Electric shock injury causing Guillain-Barre like syndrome-A case report

MARIAN JUDE VIJAY A
Department of Neurology,
MADRAS MEDICAL COLLEGE AND GOVERNMENT GENERAL HOSPITAL

Abstract:
Electrical injury can cause serious multi-organ involvement leading to death or mild symptoms which can be transient. The neurological manifestations are due to involvement of brain, spinal cord and peripheral nervous system. The symptoms can occur immediate to electrical injury or may be delayed and progressive. The injury could be due to heating of tissues which is in immediate contact or due to electricity itself which can cause injury remote to the area of contact. We present a case of 32year old female who had accidental contact with a live wire. Immediately after the event she had benumbed sensation involving the right upper limb which was in contact followed by glove and stocking type of benumbed sensation. Two days later she developed motor weakness initially involving lower limbs both proximal and distal which progressed slowly to a bed ridden state over a period of one month with minimal upper limbs involvement. On examination she had flaccid weakness with generalized areflexia. There was Glove and stocking sensory loss for all modalities. VII and IX,X cranial nerves were mildly affected bilaterally. The weakness after the initial progression for one month, was static for 3 to 5 days followed by gradual improvement to near normal strength around the second month of electrical injury. NCS showed predominant demyelinating polyradiculoneuropathy with minimal axonal changes also. She was treated with injection methylprednisolone 1gram for 5 days followed by oral prednisolone. We are reporting this case because of the rare presentation of Guillain-Barre like syndrome following electric shock.

Keyword: "Guillain-Barre syndrome","Electric shock injury"

Introduction:
Neurological manifestations of electrical injury can be categorized based on the voltage as High voltage injuries and Low voltage injuries. The factor that governs the damage to the nervous system is not simply the voltage but also the time duration the victim was in contact and the amount of current or amperage.

Mechanisms:
1. Thermal injury- due to heating of tissues causing coagulation necrosis
2. Direct effects of electricity itself

An Initiative of The Tamil Nadu Dr. M.G.R. Medical University
University Journal of Medicine and Medical Sciences
The neurological manifestations of electrical injury can be classified based on the onset.

Immediate effect Due to direct heating of nervous system (thermal injury)
Delayed effect 1 day to 6 weeks (1 week on average)

Probably due to the effect of electricity itself

Late effect After many years Due to anterior horn cell damage

The neurological manifestations depend on which part of nervous system is affected

The neurological manifestations following electrical injury can cause damage to central or peripheral nervous system depending on the path with which it traverses through human body. Electricity travels in the body along the shortest path to ground whatever the part of body which is in contact with the ground. If a person is in contact with one terminal of electricity but not with the ground like wearing a foot wear made of non conductor of electricity or standing over a wooden stand electrical injury is unlikely. Its passage through humans can often be determined by identifying entry and exit burn wounds. When its path involves the nervous system, neurological damage is likely among survivors. When electric current passes through the head

It can cause immediate unconsciousness, confusion, disorientation, seizures, headache and transient focal deficits. Among the survivors, recovery generally occurs within a few days. Residual memory and other cognitive disturbances are common. when the electric path involves the blood vessels, it causes coagulation necrosis of part of the vascular wall causing intramural thrombosis or aneurysmal distention and rupture. When the path of the current involves the spinal cord

Transverse myelopathy may occur immediately or within 7 days or may progress for several days. The disorder eventually stabilizes, after which partial or full recovery occurs. The current pathway is across the cervical cord from one arm to the other resulting in muscle atrophy in the arms accompanied by an upper motor neuron deficit in the legs associated with sensory and sphincter disturbances.

Segmental muscle atrophy may occur within a few days or weeks of electrical injury of the spinal cord. Whether this relates to focal neuronal damage or has an ischemic basis is uncertain. Occasional reports have suggested the occurrence of a progressive disorder simulating amyotrophic lateral sclerosis after an electrical injury. It can cause mononeuropathy in the region of an electrical burn probably related to thermal injury causing coagulation necrosis or polyneuropathies remote to the site of electrical injury where pathophysiology is not well understood. We are reporting a 32 year old female patient who developed polyradiculoneuropathy resembling Guillain-Barre syndrome after contact with a live wire

Case report:
32 year old female sustained electric shock from domestic cause while operating a live wire. She was in contact with electricity for a very few seconds and it is an alternating current of 240 volts.

Immediately following electric shock she noticed benumbed sensation involving right upper limb which lasted for 5 minutes. After 5 minutes that symptom disappeared but she developed another sensory symptom in the form of benumbed sensation involving both hand and feet areas. She also had burning
sensation over the same area, difficulty in appreciating the textures of things which ever in contact with her hands and felt ground like cotton wool.

Two days after electrical injury she developed weakness of lower limbs in the form of difficulty in stepping up and getting up from sitting/squatting position and difficulty in insinuating toes into the foot wear. The motor weakness progressed over a period of one month involving upper limbs, trunk and neck muscles leading to a bedridden state. She had transient mild difficulty in swallowing liquids during the period of maximum motor weakness. The weakness after the initial progression over a period of one month, was static for 3 to 5 days, then followed by gradual improvement to near normal strength around the second month of electrical injury.

No postural giddiness, bladder/bowel involvement or sweating abnormalities. No amnesia of the event or other HMF abnormalities. No history of diarrhoea, respiratory infection in the recent past.

On general examination she was not anemic or jaundiced. Her vitals, cardiovascular, respiratory and abdominal examination were normal. On local examination there was no evidence for entry & exit wounds following electric shock. Higher mental assessment was normal including memory and there was no amnesia for the event. Her Mini Mental Status Examination was 30/30. Cranial nerve examination showed minimal bifacial LMN motor weakness and mild IX,X nerve involvement with diminished gag reflex. Motor examination showed no wasting but decreased tone in all 4 limbs. Her power examination during maximum disability according to MRC grading was 4 in upper limbs both proximally and distally and in the lower limbs the proximal power was 2 and distal power was 4. She had generalized areflexia with bilateral plantar flexor response. Sensory system examination showed a graded sensory loss to all modalities in glove & stocking pattern.

On follow up after 2 months her upper limb power improved to 4+ and lower limb power to 4. No cranial nerve deficits and sensory system also showed improvement.

Complete blood count, renal function test, liver function test were normal. Elisa test for HIV, serum VDRL, serum B12 level, Thyroid function test and neuro imaging (MRI) of brain and spinal cord were normal. Serum protein electrophoresis was also normal. IGg anti ganglioside antibodies to GM1 ,GM2,GM3,GD1a,GD1b,GQ1a,G Q1b were negative. Cerebrospinal fluid(CSF) analysis showed slightly elevated protein of 60mgs/dl with acellularity and other parameters including sugar were normal.

**Sensory nerve conduction study**

Both upper and lower sensory nerve action potential(SNAP) – not obtained

Nerve conduction study shows- prolonged distal latency, slowing of conduction velocity, conduction block with mild decrease in amplitude. Conduction slowing which is minimal in comparison to gross prolongation of distal latency and this is suggestive of distal demyelination. F-waves were not obtained. Sensory nerves were not stimulatable.

She was treated with injection methylprednisolone 1gram for 5days owing to her worsening motor weakness followed by oral prednisolone.

**Discussion:**

Our patient had Guillain-Barre like syndrome following electric shock with predominant...
demyelinating polyradiculoneuropathy with some axonal changes also. She had sensory symptoms, followed by motor symptoms and cranial nerve involvement. The illness progressed for one month, then static for 3 to 5 days followed by gradual improvement to near normal strength around the second month of electrical injury. Patient was managed initially with symptomatic treatment. As the patient had progressive motor weakness and NCS showed predominant demyelination with axonal changes she was treated with pulse methyl prednisolone -1gm IV for 5 days followed by oral prednisolone.

Thermal damage typically occurs due to nerve involvement within the zone of local injury where the body part is in contact with electricity. Peripheral nerve dysfunction may occur outside the zone of local injury which is probably due to the effects of electricity itself. When electricity passes through a solid conductor, heat is generated in proportion to the current strength, the duration of the current flow, and the resistance of the conductor and this accounts for the burns and local coagulative tissue injury often seen at the points of electrical entry and exit. When electrical contact is brief, thermal injury and burns may be minimal but non-thermal injury may still cause damage by direct electrical effects causing electro conformational changes in membrane proteins. Nerve axons being the least resistance conductor of electricity causing free flow of electricity just like electric cables without production of much heat are thus especially susceptible to this type of non-thermal damage, which may disrupt peripheral nerve axons in isolation and in the absence of significant damage to surrounding tissue. Thermal damage causes coagulation necrosis and the recovery is poor. Electricity causes electro conformational changes in peripheral nerve leading to dysfunction and recovery is better. Thus the probable cause of peripheral nerve injury in our case is due to the effects of electricity itself. Since our patient was exposed to a low voltage current there is no evidence of thermal mediated local injury. The pathophysiology may be related by direct electrical effects causing electro conformational changes in nerve membrane proteins, causing damage to myelin and axons.

**Conclusions:**

We are presenting this case for its rare presentation of Guillain-Barre like syndrome following electric shock. The pathogenesis of Guillain-Barre like syndrome in our case is probably related to electricity itself rather than thermal injury. The pathophysiology probably due to electro conformational changes in nerve membrane proteins causing damage to myelin and axons. The good prognosis in our case can be attributed to non-thermal mediated electrical injury.

**References:**


Motor nerve conduction study

<table>
<thead>
<tr>
<th>Nerve</th>
<th>Distal Latency</th>
<th>Amplitude Distal/Proximal</th>
<th>Conduction velocity</th>
<th>F-wave</th>
</tr>
</thead>
<tbody>
<tr>
<td>Median</td>
<td>Right 22.08</td>
<td>4.3/1.8</td>
<td>44.28</td>
<td>Absent</td>
</tr>
<tr>
<td></td>
<td>Left 19.58</td>
<td>3.7/2.7</td>
<td>27.43</td>
<td>Absent</td>
</tr>
</tbody>
</table>

Ulnar
- Right 10.83 3.6/3.3 37.74 Absent
- Left 11.67 4.1/1.8 44.28 Absent

Peroneal
- Right 18.90 1.1/0.5 37.91 Absent
- Left Not stimulable Absent

Tibial
- Right 17.50 2.1/1.1 34.55 Absent
- Left 14.60 2.2/1.1 41.48 Absent

Sensory nerve conduction study

Both upper and lower sensory nerve action potential (SNAP) – not obtained


