CASE REPORT

Guillain-Barre syndrome-like illness in association with electrical shock injury

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SUMMARY

A 19-year-old man presented 4 weeks after an electrical shock injury with gradual onset limb weakness, altered sensation in the peripheries and respiratory difficulty. There was immediate tingling of the fingers following the electrical injury that persisted. He subsequently had transient facial weakness responsive to oral steroids before the development of further limb symptoms. On admission the clinical picture and investigation findings, including neurophysiology, cerebrospinal fluid examination and MRI were consistent with a Guillain-Barre syndrome. He was managed with a course of intravenous immunoglobulin and extensive physiotherapy and occupational therapy. He made an initial modest improvement but worsened again over the subsequent 4–6 weeks. Further investigation identified no ongoing active disease and he is left with a significant residual deficit.

500 V after the cable was prematurely tested. The contact with the cable lasted seconds and there was no loss of consciousness or amnesia regarding the event. Immediately following the electrical shock he reported of a tingling in the tips of the digits on both hands which persisted. One week later he developed a unilateral facial weakness on the left side and was treated with steroids for a presumed Bell's palsy. During the course of the steroid treatment the facial weakness improved. Two weeks prior to admission he described progressive weakness and ascending paraesthesia in all four limbs and face. There was no history of recent infection or vaccination.

On admission he had bilateral facial weakness. The remainder of the cranial nerves were intact. There was flaccidity of the limbs with distal weakness and areflexia. Sensation to pin prick and light touch was reduced to the midforearm and leg. Vibration and proprioception were maintained. Forced vital capacity was reduced to 1.8 L/min.

Examination of the palms identified two small black macules and a hard papule on the left that had appeared following the electrical injury (figure 1). There was a linear lesion on the right anterior shin that had also developed postelectrical shock (figure 2). It was felt that these represented points of entry and exit of the electrical injury, given that the cable was held in the hands and that he was kneeling on the ground when the injury occurred.

BACKGROUND

The association of acute nerve injury following electrical shock is well recognised. Less common, however, is the development of subacute symptoms. We reported a case of a male patient who developed symptoms compatible with Guillain-Barre syndrome (GBS) following electrical shock injury.

CASE PRESENTATION

A 19-year-old right-handed male electrician presented with weakness and sensory loss. One month earlier he had sustained an electrical injury at work. He described holding an electrical cable with both hands while kneeling on the ground when he received an electrical shock of approximately

INVESTIGATIONS

Nerve conduction studies were consistent with an acute inflammatory demyelinating polyneuropathy. Routine bloods including full blood count, plasma

Figure 1 Black macules and papule to hand.

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Figure 2 Linear lesion to shin, presumed 'exit point'.

viscosity, renal liver bone profile, thyroid function, vitamin B₁₂ and folate and C reactive protein were normal. Serology for borrelia, campylobacter antibodies and an HIV screen were negative. Antiganglioside antibodies were not identified. MRI of the brain and cervical spine was normal. Spinal fluid analysis demonstrated an elevation in cerebrospinal fluid (CSF) protein of 1430 mg/L (150–450). All other indices were normal (cell count 0) and oligoclonal bands were not detected.

DIFFERENTIAL DIAGNOSIS

The initial clinical picture combined with the neurophysiology indicated an acute demyelinating polyneuropathy. This is in keeping with the Guillain-Barre syndrome developing subsequent to a significant electrical shock injury.

The other differentials considered were neuropathies developing in the context of sero conversion of HIV, neuroborreliosis, vitamin deficiency, diabetes, hypothyroidism and hyperthyroidism and drugs.

TREATMENT

The patient was managed with a course of intravenous immunoglobulin. His forced vital capacity was monitored regularly and he was reviewed by anaesthetists in anticipation of a possible admission to intensive care if his respiratory function declined. Extensive physiotherapy and occupational therapy was the mainstay of the treatment.

OUTCOME AND FOLLOW-UP

This patient initially made a slow but steady recovery, with respiratory function normalising and strength and stamina improving.

Unfortunately following discharge he appeared to worsen over the subsequent 4–6 weeks with significant deterioration in strength. There was minimal improvement in symptoms following a further course of immunoglobulins. It was postulated that this could represent chronic demyelinating peripheral polyradiculoneuropathy but again there was no improvement with subsequent steroids and immunosuppression. Repeat spinal fluid analysis was entirely normal. Repeat neurophysiology was performed 6 months after his initial presentation. These demonstrated the presence of a predominantly motor demyelinating neuropathy with some axonal loss. We also went on to record brain stem responses (normal) and somato-sensory evoked potentials (difficult to interpret due to lack of response from peripheries). Sural nerve biopsy was carried out and demonstrated an axonal injury and to a lesser extent demyelination. There was no evidence of ongoing inflammation or demyelination. These results suggested a quiescent process.

At present this patient is able to ambulate with bilateral assistance, but continues to have significant disability. Prognosis for any further improvement in the future is poor, given the lack of meaningful progression following relapse.

DISCUSSION

The interesting question in this case is whether the electrical injury he sustained induced the apparent GBS he presented with or whether it is entirely unrelated. Equally the question is raised as to whether this is a true case of GBS, with atypical features or whether the electrical insult resulted in a GBS-like presentation.

GBS was first described in 1916 and is a common cause of neuromuscular paralysis, with several subtypes, of which acute demyelinating polyneuropathy is one. The reported incidence within Europe is 1.2–1.9 cases/100 000 (0.6–4 cases/100 000 worldwide).¹ It is commonly a postinfectious syndrome, most frequently associated with *Campylobacter jejuni* and studies have demonstrated that it is often secondary to an infection-induced immune response resulting in damage to peripheral nerves.² Antiganglioside antibodies have been implicated in this process. There are also reports of GBS following vaccinations, operations and stressful events.

In this case many of the clinical features for GBS are present; progressive weakness, areflexia, bilateral facial weakness, high CSF protein and typical neurophysiology features. This combined with a failure to find any alternative diagnosis on further examination strongly supports the initial diagnosis of GBS. However, against this is the subsequent worsening of his symptoms and lack of continued improvement. GBS generally has a good prognosis if treated appropriately. However, it remains a fatal disease in 4% of cases and only 60% of sufferers recover full strength in 1 year.³ Literature describes approximately 14% of cases experiencing significant disability³ which is consistent with our case. A diagnosis of chronic inflammatory demyelinating polyneuropathy was considered following the worsening of his symptoms, but the lack of evidence of ongoing inflammation and poor response to further treatment do not support this.

Electrical insults are documented as causing various peripheral and central neurological complications that can present acutely following the injury, but may also be of delayed onset. However, there is little in the literature regarding GBS-like illness following an electrical injury.

Kalita *et al*⁴ documents a 30-year-old woman who made contact with an electrical wire. She was found initially unconscious and sustained amnesia to the event and 35% burns to the body. A lower limb weakness with sensory loss developed but resolved without intervention over the subsequent 4 months. In another, Ratnayake *et al*⁵ describe a 23-year-old man who made contact with a 20 kV cable. Similar to the previous case report he had no recollection of the event and sustained significant burns. Examination identified a paresis of both lower limbs and the left arm. Areflexia was also evident. Nerve conduction studies demonstrated no motor response or sensory potentials. A gradual improvement with rehabilitation was made in this case. Thaventhiran *et al*⁶ documents a 24-year-old man who made deliberate contact with a 200 000 V supply, resulting in 20% full-thickness burns to the body. He experienced a flaccid tetraparesis, areflexia and loss of sensation distally. Neurophysiology identified a complete denervation in the limbs. There are also reports of those who have sustained similar neurological sequelae following a lightning strike. For example a 19-year-old man was found unresponsive and with 20% burns after being struck by lightning. A flaccid quadraparesis and

areflexia were subsequently identified and with extensive rehabilitation he has made some recovery. One particular publication by Therodouro *et al*⁷ bears a more striking resemblance to our case. They describe a 51-year-old man who was struck by side lightning and initially found unresponsive and pulseless. Entry and exit wounds were evident as burns. He was amnesic to the event itself. Thirteen days into admission he developed a unilateral facial paralysis followed by the development of a weakness to the previously unaffected side. He then presented with progressive weakness and tingling in the limbs and was diagnosed with GBS with examination demonstrating quadraparesis and depressed reflexes. He was treated with immunoglobulin therapy and some recovery was noted.

Although these reports bear some resemblance to the clinical presentation of our case there are several significant differences.

Learning points

- ▶ Guillain-Barre syndrome (GBS) may not always be secondary to an infective process.
- ▶ It should be considered following electrical injury if the clinical history and findings support the diagnosis.
- ▶ Electrical shock injury may trigger an inflammatory response similar to GBS.

In all of the reported cases the electrical insult was of a higher voltage and there was amnesia to the events as well as gross soft tissue injury secondary to the electrical insult itself. There are no widely available case reports documenting similar symptoms with minimal soft tissue injury and full awareness of the event such as in our case.

Contributors This case report has been written and researched by MLA. MLA received guidance and advice from JO.

Competing interests None.

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