been reported in the paediatric age group.⁶

Our case presented with in a short duration of an accidental low voltage electrical injury with home appliances in an altered mental state with severe respiratory distress and expectoration of pinkish frothy secretions from endotracheal tube suggestive of pulmonary oedema. This was further corroborated by chest x-ray findings of bilateral diffuse involvement. Cardiac dysfunction was ruled out by the absence of significant ECG changes with normal cardiac enzymes and normal echocardiographic study. As there was no history suggestive of any previous illness like pneumonia or aspiration, sepsis- and aspiration-induced pulmonary oedema was also ruled out. In view of the rapidity of the onset of symptoms and altered state of consciousness, the possibility of neurogenic pulmonary oedema was considered.

Non-cardiogenic pulmonary oedema of neurogenic origin was first reported by Shanahan⁷ about a century back in a patient with epilepsy. As the name suggests, neurogenic pulmonary oedema is triggered by a number of neurological conditions such as head trauma, seizures, intracranial haemorrhage and intracranial surgery.⁸ The entity has scarce epidemiological data with divergent theories of its pathogenesis like severe sympathetic discharge (the 'catecholamine storm' and the 'blast theory'), temporary neural commotion or either a mechanical or electrophysiological disconnection of the central nervous system vasomotor centres leading to the destabilisation of the pulmonary autonomic nervous system or a hydrostatic mechanism.^{6-7 9}

According to the severe sympathetic discharge theory, the most important vasomotor centres for development of neurogenic pulmonary oedema; the A1 and A5 groups of neurons, nuclei of the solitary tract, the area postrema, the medial reticulated nucleus and the dorsal motor vagus nucleus in the medulla oblongata gets over activated causing enhanced secretion of catecholamines from peripheral sympathetic nerve endings, which leads to peripheral vasoconstriction, an increase in systemic vascular resistance and subsequently to an increase in systemic BP together with the augmentation of central blood volume and a reduction in the compliance of the LV. These changes are followed by the constriction of the pulmonary veins, an increase in pulmonary capillary hydrostatic pressure, damage to the alveolar wall and leakage of fluid into the interstitium and intra-alveolar space and haemorrhage resulting in the

typical picture of neurogenic pulmonary oedema.^{9 10} The other school of thought is that it is not the increased lung capillary permeability but it is the increased pulmonary hydrostatic pressure which causes neurogenic pulmonary oedema.¹¹ On autopsy, the lungs with pulmonary oedema grossly show an increase in weight with dark red appearance and histologically demonstrate diffuse oedema of alveoli and septae accompanied by detachment of epithelial cells and unremarkable haemorrhage in alveoli as well as focal hyaline membrane formation.¹²

The management of non-cardiogenic pulmonary oedema consists of supportive care and adequate oxygenation in the form of either non-invasive continuous positive airway pressure or mechanical ventilation. The condition has a high risk of mortality (7%) if left unrecognised and untreated and therefore must be kept in mind while managing a patient with electrocution.⁹

Learning points

- ▶ Neurogenic pulmonary oedema is an uncommon complication which may occur following accidental electrocution.
- ▶ Management is mainly supportive.
- ▶ Early recognition and management are key determinants of outcome/clinical recovery.
- ▶ Parents should be educated time and again about the importance of prevention of injuries of any kind as these are best prevented.

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Competing interests None.

Patient consent Obtained.

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