Blunt chest trauma: a clinical chameleon

Kaveh Eghbalzadeh,1 Anton Sabashnikov,1 Mohamed Zeriouh,1 Yeong-Hoon Choi,1 Alexander C Bunck,2 Navid Mader,1 Thorsten Wahlers1

ABSTRACT
The incidence of blunt chest trauma (BCT) is greater than 15% of all trauma admissions to the emergency departments worldwide and is the second leading cause of death after head injury in motor vehicle accidents. The mortality due to BCT is inhomogeneously described ranging from 9% to 60%. BCT is commonly caused by a sudden high-speed deceleration trauma to the anterior chest, leading to a compression of the thorax. All thoracic structures might be injured as a result of the trauma. Complex cardiac arrhythmia, heart murmurs, hypotension, angina-like chest pain, respiratory insufficiency or distention of the jugular veins may indicate potential cardiac injury. However, on admission to emergency departments symptoms might be missing or may not be clearly associated with the injury. Accurate diagnostics and early management in order to prevent serious complications and death are essential for patients suffering a BCT. Optimal initial diagnostics includes echocardiography or CT, Holter-monitor recordings, serial 12-lead electrocardiography and measurements of cardiac enzymes. Immediate diagnostics leading to the appropriate therapy is essential for saving a patient’s life. The key aspect of the entire management, including diagnostics and treatment of patients with BCT, remains an interdisciplinary team involving cardiologists, cardiothoracic surgeons, imaging radiologists and trauma specialists working in tandem.

BACKGROUND
Blunt chest trauma (BCT) is the second leading cause of death after head injury in vehicular accidents.1 The incidence of this trauma has been inconsistently reported in the literature; however, a rough estimate of around 15% of all trauma admissions to emergency departments seems reasonable.2 3 Similarly, analogous inhomogeneity exists in terms of mortality rates described in previous studies, ranging between 9% and 60% and exceeds 50% within the first 24 hours.4–6 The main reasons for this disparity seem to be its versatile presentation patterns with involvement of various organs and tissues within the chest and the presence of a wide range of differential diagnoses. Approximately 90% of BCT cases are managed by insertion of a chest tube before any other consideration, whereas the remaining 10% require direct surgical interventions.6–9

The aim of this article was to present a comprehensive overview of BCT with a main focus on cardiac involvement, as this category is usually associated with the most life-threatening complications.3 9 Even though BCT represents a relatively rare condition, its high clinical impact explains the need for accurate diagnostics and early management in order to manage serious complications and death in a number of cases (see table 1).

Mechanisms of BCT
BCT typically occurs after direct impact to the anterior chest caused by high-speed sudden deceleration or compression of the thorax, usually in motor vehicle accidents. Generally, mobile intra-thoracic viscera are injured by less-mobile skeletal structures of the thorax. Massive compression of the anterior chest can lead to heart contusions of varying severities as well as injuries to the ascending aorta, the innominate artery and the thoracic vertebrae.4 In cases of ostial fractures of the sternum (figure 1), sharp bone margins may tear the right ventricular chamber or the ascending aorta due to their direct anatomical neighbourhood to the sternum.9 10 However, apart from the sternum, fractures of other skeletal structures, such as ribs and thoracic or lumbar vertebrae, are often involved in this form of trauma.11 Naturally torsion or traction forces are seen as the common pathomechanism for damage to the heart and the thoracic aorta during BCT. As the ascending aorta is directly fixed to the chest wall, a tear between the mobile part of the ascending aorta and aortic arch and stabilised part of the descending aorta can be frequently observed. The proximal descending aorta is fixed to the pulmonary artery through ligamentum arteriosum, the remnant of the ductus arteriosus, and therefore the least mobile part of the aorta. Typically this tear junction is located directly adjacent to the isthmus.12 High intraventricular pressure caused by trauma may injure cardiac valves and the myocardium itself depending on the cardiac cycle.2 The most vulnerable cycle might be end-diastole when the impact of the BCT increases intraventricular pressure due to the mechanical compression of the distended ventricles.13 A complete heart block during BCT can be caused by either electrical or structural injury to the heart.14 In contrast to contusio cordis with predominantly structural character of the damage, commotio cordis might cause sudden death after the blunt impact on the precordium with subsequent ventricular fibrillation even without any evidence of structural injury to the myocardium.14 15 However, cardiac chamber or great vessel ruptures may occur not only as a result of BCT, but a growing number of reports also describe isolated coronary artery injuries including ruptures, dissections and other types of injuries.16

DIAGNOSTICS
There is currently no gold standard for the diagnostics of BCT, which includes a wide range of possible presentations with individual symptoms. Complex cardiac arrhythmia, heart murmurs, hypotension, angina-like chest pain, dyspnoea or distention of the jugular veins may, however, indicate potential
cardiac injury. Whereas on admission to the emergency department a large proportion of patients with BCT do not show respiratory insufficiency and cardiac symptoms indicative of BCT, clinicians should be prepared for rapid changes in clinical condition of such patients as various complications can first occur within 72 hours. Therefore, the relevance of complete diagnostics including CT scan should not be underestimated after typical trauma mechanisms, even when the first impression on patient’s clinical condition does not indicate severe injury.

Whenever possible patients should first undergo physical examination, Holter-monitor recordings, serial 12-lead ECG and measurements of cardiac enzymes. Cardiac enzymes, such as creatine kinase MB (CK-MB), might be elevated depending on the severity and localization of the trauma and are therefore not always specific. Furthermore, in some cases cardiac enzymes might even not be elevated despite significant cardiac injury, such as in case of a cardiac contusion. Arrhythmia in general is a very unspecific sign and may also not always be associated with the presence of a BCT.

In terms of radiologic imaging a plain chest radiograph is considered a sufficient indication for inserting a chest tube in presence of a pneumothorax. However, the radiographic examination shows no pathological findings in up to 44% of patients suffering an aortic injury. On the basis of the Guidelines of the Eastern Association for the Surgery of Trauma (EAST) every patient presenting with specific chest radiograph pathologies, such as widened mediastinum, tracheal deviation and loss of aortopulmonary window (figure 2) after a high-speed deceleration trauma or suspicious of an aortic injury should undergo a CT scan of the chest using intravenous contrast, which is superior to catheter-based angiography. On the other hand, catheter-based angiography shows highest sensitivity in cases of coronary artery involvement after BCT. Also, a great amount

<p>| Table 1 | Overview of the most important injuries after BCT |</p>
<table>
<thead>
<tr>
<th>Relative incidence</th>
<th>Clinical presentation</th>
<th>Imaging findings</th>
<th>Acute management</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blunt aortic injury</td>
<td>~1% of all vehicular high-speed accidents</td>
<td>Lethal, when ruptured; symptomless; mid-scapular back pain; malperfusion syndrome</td>
<td>X-ray: loss of aortopulmonary window; CT scan see figure 3</td>
<td>Systolic BP &lt;120 mm Hg; surgery or EVR</td>
</tr>
<tr>
<td>Myocardial contusion</td>
<td>Up to 24% of all BCTs (from autopsy reports)</td>
<td>Symptomless up to therapy-refractory hypotension/low-cardiac output; arrhythmias</td>
<td>No specific imaging findings</td>
<td>(1) 12-Lead ECG, CK+CK-MB, Holter monitor; (2) fluid and vasopressor; (3) mechanical circulatory support</td>
</tr>
<tr>
<td>Valvular rupture</td>
<td>&lt;100 case reports</td>
<td>Typical symptoms of valve insufficiency, depending on the affected valve</td>
<td>Valve rupture in electrocardiography</td>
<td>Surgical valve repair or replacement</td>
</tr>
<tr>
<td>Coronary dissection</td>
<td>&lt;100 case reports</td>
<td>Symptoms of MI, pericardial effusion, low-cardiac output</td>
<td>True and false lumen in angiography or coronary CT angiography</td>
<td>(1) Coronary stenting; (2) surgical revascularisation</td>
</tr>
</tbody>
</table>

BCT, blunt chest trauma; BP, blood pressure; CK-MB, creatine kinase MB; EVR, endovascular repair; MI, myocardial infarction.
The typical mechanism of BAI is generally an abrupt deceleration generating maximum shear forces on the transition zone between the fixed and the mobile aorta, usually in the region of the aortic isthmus (figures 3 and 4)."12 Over 90% of aortic ruptures are located in this region, whereas other locations including ascending, distal descending or abdominal aorta have been reported in individual cases.18,29 After the landmark study by Parmley et al angiography was considered the gold standard in diagnosis of the BAI for more than 40 years.27 Thereafter, some studies favoured transoesophageal echocardiography to be first-line imaging modality for the evaluation of trauma in patients suspicious of BAI due to its portability and diagnostic accuracy.30 Nowadays, the imaging method of choice is helical CT scan with an estimated sensitivity of 100% compared with 92% for angiography (figure 3).8,20,28 When BAI cannot be treated first in high-risk patients suffering from other major associated injuries or severe comorbidities, Fabian et al showed that antihypertensive therapy with a targeted systolic blood pressure of less than 120 mm Hg results in a significantly improved outcome for patients undergoing delayed repair of the aorta.31 In patients showing evidence of hypovolaemia and contained BAI, the major source of blood loss cannot be the aorta as that could prove to be fatal. For this reason, for patients with BAI and major associated injuries resulting in hypovolaemia, delayed repair with controlled systolic blood pressure is a valuable therapy option.

Repair of the aortic disruption can be performed either surgically via left thoracotomy with an incision in the fourth intercostal space using unilateral ventilation of the right lung or interventionally through endovascular repair.19,32 Perioperative and postoperative complications of the surgical approach can be reduced by using a left atrium to left femoral artery bypass with a centrifugal pump, through a venoarterial cardiopulmonary bypass or a Gott shunt inserted between the ascending and the thoracic descending aorta.31 Endovascular repair is performed by positioning a stent graft using a guide wire under angio- graphy.34 Figure 5 shows an aortic tear treated by a thoracic endovascular stent graft. According to the guidelines for the evaluation and management of blunt traumatic aortic injury by endovascular repair as a less invasive approach (EAST) is considered to carry a significantly lower risk of blood loss, mortality, paraplegia and a comparable risk of procedure-related stroke.37 The overall mortality was lower at 7.9% as reported in 37 studies involving 2588 patients for the endovascular repair cohort compared with 18.9% for the surgical approach (risk ratio (RR)

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**Figure 3** Three-dimensional rendering, curved reformation and an axial CT image of a severe laceration of the aortic arch with formation of a pseudoaneurysm after a velocity accident of a motorcyclist. This injury was successfully treated by thoracic endovascular aortic repair procedure. A concomitant compression fracture of the sixth thoracic vertebra resulted in spinal cord injury and incomplete paraplegia.

**Figure 4** Stented aneurysm of the aortic arch. The male patient suffered a blunt aortic injury during a motorcycle accident. A tear is directly adjacent to the isthmus. Unfortunately, the patient died before arrival to the hospital.
Myocardial contusion

The second most frequent injury leading to death after BCT is myocardial contusion. The right ventricular wall is anatomically in close proximity to the dorsal sternum and represents the most vulnerable structure in cases of myocardial contusion. Compared to myocardial concussion, where only an abnormal myocardial wall motion with no anatomic or cellular injury is present, a myocardial contusion is associated with a manifest myocardial tissue damage. However, myocardial concussion should not be considered a benign entity, as it may cause severe rhythm disturbances. Autopsies of patients who died of BCT revealed myocardial contusion in up to 24% of the cases. Postmortem autopsy is the only way to retrospectively ensure the definite diagnosis of this category of BCT, whereas clinical diagnosis is often difficult and therefore the incidence is reported with wide discrepancy.

Although myocardial damage can also result in ECG changes or myocardial enzyme release, such as troponin and CK-MB, these findings remain unspecific in most cases of BCT including myocardial contusion. Echocardiography is considered to be of higher specificity as echocardiography findings were shown to correlate with complications in a large meta-analysis by Maenza et al. Unfortunately, the sensitivity of echocardiography was reported to be considerably lower compared with ECG and enzyme kinetics. Furthermore, Hammer et al. analysed CT findings after myocardial contusions and reported on poor sensitivity of specific CT findings, such as myocardial hypoenhancement. Therefore, CT scan is not recommended as a reliable screening tool for detecting myocardial contusion. The pathomechanism of cardiac contusion is based on the myocardial damage at the cellular level. Histologically, red blood cell extravasation into and between myocardial muscle fibres and a selective necrosis of the fibres is observed. This phenomenon might cause enzyme release, which can be identified via a blood test. Necrosis of myocardial muscle fibres is characterised by infiltrates of polymorphonuclear leucocytes. Haemorrhage is generally absorbed and the contusion heals by patchy and irregular scar formation. In contrast, repair of ischaemic myocardial injury shows generalised fibrosis. Consequently, the diagnosis of the non-penetrating pathology termed myocardial contusion can only be ensured by histological examination postmortem. In case of an existing penetration the diagnosis of myocardial contusion cannot be made.

Depending on the amount of myocardium involved, the damage at the cellular level might cause reduction in cardiac output potentially leading to cardiogenic shock in the worst-case scenario. In such circumstances symptom-oriented conservative treatment or implementation of mechanical circulatory support as appropriate should be considered. Moreover, constant monitoring of vital parameters, including Holter-monitoring should be performed on intensive care unit for the first 24 hours after admission to identify possible arrhythmic complications. Myocardial enzyme release markers, such as troponin and CK-MB, should be frequently performed as in patients with myocardial infarction (MI). As coronary involvement might equally lead to enzyme release and chest pain can be interpreted as being secondary to the trauma, Holter-monitor recordings and repetitive serial 12-lead electrocardiograms are the appropriate examinations to differentiate between both pathologies. Whereas the injury itself is extremely difficult to identify complicating the establishment of the correct diagnosis and therapeutic strategy, most associated symptoms caused by myocardial contusion, such as cardiogenic shock or arrhythmias, can be treated independent from the cause. In cases of low-output syndrome or cardiogenic shock, continuously performed echocardiography represents an essential tool to evaluate the response to fluids and to facilitate the management of vasopressors versus afterload reduction therapy.

Valve rupture

Valve rupture after BCT is rare and unusual. However, its incidence cannot be accurately quantified. After its first description in 1830 by Plenderleith this phenomenon has inconsistently been described in the literature, mainly in form of case reports and case studies accounting for less than 100 cases reported by 2002. Studies with larger patient cohorts have also been limited to a low number of individual cases of BCT with involvement of valve injuries. In spite of its infrequent presentation, this category of injury is of high clinical relevance and interest for clinicians as appropriate and promising therapy options can be offered.

Aortic valve is the most frequently injured valve followed by mitral and tricuspid valves. Due to higher-pressure conditions in the left heart, the valves from the left cardiac chambers are more likely to be affected, whereas the cardiac cycle has its particular risk of developing an injury. At that time sufficient valve is closed and is subjected to maximal tension, whereas the pressure in the empty left ventricle is low. A sudden increase in intra-aortic pressure due to deformations caused by an external impact transmitted through the chest wall may lead to excessive forces directed towards the closed aortic valve. In combination with low left ventricular end-diastolic pressure, such an enormous pressure aortoventricular gradient may potentially destruct all leaflets. As valve rupture usually causes an insufficiency of the involved valve, characteristic clinical signs of valve pathology may be present, such as wide pulse pressure.

Figure 5  CT angiography (CTA) in axial and coronal orientation showing a small aortic tear in the distal aortic arch after a car accident with unknown velocity. The lorry driver was stuck in his vehicle and required technical rescue. Concomitant injuries involved open head trauma, bilateral dissection of the internal carotid artery and serial rib fractures of the 1st to fifth rib on the left side. On the CTA of the initial trauma scan, a large haematoma abutting the aortic arch was noticed causing broadening of the upper mediastinum. The aortic tear was successfully treated by a thoracic endovascular stent graft (thoracic endovascular aortic repair) and the haematoma resolved.
amplitude in case of aortic regurgitation. In the same way, atrio-
ventricular valves may be injured due to a massive increase in 
intraventricular pressure during systole. Therefore, depending 

on the valve affected, the clinical picture of a new regurgitation 
caused by a tear in a leaflet or papillary muscle is usually seen 
on admission.42 As with regurgitations of any other aetiology, 
diagnosis can be made via clinical examination, auscultation of a 
cardiac murmur and using echocardiography with Doppler and 
colour flow imaging.42 Moreover, haemodynamic condition on 
admission may strongly correlate with the severity of the valve 
pathology and also with the accompanying injuries of the patient 
with trauma. Interestingly, aortic and mitral insufficiency after 
trauma can also lead to an insidious progress of cardiac failure 
over several weeks, whereas in case of tricuspid insufficiency this 
process may even take several years.43 Patients with acute heart 
failure due to the valve destruction should immediately undergo 
surgical repair, if full heparinisation is justifiable depending on 
the severity of other injuries, as valve replacement is associated 
with excellent long-term outcomes.41

Coronary involvement

Involvement of coronary arteries with subsequent MI after BCT 
is a rare phenomenon, which has only been reported on in single 
case reports or case studies. Analysing available evidence on this 
topic Christensen et al found a total of 77 cases of acute MI due to 
coronary injuries after BCT.44 Due to its anatomical position on 
less protected anterior ventricular wall left anterior descending 
(LAD) artery has been shown to be the most commonly injured 
coronary artery accounting for 76% of all coronary injuries.44 The 
second most commonly injured coronary artery is the right 
coronary artery (RCA) with an incidence of 12%.45 A possible 
explanation for a relatively frequent RCA involvement is the 
fact that with every heart beat it moves anteriorly towards the 
sternum during systole. Another hypothesis postulates that rapid 
deceleration may also lead to a disruption of the RCA from its 
junction with the aortic root.46 Coronary artery injuries after BCT 
mainly include intimal tears, coronary dissections or 
ruptures, ruptures of pre-existing plaques and occlusions due to 
external epicardial haematoma.44 Coronary artery ruptures can 
be also fatal due to acute pericardial tamponade (figure 6). In 
some cases normal appearance of coronary arteries in coronary 
angiogram may obscure the evidence of MI, whereas coronary 
spasm or spontaneous clot lysis may be responsible symptoms of 
ischaeemia. In patients suspected of MI after BCT usual diagnos-
tics with a serial 12-lead ECG and cardiac enzyme control should 
be performed. However, angina-like chest pain is generally not 
reported as a classical symptom after BCT as the symptoms might 
be partially concealed by other injuries of the thorax.44 Percuta-
neous coronary intervention with stent placement is the optimal 
therapy for uncomplicated LAD and RCA lesions.47 Surgical 
myocardial revascularisation should be performed in cases of 
an unprotected left main coronary artery disease, contraindica-
tions for antplatelet therapy and history of bleeding diathesis.48 
As patients with BCT regularly present with other concomitant 
injuries, the indication for stenting is limited due to the need 
for antplatelet therapy, which may cause bleeding complications. 
However, patients undergoing myocardial revascularisation 
need a systemic anticoagulation with heparin representing 
the optimal therapeutic strategy.49

Other cardiac injuries

Apart from more frequently described cardiac injuries after BCT 
mentioned above, there are still several other impressive clinical 
manifestations, which are exciting cases because of their rarity. 
Ruptures of cardiac chambers, or intrapericardial veins generally 
cause an acute tamponade leading to death before admission to 
the emergency department in most cases.2 The absolute minority 
of patients who survive rupture of a cardiac cavity usually have 
a tear in a low-pressure chamber.48 Moreover, some cases of 
asymptomatic haemopericardium leading to a constrictive 
pericarditis have been described in literature.9 38 A classical 
clinical presentation of a protracted constrictive myocarditis is 
severe diastolic dysfunction with indication for surgical 
pericardiectomy.50

CONCLUSION

BCT can cause a wide range of clinical presentations and inju-
ries, making the diagnostics extremely challenging. Patients may 
initially appear haemodynamically stable and asymptomatic on 
admission, however suffering a life-threatening disease, which 
can lead to death, unless the problem is correctly recognised and 
appropriate timely therapy commenced. Due to the variety of 
presentations and severity of injuries, there are no fixed algo-

rithms to identify the disease. However, simple clinical exam-
ination, ECG, measurement of the cardiac enzymes and most 
importantly an immediate transthoracic echocardiography as 
easily available and rapid imaging option can save lives. Other 
specific techniques, such as angiography or CT scan of the 
chest, are often mandatory to confirm the diagnosis and initiate 
appropriate therapy. Finally, the most important aspect of the 
entire management of patients with BCT is an interdisciplinary 
team involving cardiologists, cardiothoracic surgeons, imaging 
radiologists and trauma specialists in order to emerge victorious 
against this clinical chameleon.

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responsible for the imaging data collection and interpretation.

Competing interests None declared.

Figure 6 A lethal pericardial effusion, which can potentially be caused 
by any blunt chest trauma independent from the underlying pathology. 
The heart is also presented in sections to demonstrate the extent of the 
tamponade.

REFERENCES


