Challenges in the diagnosis of blunt cardiac injuries

Dipti Agarwal · Subhash Chandra

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Abstract

Introduction Blunt thoracic injuries (BTIs) are directly responsible for 20–25% of all deaths, worldwide. Involvement of heart in BTIs is largely underestimated and ignored, but reasonable estimate would be around 15%. This study was planned to emphasize on clinical-presentation and diagnosis of blunt cardiac injuries (BCIs).

Results Clinical presentation of BCIs, varied from mild chest discomfort to haemodynamic shock secondary to rapid exsanguinations. Non-specific presentation with associated injuries diverts physician's attention and delayed appearance of clinical features makes diagnosis further difficult. Cardiac markers and ECG are not specific, but high sensitivity of 100% could be reached using combination of elevated cardiotroponin levels and alterations in ECG. Transoesophageal or transthoracic echocardiography, angiography, intravascular ultrasound and nuclear scan have proven to detect cardiac injuries in BTIs, but lack specificity.

Conclusion Patients with suspicious-ECG finding need cardiac-monitoring for at least 24 hours. Haemodynamically stable young (<55 years) subjects, without underlying cardiac diseases and with normal-ECG and cardiac marker, could be discharged safely.

Keywords Blunt cardiac injuries · Cardiac rupture · Cardiotroponin · Electrocardiography

Introduction

Blunt thoracic injuries (BTIs) include spectrum of injuries caused by non-penetrating mechanism. Majority of it is result of motor vehicle accidents (MVAs) but other common causes include sports, falls and assault injuries. Unintentional injuries remain the 5th leading killer of in the United States of America contributing 117,809 deaths [1]. In India, MVAs is the 8th leading cause of death in all age groups and contributes to 2% of deaths from all causes and decreases life expectancy by 2-year [2]. BTIs are becoming more of importance in general practice due to increasing incidence of MVAs. A heightened level of suspicion and early identification of the blunt cardiac injury (BCI) is vital. Reliable detection however is challenging, as there are still no clear cut diagnostic criteria for BCI [3].

Hence, the present study was focused to determine clinical challenges and pit falls in diagnosis of BCIs in blunt chest trauma.

Definitions

“Blunt thoracic injuries”, any injury sustained to the chest from blunt force, which may be related to MVAs, or mis-haps, falls or jumps, blows or crush injuries from animals, blunt objects or unarmed assailants. BCI is injury to heart by non-penetrating mode. The combination of impact against a steering wheel followed by sudden deceleration is the commonest cause. “Myocardial contusion” was used as a general term encompassing the gamut of myocardial

D. Agarwal¹ · S. Chandra²

¹Mayo Clinic, Rochester, MN, USA

²All India Institute of Medical Sciences,
New Delhi, India

S. Chandra (✉)

E-mail: subhash_doc84@yahoo.com

injuries. The lack of specification in use of this term leads to its variable prevalence reporting. It implies to necrosis with or without haemorrhage into the myocardium [4]. Myocardial rupture (or heart rupture) is a laceration or tearing of the walls of the ventricles or atria of the heart, of the interatrial or interventricular septum, of the papillary muscles or chordae tendineae or of one of the valves of the heart.

Epidemiological patterns

Traumatic myocardial injuries present in up to 55% of BTIs, though, the rate of cardiac injury varies widely, depending on diagnostic criteria [5]. The commonest form of cardiac injury is myocardial contusion. The reported incidence depends on the diagnostic modality and criteria used and ranges from 5–71% [6–9]. Whereas, most catastrophic is myocardial rupture. Parmley et al. [9] from the United States of America reported an autopsy study of 546 non-penetrating cardiac trauma cases in 1956, found that 64% had at least one chamber rupture, 20% had two or more, 23% had myocardial contusion, 6% had pericardial lacerations, 4% had papillary muscle rupture, 2% had valvular rupture, and 1% had a coronary artery laceration [9]. For patients with blunt traumatic rupture of the heart, the risk of having more than one chamber rupture is over 20% [10]. Among the patients with rupture of cardiac chamber, a few were alive [11].

In a series reported by Kulshrestha et al. [12] 1,012 patients have sustained BTIs of various magnitude and 102 had cardiac injuries among 3,653 trauma death autopsies; 141 (13.9%) died primarily of chest injuries (thoracic deaths) and out of thoracic deaths, 44 (31.2%) were result of cardiac injuries [12]. Of 102 cardiac injuries, 33 patients died on the spot, 58 died during transportation and only 11 reached hospital [12]. Cardiac injuries accounted for 3.3% and 8.2% of total chest injuries in clinical and autopsy groups, respectively [12]. The lower incidence of cardiac injuries in the clinical series is due to higher pre-hospital mortality in cardiac injuries compared with chest injuries without cardiac involvement. Twenty-four percent of chest injuries with cardiac involvement reached to hospital where as only 10.8% of patients with cardiac injuries reached to hospital [12].

Since there are no standard diagnostic criteria for BCI, the true incidence of BCI remains unknown [13].

Clinical presentation

Cardiac injuries with BTI have variable clinical presentation. Signs and symptoms range from being asymptomatic to mild chest discomfort, shortness of breath, anxiety, cyanosis and palpitations to signs of cardiac tamponade

(e.g. muffled heart tones, jugular venous distension (JVD) and hypotension) and haemodynamic shock due to rapid exsanguinations. The most common symptom is non-specific chest pain arising from non-cardiac chest structures [14]. BCIs are result of high-velocity impact blunt injuries, which invariably gives wounds to thoracic cage. Physical exam findings concerning for cardiac injuries are often the result of cardiac dysfunction, and they are typified by dysrhythmias, global cardiac ischaemia, or low cardiac output and hypotension [11]. The findings relevant to cardiac injury are hypotension, JVD, tachypnea, wheezing, rhonchi, chest abrasion or ecchymosis, imprints of a seatbelt or steering wheel, chest tenderness, palpable crepitus, flail chest, fractures of the rib(s), sternum or clavicle, distant heart sounds, S3, tachycardia, bradycardia, rub or murmurs [15].

Clinical course

The spectrum of BCI ranges from clinically insignificant myocardial contusion to full thickness cardiac rupture. There are two distinct clinical courses of significant BCI;

- a) Acute type: Acute type is result of rupture of a chamber, which is highly fatal and leads to immediate loss of life or in the next few hours, if not treated immediately [16].
- b) Subacute type: Diminished cardiac reserve of contused heart places patient at the risk of death secondary to cardiovascular complications remote from the site of injury [16].

Outcome

Major outcomes of BCIs and course of injury are shown in Fig. 1, which range from uneventful resolution to death at the scene or delayed death [17–26]. Immediate death from rupture is due to tamponade, extracardiac hemorrhage or intracardiac shunt.

Pathophysiology

BCIs are result of high impact BTIs. Following are the mechanisms which can lead to BCIs.

- Direct precordial impact
- Crush injury resulting from compression between the sternum and spine
- Deceleration or torsion causing a tear in the heart at a point of fixation, such as between the right atrium and vena cava
- Hydraulic effect resulting in rupture, such as that seen during an abrupt abdominal compression that results in significantly elevated venous pressure that is transmitted to the right atrium or ventricle

Fig. 1 Outcome of BCIs; trivial to death at the scene [17–26].

- Blast injury [7, 16, 27, 28].

BTIs can also lead to penetrating cardiac injuries in the form of broken rib or sternum can lacerate the heart [8, 25]. The risk to the valves varies with the different events of the cardiac cycle. Atrioventricular (AV) valves are most commonly injured in isovolumetric contraction stage secondary to sudden rise in ventricular pressure, when valves are closed [29]. In early diastole, the aortic valve is vulnerable to injury because of lack of support by otherwise empty left ventricle and retrograde aortic wave produced due to chest or abdominal compression. In early systole, a competent AV valve may be damaged because of an increase in ventricular pressure [30].

Histopathology

As said before, contusion is the commonest cardiac injury, the true diagnosis of it can only be established by direct inspection of myocardium [4]. Contused myocardial tissue is histologically characterised by intramyocardial haemorrhage, oedema and necrosis of myocardial muscle cells, which are similar to the findings of acute myocardial infarction (MI) [4]. Nevertheless; there are distinct differences between myocardial contusion and infarction [4, 31, 32].

- In contusion, there is a distinct boundary between the normal and contused tissue, whereas in MI there is gradual transition from the normal tissue.

- Moreover, contusion is usually confined to the traumatised muscle bundles, whereas infarction is distributed along the vascular supply of the infarct-related artery.
- Also, in the former there is patchy necrosis and haemorrhage in a pyramidal fashion, whereas infarction causes generalised coagulation necrosis.
- Finally, the repair of cardiac contusion is usually in the form of patchy and irregular fibrosis, contrary to the generalised fibrosis after MI.

Myocardial contusion is often associated with injury to coronary artery possibly in the form of dissection, spasm, thrombosis or laceration which leads to MI; lesser damage could lead to delayed stenosis or no sequelae at all. Very severe injuries results in cardiac rupture, which include free wall, septum, valves or pericardium.

Laboratory investigation

Despite numerous investigations, no consensus has been reached on how to approach patients with suspected or manifest myocardial injury in BTI.

Radiography

Chest X-ray, which is routinely obtained for BTIs, serves as a screening test rather than being a definitive test. It

may detect fractured ribs, flail chest and globular shaped cardiac silhouette, but is missed many times in plane X-ray and picked up with greater detail on computed tomography [33].

Electrocardiograph (ECG)

Admission or emergency room 12-lead ECG for all patients suspected of having BCI has level-1 recommendation [34]. If on admission ECG is abnormal (arrhythmia, ST changes, ischaemia, heart block, unexplained ST), the patient should be admitted for continuous ECG monitoring for 24–48 hours [34]. Most commonly ST segment elevation changes represent myocardial contusion [35]. However, significant ST segment elevation changes may be caused by coronary artery spasm, thrombosis, dissection of the aorta with propagation to the coronary arteries, or coronary artery dissection [35]. Unfortunately, no single

ECG finding is pathognomonic for BCI [36]. Non-specific changes, including sinus tachycardia, are present in upto 80% of the ECGs obtained in patients with suspected BCI [36, 37]. Dysrhythmias requiring treatment also can be detected by the admission ECG. Using any ECG changes, including sinus tachycardia, bradycardia, conduction delays, or atrial or ventricular dysrhythmias, can provide a sensitivity of 100%, a specificity of 47%, and a negative predictive value of 90% in the detection of BCI-related complications that require treatment [37]. The most common finding is sinus tachycardia, followed by premature atrial or ventricular contractions [13]. As shown in Fig. 2, in young haemodynamically stable patient, a normal ECG precludes the need for further evaluation to detect BCI [14, 20, 25, 38]. However, a small number of patients with a normal ECG can encounter cardiac complications related to BCI [38]. These patients often have other associated finding like unexplained hypotension, old age, multiple chest wounds or underlying cardiac disease.

Fig. 2 Admission ECG interpretation in BCI

Cardiac markers

The cardiac markers are also not very specific, as they merely indicate cardiac necrosis but not the mechanism of injury. It may be because in myocardial contusion injury to myocytes is not sufficient to release, and same markers are also released in injury to skeletal muscles. Historically, creatine kinase (CK) with myocardial type B (MB) fractions was utilised in order to assist in the detection of BCI or patients at risk for serious sequelae. Illig et al. [39] found that CK-MB analysis failed to predict complications secondary to BCI which was further confirmed by Biffi et al. [39, 40]. Lactate dehydrogenase-1 (LDH-1) to total LDH ratio is raised in all the patients with myocardial death in 24 hours but LDH-1/LDH-2 ratio rises within 9 hours of myocardial insult [41, 42]. Cardiac troponins are most specific amongst conventional markers as they differ from the skeletal isoform by a unique aminoterminal sequence of 31 amino acids never expressed by skeletal muscle [43]. Cardiac troponin-I has shown maximum sensitivity (23–100%) and specificity (85–97%) in detecting clinically significant BCI [34–46]. Velmahos et al. [47] concluded that, 100% negative predictive value was reached on combining ECG with troponin-I at 8 hours. Hence, in the absence of other injuries or haemodynamic instability, patients with normal ECG and troponin-I can be discharged [47].

Echocardiogram

It is an anatomical assessment of heart, detection of pericardial effusion, myocardial contusion, valvular disruption and wall motion abnormalities assist in diagnosis of BCI. Main findings in contusion include wall motion abnormalities in upto 25%, increased echo brightness, and increased end-diastolic wall thickness of the contused segment [48]. Focused assessment with sonography for trauma (FAST), which is a bed side non-invasive procedure, being popular lately [49]. Four sonographic windows (subcostal, right upper quadrant, left upper quadrant, and suprapubic) are used to detect the presence of significant amounts of free fluid. The pericardial window allows ample visualisation of the pericardium and heart, to rule out pericardial effusion and tamponade. Rozycki et al. [50] conducted a study of 247 penetrating chest injury patients, demonstrating a 100% sensitivity and specificity for haemopericardium with the use of FAST [50]. Rozycki et al. [51] have also utilised FAST to minimize emergency department to operating room times to an average of 12.1 minutes. Abnormalities such as wall motion defects and valvular incompetence are readily recognised. Nagy et al. [52] in a prospective analysis of 315 patients, ascertained that the echocardiogram has no added diagnostic utility for patients with normal ECG and blood pressure on admission. Also, the Eastern Association

for the Surgery of Trauma recommends selective use of echocardiography for patients, who are haemodynamically unstable or suffer cardiac complications [33].

Transoesophageal echocardiography (TOE) is now recognised as an excellent technique for the evaluation of cardiac injuries after trauma. It provides high quality images of the right ventricle and the left ventricle as well as the aorta. The introduction of biplane and multiplane, TOE has allowed for better image acquisition and improved diagnostic capabilities. TOE was found to be superior to EKG, cardiac enzymes and transthoracic echocardiogram (TTE) for the diagnosis of cardiac contusion [48, 53]. TOE is also found safe and feasible specially in presence of severe chest wall injury, pleural tubes and mechanical ventilation, where TTE is not feasible. Chirillo et al. [53] found that although 98% of blunt chest trauma patients successfully underwent TOE, TTE was feasible in only 38%.

The use of TOE may be limited by the need for experienced operators and the difficulty of the procedure with associated head and neck trauma, however, it is still considered by many to be the test of choice for diagnosing cardiac injuries after trauma.

Angiography and intravascular ultrasound (IVUS)

The coronary vascular status should be elucidated immediately using angiography, when the chest trauma patients complain of ongoing, deep-seated chest pain with elevated cardiac enzyme levels and an abnormal ECG, to rule out the possibility of coronary artery disruption and MI [54]. If a coronary lesion is confirmed angiographically and ongoing myocardial ischaemia is considered and bypass surgery or coronary angioplasty may be indicated. A complete angiographic investigation is also justified as the degree of severity of the visceral lesions and their possible association with tears in the wall of the great vessels may be underestimated.

IVUS is a newer imaging modality for better visualisation of abnormalities of the multilayered vascular wall structure in comparison with the planar luminogram obtained by coronary angiography, especially useful in situations where later is considered unreliable; such as for the lumen of ostial lesions or regions with multiple overlapping arterial segments [55]. It provides a cross sectional view of specific portion of coronary artery. It is also useful in detecting mild atherosclerotic lesion, assessing angiographically intermediate lesions and a coronary stenosis before and after catheter-based interventions [54]. Left main coronary artery lesions, which are often difficult to quantify using angiography due to overlapping branches, diffuse disease or ostial lesions, best suited for a study with IVUS [54]. The primary disadvantages of IVUS being used routinely are its expense, time consuming procedure, and the fact that it is considered an interventional procedure, and should

only be performed by angiographers that are trained in interventional cardiology techniques. In addition, there may be additional risk of plaque rupture imposed by the use of the IVUS catheter.

F-fluorodeoxyglucose positron emission tomography (FDG-PET)

FDG-PET scan is the gold standard to assess the myocardial viability to the exact extent and location of the contused myocardium, which results in perfusion-metabolism mismatch [56, 57]. Defect on FDG-PET suggests non-viable infarcted myocardium. FDG-PET may be useful in early decision making for patients with blunt chest trauma in a case with indistinct laboratory and imaging findings [56]. FDG-PET can also be used to predict post-revascularisation improvement in left ventricular function, symptomatic relief, and long-term survival [58]. FDG-PET has several advantages over single photon emission computed tomography (SPECT), including higher sensitivity and specificity, as well as the ability to measure myocardial blood flow and myocardial metabolism in absolute terms.

Cardiac magnetic resonance (CMR)

CMR has demonstrated excellent capability in the fine depiction of functional and morphological damage at myocardial level [54]. Functional and morphologic information derived from CMR appears theoretically to be helpful in defining the nature and severity of myocardial involvement at presentation, as well as during follow-up of patients with cardiac contusion [59]. Infarcted myocardium demonstrating delayed hyper-enhancement, and delayed contrast-enhanced cardiac MRI may offer means to differentiate myocardial injury secondary to contusion from peritramatic infarction [60]. Patients with post-traumatic myocardial dysfunction, but normal MRI could be spared unnecessary coronary artery catheterisation [60].

Myocardial perfusion scintigraphy

^{99m}Tc pyrophosphate scan can detect the myocardial necrosis but has low sensitivity and higher false negative rate [61]. Since, there is a higher likelihood of injury of the right ventricle and the right ventricle has a lower mass relative to the left ventricle, the scintigraphic detection of right ventricular necrosis is difficult [14]. 201-thallium SPECT is more precise in the detection and localisation of small transmural and non-transmural defects of the left ventricular wall [62]. Twenty-four-hour late 201-thallium imaging, with satisfactory image quality, enhances the detection of myocardial viability [63].

Pericardiocentesis

Pericardiocentesis, as a diagnostic modality, may be exam of preferred in lack of equipment or radiology personnel. False positives are result of aspiration of blood directly from the cardiac chamber and false negatives are result of immediate clotting of the aspirated blood [64]. Currently, the recommended approach is USG-guided-subxiphoid drainage [65]. Emergency pericardiocentesis gives temporary relief and time for surgical intervention in cardiac tamponade [65].

Other investigations

Radionuclide angiography (RNA) shows wall motion abnormality of injured myocardium [66]. Intracardiac structures cannot be visualised in RNA in the presence of arrhythmias. Routine use of RNA was proved not to be useful in the evaluation of patients with BCI [66, 67].

Susceptible population

A number of studies reveal that young adult age group (16–45 years) comprises a substantial number of deaths due to MVA, which happens to be the commonest cause of BTI leading to injury to the heart. Wall et al. [68] in a series of 711 patients found mean age of 31 years and 90% being male. Christensen et al. [69] noticed that 82% of acute MI following BCI were in patients below 45 years of age.

Pitfalls in diagnosis

Diagnosis of BCI is difficult to make due to:

- 1) Associated injuries which divert physician's attention: Failure to diagnose the infarct at the time of the accident is probably because other traumatic lesions (fractured ribs, pulmonary contusion, haemothorax, pneumothorax, transection of aorta, etc.) are responsible for the pain and state of shock masking the symptoms of the infarct [70, 71].
- 2) Lack of physical findings: Volume of heart sounds is difficult to assess in noisy environment, distended neck veins may be absent because of hypovolaemia, pulsus paradoxus may be absent and tension pneumothorax may mimic tamponade [70].
- 3) Lack of specificity of non-invasive tests available in an emergency room: Changes in chest radiogram, ECG, ECHO are non-specific and more specific investigations are usually not available in emergency room [62, 70].
- 4) Delayed appearance of clinical signs and symptoms: The clinical outcome of BCI may present late as is

seen in chronic pericarditis (CP). Brown et al. [72] reported that the period between injury and diagnosis of pericardial haematoma producing symptoms of CP ranged from 3–20 years. Thus, the history of trauma remote from the onset of CP is often overlooked and it becomes difficult to make sure that it is a delayed response to earlier chest injury.

Suggestions

Diagnosis of BCIs need high level of vigilance and detailed investigations at slightest suspicion. This can help detect the large pool which goes undiagnosed otherwise and gives immediate and delayed ill effects which could be avoided by diagnosing early. In patients with suspicious ECG finding, at least 24 hours cardiac monitoring and a follow up ECG should be done. Haemodynamically stable young (<55 years) subjects without underlying cardiac diseases with normal ECG and cardiac troponin-I at 8 hours, could be discharged safely. Physicians in primary care centre should refer the cases to tertiary care at earliest, if needed. Healthcare professionals should be trained in managing acute chest injuries including cardiac involvement through periodical CME programmes.

Conflict of interest The authors do not have any disclosable interest

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