Conclusion

Heart rate variability may be a useful modality for understanding the pathophysiology of periodic lethal arrhythmias and for judging the efficacy of treatments. However, these findings should be duplicated and confirmed in larger cohort.

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Aortic dissection provoked by electrical storm

Elektriksel fırtına nedeni ile gelişen aort disseksiyonu

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Introduction

Acute aortic dissection is the most common catastrophe of the aorta. Although maneuvers that increase intra-thoracic pressure (ITP) have been reported to trigger aortic dissection, there is no report of aortic dissection in the setting of multiple automated implantable cardioverter defibrillator (AICD) shocks. We report and discuss the case of a man who developed aortic dissection following multiple AICD shocks.

Case report

A 76-year old man with a history of congestive heart failure (CHF) was brought to the emergency department after having multiple AICD shocks.

Two weeks earlier, the patient was admitted with shortness of breath and was found to be in CHF exacerbation and atrial fibrillation. He was treated medically. Transthoracic echocardiogram (TTE) and transesophageal echocardiogram (TEE) studies revealed a left ventricular ejection fraction of 20-25%, dilated atria, mild aortic regurgitation, without evidence of thrombus or aortic dissection (Fig. 1. Video 1. See corresponding video/movie images at www.anakarder.com). He underwent dual chamber AICD (Medtronic Virtuoso DR) implantation with right ventricular defibrillation impedance of 29 ohms, pacing impedance of 392 ohms and threshold of 1 volt at 0.4 millisecond.

On this admission, he denied palpitations, chest pain, dizziness or loss of consciousness. He was alert and oriented, blood pressure (BP) was 142/88 mmHg and his heart rate was irregularly irregular at 132 beats/min. The rest of the physical examination was unremarkable. His electrocardiogram (ECG) revealed atrial fibrillation with a rapid ventricular response of 130 beats/min and left ventricular hypertrophy. Chest X-ray showed mild cardiomegaly. Laboratory tests were unremarkable.

On the 3rd hospital day, he experienced 3 episodes of AICD shocks accompanied by jerky movements that coincided with unsustained ventricular tachycardia on telemetry. Subsequently, his BP increased to 170-185/95-110 mmHg from baseline of 120-145/75-88 mmHg, and was difficult to control with multiple antihypertensive medications. Within 24 hours of the electrical storm, he complained of non-radiating pre-

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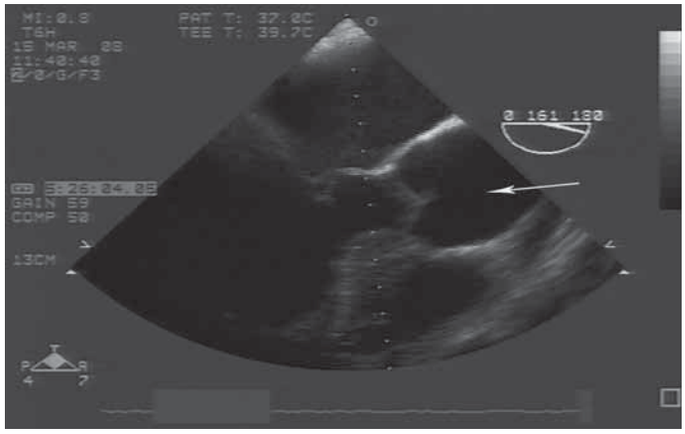

Figure 1. Transesophageal echocardiography, long-axis view, shows ascending aorta with no evidence of dissection. The arrow points to the ascending aorta Video 1. Transesophageal echocardiography, long-axis view, shows the motion image of ascending aorta with no evidence of dissection at different stages of cardiac cycle

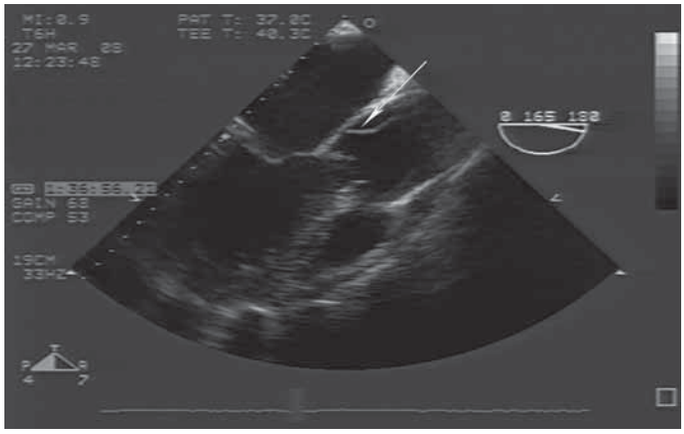

Figure 2. Transesophageal echocardiography, long-axis view, shows aortic dissection intimal flap immediately distal to the aortic valve in the ascending aorta. The arrow points to the intimal flap. Video 2. Transesophageal echocardiography, long-axis view, shows the motion image of aortic dissection in the ascending aorta at different stages of cardiac cycle

cordial chest pain that improved with sublingual nitrate and aspirin. The ECG revealed no changes and a repeat troponin was 0.30 ng/ml (normal: 0.02–0.39 ng/ml). Treatment for acute coronary syndrome was promptly initiated. Six hours later, his BP was observed to be 165/85 mmHg in the left and 84/40 mmHg in the right arm. There was elevation of creatinine to 1.9 mg/dL (normal: 0.7–1.3 mg/dL) and blood urea nitrogen to 29 mg/dL (normal: 7–25 mg/dL). Aortic dissection was suspected and TEE revealed type A aortic dissection originated from the ascending aorta, and extended to distal descending aorta (Fig. 2. Video 2. See corresponding video/movie images at www.anakarder.com). In view of his co-morbidities, he was treated medically, but he died within 72 hrs of diagnosis.

Discussion

In the AICD literature, electrical storm, defined as ≥ 3 AICD shocks prompted by ventricular tachyarrhythmia, portends an increased risk of death, particularly within the first three months following the event (1). Although multiple defibrillations by AICD have been reported to result in myocardial damage (2), we are unaware of a prior report of aortic dissection in the setting of electrical storm.

Stimulation of thoracic and abdominal muscles during AICD discharges has been previously reported (3), and this may increase ITP and peripheral BP. Alternatively, the pain from a shock can increase BP indirectly. In prior studies, novel factors including weight lifting and sneezing, have been reported to trigger aortic dissection (4, 5). Both maneuvers increase ITP and peripheral BP (4, 5). In our patient, the sudden and recurrent stimulation of skeletal muscles, evidenced by jerky movements, was likely associated with increased ITP, which in addition to pain from the shocks resulted in acute elevation of BP. We believe that the combination of increased ITP and acute BP elevation created a tension on the aortic wall that triggered the intimal tear.

Aortic dissection is often life threatening. In untreated patients, the mortality rate is 25% within 24 hours, 70% by 2 weeks and 90% after 2 weeks (6). In over 38% of cases, the diagnosis is missed at the first evaluation (7). Although chest pain is present in about 90% of patients with acute aortic dissection (8), painless aortic dissections have also been reported, most especially in patients with CHF (9). Chest pain preceded the changes in pulse and blood pressure in the case described above. The major clue to aortic dissection in our patient was inter-arm blood pressure and radial pulse difference.

Conclusion

Aortic dissection should be recognized as a possible complication of multiple AICD shocks.

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