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Central Hyperadrenergic State After Lightning Strike

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Abstract

Objective—To describe and review autonomic complications of lightning strike.

Methods—Case report and laboratory data including autonomic function tests in a subject who was struck by lightning.

Results—A 24-year-old man was struck by lightning. Following that, he developed dysautonomia, with persistent inappropriate sinus tachycardia and autonomic storms, as well as posttraumatic stress disorder (PTSD) and functional neurologic problems.

Interpretation—The combination of persistent sinus tachycardia and episodic exacerbations associated with hypertension, diaphoresis, and agitation were highly suggestive of a central hyperadrenergic state with superimposed autonomic storms. Whether the additional PTSD and functional neurologic deficits were due to a direct effect of the lightning strike on the CNS or a secondary response is open to speculation.

Keywords

hyperadrenergic; PTSD; neuropathy; tachycardia

High electric forces produced by lightning can cause human injury in several ways: direct strike, contact injury, ground current, upward streamer, and blast injury [1]. Survivors often suffer from a variety of long-term physical morbidities involving multiorgan derangement, with the most dramatic effect on nervous and cardiovascular systems. Multiple psychiatric disabilities have also been reported [1, 2]. Neurological complications are usually due to central nervous system (CNS) damage [3], and rarely due to peripheral nerve lesions [4]. Involvement of the autonomic nervous system (ANS) has been recognized but sparsely described [4–7]. We report a case of hyperadrenergic state with episodic autonomic symptoms (autonomic storms), plus post-traumatic stress disorder (PTSD) manifest as neuropsychological and severe functional impairment that occurred in a previously healthy young male following lightning injury [6, 8–10].

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Case

A 24-year-old man was struck by lightning while putting a hitch on a truck. The lightning hit the truck and threw him back several feet. He did not lose consciousness but sustained a burn in the first web space of his left hand. He was immediately confused and disoriented and was taken to an emergency room for evaluation. Initial evaluation revealed sinus tachycardia of about 140 beats per minutes (bpm). Tachycardia persisted subsequently, ranging between 120 and 140 bpm during the day on Holter monitor. Heart rate was documented to decrease during the night, but remained persistently above 90 bpm. P-wave morphology was consistent with sinus tachycardia. Cardiac pathology was excluded with normal electrocardiography (ECG), echocardiography, and cardiac enzymes. Subsequent magnetic resonance imaging (MRI) of the heart was normal with no evidence of atrial scar.

By 1–2 months, he began to experience palpitations and episodes of severe electric shock like pain on wrist, elbow, and shoulder. These shocks seemed to be centered around the joints and lasted about 5–10 seconds. He also noted some tingling of the toes and fingers. He complained of the loss of ability to distinguish between hot and cold stimuli throughout his body. Tachycardia persisted between 120 and 130 bpm with superimposed episodes of tachycardia to 160 bpm associated with diffuse diaphoresis, hypertension, and agitation lasting between one hour to one day. He recalls BP recordings of at least 160/115 mm Hg recorded on a number of occasions.

By the 3rd month, he complained of symptoms of PTSD. He had persistent anxiety and insomnia. He felt agitated and too “high” to sleep. He had problems with both sleep onset and maintaining sleep; he was unable to attain deep satisfying sleep level. His partner thought that he had short periods when he stopped breathing. He had recurrent flashbacks to the event and reacted with anxiety to weather storms and lightning with an urge to hide in a closet, and desire to have the drapes closed. He also developed problems with agitation and poor frustration tolerance. His bouts of tachycardia often occurred independently of these posttraumatic stress symptoms. At about the same time, he complained of a loss of fine motor skills with impaired use of his hands so that he could not write or perform other activities involving his fine motor functions. He also experienced occasional episodes of headache and slurred speech, intermittent difficulties with his memory, and periods of derealization.

The patient was evaluated at Mayo Clinic 3.5 months following lightning strike. Neurologic examination done at 3.5 and again 5 months after the event revealed several abnormalities. This included prominent action tremor of the hands, with neurophysiologically-documented co-contraction of agonists and antagonists. Despite normal muscle strength in all limbs, he had greatly impaired fine movements of his hands, but performed better when distracted. For instance, he was easily able to put on his socks, but performed poorly on formal testing of hand dexterity and function. His sensory examination suggested functional sensory impairment. He complained of difficulty walking. On formal testing, he was able to walk on toes and heels and had variable balance. His tendon reflexes were hypoactive at the knees. Cranial nerve function was intact.

Laboratory Evaluation

Autonomic function tests confirmed persistent tachycardia with essentially normal cardiovagal, sudomotor, and adrenergic reflexes. Autonomic function tests and their interpretation are summarized in Table 1. The impaired heart rate variation to deep breathing (Table 1) was thought to be related to hyperadrenergic suppression of cardiovagal function. Specifically, appropriate slowing of heart rate with baroreflex activation was observed [11, 12]. Thermoregulatory sweat test showed intact sweating except for minor sweat loss at the

toes, consistent with a mild length-dependent neuropathy. Plasma catecholamines showed an increased supine norepinephrine of 1605 pg/ml (Table 1) Following treatment with clonidine for 3 days at 0.4 mg/day, a repeat measurement was normal (supine, 77 pg/ml; standing 201 pg/ml). Electromyography (EMG) showed abnormalities limited to a reduced medial plantar sensory action potential amplitude and minor distal motor unit changes consistent with a minimal neuropathy. Quantitative sensory testing showed normal touch-pressure sensation, but cooling and heat/pain could not be adequately tested since the patient had an episode of diaphoresis preventing accurate testing. Brain MRI revealed nonspecific foci of increased T2 signal, and cervical spine MRI showed a right paracentral disk protrusion with an annular tear at C7-T1 without associated spinal cord compression. Obstructive sleep apnea was diagnosed on sleep study, showing REM-associated sleep apnea that disappeared with positive pressure. Treatment with continuous positive airway pressure was initiated. On formal neuropsychological testing, the patient's cognitive performance suggested a psychological rather than neurological cause for his memory complaints. CT imaging of the abdomen and serial urine metanephrine levels were obtained to exclude pheochromocytoma.

Diagnostic Formulation and Management

This gentleman presented with a combination of persistent sinus tachycardia with superimposed episodes of hypertension, diaphoresis, and agitation, highly suggestive of a central hyperadrenergic state with superimposed autonomic storms. This was confirmed with autonomic function testing, as well as the presence of abnormally high plasma norepinephrine levels (in the absence of pheochromocytoma), and the remarkable response to clonidine (0.4 mg/d) with plasma norepinephrine reverting to normal within one week of treatment.

Our patient also had recurrent flashbacks, newly acquired fear of weatherstorms, insomnia, and agitation as core symptoms of posttraumatic stress disorder (PTSD). PTSD was not associated with the level of tachycardia or plasma norepinephrine seen in this patient, so PTSD could not be responsible for his hyperadrenergic state [13–15]. On the other hand, patients with PTSD may have functional neurological symptoms, either as posttraumatic dissociative phenomena or as comorbid functional neurologic disorders. This patient's functional symptoms included memory difficulties, lancinating pain in the joints (atypical for neuropathic pain), and variable fine motor and gait deficits.

After failing traditional physical and occupational therapy approaches, a program of psychotherapy and functional rehabilitation [16], begun approximately 4.5 months after lightning strike resulted in gradual improvement in his activities of daily living. Approximately 4.5 months into his program of intensive psychotherapy and rehabilitation, clonidine was added and resulted in significant improvement of his hyperadrenergic state. Although he continued to have hyperadrenergic symptoms, they had a less troublesome effect on his level of function. Clonidine also reduced his PTSD symptoms. In the last few years, alpha-adrenergic agents, such as prazosin and clonidine, have emerged as a new pharmacologic treatment for PTSD-related agitation and insomnia [17–19]. Psychotropics were not needed after clonidine was prescribed.

Discussion

The combination of persistent sinus tachycardia and superimposed episodes of diaphoresis, exacerbated hypertension and agitation were highly suggestive of a central hyperadrenergic state with superimposed autonomic storms. Abnormal autonomic function testing, markedly elevated plasma and urine norepinephrine levels (in the absence of pheochromocytoma), and the remarkable response to clonidine (0.4 mg/d) with plasma norepinephrine reverting to

normal within one week of treatment support this notion. Coupled with this was PTSD, which was presumably the substrate for his persistent anxiety, flashbacks, avoidance of weather-storms, and hypervigilance; this markedly impaired his social and occupational functioning. Functional (nonorganic) neurologic symptoms, including the cognitive and motor complaints reported by this patient, are common in individuals exposed to life-threatening events.

Pheochromocytoma was excluded by abdominal imaging and serial urinary catecholamines, plus the dramatic response to clonidine.

Lightning strike is associated with a fatality rate of about 8–10%, and the majority of survivors suffer from long term disabilities with some unique characteristics [5]. The primary cause of death following lightning is cardiac, mainly ventricular fibrillation or tachycardia [1]. Respiratory arrest may result from central nervous system injury with loss of respiratory drive, prolonged paralysis or tetanic contraction of respiratory muscle, or combined cardiorespiratory arrest [7]. Cardiac abnormalities include arrhythmia, conduction abnormalities, and myocardial damage [7]. Most arrhythmias occurs in the immediate aftermath of the strike, but delayed ventricular arrhythmia of up to 12 hours late may occur [20]. Survivors may suffer from ectopic atrial rhythms, inappropriate sinus tachycardia, ECG abnormalities, blood pressure changes, vasovagal syncope, and other cardiac abnormalities due to autonomic dysregulation [1, 6, 8].

Lightning injury may cause several types of CNS injury. The first type is characterized by immediate but transient neurologic deficits. This commonly includes a transient loss of consciousness, often with confusion, amnesia, headache, paraesthesia, weakness and keraunoparalysis for a period of time following restoration of consciousness. The second pattern of neurologic deficits occurs immediately but with prolonged or permanent sequelae. These include intracranial hemorrhage, post-arrest cerebral infarction, and spinal and peripheral nerve injuries. The spinal injuries have been ascribed to hypoxic or ischemic injury. The relationship of peripheral neuropathy to lightning strike is poorly documented. Some of the permanent sequelae comprise possible delayed neurological syndromes such as motor neuron disease and movement disorders, comprising a third type of complication. A fourth type is acute effects that occur as a result of trauma from falls and blast, and include subdural and epidural hematoma and subarachnoid hemorrhage [1].

A variety of autonomic complications have been ascribed to lightning strike, including cardiovascular, gastrointestinal, urogenital, respiratory, sleep, and thermoregulatory dysautonomia [6, 8–10]. Lightning strikes that are sufficient to induce neurologic symptoms usually include damage to the CNS [6, 9]. Limbic lobe and hypothalamus injury result in dysautonomia and are structures that could be affected by lightning [6], although documentation of hypothalamic or limbic lobe injury is sparse.

There is limited precedent for the clinical picture of our patient, with a hyperadrenergic state and autonomic storms occurring post lightning injury [1]. Apart from direct destruction of brain and spinal cord, the hyperadrenergic state, with norepinephrine-induced intense vasoconstriction, has been touted as being a possible cause of paraplegia [6, 21]. A case of idiopathic orthostatic hypotension and two cases of postural tachycardia syndrome have been reported post lightning injuries [5, 22]. Our case is indicative of hyperadrenergic dysautonomia with possible central origin after lightning injury. Many of the clinical manifestations including tachycardia, hypertension, and palpitations can be explained by autonomic dysfunction, which improved with clonidine treatment.

This patient had a minimal axonal neuropathy. The relationship of this neuropathy to lightning strike is highly questionable. While peripheral nerve injury following lightning

may occur due to cell damage caused by electroporation, joule heating or combination of both [4], this patient has a minimal length-dependent axonal neuropathy with features of a very chronic process.

Neuropsychological deficits associated with lightning injury may include disturbances of attention, concentration, verbal memory, new learning, language, awareness, emotional, sensory, and visuospatial functions [23]. Individuals who have been struck by lightning often suffer from neuropsychiatric disorders including PTSD, depression, panic and other anxiety disorder, personality changes, functional neurologic (conversion) disorders, adjustment disorders, emotional lability, sleep disturbance, and cognitive impairment [24], and experience reduced functional capacity and inability to handle any stressful situations [23]. Early neuro-psychiatric intervention, serial multidisciplinary assessment for systemic, neurological and neuropsychological effects, traumaspecific psychotherapy along with the rehabilitation program is recommended for neuropsychological impairment [23]. Clonidine appeared to exert two therapeutic actions, reducing the patient's centrally mediated autonomic and adrenergic hyperactivity and his PTSD-related agitation and sleep disturbance.

The clinical outcome of individuals struck by lightning is variable, with some patients returning to premorbid status and others experiencing persistent symptoms [23]. This case illustrates the benefits of a coordinated approach to the evaluation and treatment of neurologic, cardiac, psychiatric, and functional sequelae of lightning injuries.

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Table 1**Autonomic Function Tests**

Test	Result	Normal Values*	Interpretation
Plasma Catecholamines 2/27/12	NE: Supine 1685; E: supine, 28pg/ml; Dop: supine 109 (all in pg/ml).	NE: 70–750 pg/ml E: 0–110 pg/ml Dop: <30 pg/ml	Increased NE Normal E Increased dopamine
Plasma Catecholamines	NE: sup/std, 77/201 E: sup/std, 12/14 Dop: sup/std, <10/11	NE: 70–750/200–1700 E: 0–110/0–140 Dop: <30/<30 All in pg/ml	Normal for all values
24h Urine Catecholamines	NE: 272 E: 17 Dop: 657 All in µg/24h	NE: 15–80 E: 0–20 Dop: 65–400 All in µg/24h	Increased NE and Dopamine
24h Urine Metanephrines	Metanephrine: 162 Normetanephrine: 996 Total Metanephrines: 1158 All in µg/24h	Metanephrine: <400 Normetanephrine: <90 Tot Metanephrines: <1300 All in µg/24h	Increased Normetanephrines
QSART	Volumes of 2.76, 0.19, 3.68, 2.68 µL/cm ²	Recorded volumes were at 90, 9, 91, 91st percentile	Normal postganglionic function
HRDB	9.0 bpm	>14 bpm	Mildly reduced cardiovagal function
VR	1.66	>1.59	
HUT	Normal response	Modest fall SBP (<30 mm Hg) and mild increase DBP	Normal tilt
BRS_v1 (BP fall)	1.82 ms/mm Hg	>3.5 ms/mm Hg SBP	Reduced
BRV_v2 (BP rise)	8.34 ms/mm Hg	>5 ms/mm Hg SBP	Normal
PRT	2.0 seconds	<5 seconds	Normal BP recovery time
TST%	2% anhidrosis, affecting toes bilaterally	Normal sweating	Mild DSFN
Polysomography	REM-related sleep apnea, corrected by positive pressure		Mild obstructive sleep apnea

* normal values for hypertensive male of patient's age; NE, norepinephrine; E, epinephrine; Dop, dopamine; QSART, quantitative sudomotor axon reflex test; HRDB, heart rate response to deep breathing; BRS-v1, baroreflex sensitivity, defined by slope of heart period to systolic BP change during early phase II of Valsalva maneuver; BRS_v2, baroreflex sensitivity, defined by slope of heart period to systolic BP change during early phase IV of Valsalva maneuver; PRT, blood pressure recovery time; TST%, percent of total body anhidrosis on the thermoregulatory sweat test.