

# New Onset Postural Tachycardia Syndrome Following Lightning Injury

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*Lightning injuries are one of the top three causes of death from environmental causes. Survivors of lightning injuries may be left with injuries, which may affect either the somatic or autonomic nervous systems. We report on two previously healthy patients who developed severe forms of postural tachycardia syndrome following a lightning injury, and review the literature on the subject. (PACE 2007; 30:1036–1038)*

**autonomic dysfunction, lightning injury, postural tachycardia syndrome**

## Introduction

Lightning is a major source of both injury and death, and is one of the top three causes of death due to environmental causes (the others are floods and extreme temperature). Lightning strikes approximately 400 people per year and causes an average of 66 deaths per year in the United States alone.<sup>1,2</sup> Approximately 10% of all individuals struck by lightning will die, and the rest frequently suffer from a variety of injuries, often chronic in nature.<sup>2,3</sup> We report two cases of severe postural tachycardia syndrome (POTS) that occurred in previously healthy patients following a lightning injury.

## Case Reports

### Case 1

An 18-year-old woman was standing on an athletic field with her college soccer team when a bolt of lightning struck the ground adjacent to them. She had no history of syncope, near syncope, dizziness, or Meniere's disease. The patient was thrown several feet, while one of her teammates a few feet away was killed instantly. When paramedical personnel arrived, she was bradycardic, hypotensive, and apneic. She was intubated, given IV fluids, and transported to a local hospital. By the time of her arrival to the emergency department, she had regained consciousness, and was normotensive and in normal sinus rhythm. Computed tomography (CT) of the head was normal; a 12-lead electrocardiogram (EKG) showed only nonspecific ST-T changes in II, III, and AVF. An echocardiogram was normal, serum troponin levels were normal, as were serum electrolytes and complete blood count (CBC). She was admitted to the intensive care unit for observation, extubated,

and discharged 2 days later. Several days after discharge, the patient began to experience significant fatigue and weakness as well as cognitive impairment. Over the next several months, she began to develop progressive dyspnea on exertion and memory loss. Tests for Lyme disease were negative. Six months after the accident, she was functionally disabled and was referred to our center for evaluation. On presentation, she appeared pale and anxious. Her physical examination was remarkable for a 40 beats/min increase in pulse rate going from sitting to standing, along with a 20 mmHg fall in systolic blood pressure. Repeat echocardiogram and EKG were normal, as were repeat serum electrolytes and CBC. On baseline 70% tilt table testing, she went from an initial heart rate of 78 to 155 beats/min within 7 minutes of upright posture (consistent with a diagnosis of POTS). The tilt protocol employed is described elsewhere.<sup>4</sup> Blood pressure fell from an initial value of 110/80 to 85/60 mmHg over the same period. At this point, she developed significant bluish discoloration of the lower extremities (acral cyanosis). The patient reported that the test reproduced her feelings of lightheadedness, near syncope, and dyspnea. She was later treated with a combination of fluorocortisone, midodrine, and fluoxetine. Although improved, she continued to suffer from exercise limitations and cognitive impairment over a 2-year follow-up period.

### Case 2

A 25-year-old woman personal trainer was speaking on a land line telephone inside a gym. She had no history of syncope or near syncope, dizziness, or Meniere's disease. Lightning struck the building she was in and traveled down the phone line, causing the phone to melt in her hand. She was thrown several feet to the floor. The patient was then taken to the emergency department, where she complained of headache and tinnitus. An EKG and CT scan of the brain were unremarkable, and the patient was discharged. Several weeks after the event, she began to experience episodes of lightheadedness, palpitations,

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syncope, and near syncope. She had significant complaints of dyspnea on exertion and exercise intolerance. She also began to experience symptoms of cognitive impairment with impaired memory and concentration. A stress echocardiogram was normal. She underwent a comprehensive cardiac electrophysiologic study after a Holter monitor recorded heart rates of 150–160 beats/min, but it showed no abnormalities. Tests for Lyme disease were negative. Magnetic resonance imaging of the brain was normal as was an EKG. On physical examination, she appeared tired and anxious. Physical examination was remarkable for a 40 beats/min postural increase in heart rate, accompanied by a 12 mmHg fall in systolic blood pressure. During 70-degree head upright tilt table testing (the protocol is described elsewhere),<sup>4</sup> her heart rate increased from 79 to 182 beats/min. Her blood pressure stayed at 99/58 to 102/69 mmHg. At 7-minutes upright, she became syncopal with hypotension and accompanying convulsive activity. She was ultimately treated with a combination of 200 mg of modafinil twice daily and erythropoietin 20,000 IU subcutaneously each week. Although improved, she continues to suffer significant functional impairment with cognitive problems and exercise intolerance, over a 2-year follow-up period.

### Discussion

Lightning strikes can cause a variety of injuries resulting in blunt head injuries as well as cardiac and neurologic damage. While lightning strikes may be lethal, four to five times as many people suffer nonlethal injuries.<sup>1</sup> It is estimated that as many as 76% of lightning strike victims will suffer long-term problems such as peripheral neuropathy and cognitive impairment.<sup>2,3</sup>

Lightning occurs as a result of a cold high-pressure front that moves over a moist, warm low-pressure zone.<sup>5</sup> The friction caused by the moving air particles within a cloud results in ionization and other energy alteration. Lightning occurs when a “leader stroke” from a cloud makes a slow jagged, irregular path to the earth (which has the opposite charge).<sup>2,5</sup> Lightning is neither truly direct nor alternating current. It is a unidirectional, massive, current impulse with several strokes back to the cloud.<sup>5</sup> A bolt of lightning can exceed 200 million volts, an amount far greater than the highest tension electrical wire.<sup>1</sup> A lightning strike is not always fatal as the current flow occurs for only a millisecond or less, and much of the current often flows over the body rather than through it. Because of the brief period of contact, lightning usually does not cause burns, but may cause arborescent red lines (“lightning disguises”) that indicate the path of lightning over the surface of the body.<sup>2</sup> The current flow through the resistance of

tissue produces heat, which is the cause of many of the resultant injuries.<sup>6</sup> However, some neurologic symptoms may begin months after the lightning strike occurs. This is thought to be caused by current induced perforations in the plasma membrane between the intracellular and extracellular areas, resulting in a disturbance in cellular metabolism that would lead to delayed rather than immediate cell death (a process called “electroporation”).<sup>3</sup>

Lightning injury may occur in five different ways:<sup>5</sup> The first type is caused by a direct strike (35% of injuries); the second involves a side flash (or splash) from an adjacent object (30% of injuries); the third is a contact injury (when a person touches an object that is part of a lightning current circuits: 1%–2% of injuries); the fourth involves ground current effects as current spreads through the ground from the site of the strike (30% of injuries); last are injuries caused by blunt trauma, occurring because of both current induced muscle contractions as well as the lightning’s ability to cause superheating of the air, resulting in its sudden expansion followed by an implosion as the air rapidly cools (30% of injuries). Although this explosion and implosion of air rarely leads to burns, the concussive effect can cause trauma to organs such as the heart, liver, brain, and spleen.<sup>6</sup>

Injuries to individuals using telephones (landlines) from lightning are relatively common. The telephone serves as a conduit for the charge to enter or escape from the building (as well as pass through the person). There appears to be no inherent lightning danger in cellular phones.<sup>2,5</sup>

Although the immediate effects of lightning injury are familiar to most physicians (such as ventricular fibrillation, stroke, and respiratory onset), many are unaware of the devastating long term effects of lightning injury.<sup>3</sup> In addition to the effects on the somatic nervous system, resulting in chronic pain and paralysis and central nervous system effects that produce cognitive impairment, the autonomic nervous system may also be affected after lightning injury.<sup>7</sup> However, the autonomic nervous system dysfunction that occurs as a consequence of lightning injury has not been as fully reported (or understood).

Jobst et al. describe autonomic nervous system dysfunctions secondary to lightning injuries resulting in hypertension, vasovagal syncope, and a complex regional pain syndrome.<sup>8</sup> The authors however do not describe POTS. Indeed no literature exists regarding the development of chronic POTS following lightning injury.

POTS is a type of autonomic disturbance, which is characterized by the body’s inability to maintain adequate peripheral vasoconstriction in the face of orthostatic stress, thereby allowing excessive pooling of blood to occur in the lower half

of the body when upright. The body compensates by a reflex mediated increase in heart rate and myocardial contractibility, which, while sufficient to prevent syncope, is often not adequate to meet the circulatory needs of the body.<sup>9</sup> Patients experience palpitations, dyspnea on exertion, exercise intolerance, fatigue, and lightheadedness. Severe forms of POTS can be quite disabling, creating a state of functional impairment not dissimilar to that seen in chronic congestive heart failure or chronic obstructive pulmonary disease. Therapy for POTS involves reconditioning to strengthen the skeletal muscle pump's ability to augment venous return as well as pharmacotherapy to restore peripheral vasoconstriction and postural normotension. De-

tails on the diagnosis and management of POTS can be found elsewhere.<sup>9,10</sup>

Both patients reported here were previously healthy but developed severe forms of POTS after suffering lightning injuries. The mechanism by which lightning injury causes POTS is not fully understood. Prompt recognition of POTS when it occurs following lightning injury is critical to proper evaluation and management. Further observations will help clarify the incidence of POTS following lightning injury.

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