

Myocardial contusion: emergency investigation and diagnosis

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Cardiac contusion is an infrequent but occasionally serious complication of deceleration injury. According to ATLS teaching, the true diagnosis of contusion can only be established by direct inspection of the myocardium. The clinically important sequelae of myocardial contusion are hypotension and arrhythmia. Despite recent advances in investigative techniques, myocardial trauma remains an important diagnostic and management challenge. This paper presents an evidence-based review of the topic.

INCIDENCE

The exact incidence of cardiac contusion is unknown as diagnostic criteria vary. Postmortem evidence of myocardial contusion is present in 14%¹ of immediate fatalities from blunt injury.

PATHOPHYSIOLOGY

Cardiac contusion is caused by a deceleration force affecting the viscoelastic chest wall causing (1) direct pressure on myocardium and (2) indirect effects secondary to increased intrathoracic pressures with shearing stresses. Experimental evidence shows a sternal blow can reduce the AP diameter of the thorax by 50%. Tenzer² reported a spectrum of myocardial injury from blunt trauma ranging from myocardial concussion without cellular damage to severe contusion. Severe contusion is characterised by patchy and irregular myocyte necrosis associated with epicardial haemorrhage extending in pyramidal fashion intramurally or even transmurally. The right ventricle (RV) is most commonly injured because of its position behind the sternum. Left ventricular (LV) output can fall in the absence of direct injury because of reduced preload. Reduced LV compliance from intraventricular septum shift also contributes to biventricular failure. The resultant drop in cardiac output may be up to 40% and can persist for several weeks.

COMPLICATIONS

Ventricular dysfunction and arrhythmia are the two most serious complications. Arrhythmia usually occur within 24 hours (91% within 48 hours). The need for intervention has been reported at between zero to 20%,³ depending upon diagnostic criteria and intervention threshold. Coronary vessel and valvular injury are associated with cardiac contusion but are regarded as separate entities.

DIAGNOSIS

The definitive diagnosis of myocardial contusion has proved complex as this can only be made on gross or histological examination of the heart. No *in vivo* test has yet been clearly shown to identify injury or to predict those associated with potentially lethal complications.

A meta-analysis⁴ of blunt cardiac trauma prior to 1994 concluded that an abnormal ECG on admission or a raised CK-MB isoenzyme correlated with clinically significant cardiac complications. Correlation with transthoracic echo (TTE) was less conclusive. The 41 studies (4861 subjects) in the analysis included heterogeneous populations and varied in findings considered clinically significant.

CREATININE KINASE

In 1994 Biffi *et al*⁵ demonstrated that "cardiac enzymes" were irrelevant in suspected myocardial contusion. Some 5%–7% of skeletal muscle creatinine kinase (CK) can be in the MB isoenzyme form. After injury both CK-MB and CK-MM fractions are produced as part of the normal inflammatory response. Similarly increased CK-MB fractions are also seen in inflammatory muscle disease or after exercise. The tongue and diaphragm, frequently damaged in deceleration type injuries, are also inherently rich in the MB isoenzyme. Others^{6,7} have confirmed Biffi's conclusions, most recently Swaanenburg *et al*⁸ (1998) who demonstrated a rise in CK-MB activity in five and CK-MB mass in 35 of 51 trauma patients with no evidence of thoracic trauma.

In conclusion, CK-MB studies are non-specific and do not predict cardiac contusion or its complications.

CARDIAC TROPONIN

Serum cardiac troponin is highly specific for myocardial injury. Cardiac troponins differ from the skeletal isoform by a unique aminoterminal sequence of 31 amino acids never expressed by skeletal muscle. They are myocardial regulatory contractile proteins only released into the circulation after loss of myocyte membrane integrity.

Animal studies⁹ on myocardial injury have demonstrated that maximal cTnI concentration and area under the cTnI curve increase with the power of kinetic energy. This suggests it may be

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possible not only to diagnose myocardial contusion but also to stratify patients according to cTn level and possibly to predict those in whom complications are more likely. Studies in humans^{10–11} have shown that cardiac troponins demonstrate a high sensitivity and specificity for cardiac injury. Bertinchant's¹² conflicting results showed that although troponins showed improved specificity compared with conventional markers, their sensitivity was poor. Sensitivity, specificity, and negative and positive predictive values of cTnI and cTnT in predicting myocardial contusion were 23%, 97% and 77%, 75% and 12%, 100% and 74%, 100% respectively. This may reflect over diagnosis of myocardial contusion within the study population as diagnosis was based on echocardiographic and/or ECG criteria.

A small study¹³ has questioned the specificity of cTnI. Six of 17 patients with multiple trauma admitted to ICU with no transoesophageal echocardiography (TOE) abnormalities had raised levels. The finding could represent a period of severe hypotension causing subendocardial injury. The authors suggested that raised troponin levels could therefore represent a surrogate marker for the development of the systemic inflammatory response syndrome. It could equally represent minor cardiac contusion without wall motion abnormalities suggesting cTnI is more sensitive than TOE.

In summary, cardiac troponins seem to be highly specific and sensitive for myocardial injury. They may offer the possibility of stratification of severity and risk of adverse outcome. Increased levels could however be secondary to non-significant cardiac damage or secondary to a period of grade IV shock. Further prospective studies are necessary to clarify the situation.

ELECTROCARDIOGRAPHY

Electrocardiographic (ECG) changes have been widely used to try and predict myocardial contusion. Maenza's⁴ meta-analysis concluded that an abnormal ECG on admission correlated with clinically significant cardiac complications.

Schick *et al*¹⁴ in experiments on rabbits demonstrated transient non-specific ST segment and T wave changes after blunt thoracic trauma, which correlated with RV contusions at necropsy.

Viano *et al*¹⁵ in experiments on pigs demonstrated SA nodal disturbance, AV junctional dysfunction and intraventricular conduction defects after blunt thoracic trauma. They also demonstrated precipitation of ventricular fibrillation in 4 of 12 subjects. They did not comment on ECG changes other than those affecting the conducting system. Potkin *et al*¹⁶ demonstrated ECG abnormalities in 70 of 100 human patients with severe blunt thoracic trauma. They described non-specific ST segment and T wave changes consistent with those reported by Schick *et al* in rabbits. These ECG changes were not predictive of cardiac complications or of myocardial contusion at necropsy in the 15 patients who died.

ECGs are intrinsically unlikely to be sensitive in the diagnosis of myocardial contusion as they largely reflect atrial and LV activity, the LV mass electrically overshadowing the RV.

In summary, the ECG changes observed in animal studies are non-specific. Similar changes may occur in humans as a reflection of a number of metabolic abnormalities associated with significant trauma. The ECG can only be interpreted after exclusion or correction of hypoxia, hypovolaemia, acidosis and electrolyte imbalances.¹⁷

TRANSTHORACIC ECHOCARDIOGRAPHY (TTE)

TTE demonstrated wall motion abnormalities correlate with clinically significant injuries.⁴ Characteristic findings after deceleration injury to the thorax are: segmental wall motion abnormalities representing regional hypokinesia and RV dilatation.¹⁸ TTE is readily available but associated thoracic and

Key points

- There remains no sensitive diagnostic test for myocardial contusion.
- TOE remains a useful screening tool in those who develop cardiac complications or unresponsive hypotension after severe deceleration chest trauma.
- Treatment includes supportive care and the appropriate use of inotropes and antiarrhythmics.

pulmonary injuries often obscure the ultrasonic window. Sub-optimal examination varies from 13%–39%. Recent studies have shed doubt on the specificity of echocardiography.

TRANSOESOPHAGEAL ECHOCARDIOGRAPHY (TOE)

TOE is less widely available but more reliable. Garcia-Fernandez *et al*¹⁹ produced good images in all 117 patients selected on the basis of clinical suspicion of myocardial contusion. They demonstrated ventricular dysfunction in 42% (> 60% RV) and interestingly showed no correlation with CK-MB or ECG changes. They also demonstrated that Injury Severity Scores failed to predict cardiac injury. Similar results were obtained by Weiss *et al*²⁰ who produced good images in 81 patients with no complications from the procedure. Concurrent ECG changes were neither specific nor sensitive for echocardiogram findings. Injury Severity Scores failed to predict cardiac injury although it was noted that the presence of myocardial dysfunction after blunt trauma was associated with a higher average Injury Severity Score (33 v 27) and that this was reflected in mortality rates (27% v 9%).

TOE is not widely available and is not simple enough for use as a screening test. Furthermore, wall motion abnormalities may not necessarily represent myocardial injury. Moomey *et al*²¹ demonstrated in animal studies that after profound lung injury although arterial blood pressure, cardiac output and heart rate remained in the normal range, RV afterload rose and ventricular contractility fell.

RADIONUCLEOTIDE PERFUSION SCANS

Isotope scans have shown no correlation with clinical outcome⁴ and even in experimental conditions have only accurately demonstrated full thickness contusions.²²

MANAGEMENT

Cardiac contusion is seen in the setting of significant blunt chest trauma. Diagnosis is dependent upon a high index of suspicion. Cardiac contusion is usually associated with significant extracardiac trauma. Significant deceleration forces are necessary to injure the centrally placed structure. ATLS principles dictate that shock of hypovolaemic origin should be treated with fluid resuscitation. Spinal and obstructive causes need to be considered. Persistent hypotension with no clear haemorrhage is suspicious for myocardial injury. This is especially true with associated significant chest wall and pulmonary injury. Early central venous catheter insertion and pressure monitoring is vital. This excludes inadequate fluid resuscitation and allows guided fluid management. The myocardium tends to be very sensitive to inotropes after injury. Careful inotropic support may be necessary.

Cardiac arrhythmias, after trauma, can be indicative of myocardial injury. They can also occur secondary to the altered biochemistry or acid base balance associated with hypoxia and tissue hypoperfusion. Underlying cardiac disease can be exacerbated by trauma. Some patients will have had a primary cardiac event as the precipitating cause for the injury. The treatment of arrhythmias after major trauma is expectant and supportive as per ALS periarrest guidelines. All patients with ECG morphology or rhythm disturbances should have cardiac

monitoring for at least 24 hours. Biochemical abnormalities should be actively managed. Early echocardiography is the imaging investigation of choice. Anaesthesia and DC cardioversion are avoided where possible.

CONCLUSIONS

Myocardial contusion is a well defined pathological entity following deceleration trauma. Severe contusion results in ventricular dysfunction and malignant arrhythmia. Diagnosis is based on ECG findings.

Diagnosis in less severe cases is more difficult. ECG and CK abnormalities are non-specific and do not predict cardiac contusion or its complications. TTE is insensitive. TOE has proved sensitive in detecting ventricular dysfunction but this is not specific for myocardial injury. Troponins seem to be reliable indicators of myocyte injury but their utility has yet to be proven in risk stratification of such patients.

In patients with less severe blunt chest injuries, cardiac contusion, should only be sought in those demonstrating clinical signs or patients with a history of coronary artery disease. The incidence of injury associated with mild/moderate blunt chest trauma is unclear because of the lack of a gold standard investigation. Complications are rare and routine observation or investigation is not always warranted.

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