Delayed Presentation of Blunt Traumatic Rupture of the Left Ventricle: Successful Surgical Repair

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Abstract
Blunt traumatic cardiac rupture is rare and is usually associated with a high mortality rate. A case of delayed cardiac rupture with survival is described in a woman sustained blunt chest trauma during a motor vehicle accident. Preliminary investigations at the time of admission did not show any evidence of hemopericardium. The patient deteriorated suddenly 48 hours postinjury, due to a delayed left ventricular rupture which was successfully repaired. Reports of survival after this type of blunt cardiac injury are rare since survival rates are low. Early diagnosis is essential to optimize the patient’s opportunity for survival.

Keywords: Delayed cardiac rupture; Blunt trauma; Emergent thoracotomy; Delayed repair

Introduction
Cardiac rupture secondary to blunt trauma is encountered in 0.045% of patients who present to the hospital alive, [1] and has an overall survival of only 10% [1]. It is found in more than a third of patients who sustain fatal blunt thoracic trauma [2]. This suggests that while blunt traumatic cardiac rupture is a relatively uncommon clinical finding, it is associated with a high mortality rate. A case of blunt chest trauma is presented where the preliminary investigations at the time of admission did not show any evidence of cardiac injury, but the next day the patient deteriorated suddenly due to a delayed rupture of the left ventricle which was successfully repaired [3–6].

Case Description
A 76-year-old female was the restrained driver in a front end motor vehicle crash. She arrived in the trauma receiving area awake, alert and hemodynamically stable. The physical examination was unremarkable except for a seatbelt sign on the right side of her chest. There was no evidence of rib fractures or sternal tenderness. The chest X-ray demonstrated a normal mediastinal width, normal aortic notch, midline trachea and no evidence of rib fractures. Computed tomography of the brain and abdomen were unremarkable. The patient was admitted to the floor for observation.

On the second post-injury day the patient’s condition started deteriorating. She was hypotensive, diaphoretic and tachycardic. Re-assuscitation was initiated and she was transferred to the surgical intensive care unit. An electrocardiogram demonstrated decreased voltage in all leads. The pulmonary capillary wedge pressure was 2 mmHg. A bedside echocardiogram demonstrated a large pericardial effusion and a defect at the apex of the heart. She was taken to the operating room where she underwent a left anterior thoracotomy and pericardiectomy. The anterior wall of the left ventricle was found to have a 3 cm defect which was closed with horizontal mattress sutures. Cardiopulmonary bypass was not required for the procedure. Postoperatively the patient remained hemodynamically stable despite a hospital course complicated by a calculous cholecystitis and pneumonia. She was discharged to a rehabilitation facility on her 67th postoperative day.

Discussion
The rate of cardiac injury following blunt thoracic trauma varies widely and is estimated to be around 15% [7]. Blunt cardiac injuries are a broad diagnostic spectrum that includes myocardial contusion, pericardial or coronary artery laceration, valvular injury and acute rupture of the papillary muscles, atria or ventricles [8]. Cardiac rupture is the most extreme manifestation of blunt cardiac injury with the majority of patients dying before receiving medical attention, and 81% mortality in those that arrive to the emergency department [9]. However improvements in prehospital care and rapid transport have resulted in blunt cardiac rupture patients arriving at the hospital for evaluation.

Motor vehicle accidents are the most common cause of acute cardiac rupture after blunt trauma, followed by motorcycle crashes, pedestrians struck by automobiles, crush injuries and falls [8]. Classically these patients arrive with symptoms that characterize cardiac tamponade or exsanguination, but patients can have a varied clinical presentation. The circumstances of the injury may be the only clue to such an injury given the absence of external signs of chest trauma.

Sometimes blunt traumatic injury to the chest wall is confined to the soft tissues, leaving the rib cage and sternum intact [10]. In a retrospective review, Nan et al. proposed an algorithm for the diagnosis and treatment of blunt cardiac rupture, [11] but to date there is no standardization on how to diagnose such an injury and as such, it remains associated with a high mortality rate. Even the diagnosis of cardiac contusion can prove difficult as such lesions are usually well tolerated and clinically significant findings may be absent or transient. Electrocardiogram changes and the reliability of cardiac enzyme levels has long been the subject of considerable debate [7].

The pathophysiology of blunt cardiac rupture is directly related to one of several potential mechanisms of injury: (a) direct impact or blow to precordium resulting in cardiac compression between chest wall and vertebral column; (b) momentum injury caused by rapid deceleration resulting in tears secondary to shearing forces occurring at the attachment of free and fixed points of the movable intrathoracic viscera [12]. Additionally, the presence and type of blunt cardiac rupture will vary depending upon the cardiocycle and the volume of blood present in the heart at the time of injury [13].

Delayed blunt cardiac rupture has been postulated to originate as a myocardial contusion sustained during high-speed deceleration [7].
Severe precordial impaction to the chest wall or compression of the heart between the spine and anterior chest wall initiates the myocardial contusion. Myocardial contusion is characterized by patchy areas of muscle necrosis and hemorrhagic infiltrate that often disrupt the muscle fibers [8]. While usually proceeding along a benign course, myocardial contusions can have any number of sequelae, especially if progression to transmural necrosis occurs. Subsequent rupture of contused, necrotic myocardial tissue is relatively rare and is indicative of a severe and extensive myocardial injury. On autopsy a delayed rupture is identified as ragged lacerations with necrotic edges [7]. This typically occurs during the second week after the initial injury but may be delayed longer if an aneurysm forms [8].

DesForges published the first report of a successful blunt cardiac rupture in 1955, which involved the right atrium. Given its thin wall and more anterior position, the right atrium is the most common site for cardiac rupture in survivors of blunt trauma, with an incidence of 40-50% [9]. Rupture of the left ventricle, as was repaired in the case presented, is the least frequently seen site for cardiac rupture, with an incidence of 9-13% [9]. Left ventricular rupture was identified as the most common cardiac injury pattern identified among mortalities after blunt thoracic trauma [3]. Blunt trauma resulting in a delayed left ventricular rupture is exceedingly rare, and the literature is scant, with only two case reports of successful repair [5,6]. Successful surgical repair and survival of these patients remains uncommon.

**Conflict of Interest**

The authors report no conflicts of interest.

**References**


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