Atrial Fibrillation due to Blunt Cardiac Injury: Case Report

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ABSTRACT
Introduction: Blunt cardiac injury secondary to chest trauma is an important cause of emergency department admissions. It can range from asymptomatic myocardial contusion to significant dysrhythmia, acute heart failure, valvular injury, or cardiac rupture.

Case Report: A 38-years-old patient was admitted to the emergency department with atrial fibrillation (AF) that occurred after a car accident. The patient had chest trauma caused by striking the steering wheel during the accident. The AF rhythm of the patient reverted to the normal sinus rhythm at the 3rd hour after trauma.

Conclusion: Emergency medicine professionals should keep in mind arrhythmias due to blunt chest trauma and take electrocardiograms of patients to be able to provide treatment in time.

Keywords: Atrial fibrillation, cardiac injury, chest trauma

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Introduction
Blunt cardiac injury (BCI) encompasses different clinical circumstances, such as gradual myocardial injuries, great artery injuries, cardiac tamponade, septum or wall ruptures, pump failure, conduction disturbances, and cardiac arrhythmias (1). The incidence of BCI in multiple traumas is 10%, but it ranges between 8% to 71% in blunt chest trauma patients, depending on the different diagnostic criteria. However, the real incidence is not known because there is no gold standard of diagnosis. BCI occurs mainly due to motor vehicle accidents (2).

The incidence of arrhythmias due to cardiac injuries is 24%-73%, whereas arrhythmia without any cardiac injuries is rare, and the accurate incidence is unknown (3). Various dysrhythmias such as atrial fibrillation (AF), ventricular and supraventricular premature beats, supraventricular tachycardia, ventricular tachycardia, and ventricular fibrillations (VF) after BCI have been reported. Cardiac arrhythmias without BCI could also be seen due to the electrical disturbance trauma. These arrhythmias happen after any strike to the anterior face of chest or left precordial area inflicted by the steering wheel, fists, or a ball (4).

In the literature, AF is reported as the most commonly seen arrhythmia in trauma patients. It is frequently a result of the cardiac complications (myocardial contusion, atrial rupture, cardiac tamponade, etc.) after blunt chest trauma (1). As far as we know, AF without any cardiac injury is rarely reported. In this paper, we aimed to present an atrial fibrillation case that occurred after a blunt chest trauma in which no cardiac injury was determined.

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Case Report
A 38-years-old male patient was admitted to our emergency department (ED) after a car accident that happened 1 hour before. The patient explained that he did not wear a seat belt, and the steering wheel struck his chest during the accident. He suffered from chest and knee pain at the ED admission. The patient was conscious, and his Glasgow Coma Scale was 15 points. His blood pressure was 140/75 mmHg, his pulse rate was 123 beats per minute (bpm) and arrhythmic, his respiratory rate was 24/minute, and his arterial oxygen saturation was 95%. On physical examination, there was tenderness and hyperemia on sternum and right 5th and 6th ribs. There were three lacerations that were roughly 2 cm long and involving cutaneous and subcutaneous tissue placed on the right knee. The other examination findings were normal. After the tachyarrhythmia was detected by monitoring and on physical examination, 12-lead electrocardiogram (ECG) was performed immediately. The ECG demonstrated a normal QRS axis but showed AF with a heart rate of 123 bpm (Figure 1). The patient did not have any previous organic disorders, including cardiac disease, hypertension, and arrhythmias. He was stable hemodynamically at the ED admission, so the radiologic studies of trauma were performed. Brain computerized tomography (CT) and cervical, chest, lumbosacral, and pelvic X-rays revealed no pathologic signs. On contrast enhanced thorax CT, we found the fracture of the 5th rib, small pneumothorax, and apical lung contusion in right hemithorax. The right patella sectional fracture was revealed on X-ray. Laboratory studies of the patient showed that the white blood cell count was 20×10³/mm³, hemoglobin was 16.1 g/dL, hematocrit was 47.8%, and platelet was 294×10³/mm³. Other laboratory findings were within normal ranges. Written informed consent was obtained from the patient.

The patient was referred to the cardiology department, and cardiac enzymes were studied. Transthoracic echocardiography (ECHO) was performed in ED, and it revealed normal cardiac functions. Creatinine kinase was 7 μg/mL (normal range, 0-24 μg/mL), myoglobin was 41 μg/mL (normal range, 14.3-65.8 μg/mL), and troponine I was 0.010 μg/mL (normal range, 0.010-0.023 μg/mL). Cardiology department did not suggest any antiarrhythmic drug, but cardiac enzymes, ECG follow-up, and low molecular weight heparin application were suggested. The ECG of the patient reverted to normal sinus rhythm on the 3rd hour after trauma (Figure 2). Oxygen (8-10 L/min) treatment for pneumothorax and daily chest X-ray for evaluating pneumothorax and contusion progression were suggested by a thoracic surgeon. The patient transferred to the orthopedics ward for his right patellar fracture. He was operated and discharged on the 13th day after full recovery. During this time, cardiac enzymes, cardiac rhythm, and ECG follow-up were normal. After discharge, on the 1st and 6th month, ECG showed normal sinus rhythm each time during control examinations.

Discussion
Cardiac arrhythmia cases were reported as a posttraumatic complication in the literature. In patients with posttraumatic arrhythmia, clinicians primarily consider cardiac injuries such as myocardial contusion, cardiac failure, and cardiac tamponade. However, various cardiac arrhythmias without BCI can also be seen due to the electrical disturbances that occur after trauma (1, 3). In this paper, we discussed an AF case that occurred after a car crash. No cardiac injury was determined, and the rhythm reverted to normal sinus rhythm spontaneously on the 3rd hour after the accident without the use of any antiarrhythmic drug.

There are a lot of factors that can cause cardiac arrhythmia in trauma patients, such as abnormal tissue perfusion, vagosympathetic...
Cardioversion and anticoagulant therapy was not performed in our ED due to an increased damage size and hemorrhage risk (3, 5). In our case, we did not reveal any cardiac damage and great artery injury in ECHO and dynamic-contrast-enhanced CT. Furthermore, during the first 24 hours, cardiac enzymes (troponin I, creatinine kinase, creatinine kinase MB) showed that the myocardial injury did not increase. Thus, we speculated that AF occurred secondary to the disturbance of the electrical conduction system.

Any mechanical stress to cardiac tissue can affect the cardiac electrical system. The activation of mechano-sensitive ion channels leads to early ventricular depolarization (6). If the depolarization coincides with sensitive time of cardiac cycle, ventricular fibrillation may be triggered. Similarly, if atrial pressure and wall tension increase, mechano-sensitive ion-channels will be activated, and AF will occur (7). In previous studies, it was reported that if a low-energy chest wall impact occurs during the vulnerable window of atrial repolarization, the impact-induced elevated atrial pressure will trigger a premature atrial depolarization and the initiation of AF, similarly to the VF-triggering mechanism (8). In our case the patient did not wear a seat belt, and he sprang forward during crash so that the steering wheel struck the sternum and the right face of the chest. After the strike, pain and hyperemia in the sternum, fracture of the right 5th rib, pneumothorax, and lung contusion occurred. The AF rhythm was initiated within the first 30 minutes. In our case, the mechanism of AF was probably either of them, but we cannot determine exactly which one.

Arrhythmias generally develop within the first 2 hours after trauma, like in our case, or 24 to 48 hours after trauma. Also, some arrhythmias may rarely develop day after day. Therefore, the ECG monitoring should be done for 2 days following trauma (9, 10). For this reason, we followed up the patient for at least 48 hours.

Posttraumatic cardiac injury might be detected by using ECG, ECHO (especially transesophageal), cardiac enzymes, and contrast enhanced CT. In a study, it was reported that a normal sinus rhythm ECG had a 98% negative predictive value in blunt chest trauma patients. Creatinine kinase, creatinine kinase MB, troponine I, and troponine I are diagnostic markers of cardiac damage, but the most sensitive marker is troponine I. The patient whose ECG and cardiac markers are normal can easily be discharged from ED. If there is an abnormal rhythm on ECG and suspected myocardial injury, 24-hour monitoring, serial physical examinations, and cardiac enzymes levels follow-up are suggested (11). The management of trauma-caused AF is the same as non-traumatic AF. Asymptomatic and hemodynamically stable AF patients like in our case could revert to normal sinus rhythm without any treatment. If the rhythm does not recover spontaneously, antiarrhythmic drugs are suggested. In stable patients, cardioversion and anticoagulation should not be started in ED due to an increased damage size and hemorrhage risk (3, 5). Cardioversion and anticoagulant therapy was not performed in our case because the patient was stable. Low molecular weight heparin was applied as the prophylaxis of venous thrombosis.

Conclusion
Patients who are admitted to ED due to trauma, especially due to multiple trauma after a car accident, should have their ECG promptly evaluated. If a patient suffers blunt chest trauma, the patient must be monitored, and ECG and cardiac enzymes should be followed up for at least 24 hours. Thus, arrhythmias will be treated on time and clinicians will not miss cardiac injuries and arrhythmias.

Informed Consent: Written informed consent was obtained from the patient who participated in this study.

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