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Atrial Fibrillation due to Blunt Cardiac Injury: Case Report

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ABSTRACT

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> 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. BCIs occur 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.

This study was presented in 3rd National Emergency Medicine Congress, 2016, Antalya, Turkey.

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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

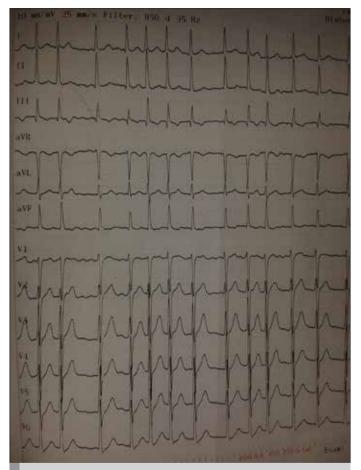


FIGURE 1. The initial ECG of patient which showed atrial fibrillation rhythm.

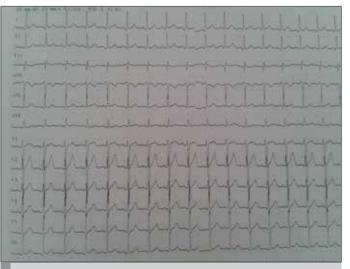


FIGURE 2. The normal sinus rhythm ECG of patient on the 3rd hour after the trauma.

the ED admission. The patient was conscious, and his Glasgow Coma Scale was 15 points. The patient's 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), myoglobine 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 stimulus, electrical gradient difference between normal and injured tissue, aberrant conductions occurred after myocardial damage, electrolyte imbalance, catecholamine discharge, pain, and anxiety. Besides, myocardial local hypoxia and ischemia due to increased intravascular rouleaux formation were also thought to be the cause of arrhythmias (5, 6). 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 (troponine 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 T, 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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References

- El-Chami MF, Nicholson W, Helmy T. Blunt cardiac trauma. J Emerg Med 2008; 35: 127-33. [CrossRef]
- Feghali NT, Prisant LM. Blunt myocardial injury. Chest 1995; 108: 1673-77. [CrossRef]
- Ismailov RM, Ness RB, Redmond CK, et al. Trauma associated with dysrhythmias: results from a large matched case-control study. J Trauma 2007; 62: 1186-91. [CrossRef]
- Ota K, Bratincsak A. Atrial Fibrillation Induced by Commotio Cordis Secondary to a Blunt Chest Trauma in a Teenage Boy. Pediatrics 2015; 135: 199-201. [CrossRef]
- Darok M, Beham-Schmid C, Gatternig R, Roll P. Sudden death from myocardial contusion following an isolated blunt force trauma to the chest. Int J Legal Med 2001; 115: 85-9. [CrossRef]
- Bode F, Franz MR, Wilke I, Bonnemeier H, Schunkert H, Wiegand UK. Ventricular fibrillation induced by stretch pulse: implications for sudden death due to commotio cordis. J Cardiovasc Electrophysiol 2006; 17: 1011-7. [CrossRef]
- Ninio DM, Saint DA. The role of stretch-activated channels in atrial fibrillation and the impact of intracellular acidosis. Prog Biophys Mol Biol 2008; 97: 401-6. [CrossRef]
- Ellis DY, Hutchinson NP. Synchronized direct current cardioversion of atrial fibrillation after blunt chest trauma. J Trauma 2000; 49: 342-4. [CrossRef]
- Illig KA, Swierzewski MJ, Feliciano DV, Morton JH. A rational screening and treatment strategy based on the electrocardiogram alone for suspected cardiac contusion. Am J Surg 1991; 162: 537-44. [CrossRef]
- 10. Sakka SG, Huettemann E, Giebe W, Reinhart K. Late cardiac arrhythmias after blunt chest trauma. Intensive Care Med 2000; 26: 792-5. [CrossRef]
- Velmahos GC, Karaiskakis M, Salim A, Toutouzas KG, Murray J, Asensio J, et al. Normal electrocardiography and serum troponin I levels preclude the presence of clinically significant blunt cardiac injury. J Trauma 2003; 54: 45-50. [CrossRef]