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A Case of Multiple Cranial Nerves Palsy Post Electrocution

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ABSTRACT

Multiple cranial neuropathies are uncommon but not rare. Localization also depends on the etiology. Electrocution can cause neuropraxia, axonal damage, and necrosis of soft tissue and bones. We are presenting a rare case of post electrocution injury secondary infection of the head leading to the right side all cranial nerves palsy. Our patient suffered an electrocution injury. Post electrocution injury, he developed right sided multiple cranial nerve palsy. CT head was suggestive of skull base osteomyelitis. MRI brain images showed the right cavernous sinus involvement, soft tissue enhancement at the eight sphenoid sinus, right mastoiditis, soft tissue enhancement in the right infra-temporal fossa and sphenoid wings. *Escherashia Coli* and *Candida* were isolated from purulent ear discharge. This could be secondary infection. This is a very rare case where a patient developed all right-sided Lower Motor Neuron type cranial nerve palsies due to infection following electrocution. However, other etiology cannot be ruled out.

KEY WORDS: Electrocution injury, Multiple cranial neuropathies, Cavernous Sinus Thrombosis, *Escherashia coli, Candida albicans*

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Background: Multiple cranial neuropathies are uncommon but not rare. Most common causes are tumor, vascular disease, trauma, infection autoimmune disorders like Guillain-Barré syndrome and Miller Fisher syndrome.(1) Localization also depends on the etiology. Most common site is cavernous sinus, clivus/base of skull lesion and subarachnoid space involvement. Few systemic disease like Diabetes Mellitus type 2, Sjogrens syndrome, Wegener's Granulomatosis and HIV infection, make the individual prone to multiple cranial never palsy directly due to secondary infections.(2) We are presenting a rare case of post electrocution injury, secondary infection of head leading to right side all cranial nerves palsy.

Case presentation: 53 year male, an old case of Diabetes Mellitus type 2 on regular oral hypoglycemic drugs for last ten years with good glycemic control, suffered electrocution injury with an entry wound on the right hand and exit wound on at the right ala of nose. Two days after the electrocution injury, he developed purulent ear discharge in the right ear associted with moderate to severe headache. It was followed by diplopia, progressive diminution of vision and difficulty in opening the right eye. He also developed absence of sensations on the right side of the face, right sided asymmetry of face, mouth deviated towards the left side, loss of naso-labial folds on the right side and coughing while eating and neck weakness while turning towards left shoulder.

On examination he was not able to smell from the right side of the nostril which was suggestive of olfactory nerve palsy. Right sided optic nerve involvement leading to decreased visual activity from right eye. He had complete right sided unilateral ophthalmoplegia with ptosis due to LMN type oculomotor, trochlear and abducens cranial nerves palsy. He had absence of sensation on the right side of the face due to Involvement of V1 and V2 divisions of trigeminal nerve. Asymmetry of face with mouth deviated towards the left side, loss of naso-labial folds on the right side because of right LMN Facial palsy. Rinne's test showed air conduction more than bone conduction in right ear and Weber's test was lateralized to left side which was suggestive of right sided sensorineural hearing loss. Right sided soft palate was sagging and absent

gag reflex is suggestive of glossopharyngeal and vagus nerve palsy. Hypoglossal nerve palsy leads to weakness in turning of head towards left shoulder. While shrugging of shoulder it was reduced in right side as compare to left due to Spinal Accessory nerve palsy.

His hemogram was normal. Fasting and postprandial blood sugars were 128 and 118 mg/dL respectively. Cerebrospinal Fluid (CSF) study showed 50 mg/dl of protein and sugar was 86 with random blood sugar was 145 mg/dL. No organism was seen in CSF staining with Gram and Potassium Hydroxide (KOH) mount. CSF culture was sterile. NCCT head images were suggestive of skull base osteomyelitis. MRI brain images showed the right cavernous sinus is widened showing enhancement of soft tissue component on contrast MRI, soft tissue enhancement seen at the sphenoid sinus, clivus/base of skull, fluid in mastoid air cells suggestive of mastoiditis, soft tissue enhancement in the infra-temporal fossa and sphenoid wings. *Escherashia coli* have been isolated from purulent ear discharge culture. Staining of pus was suggestive of *Candida albicans*. He was managed with injectable antibiotics covering gram positive and negative bacteria (Meropenem and Teicoplanin) and anti-fungal (Amphotericine) for a period of 4 weeks as per sensitivity pattern of pus cultures. Repeat imaging was done in view of no improvement. Because of extensive tissue necrosis surgical drainage of sphenoidal sinus or debridement was done. Neurological deficit did not improve. However, this patient had significant improvement in headache.

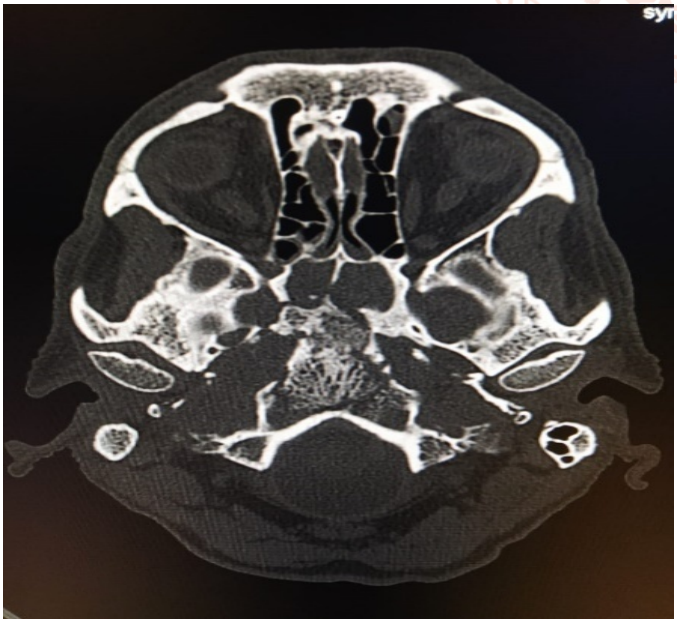

Image 1: NCCT head suggestive of skull base osteomyelitis.

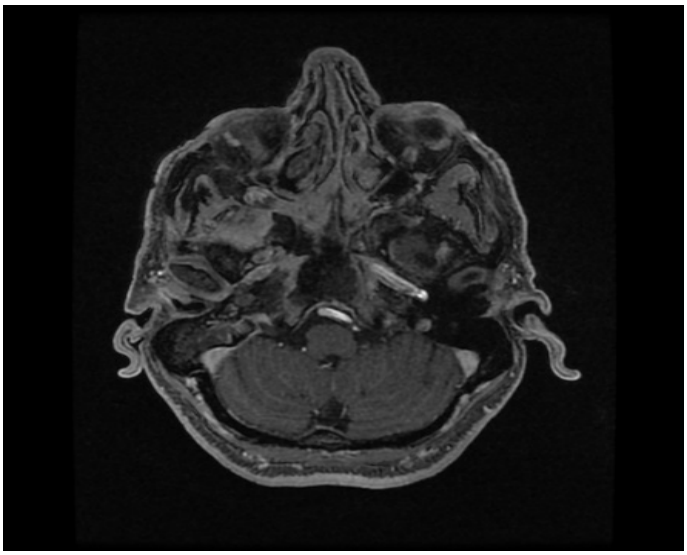

Image 2: T1W MRI showing soft tissue enhancement in infra-temporal fossa and sphenoid wings.

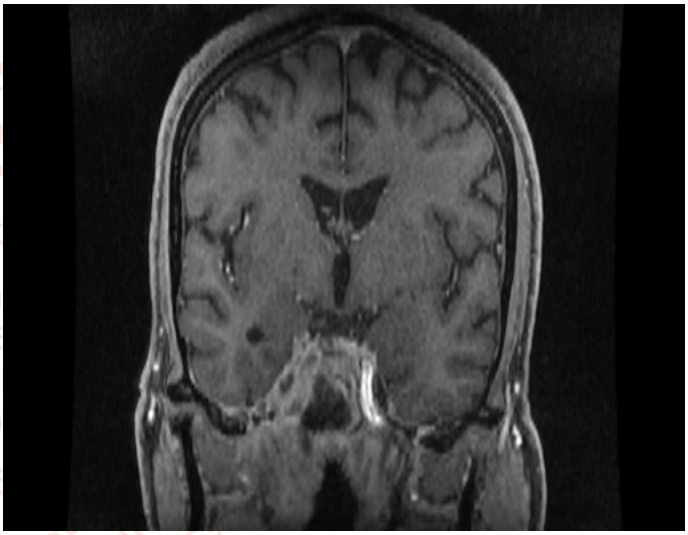

Image 3: Contrast T1W MRI showing the right Cavernous sinus is widened and enhancement of soft tissue component.

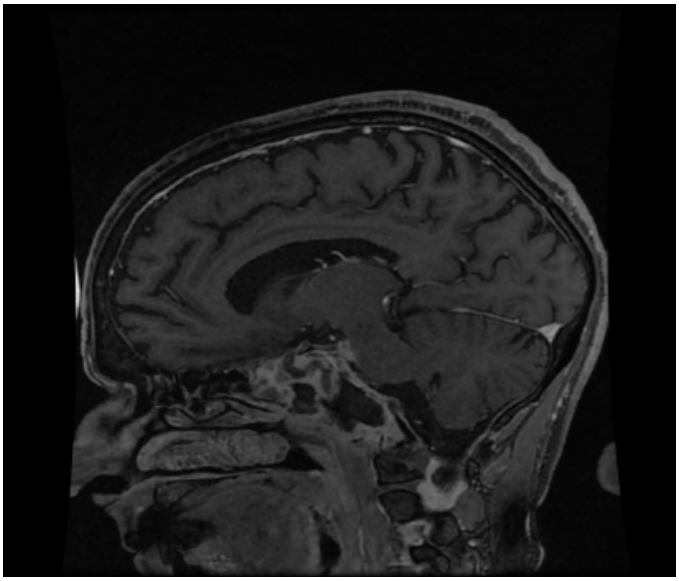

Image 4: Contrast T1W MRI showing soft tissue enhancement seen at sphenoid sinus, clivus/base of skull.

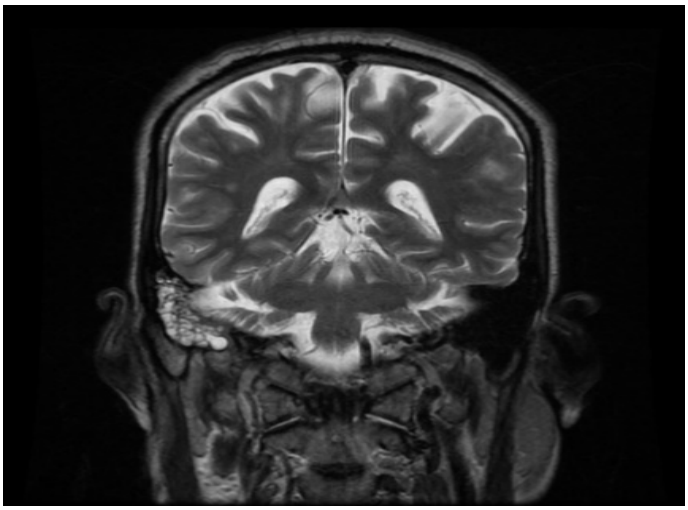

Image 5: T2W MRI showing fluid in mastoid air cells suggestive of mastoiditis.

Discussion: Localization and causes of multiple cranial neuropathies are very diverse. It reflects involvement in cavernous sinus lesions, clivus-skull base lesion, brain stem and meningeal involvement.[1] The cavernous sinus is the most frequent site of involvement, particularly by tumors and trauma. Brainstem involvement was dominated by vascular causes. Clivus-skull base is most commonly due to tumors and subarachnoid space is due to meningeal infection. Diabetes itself is the leading cause of multiple cranial nerves palsy. It most commonly involves facial and oculomotor cranial nerves.

In this case report, individual was an old diabetic patient who had an electrocution injury leading to wound at face and secondary intracranial infection. His infection progressed to mastoiditis, sinusitis, skull base osteomyelitis, and finally cavernous sinus thrombosis leading to all cranial nerve palsies. Oculomotor, Trochlear, Trigeminal and Abducens cranial nerves are located within the dural sleeve of cavernous sinus. Infections of the face, including the nose, ear, orbits, tonsils, and soft palate can also spread to the cavernous sinus. Vestibulocochlear palsy was caused due to mastoiditis, infra-temporal fossa and sphenoid wings involvement. His lower cranial nerves were involved due to clivus/skull base osteomyelitis.

Most common pathogens causing such malignant infection of cavernous sinus thrombosis are bacteria like Methicillin Resistant Staphylococcus Aureus (MRSA), Streptococcus and rarely gram negative-bacteria, anaerobes and fungi like Aspergillus and Mucormycosis.[3] However, our patient's pus showed *Escherashia coli* and Cadidia. Both these organisms were not known for such a fultminant disease with so extensive necrosis. Due to extensive damage of soft tissue and skull base infection he had persistent deficits. As only the right sided cranial nerves are involved it could also be considered that it had occurred because of the late complication of electrocution. Electrocution can cause neuropraxia, axonal damage, thermal injury to perineural tissue and necrosis of soft tissue and bones, vascular injury, and histological or electrophysiological changes.[4] If it would be due to pure infection then some of the left sided cranial nerves should have some involvement which was not there in our case.

Conclusion: Multiple LMN type cranial nerve palsies can develop due to electrocution. Henceforth, clinicians should rule out all other etiologies in any case presenting with multiple cranial nerve palsy. In our case we found that secondary infection due to poor glycemic control in post electrocution wound at face. We conclude that his multiple cranial nerve palsy is due to electrocution.

Abbreviation:

Cerebrospinal Fluid (CSF),

Potassium Hydroxide (KOH),

Lower Motor Neuron (LMN),

Methicillin Resistant Staphylococcus Aureus (MRSA)

Declarations

1. Funding: No funds were received from any government or private organisation.
2. Conflict of interest: The authors of this study have no conflict of interests to disclose
3. Ethics approval: This study contains retrospective data and therefore does not need a formal approval by the local Ethical Committee.
4. Availability of data and material: From medical records of hospital.
5. Authors' contributions: All authors have read and approved the manuscript, and ensure that this is the case. KA was treating when patient, RS was neurologist and wrote the article and did the research necessary for its production, AK was resident medicine and elaboration of the case description.

References:

[1] James R K. Multiple cranial nerve palsies: analysis of 979 cases. Arch Neurol. 2005 Nov;62(11):1714-7.

[2] Khadilkar S V, Harish A H, Prasad P B, Jaggi S, Muzumdar G, B. K. Smruti et al. Adult male with multiple cranial nerve palsies. Ann Indian Acad Neurol. 2012 Apr-Jun; 15(2): 96–100.

[3] Weerasinghe D and Lueck C J. Septic Cavernous Sinus Thrombosis. Case Report and Review of the Literature. Neuroophthalmology. 2016 Dec; 40(6): 263–276.

[4] Kim H M, Yeong A K, Kim J S, Lim S H, Hong B Y. Neurological Complication After Low-Voltage Electric Injury. A Case Report. Ann Rehabil Med. 2014 Apr; 38(2): 277–281.