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CASE REPORT

Postural Orthostatic Tachycardia Syndrome. A Rare Complication Following Electrical Injury

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We report on two previously healthy patients who developed severe form of postural orthostatic tachycardia syndrome (POTS) following an electric injury. Both the patients developed symptoms of orthostatic intolerance in the form of dizziness, fatigue, lightheadedness, and palpitations, weeks to months after electrical injury. Orthostatic intolerance produced considerable functional impairment in these patients. Early recognition of POTS when it occurs after an electrical injury allows for prompt evaluation and management to occur. (PACE 2009; e1–e3)

postural tachycardia syndrome, electrocution

Introduction

The widespread use of electricity and the application of electrically powered machinery have led to an increase in the number of electrical injuries. Electrical injuries now comprise approximately 3–7% of all burn injuries each year.[1–3] The first recorded death caused by electrical current from an artificial source was reported in 1879, when a carpenter in Lyons, France, inadvertently contacted a 250 V AC generator.[4] The first U.S. fatality occurred in 1881, when a local inebriate, Samuel W. Smith, passed out onto a similar generator in front of a crowd in Buffalo, New York. The apparent painlessness of his death impressed the crowd and there after electrocution became considered a more "humane" mode of execution. In 1890 William Kemmeler was the first man to be put to death in New York State's electric chair.[3] In adults, most electrical injuries occur at work places and constitute fourth leading cause of work-related injuries.[3] An electrical injury has numerous neurological consequences including transient loss of consciousness, transient paralysis, injury to peripheral and cranial nerves, and autonomic peripheral neuropathy.[5–7] There has been no report of postural orthostatic tachycardia following electrical injury. We report two cases of severe postural tachycardia syndrome (POTS) that occurred in previously healthy patients following an electrical injury.

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Case 1

A 62-year-old man suffered an electrocution injury at his place of employment when he placed a faulty electrical cord into an electrical outlet. He had no prior history of syncope, near syncope, dizziness or Meniere's disease. Upon placing the defective cord into the outlet there was a sudden large arc of electricity and he was thrown to the floor unconscious. Paramedical personnel found him pale, hypotensive, and bradycardic and transported him to a local hospital. There he was admitted and treated for significant burn injuries to his left hand. A 12-lead electrocardiogram (EKG) showed only minor ST-T wave changes in lead II, III, and aVF. Serum troponins and electrolytes were normal as was a complete blood count (CBC). Several days after the incident, the patient began to experience significant fatigue, palpitations, weakness, and lightheadedness. Over the next several months, he started to experience periods of dyspnea on exertion, cognitive impairment, and aphasia. By the approximately 5 months after the injury, he was functionally disabled and was referred to our center for evaluation. On physical examination, he appeared pale and anxious. His physical exam was remarkable for a 35 beat per minute increase in pulse rate going from sitting to standing; along with a 30-mmHg fall in systolic blood pressure down to a level of 70/45 mmHg. Repeat EKG, echocardiogram, and magnetic resonance imaging (MRI) of the brain was normal as were CBC and serum electrolytes. Neurologic function was normal. Adrenal function testing was normal. Tilt table testing demonstrated a 30 beat per minute increase in the heart

rate within the first 5 minutes of upright tilt followed by a 20 mmHg fall in blood pressure, reproducing his symptoms of lightheadedness, palpitations, and dyspnea. Therapy with fludrocortisone, midodrine, and methylphenidate was ineffective. His orthostatic symptoms have improved with duloxetine and pyridostigmine; however, his aphasia and cognitive impairment have continued over a 2-year follow-up period.

Case 2

A 43-year-old man was working in a company that manufactured acoustic speakers when he touched a circuit that he thought the electrical current to have been shut off. He experienced tetany and initially was unable to break contact with the current, until the co-workers shut off the power. Paramedical personal found him unconscious and he was transported to the local hospital. He was treated for burn injuries to both hands and was later released. Several weeks after the injury he began to experience symptoms of lightheadedness, palpitations, and dyspnea on exertion. Symptoms occurred only while upright and were relieved by recumbency. Evaluation at that time included an EKG , MRI of the brain, CBC, and electrolytes, all of which were normal. A persentine stress test with sestamibi imaging was normal. His symptoms began to worsen and he began to suffer from periods of near syncope, diaphoresis, and cognitive impairment, after which he was referred to our center for evaluation. On physical examination, he displayed a sitting blood pressure of 110/70 mmHg, with a pulse of 86 beats per minute. Upon standing, his blood pressure was 100/50 mmHg and his pulse was 120 beats per minute. During head upright tilt table testing, he displayed a 45 beat per minute increase in heart rate in the first 5 minutes of the upright posture, associated with a 20 mmHg fall in systolic blood pressure, with reproduction of his symptoms. Prior to the electrical injury, he had no history of syncope, near syncope, dizziness, or Meniere's disease. He was tried on a number of different medications to control his symptoms, ultimately achieving the best response with a combination of midodrine, pyridostigmine, and propranolol. Although improved on medications, he nonetheless continued to experience functional and cognitive impairment over a 3-year follow-up period.

Discussion

Electrical injuries continue to be the cause for many fatalities, considerable morbidity, and almost 500 deaths in the United States each year.[8,9] Most of these deaths (300) occur at work places. The most important determinant of the electricity-induced injury is the amount of current flowing through the body of the victim. To understand the mechanism of injury, Kouwenhoven[9] describes factors like voltage, resistance (R), amperage (I), and type of current, current pathway, and duration of contact. Current is described as the flow of electrons. The force or pressure that drives the current is called voltage. Electrical injuries have been classified either as high voltage (>1,000) or low voltage (<1,000). In high transmission line it usually exceeds 100,000 V and in distribution lines it is reduced to 7,000–8,000. Before delivery to home, it is further reduced to 110 V in USA.[10]

Cooper[11] described four types of injuries resulting from electricity. These include direct contact with electrical source that leads to passage of current through the body. The current flow through the resistance of tissue produces heat, which is the cause of further injuries. At the cellular level the electrical current traveling through the body can lead to disruption by electroporation (perforation in the plasma membranes between the intracellular and extracellular membranes) and electroconfirmational protein denaturation (changes in conformation of proteins and subsequent denaturation).[12] Both electroporation and electroconfirmational protein denaturation may be responsible for delayed rather than immediate cell death. Some neurological symptoms may begin months after the electrical injury. Other types of electrical injuries include arc injuries, blunt injuries, and thermoacoustic effect. As electrical energy flows through the body it can be transformed into other types of energy such as thermal, mechanical, and acoustic energy, which can cause considerable tissue damage.

There are multiple neurological manifestations of electrical injuries; however, effects on autonomic nervous system are not well understood. Jobst et al.[13] described a syndrome of hypertension, vasovagal syncope, and reflex sympathetic dystrophy following lighting injury. Two cases of severe POTS have been reported following lightning injury from our center.[14] However, till date there has been no report of electrocution-induced POTS.

POTS is a form of autonomic nervous system disturbance that often occurs due to the body's inability to maintain adequate amounts of peripheral vasoconstriction in the presence of orthostatic stress, thereby resulting in an abnormal degree of pooling of blood into the lower half of the body while upright. This shift is then compensated for by a reflex-mediated increase in the rate and myocardial contractility, which although adequate enough to prevent syncope is not completely sufficient to meet the body's circulatory

requirements. Patients suffering from POTS experience palpitations, severe fatigue and dyspnea on exertion, lightheadedness, and exercise intolerance. Severe forms of the disorder can be very disabling, resulting in a state of functional impairment not unlike that seen in congestive heart failure. Treatment usually includes physical reconditioning to strengthen the skeletal muscle pump's ability to increase peripheral venous return to the heart, as well as pharmacotherapy to attempt to increase peripheral vasoconstriction. Details on the diagnosis and management of POTS can be found elsewhere.[15] Both of the patients reported here were previously healthy and developed symptoms of POTS only after suffering an electrical injury. The mechanism by which electrical injury can cause POTS is unknown. Early recognition of POTS when it occurs after an electrical injury allows for prompt evaluation and management to occur. Further studies will be necessary to better define the mechanism that cause POTS following electrical injury.

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