Electrical Injury-Induced High-Degree Atrioventricular Block Requiring a Permanent Pacemaker

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ABSTRACT

Electrical injury may lead to a conduction disorder of the heart. We report here on a 36-year-old man, who was treated with a permanent pacemaker, after an electrical injury induced high-degree atrioventricular block and clinical manifestations (dizziness and dyspnea). (Korean Circulation J 2006;36:767–770)

KEY WORDS : Electric injuries ; Heart conduction system.

Introduction

Electrical injuries account for a considerable amount of morbidity and mortality, and most deaths are caused by arrhythmia at the time of exposure to the electrical source.1) Electrical injury has a predilection for the sino-atrial and atrioventricular nodes.2) This increases the difficulty of recognizing subtle damage because the performance of natural pacemakers can be capricious even in healthy people and some prolongation of atrioventricular conduction delay may be accepted as a normal variant. Also, there is diagnostic difficulty associated with disease of the sinus node and the possible delay between the electrical injury to the heart and the clinical manifestations.3) We report her on a case of the injury to the heart conduction system after electrical injury and this is the first such case report from Korea.

Case

A 36-year-old man, an inspector of the construction company, was referred to our hospital because of a dizziness and dyspnea on exertion. He received an electrical shock on his left hand during his usual work, and then he fell down about 1 meter on the ground 5 days before hospitalization. The welding voltage was 380 volt (alternating current). At that time, there were no external wound and symptoms. Five hours after the electrical injury, he felt dizziness during driving and also dyspnea on exertion, but at that time he did not visit the hospital. On admission 5 days after the injury, his blood pressure was 120/80 mmHg and his pulse rate was 36 beats/min. A physical examination revealed no abnormal findings. The blood tests were within normal limits, including the cardiac enzymes. The chest radiography was normal. The electrocardiogram showed 2 : 1 atrioventricular block, a prolonged PR interval and complete right bundle branch block (Fig. 1B). Eight months prior to admission, his previous electrocardiogram showed normal sinus rhythm and no abnormal findings (Fig. 1A). The transthoracic echocardiography showed normal left ventricular wall motion and no abnormal findings. Electrocardiographic monitoring showed intermittent 2 : 1 atrioventricular block, Wenchebach phenomenon and a high-degree, complete atrioventricular block with mostly first degree AV block (Fig. 2). The exercise electrocardiogram test done with the modified Bruce protocol induced high-degree AV block from the first degree AV block with 180 beat/min for the atrial rate (Fig. 3). Electrophysiologic testing showed that the AH interval, HV interval, sinus node recovery time and corrected sinus node recovery time were 220 msec, 195 ms, 1048 msec and 245 msec, respectively. The AV block cycle length was 690 msec. HV block developed after intravenous isoproterenol infusion (maximal sinus cycle length: 540 msec) (Fig. 4). A permanent pacemaker (Kappa KVDD901, VDD type, Medtronic®, Minneapolis, USA) was implanted into the patient based on the above results. The ECG without the function of the pacemaker still showed high-degree AV block 2 months after pacemaker implantation (Fig. 5).
Several mechanisms have been proposed to account for the myocardial and conducting tissue damage seen after electrical injury. These include the induction of coronary artery spasm, direct thermal injury, ischemia that's secondary to arrhythmia-induced hypotension, chemoreceptor stimulation that produces acute hypertension, catecholamine mediated injuries and vascular injury. Death following an electrical shock is usually secondary to cardiac complications, although respiratory arrest or multisystemic complications can occur. The cardiac complications include sudden death secondary to ventricular fibrillation or asystole, myocardial injuries and immediate or delayed arrhythmias. The immediate electrical effects will depend on the physical properties of the electricity itself. These effects may include sinus node dysfunction, atrial fibrillation, ventricular fibrillation and asystole. Electricity, even when it is lower than 80 volt, can cause of death. For this kind of death, the patients generally present with electrical marks mostly on the chest or head, severe electrical injury and moisture at the contact site, and a hot and humid accident environment is usually present.

Experimental studies have shown that alternating current is more dangerous than direct current. The consequences of alternating current injury are exacerbated by its ability to produce tetany. Alternating current changes the polarity of the cellular membrane, causing release of acetylcholine at the motor-end-plate.

The incidence of ventricular fibrillation following alternating current injury is inversely proportional to the voltage, whereas the incidence of atrial fibrillation

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Fig. 3. Electrocardiograms during the treadmill test. A: before exercise, the electrocardiogram showed first degree atrioventricular block with right bundle branch block. B: eight minutes after exercise, the electrocardiogram showed escape rhythm and a worsened atrioventricular block with a lower heart rate (50 beat per minute) than before exercise. C: fifteen minutes after exercise, the sinus rate was about 180 beat per minute. D: five minute after rest, 2:1 atrioventricular block had developed.

Fig. 4. Electrophysiologic test. A: baseline intracardiac electrocardiogram showed prolonged AH and HV intervals. HV block was seen in B after isoproterenol intravenous infusion and in C with atrial pacing with a 690 msec pacing cycle length. RA: right atrium, Stim A: atrial stimulation, A: atrial activity, H: his bundle activity, V: ventricular activity.

Fig. 5. Electrocardiogram after permanent pacemaker implantation (A) showed good sensing and pacing of the pacemaker, and the electrocardiogram with the pacemaker off (B) still showed high-degree atrioventricular block 2 month after pacemaker implantation.
and ventricular tachycardia are directly proportional to the voltage. High voltage (500 volt) may cause sudden death from asystole but the more commonly cause is severe tissue damage. Low voltage injury may also cause sudden death, and this is usually from ventricular fibrillation. Household voltage (110-350 volt) is associated with arrhythmias and conduction disturbances in 11-39% of cases, but these type of patients seem particularly prone to ventricular fibrillation. In contrast, atrial fibrillation has been more commonly reported following higher voltage injury.

The conductive tissue is the the tissue responsible for generating and regulating electrical activity within the heart, and it seems most vulnerable to electrical insult. The reasons for this special vulnerability of the sinus and atrioventricular nodes are not clear. The possible reasons are followings. First, most tissues that develop for the generation and regulation of electrical activity have ionic channels, and control of these channels may be disrupted by exposure to alternating current injury. Second, ischemia or infarction following electrical injury appears to predominately affect the distribution of the right coronary artery. This may happen because its close proximity to the surface of the chest makes it vulnerable to electrical injury. The long term effects on conductive tissue and myocardial function are less clearly documented. Pathological, experimental and clinical evidence have disclosed the need for a greater awareness of the potential long term cardiac complications of electrical injury.

This case showed that the atrioventricular node and the right bundle branch can be jeopardized after electrical injury.

REFERENCES