Commotio Cordis as a Rare Cause of Traumatic Cardiac Arrest in Motorbike Crashes: Report of a Case

CHUN-CHIEH YEH, CHI-HSUN HSIEH, YU-CHUN WANG, PING-KUEI CHUNG, and RAY-JADE CHEN

Department of Surgery, Trauma and Emergency Center, China Medical University Hospital, No. 2 Yuh-Der Road, Taichung 404, Taiwan

Abstract
It is futile to attempt resuscitation in a blunt injury patient with no vital signs upon arriving at the emergency department. Therefore, it is recommended that resuscitation be withheld in any blunt trauma patient without vital signs while emergency medical technicians arrive at the scene of the accident. This report presents a case of a blunt torso trauma patient who lost vital signs at the scene and still received cardiopulmonary resuscitation until recovery of spontaneous circulation at the emergency department. The patient was later diagnosed with commotio cordis, and survived to be discharged without any neurological sequelae. Therefore, aggressive resuscitation should be continued until a diagnosis and differential diagnosis of blunt trauma-related cardiac arrest are made by a thorough examination in the emergency department.

Key words Commotio cordis · Traumatic cardiac arrest · Blunt torso trauma · Motorbike crash

Introduction
Due to advances in prehospital care of the critically injured, the number of transferred trauma patients in extremis has increased gradually in recent decades. Even under aggressive resuscitation, it was very rare to save a blunt injury patient diagnosed with traumatic cardiac arrest when arriving at the emergency department. It is therefore recommended that resuscitation may be withheld in any blunt trauma patient without vital signs while emergency medical technicians (EMTs) arrive at the scene of the accident. However, there is a rare subtype of blunt trauma-related disease, commotio cordis, which can lead to sudden death in young people injured by a blast or blunt trauma to the anterior chest. Patients may be saved with timely resuscitation and electric defibrillation. This report describes one patient diagnosed with commotio cordis in a motorbike crash. The patient lost his vital signs at the scene, and survived to be discharged without any neurological sequelae.

Case Report
A 17-year-old male patient suffered from blunt torso trauma in a motorbike crash where he had been hit by the handlebar of a scooter. Emergency medical technicians arrived at the scene 7 min later. No vital signs were detected at the scene and the EMTs still performed prehospital cardiopulmonary resuscitation (CPR) immediately. En route to the hospital, the first documented cardiac rhythm was a ventricular fibrillation (Fig. 1), and electric defibrillation was performed twice (180J biphasic waveform defibrillation) with the assistance of an automated electric defibrillator (AED). Asystole was noticed after electric defibrillation, and chest compression was continued for more than 11 min in the ambulance. Upon arrival at the emergency department, there were no detectable vital signs including pulse, respiratory effort, or organized cardiac electric activity. The patient was intubated and given 1500 ml crystalloid. No blood transfusion was attempted before vital signs returned, due to the expected poor prognosis of the patient with traumatic cardiac arrest in a blunt torso trauma. The initial laboratory data showed extreme metabolic acidosis (arterial blood gas analysis: pH 6.992; HCO₃⁻ 8.3 mmol/l; PaCO₂ 33.9 mmHg; base excess, –21.3 mmol/l), elevated liver enzymes (glutamic
oxaloacetic transaminase 154 IU/l), creatine phosphokinase 440 IU/l, creatine phosphokinase MB form (CPK-MB) 117 IU/l (reference value 3–10 IU/l), troponin I 15.5 μg/l (reference value <0.5 μg/l), and hematocrit (42.6%). Because of the stable hemodynamic status following resuscitation, whole-body computed tomography was performed. A right renopedicle injury with contrast extravasation was detected, but the hematoma was mainly confined in the retroperitoneal space (Fig. 2). Since it was indicated to explore the retroperitoneal zone II hematoma based on the contrast pooling at the right renopedicle region, a celiotomy was performed immediately. A tear was found near the junction of the right renal vein and inferior vena cava. A right nephrectomy was performed directly without any attempt to preserve it in order to achieve definite damage control in such a critical condition. Temporal abdominal closure with a Bogota bag was performed to prevent subsequent abdominal compartment syndrome. During the patient’s stay in the intensive care unit (ICU), the levels of troponin I and CPK-MB continued to rise until the 3rd day. There was no structural abnormality or functional impairment detected on the following cardiac ultrasonography in the ICU. The patient was discharged on the 20th day, without neurological sequelae. The Acute Physiology and Chronic Health Evaluation II (APACHE II) score was 35. The total Injury Severity Score (ISS) was 25, and the Trauma and Injury Severity Score (TRISS)-associated mortality rate was 99%.

Discussion

A tear on the right renal vein with contrast extravasation was evident, but the extravasated blood was confined in the retroperitoneal space, which meant that the estimated blood loss was limited (Fig. 2). Besides, before regaining vital signs successfully, there had been only 1500 ml Ringer lactate solution administered. Therefore, hypovolemia-related cardiac arrest could be ruled out by the small resuscitation volume. However, blunt torso trauma is often associated with a higher frequency of extra-abdominal injury. Therefore, the physician searched for other extra-abdominal injuries, such as cardiac tamponade, cardiac rupture, tension pneumothorax, or cardiac contusion with fatal dysrhythmia, as the cause of the traumatic cardiac arrest. Cardiac tamponade, cardiac rupture, or tension pneumothorax were easily ruled out with an image study and physical examination in this case. However, no evidence of structural abnormality or functional impairment on cardiac ultrasonography and ventricular fibrillation recorded in the ambulance at the outset were clues to blunt trauma-related fatal dysrhythmia, which was compatible with commotio cordis (Fig. 1). Moreover, according to the findings of cardiac ultrasonography in this patient, the elevated levels of troponin I and CK-MB could be the result of shock-related insufficient coronary perfusion before the restoration of spontaneous circulation, but not due to structural damage to the heart.

In 1992, Mattox recommended that “blunt cardiac injury” be combined with specific descriptions for more definite identification of the injury and its sequelae. Complications of blunt cardiac injury include minor electrocardiographic (ECG) or enzyme abnormalities,