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CHARCOT'S PARALYSIS- A RARE PRESENTATION OF ELECTROCUTION

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ABSTRACT

In the spectrum of post-electrocution episodes, Charcot's paralysis is a mysterious esoteric entity. Since it is an unusual form of shock, it is usually found in a small percentage of victims. Ritenour et al describe several ways in which electric shock affects the human body. A series of normal tests and a negative radiological image seem to validate the diagnosis, while a strict observation relieves symptoms. In this case report, we present a young guy who developed a monoparesis of the right lower extremity that resolved spontaneously after electrocution, was thoroughly probed, and was retrospectively identified as a case of Charcot paralysis. In a nut shell, clinical rarity such as this requires a high level of suspicion to begin with continuous monitoring and symptomatic treatment to treat the disease.

KEYWORDS: - *Charcot's paralysis, electrocution, monoparesis, transient phenomenon*

INTRODUCTION

Electrocution by lightning or electric shock is a common threat that doctors deal with on a daily basis. Skin alterations to an entire cardiorespiratory shutdown have been observed as a result of such an occurrence. Charcot's paralysis as a result of lightning or electrocution is a rare occurrence, with just about 50 cases reported in the literature. Although the ailment progresses as a self-resolving entity, the vasomotor and sensorimotor abnormalities associated needs meticulous monitoring [1]. We offer this case to highlight the possibility of an exclusionary diagnosis in individuals who present with brief self-resolving monoparesis after primary or secondary electrocution.

CASE PRESENTATION

A 34-year-old patient with a known history of coronary heart disease diagnosed for 6 months, presented to our casualty with an alleged lower right-hand electroshock in his agricultural field due to an uninsulated electric wire following which he developed an acute paralysis of the right lower limb. The patient had no intact sensory or motor abilities in the lower right limb. There was no history of any loss of consciousness, convulsions, amnesia or other post-electrocution focal neurological deficits. The patient had no history of syncope, vomiting, dizziness or evidence of a neurogenic bladder. The patient did not experience palpitations, irregular heartbeat or shortness of breath. The patient presented to us after 18 hours of incident and was dealt with as a case of acute onset monoparesis under evaluation. On presentation, the patient's vitals recorded a pulse rate of 78 per minute and a blood pressure of 140/90 mm Hg. Systemic tests was normal. A detailed neurological examination was conducted. The patient had a complete GCS of 15 with bilateral regular, reactive and round pupils. The bulk was normal in all the limbs. Reflexes were attenuated in the right lower limb with a bilateral flexor plantar response. The tone was increased in the right lower limb with a motor power count of 0/5. With the patient being clinically stable, blood investigations were done which suggested all the parameters in the normal range. Given a possible cause of spinal origin by mechanisms of tertiary lesions, x-rays of the whole body were obtained that did not suggest any fracture, dislocation, or bone insult of any kind. X-rays of the lower right limb.

Since there were no x-ray abnormalities, any further radiological investigations were deferred. With all investigations being normal, the patient was treated with fluid

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resuscitation and maintained for observation. A follow up MRI spine was planned. At 6 hours after presentation to the centre totalling a duration of 24 hours, the post-electrocution patient had a reversal of all possible symptoms. The patient's neurological examination was completely normal at the end of the 24 hours and a diagnosis of Charcot's paralysis was made (diagnosis of exclusion). After obtaining the required clearance, the patient was discharged with an advice for follow-up after 2 weeks for review. Unfortunately patient was lost to follow up.

DISCUSSION

Charcot's paralysis, commonly referred to as lighting paralysis due to its close association with lightning strikes, may also occur as a result of electrical damage from other causes. This kind of temporary paralytic phenomenon, characterized by a self-resolving condition affecting primarily the limbs, was first described and theorized by Charcot in 1890 in his presentation. Other cases such as Boudin (1854), Duchenne (1861) and Nothnagel (1861) sporadically described this unusual clinical condition in literature. Vasomotor and sensory abnormalities associated with such occurrence are typically found to revert within 1 hour to a maximum of 24 hours within 1 hour, making it a self-limiting disorder requiring a high level of suspicion for diagnosis and is considered an exclusion diagnosis [2].

A voltage difference between two adjacent non-isoelectric potential surfaces causes a current spark, which may be lightning or an electrical charge. The resulting load intensity is determined by the difference in voltage, the amount of load shunted and the resistance to the current flow as well as the duration of contact with the source, which gradually increases the amount of damage that discharges into the body [2]. Ritenour et al. has proposed direct impact, contact injury, side flash, electric current on the ground and explosion injuries as the five methods how an electric charge can affect the human body [3].

The pathophysiology of this strange event is still a mystery. Functional disruption of the sensorimotor and vasomotor abilities of the peripheral nerves as a consequence of a high voltage current flow is the most widely accepted explanation for the sequence of symptoms. Most of the disturbances, according to Panse (1955), are a direct result of excessive vasoconstriction in response to a high voltage current influx. In a patient with Charcot's paralysis of the forearm, Keller (1917) observed that the current entered through the hand and exited around the elbow, indicating that the myelin was not in the circuit of electric discharge. Currens (1945) and Critchley (1935) proposed that sensory alterations are caused by peripheral vascular changes and that spontaneous resolution is proportional to the restoration of the same.

The passage of charge through the human body, known as electrocution, is thought to have a multisystem effect, with a wide range of clinical disclosures. The mucocutaneous system includes six different forms of burns: feathering, linear, punctate, thermal, contact, and flash burns [4]. It is pathognomonic for keraunographic markings or Lichtenberg figures. Cardiopulmonary system is the most commonly damaged of all body systems, with direct current induced cardiac arrest being the leading cause of death. However, the heart's intrinsic electrical rhythm generator causes depolarization and resumption of the pulse, which Tausig et al emphasized as an excellent survival response to advanced cardiopulmonary resuscitation and ventilator support for people who suffer cardiac arrest following electrocution [2, 5].

Acute renal failure with myoglobinuria occurs due to an increase in CK and CK-MB due to tetanic muscle contractions. It causes toxic quantities of myoglobin to be released into the circulation thereby shocking the kidneys [6]. This usually responds favourably to constant hydration and diuretic treatment. Fortunately, the patient in our situation did not suffer from such a misfortune. The neurological system is the most important system since it has the widest spectrum of symptoms, ranging from moderate tingling to full neurovascular collapse. Cherrington et al. have provided a detailed account of all the many forms of injuries that a patient could sustain as a result of such an event [7]. In 1995, he described 13 individuals who had identical symptoms and healed spontaneously with no long-term consequences.

Charcot's paralysis is invariably a post-mortem diagnosis that is frequently caused by exclusion. It falls under class 1 of the Cherrington classification, which affects the lower limbs more than the upper limbs abruptly and transiently, but can also manifest as hemiparesis, as Rahmani et al demonstrated in their work [8]. Given the way the patient presents, a diagnostic conundrum often arises, and the patient may be subjected to a battery of tests that are medically insignificant in this circumstance. In such cases, the physician must maintain a high level of suspicion so that this unusual entity can be identified early and wastage of medical resources can be avoided. If the patient does not improve or worsens after the second day, neurological imaging may be done, demonstrating a functional cause for the symptoms.

Since the patient's biochemical were all normal in our case, a decision was reached to pursue higher scans for further evaluation after an observatory period. The patient was maintained under close supervision and given enough fluid resuscitation, which is the mainstay of such a situation as a vasomotor shock is thought to be the most prevalent pathophysiology. The patient's symptoms resolved on their own within 6 hours of admission and there was full remission of his symptoms. The diagnosis of Charcot's paralysis was verified retrospectively after the electrocution. By the end of 12 hours, the patient had fully recovered with no residual neurological abnormalities, and he was discharged with advice to return in two weeks for subsequent review.

CONCLUSIONS

Charcot's paralysis is a well-known neurological condition that occurs in people who have been injured by an electric charge of some sort. Since it is self-limiting, temporary, and self-resolving with no permanent deficiencies, it should not be misinterpreted as a functional masquerader or a clinical malingerer. A vigilant attitude toward this benign ailment will not only avoid unnecessary clinical testing and

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therapeutic interventions, but also the terrifying panic of an undetected condition in the patient's and their well-wishers' minds. A high level of awareness would suffice in determining the patient's diagnosis and would go a long way in providing holistic care to a patient who requires only observation.

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