Dilated Cardiomyopathy After Electrical Injury: Report of Two Cases

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The specific etiologic factor and pathogenesis of most dilated cardiomyopathies have yet to be described definitively. Hypotheses of the etiologic factor of idiopathic dilated cardiomyopathy (DCM) abound. This report describes two patients with electrical injury in whom DCM developed after the electrical insult in the absence of other precipitating causes. Further histologic examination of myocardial tissue after electrical injury may reveal clues regarding the pathophysiology behind electrically induced DCM. Because electrical injury may be associated with myocardial dysfunction, short- and long-term evaluation of left ventricular function may be warranted.

Many theories have been proposed regarding the etiologic factors of idiopathic dilated cardiomyopathy (DCM); however, none has been established.1-5 Physical agents reported to cause DCM include heat stroke, hypothermia, radiation, and lightning strike.1,6 Electricity has been documented to cause myocardial necrosis, infarction, dysrhythmia, and contractile dysfunction, all of which may be delayed as well as persistent.7,8 We describe DCM in two patients after electrical injury: in one patient, DCM occurred after high-voltage injury, and in the other, after low-voltage injury. We believe electrical injury to be an additional physical cause of DCM.

Case Presentations

Case 1
A 24-year-old black man was brought to the emergency department after receiving an electrical shock. The shock originated in his left hand from a faulty electrical appliance connected to a standard wall outlet. During the insult, the patient had to be pulled away from the electrical device.

The patient was previously in good health and had no history of sustained hypertension, diabetes, substance abuse, or family history of heart disease. At physical examination, he was afebrile. His heart rate was 96 beats per minute; respirations were 20/min; and blood pressure, 130/100 mm Hg. Although he had no external burn marks or focal neurologic deficits, he complained of severe pain, decreased range of motion, and mild edema of the left upper extremity, as well as abdominal pain. Ketorolac was administered intramuscularly for relief of pain.

An electrocardiogram (ECG) revealed sinus tachycardia (108 beats per minute) with premature ectopic complexes and diffuse ST and T wave abnormalities more anterolaterally.

Laboratory studies revealed a white blood cell (WBC) count and electrolyte levels within the normal range; the serum creatine kinase (CK) level was 748 U/L, and serum CK-MB isozyme, 2.4% of total CK activity. Results of the routine drug and alcohol screen were negative. Three days after admission, the patient also began complaining of scotoma.

Approximately 5 weeks later, the patient was admitted to the hospital because of progressive shortness of breath, chest pain, palpitations, and epigastric pain. At that time, the patient was afebrile; respirations were 20/min, and blood pressure, 140/110 mm Hg. An ECG revealed normal sinus rhythm (94 beats per minute) with ST and T wave abnormalities suggestive of lateral ischemia.

Laboratory studies revealed the complete blood cell count and electrolyte values to be in the normal range. Findings on chest x-ray films were consistent with congestive heart failure (CHF). Two-dimensional echocardiograms showed a dilated, severely hypokinetic left ventricle. The patient was treated with furosemide administered intravenously, an angiotensin-converting enzyme inhibitor, and topical nitrates.

Approximately 5 months after the initial electrical insult, the patient was again admitted to the hospital with CHF and found to have an ejection fraction of 15%. His blood pressure was 240/140 mm Hg. An ECG revealed normal sinus rhythm (94 beats per minute), premature ventricular contractions, right atrial enlargement, and anterolateral T wave inversions. Chest x-ray film revealed bilateral interstitial and vascular prominence with cephalization, bilateral pleural effusions, and cardiomegaly.
CARDIAC catheterization revealed normal coronary arteries, elevated left ventricular end-diastolic pressure, a pulmonary capillary wedge pressure of 32 mm Hg, and severe global hypokinesis with a cardiac index of 1.4 (calculated as cardiac output in liters per minute divided by body surface area in square meters). The findings were consistent with DCM.

Endomyocardial biopsy specimens from the right ventricle revealed myocyte hypertrophy with focal interstitial fibrosis. No evidence of active inflammation or necrosis was present.

Eleven months after the initial insult and follow-up neurologic evaluation, the patient was admitted for a second cardiac catheterization and referral for cardiac transplantation. While awaiting the transplant, the patient was treated pharmacologically for CHF with a persistently depressed ejection fraction of 15% and a severely dilated left ventricle with diffuse patchy uptake and diaphragmatic attenuation revealed by thallium-201 myocardial perfusion scintigraphy. Sudden cardiac death occurred while the patient was being monitored, and he was discharged from the hospital. He denied loss of consciousness, chest pain, dyspnea, emesis, or loss of bowel and bladder control. On admission, his blood pressure was 152/90 mm Hg, and his heart rate was 108 beats per minute and regular. Findings of the physical examination were unremarkable except for burns evident on the chest, arms, abdomen, buttocks, and legs. Urinalysis was negative for myoglobin. The WBC count was elevated (13,700/μL). All other laboratory values were unremarkable.

An ECG showed borderline delay of intraventricular conduction with no serial changes. Findings on chest and spine x-ray films were unremarkable. The patient had a 20-pack-year history of tobacco use and was a mild social drinker. He had no history of cardiac disease, hypertension, or diabetes.

The patient was hydrated, and silver sulfadiazine cream was applied to his wounds. Physical therapy, occupational therapy, and psychiatric consultation for posttraumatic stress disorder were initiated. The patient had no telemetry abnormalities while monitored, and he was discharged from the hospital with appropriate follow-up.

Approximately 1.5 years after the initial injury, the patient had cardiac arrest and died. Autopsy demonstrated DCM with a markedly dilated left ventricle weighing 540 g. No coronary atherosclerosis was present. A 1-cm area of bridging of the left anterior descending coronary artery was noted. The lungs were congested.

Case 2

A 42-year-old man sustained severe first- and second-degree burns (20%) when he placed a shovel into an electrical line while digging a hole. Coworkers estimated the line to be approximately 13,000 V.

The patient was admitted to the burn unit of a local hospital. He denied loss of consciousness, chest pain, dyspnea, emesis, or loss of bowel and bladder control. On admission, his blood pressure was 152/90 mm Hg, and his heart rate was 108 beats per minute and regular. Findings of the physical examination were unremarkable except for burns evident on the chest, arms, abdomen, buttocks, and legs. Urinalysis was negative for myoglobin. The WBC count was elevated (13,700/μL). All other laboratory values were unremarkable.

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Comment

Electrical injury from lightning strike, direct current cardioversion, and both high-voltage (≥1000 V) and low-voltage (<350-V standard wall outlet) sources has been well documented in effecting multiple organ injury in muscle (cardiac and skeletal), nervous tissue, as well as most other soft tissue in its path.8-20

Cardiac morbidity and mortality associated with electrical injury has been documented to result from cardiac arrest, tachycardia, arrhythmia, conduction disturbances, myocardial infarction, and contractile dysfunction.8,12,13,21-27 The extent of cardiac involvement in electrical injuries varies with the location of the entrance and exit wounds, voltage, magnitude, current frequency, and duration.6,13,14,19,28-31

Although it is generally appreciated that electrical injuries can cause fatal arrhythmias,2 it is less appreciated that transient or permanent myocardial dysfunction with or without CHF can occur.6,22,23 Our two case presentations describe DCM in association with both high- and low-voltage electrical injuries in the absence of other precipitating etiologic factors.

Conclusion

The variability of cardiac injury in survivors of electrical trauma limits the appearance of cardiac manifestations such as DCM. The literature is replete with warnings to provide long-term follow-up of electrically injured patients to monitor for latent cardiac dysfunction and cardiomyopathy. We believe electrical injury may be associated with myocardial dysfunction and short- and long-term evaluation of left ventricular function may be warranted.

References

CASE REPORT


