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Cardiac injuries caused by trauma: Review and case reports

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ABSTRACT

Assessment of suspected cardiac injuries in a trauma setting is a challenging and time-critical matter, with clinical and imaging findings having complementary roles in the formation of an accurate diagnosis. In this article, we review the supporting literature for the pathophysiology, classification and evaluation of cardiac injuries caused by trauma. We also describe 4 cardiac trauma patients seen at a tertiary referral hospital.

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1. Introduction

Trauma is a major problem in health care and is the leading cause of death and disability in those younger than 45 years in developed countries.[1] The reported incidence of cardiac trauma varies greatly, from 8% in an autopsy study to 76% in a clinical series; this variance results from a lack of standardised diagnostic criteria.[2] Nevertheless, cardiac trauma is highly lethal, and cardiac injuries are the second leading cause of death among trauma victims, after central nervous system injuries.[3]

The primary site of myocardial injury is the right ventricle (RV) free wall, due to its anterior location within the thoracic cavity. The mitral and aortic valves are at greater risk for injury than the tricuspid and pulmonic valves, as mural pressure is higher on the left side of the heart.[1]

Cardiac trauma may be classified, based on the mechanism of injury, into 2 broad categories: non-penetrating (also referred to as blunt cardiac injury [BCI]) and penetrating.

An online database query was performed using the PubMed medical database. All relevant articles from the past 20 years were reviewed.

2. Blunt cardiac injury

BCI refers to damage sustained from blunt thoracic trauma and is the most common type of cardiac trauma.[4] Most BCIs occur with motor vehicle crashes (50%), followed by pedestrians being struck by motor vehicles (35%), motorcycle crashes (9%) and falls from a significant height (6%).[1] BCI is often part of multisystem trauma and is most commonly associated with other thoracic injuries such as rib fracture, sternal fracture, pneumothorax, haemothorax and lung contusion. External thoracic injury may not always be present, and a significant BCI may be seen in the absence of external signs of thoracic trauma.

BCI is the preferred generic term to refer to a non-penetrating cardiac injury. The term 'myocardial concussion' refers to a subset of BCI that includes wall-motion abnormalities with no proved anatomic or cellular injury. In contrast, the term 'myocardial contusion' denotes an anatomic injury or tissue damage demonstrated at surgery or autopsy, or manifested as increased levels of myocardial necrosis markers.[4]

An autopsy based assessment showed that the most common lethal cardiac injuries caused by blunt trauma were transmural rupture of cardiac chambers (64%), tears occurring at the venous-atrial confluence (33%), and coronary-artery dissection (2%).[5]

2.1. Myocardial rupture

Complete free-wall rupture, mostly of the RV, is usually fatal,

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with death occurring at the trauma scene.[2] The few patients who survive present to the emergency department with signs of profound hypotension or pericardial tamponade. A pseudoaneurysm can develop if the ruptured myocardium is sealed off by the pericardium and an organised thrombus; this pseudoaneurysm has a high risk of spontaneous rupture.[6] A traumatic ventricular septal defect (VSD), typically within the membranous portion of the septum, may occur immediately after the traumatic event or within the first few days after injury; depending on the size of the defect, there may be little haemodynamic change or it may cause cardiogenic shock.[7]

2.2. Tears at the venous-atrial confluence

A distraction-avulsion injury at the venous-atrial confluence is believed to result from rapid deceleration in the freely mobile ventricles, while the posterior veins remain fixed in place. This type of injury usually occurs at the junction of the inferior vena cava and the right atrium, and at the junction of the pulmonary veins with the left atrium.[1] This is a highly lethal injury, but small tears at the venous-atrial confluence can be contained, and the patient may be initially stable.[8]

2.3. Valvular injury

Mitral-valve injuries usually result from the rupture of a papillary muscle, a chordae or a leaflet.[9] Tricuspid-valve involvement has the same injury pattern but is less frequent and has a subtler clinical presentation. Severe tricuspid regurgitation can actually be well tolerated and may only be diagnosed months or years later.[1] Aortic-valve injuries range from mild annulus damage to laceration or detachment of the cusps leading to acute aortic regurgitation.[10] Aortic-valve involvement is often associated trauma to the with ascending aorta.[11] Pulmonary-valve injuries are rare, with very few cases of valve disruption described.[5]

2.4. Coronary-artery injury

Acute dissection of the coronary arteries can result from BCI, leading to myocardial infarction.[12] The pathophysiology of this injury tends to be direct impact, most frequently over the left anterior descending artery or the left main coronary artery, usually in a previously diseased portion of the vessel.[13] The dissection is initiated as the intima over the plaque tears, creating a flap that obstructs the blood flow.[14]

2.5. Thoracic-aorta injury

The mechanisms of blunt injury to the thoracic aorta include rapid deceleration, production of shearing forces and direct luminal compression against points of fixation, especially at the ligamentum arteriosum.[15] The aortic isthmus (junction of the mobile aortic arch and the fixed descending aorta) is the most common site of aortic injury. Many patients die of vessel rupture and rapid exsanguination, either at the scene or before reaching medical care. Other resulting injuries include mural hematoma formation, intimal tear, transection, pseudoaneurysm, and dissection.[2]

2.6. Dysrhythmias

Premature ventricular and atrial contractions are the most commonly seen dysrhythmias, but life-threatening patterns may also occur.[2] Atrial fibrillation was reported on the initial electrocardiogram (ECG) in 4% of patients with chest trauma,[16] but this arrhythmia may not be due to direct cardiac trauma because a similar incidence was shown in isolated head or abdominal injuries.[17] The development of right bundle branch block after BCI has long been reported and should raise suspicion for cardiac involvement, but it has little clinical relevance by itself, even when persistent.[16]

Commotion cordis refers to sudden death caused by ventricular fibrillation, triggered by a blunt blow to the chest, in the absence of underlying cardiovascular disease. The trauma occurs without anatomic damage to the ribs, sternum or heart, most often in children and young adults participating in recreational and competitive sports.[18] Transmission of the impact to the myocardium during cardiac repolarization, an electrically vulnerable phase of ventricular excitability, induces ventricular fibrillation.[19] Using personal chest barriers appears to be not totally protective, as athletes wearing these still die.[20]

3. Penetrating cardiac injury

In penetrating chest trauma, both ventricles are injured with similar frequency, but the RV is the most common site of entry because it forms most of the anterior surface of the heart.[3] The most frequent trauma scenario is a young man who presents with a precordial stabbing or thoracic gunshot wound.[21] The initial manifestation is commonly cardiac tamponade and, depending on the haemodynamic stability of the patient, immediate surgical intervention may be necessary without further evaluation.

Penetrating cardiac injury is one of the most lethal medical emergencies, with an estimated pre-hospital mortality rate of 94% and a subsequent in-hospital mortality rate of 50% among initial survivors.[22] The 2 most significant causes of death, as reported in autopsy studies,[23] are haemorrhagic shock and cardiac tamponade.

4. Diagnostic modalities

The symptom-based diagnosis of cardiac trauma, particularly in non-penetrating injury, is usually difficult to make because the symptoms, such as chest pain, are common in patients with thoracic trauma and often arise from a non-cardiac source. Signs of congestive heart failure, pulmonary oedema, a pericardial friction rub or a new cardiac murmur are less common but, when present, should prompt evaluation for cardiac injury.[2]

ECG should be routinely performed in all patients with suspected cardiac trauma. Although it has low sensitivity and specificity when used alone,[24] patients with abnormal ECG findings had more significant complications that required treatment.[25]

Cardiac biomarkers, namely serum cardiac troponin levels, also have an important role in cardiac-trauma screening. High-sensitivity cardiac troponin assays with improved analytical performance have lower limits of detection, allowing patient with blunt chest trauma to be safely discharged home if their levels are normal.[26] However, even these novel assays have shown cross-reactivity with diseased skeletal muscle.[27] In other words, elevated cardiac troponin in a patient with chest trauma may originate from skeletal-muscle injury, not necessarily from cardiac trauma or from associated ischaemic injury.

Transthoracic echocardiography (TTE) is a very useful tool for detecting BCI and represents the primary screening tool in unstable patients with either penetrating or non-penetrating chest trauma. Transoesophageal echocardiography may help to define intracardiac anatomy and function in some cases, such as when traumatic valve injury or a ruptured septum is present.[28]

Thoracic computed tomography (CT) allows an accurate examination of the entire chest, including the heart, pericardium and great vessels. CT is currently recommended as the initial imaging modality in haemodynamically stable trauma patients.[3]

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