Intensive Myocardial Fibrosis After High-Voltage Electric Shock Demonstrated by Delayed-Enhancement Cardiac Magnetic Resonance Imaging

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In September 2007, a 27-year-old man received a high-voltage electric shock of 3000 V while working at a railway station. He remained conscious and had no burns or shortness of breath but experienced chest pain with tingling in the left arm. His medical history was unremarkable, with no hypertension, diabetes mellitus, substance abuse, or family history of heart disease. The initial physical examination showed only a dry 1-mm lesion at the tip of the left forefinger, probably at the source point of the electric current. The most likely path was from the left hand to the feet. The ECG showed a sinus rhythm with inferior and lateral T-wave inversion. Cardiac enzyme levels were normal. The 2-dimensional echocardiogram showed hypokinesia of the inferior and anterior walls with a left ventricular (LV) ejection fraction of 50%. The coronary arteries were normal by coronary computed tomography angiography. The β-blocker atenolol 100 mg/d was initiated because of persistent sinus tachycardia at 120 bpm.

In February 2008, cardiac magnetic resonance imaging (MRI) showed mild hypokinesia with thinning of the inferior and lateral walls, no evidence of necrosis, and subnormal LV ejection fraction.

In July 2010, an evaluation for chronic shortness of breath showed New York Heart Association class II heart failure with asthenia and hypotension. The atenolol dose was decreased to 50 mg/d.

In November 2011, he was admitted for acute heart failure. An ECG showed a normal sinus rhythm with flattened T waves on the inferior and lateral leads. His serum N-terminal probrain natriuretic peptide was elevated to 3200 pg/mL. Echocardiography demonstrated an LV ejection fraction decrease to 27% (Figure, A and B) with akinesia of the inferior and septal walls, LV dilation (LV end-diastolic diameter, 34 mm/m²), and elevated LV pressures (E/A=3.1; E-wave deceleration time, 68 milliseconds). Coronary angiography was normal. Cardiac MRI showed extensive delayed subepicardial enhancement of both ventricles (Figure, C and D) suggesting nonischemic myocardial fibrosis with transmural involvement of the LV inferolateral wall.

In June 2012, his symptoms worsened precipitously. He had biventricular dysfunction with a thrombus at the LV apex (Figure, E and F). He received a heart transplantation 1 month later, that is, 5 years after the electric injury. Histological examination of the explanted heart with Sirius red staining showed biventricular disease with a normal subendocardial layer (Figure G) but diffuse interstitial fibrosis of the subepicardial layer (Figure H).

Discussion
Cardiac MRI in our patient showed both cardiac dilation and delayed diffuse subepicardial enhancement, a pattern previously described in chronic myocarditis with heart failure, particularly that caused by parvovirus B19 and human herpesvirus 6 infection documented histologically.1,2 Although viral myocarditis cannot be definitively ruled out in our patient, strong arguments support electric injury as the cause of the progressive heart failure, including the chest pain and ECG alterations at the time of injury, electric current pathway from the left forefinger to the feet, absence of other identifiable causes, and extensive delayed subepicardial enhancement of both ventricles.

Electric cardiac injuries range in severity from mild to fatal. Within hours after the injury, the ECG may show nonspecific ST and T changes, with the most common rhythm being sinus tachycardia, possibly resulting from catecholamine spillover. Ventricular fibrillation or conduction disorders are the main causes of death after electrocution. Other arrhythmias such as premature ventricular contractions, ventricular tachycardia, atrial tachycardia, sinus bradycardia, and conduction disorders have been reported in 10% to 46% of patients.3 In our patient, the ECG recorded immediately after electrocution showed sinus tachycardia with T-wave inversion on the inferior and lateral leads. Pericardial effusion and cardiac rupture have also been reported after electrocution. Acute myocardial infarction has been found in autopsy studies.3 LV dysfunction may occur and may be delayed or persistent. Dilated cardiomyopathy with LV dysfunction developed in 2 patients after electric injury.4 The diagnosis of dilated cardiomyopathy was established 5 weeks after injury in 1 patient, who died 11 months after injury while awaiting heart transplantation. The other patient had normal ECG findings immediately after the injury but died of cardiac arrest 1.5 years later and had dilated...
cardiomyopathy by autopsy. However, the cardiac effects of electric injury are debated. A retrospective study of 102 patients admitted for high- or low-voltage electric injuries over a 20-year period indicates that arrhythmias occur immediately after the injury and that patients in good health who have normal ECG findings at admission without loss of consciousness or major trauma are unlikely to develop serious cardiac dysrhythmia and therefore do not require observation in the emergency department.

Our case underlines the possibility of LV failure caused by cardiac fibrosis after electric injury. Cardiac fibrosis in our patient produced delayed enhancement by MRI and was confirmed histologically. In a previously reported case, cardiac MRI showed delayed enhancement suggesting myocardial fibrosis, but histological confirmation was not obtained.6 Delayed myocardial enhancement by MRI is a powerful tool for detecting fibrosis and may assist in patient follow-up after a high-voltage electric shock. Further studies are needed to evaluate this possibility.

Very few cases of dilated cardiomyopathy after a high-voltage electric shock have been reported to date. To the best of our knowledge, this is the first case showing progressive myocardial fibrosis with delayed enhancement by MRI after high-voltage electric shock and subsequent histological confirmation.

Disclosures

None.

References

Figure. A, Echocardiography, 4-chamber view: evaluation of the left ventricular ejection fraction with the Simpson method. B, Echocardiography, 2-chamber view: evaluation of the left ventricular ejection fraction with the Simpson method. C and D, Cardiac magnetic resonance imaging showing extensive subepicardial delayed enhancement of both ventricles, consistent with non-ischemic myocardial fibrosis (small arrows). Large arrow indicates transmural delayed enhancement of the inferolateral wall of the left ventricle, suggesting greater severity of myocardial fibrosis at this site. E, Echocardiography, 4-chamber view: apical thrombus (arrow). F, Echocardiography, 2-chamber view: apical thrombus (arrow). G, Histological examination with Sirius red staining of the subendocardial layer of the explanted heart: normal appearance. H, Histological examination with Sirius red staining of the subepicardial layer of the explanted heart: diffuse interstitial fibrosis.
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