

An Uncommon Consequence: Facial Palsy and Temporal Bone Fracture Post-electrical Injury

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How to cite this article:

Priti Sathe, Shilpa Aroskar. An Uncommon Consequence: Facial Palsy and Temporal Bone Fracture Post-electrical Injury. Indian J Trauma Emerg Pediatr. 2024;16(1-2):19-21.

ABSTRACT

An electrical injury can cause multisystem involvement depending on the type of electrical exposure and the duration of contact. Burns, cardiovascular, renal and central nervous systems involvement are common manifestations post electrical injury. Both immediate and delayed effects of electrocution are known to occur. The central and peripheral nervous system both can be involved in an electrocution injury. Focal neurologic deficits are an uncommon central nervous system manifestation following electrocution. We describe an uncommon consequence of left-sided lower motor neuron facial palsy with a temporal bone fracture in a female child following an electrical injury.

Keywords: Electrocution; Child; Facial palsy.

INTRODUCTION

Children are susceptible to electrical injuries due to their curious nature and often due to a lack of adequate prevention measures. Burns are the major cause of morbidity and mortality due to electrical injuries.¹ Cardiovascular, central nervous system and renal manifestations are common post-

electrocution.^{1,2} Focal neurologic deficits are a rare manifestation of electrical injuries in children.² Here we report a case of a 12-year-old female who sustained an electrical injury followed by left ear hemotympanum and left lower motor neuron facial palsy.

CASE DETAILS

A 12-year-old female was brought to the pediatric emergency room with an alleged history of unwitnessed electrocution on her house terrace while trying to hang her clothes for drying. Unfortunately, the metallic rod was in contact with an overhanging electrical wire. Following this, the child sustained electrical burns on the hands and feet with a left ear bleed and was rushed to our emergency room. On initial examination, she was conscious, oriented and hemodynamically stable. She had an entry wound on the palmar aspect of both hands and exit wounds on the feet with active bleeding from the left ear. There was no other systemic involvement on admission.

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Received on: 10.12.2024

Accepted on: 18.01.2024



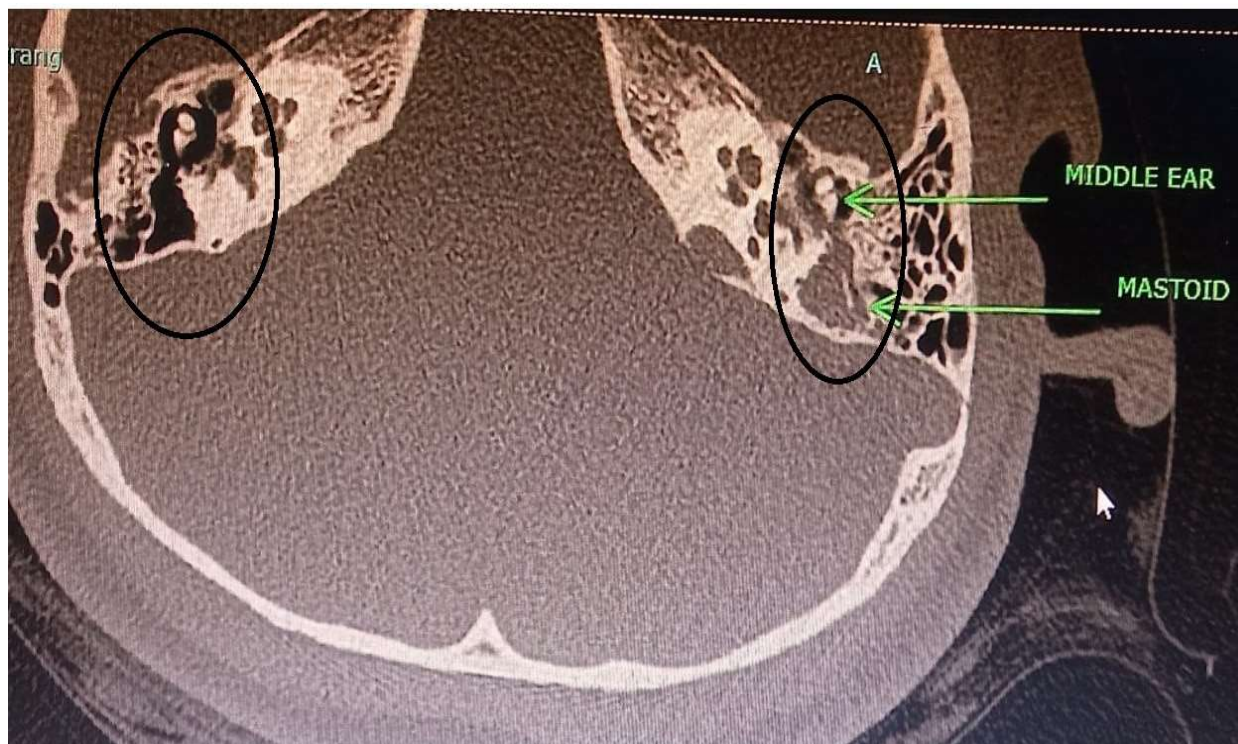


Fig. 1: High-resolution computed tomography of temporal bone showing the collection of fluid (blood) in the left middle ear for comparison right ear is also shown in the image



Fig. 2: Face photograph showing left-sided facial palsy (angle of mouth deviated towards right)

The child was admitted to the pediatric intensive care (PICU) for monitoring. Her initial investigations of renal function tests, coagulation profile and creatinine kinase were within normal limits. A plastic surgery reference was sought for the electrical burns. Wound debridement and dressing was done. Vitals were monitored including continuous electrocardiograph and no arrhythmias

were detected during the PICU stay. Otoroscopic examination revealed a clot in the left external auditory canal and hemotympanum. The ear bleed was managed conservatively with ear packing under the guidance of an otorhinolaryngologist. She was shifted to the ward after ensuring vital stability for 48 hours.

On day 4 of the hospital stay, the child developed left-sided lower motor neuron facial palsy. With left ear bleeding at the onset, the possibility of facial nerve involvement along its middle ear course was thought to be the aetiology. A high-resolution computed tomography (HRCT) temporal bone was undertaken which showed fractures of the left tympanic, petrous, and mastoid parts of temporal bones with opacification of the left middle ear cavity suggestive of hemorrhagic effusion. A computed tomography (CT) Brain was done concomitantly which showed few hemorrhagic contusions in the right frontal lobe with an undisplaced fracture of the left parietal bone. However, the child did not develop any other neurological symptoms apart from facial nerve palsy and was managed conservatively. The possible reasons for the facial nerve palsy were suspected to be nerve oedema due to an adjacent fracture or the hemotympanum causing nerve compression. She was given oral

steroid treatment (prednisolone 1mg/kg/day) with adequate eye care. A plan for middle ear decompression (myringotomy) was made, if symptoms did not improve, but eventually not required. Serial audiograms done were suggestive of left-sided severe conductive and moderate sensorineural hearing loss attributed to the clots, hemotympanum and possible inner ear damage. On follow-up after 2 months, the child showed complete resolution of facial palsy.

DISCUSSION

Children are a major group susceptible to electrical injuries because of their inquisitive nature. Low-voltage electricity, commonly used for household purposes, is the most common type of electric injury in children.¹ Electrical injuries can present with cardiac, renal and central nervous system symptoms. The central, peripheral and autonomic nervous systems can be involved in electrical injuries but nerve palsies are less common among neurological manifestations.²

The possible theories for neurological symptoms post electrocution include 1. direct electrical damage to the nerves, 2. thermal damage due to the dissipative heat produced as a result of electrical current, 3. secondary trauma post electrocution causing nerve injuries, 4. electroporation causing dysfunction of cellular ionic flux.² In our patient, the possible mechanisms were nerve compression due to hemorrhagic effusion in the middle ear or nerve oedema due to the adjacent fractures and proximity of the facial nerve.

The common causes of facial nerve palsy in children are post-infectious, malignancy, trauma, inflammatory, neurological, congenital, autoimmune and metabolic conditions. Electrical injury causing facial nerve palsy is uncommon in children.³ An injury similar to our patient has been reported in a 3-year-old child post unwitnessed electrical injury who developed right ear hemotympanum and ipsilateral LMN facial palsy.³ Unilateral facial nerve palsy has been reported in adults secondary to electrical injuries after day 4 of the injury probably due to demyelinating effect

of the electrical current.⁴ Similarly, facial nerve palsy and ipsilateral sensorineural hearing loss was reported in a 20-year-old adult post-electrical injury which was managed with oral steroids.⁵

The audiovestibular system is vulnerable to electrocution with tympanic membrane rupture, basilar skull fracture, cerebrospinal fluid otorrhea, hemotympanum and permanent deafness being the common consequences.⁶ The injury sustained by our patient described in this report is quite unusual with facial palsy as a consequence.

CONCLUSION

The intent of this case report is to underline the importance for the physicians to recognize possible facial nerve palsy in an electrical injury.

Competing interests/ Conflict of Interests: Nil

Funding/Financial Support: This research received no specific grant from any funding agency in the public, commercial or not-for-profit sectors.

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