

Aortic Valve Rupture due to High-voltage Electrical Injury: Case Report

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In the heart, the most common sequelae after electrical injury are myocardial contusion and arrhythmias. A case is presented of segmental ventricular dysfunction and severe aortic regurgitation due to laceration of the right coronary cusp of the aortic valve

The pathophysiology of electrical injury has been attributed to the conversion of electrical energy into heat. The resultant tissue damage depends on the type of current, the pathway of flow, the local tissue resistance, and the duration of contact. High-voltage electrical injuries are very dangerous because of the complications of electrical damage including that to the heart, kidneys and/or deep tissues. Electrical injuries include tympanic membrane rupture, nerve injury, vascular injury, occult abdominal injury and fractures (1). Significant cardiac complications among survivors of high-voltage electrical injury are less common, but it is a serious and often life-threatening situation (2,3). In the heart, the most common sequelae after electrical injury are myocardial contusion and arrhythmias. High-voltage electricity can also act as a countershock, putting the heart into asystole. Here, a case is presented of rupture of the right coronary cusp of the aortic valve, following electrical injury, resulting in severe regurgitation.

Case report

A 63-year-old man was admitted to the authors' clinic for acute shortness of breath and angina that had increased over the past fortnight. Two weeks ago, the patient had made direct contact with a high-voltage wire while cutting a tree, though his feet remained on the ground. His chest and left upper extremity were

caused by electrical injury. To the authors' knowledge, this is the first reported case of valvular rupture due to electrical injury.

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exposed to a high-voltage alternating current (34,500 V), causing him to fall down. The patient had remained unconscious for a period of 5-10 min. After a further period of 1-2 h, he was examined at another hospital, where no evidence was found of any electrical injury. The patient had neither a history of cardiovascular disease, nor of any coronary artery risk factors (e.g. diabetes mellitus, hypertension, hyperlipidemia, smoking) except for advanced age and male gender.

Physical examination revealed a raised jugular venous pulse, a laterally displaced apex beat, a widened pulse pressure (150/50 mmHg) and a regular pulse rate of 115 beats/min. All peripheral pulses were slightly increased in intensity. Cardiac auscultation revealed a normal S1 and decreased S2, and a grade 4/6 diastolic blowing murmur along the left sternal border. There was no sign of infective endocarditis, and the remainder of the physical examination was unremarkable.

A complete hematological and microbiological evaluation was performed in order to rule out infective endocarditis. Neither the erythrocyte sedimentation rate nor white blood cell count was elevated. Chest X-radiography revealed cardiac enlargement, while an electrocardiogram showed a normal sinus rhythm and non-specific ST-T-wave changes.

Transthoracic echocardiography (TTE) showed the presence of anteroapical and interventricular septal hypokinesis, and significant aortic regurgitation. The left ventricle was seen to be dilated, and the ejection fraction was 40-45%. The right ventricular size was increased. Transesophageal echocardiography (TEE) showed avulsion of the free edge of the right coronary cusp of the aortic valve to the ascending aorta (Fig. 1a

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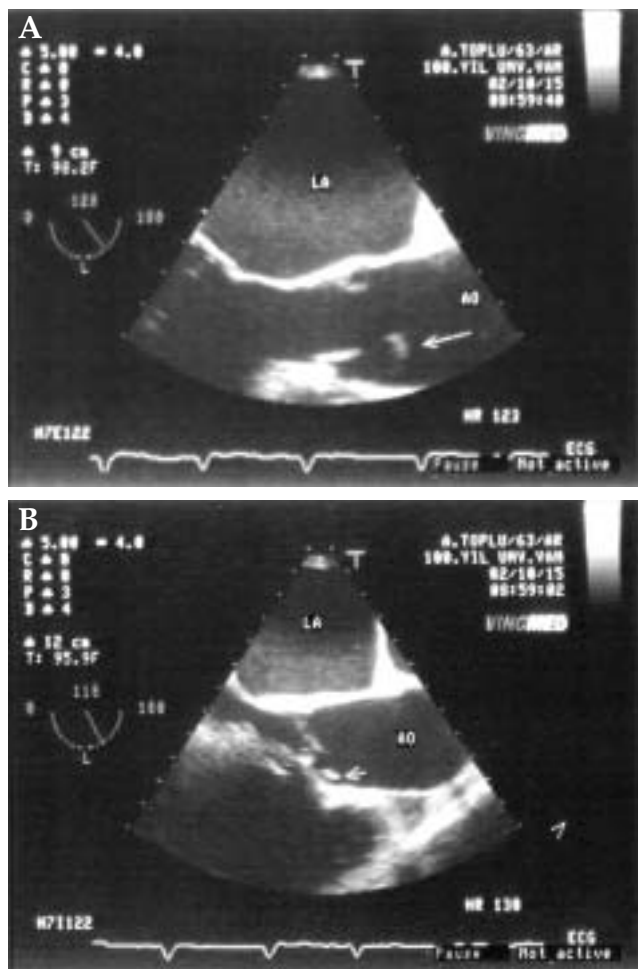


Figure 1: Transesophageal echocardiograms showing avulsion of the free edge of the right coronary cusp of the aortic valve. The arrow indicates the lacerated part of the cusp. Ao: Aorta; LA: Left atrium.

and b). TEE also demonstrated non-coaptation of the aortic valve (Fig. 2).

Due to the severity of the patient's aortic regurgitation and the signs of left ventricular segmental dysfunction, coronary angiography was performed. Cardiac catheterization showed normal coronary arteries and severe aortic regurgitation. The decision was made to operate, with the intention of repairing the valve, if possible. During surgery, the surgeon found the free edge of the right coronary cusp to be lacerated and necrotic. The laceration was treated successfully with urgent aortic valve replacement.

Discussion

Recently, Jiang and Xu (4) investigated the histomorphological changes in canine heart that occurred after electrical injury. The cardiac muscle cells in the atrial and ventricular walls were found to exhibit clear



Figure 2: Transesophageal echocardiogram showing non-coaptation of the aortic valve at systole (arrow). LA: Left atrium.

swelling and deformation, blurred cross-striation, breaking and expanding of the intercalated disk, intracytoplasmic structural destruction of the myofibrils, and mitochondrial swelling and vacuolization in the cardiac conduction system. These authors concluded that the pathomorphological characteristics of cardiac histological changes after electrical injury were similar to those of acute myocardial infarction, and consistent with this a segmented systolic dysfunction was found in the present patient. The present case was the first to be reported of aortic valve rupture due to electrical injury. The mechanism of valve rupture was thought to be the establishment of an electrical arc through the anteroapical and septal myocardium and the aortic valve.

Acute aortic regurgitation is usually the result of

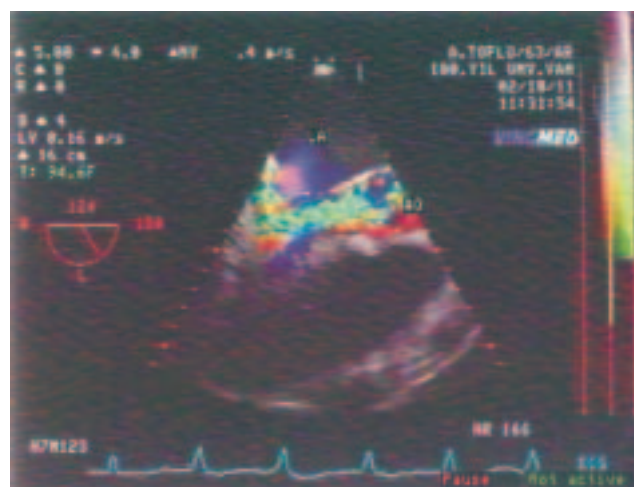


Figure 3: Color Doppler echocardiogram showing significant aortic regurgitation. Ao: Aorta; LA: Left atrium.

infective endocarditis, with perforations or defects occurring within one or more cusps. Less commonly, it occurs following acute aortic dissection, with retrograde disruption and prolapse of the commissure. Other rare causes of acute aortic insufficiency include traumatic rupture of a cusp following blunt chest injury and spontaneous rupture of a fenestrated cusp. Incompetence usually results from cusp tears or avulsion of the valve from its annulus. In the present case, infective endocarditis was considered in the differential diagnosis, but this was never established. No valvular vegetations were identified, and the blood cultures proved to be negative.

TEE following TTE was chosen in the present patient because the latter approach provides a better visualization of the valvular anatomy. TTE confirms the reality of aortic regurgitation suggested clinically by the appearance of a diastolic murmur, but confirmation of the mechanism of the lesions is based on TEE, which allows safe and rapid visualization of the mechanism of the valvular lesion, investigation of any associated lesions, and guidance of therapeutic management. TEE was valuable in identifying the traumatic mechanism of aortic regurgitation and the extent of valvular lesions in the present patient, in whom neither fibrotic

nor calcific changes of aortic and mitral valves were seen on echocardiographic examination.

In conclusion, the present case report showed that cardiac valve injuries may occur following electrical injury. Although the patient's cardiac status immediately prior to the electrical injury was unknown, the link between the onset of shortness of breath and the electrical injury was highly suggestive of a cause-effect relationship.

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