Ben Roestenburg

Blunt Cardiac Injury
90% of BCl’s are lethal and do not make it to hospital.
Case – 68 year old male

Mechanism
• Car vs Tree
• 80-100kmh
• Steering wheel impact
• Restrained driver
• No airbags
• Hit head on door pillar

Injuries
• Sternal Fractures
• Rib Fractures
• Right distal radius fracture
• Maxillary sinus fracture
• Facial lacerations
• Abrasions

Signs
• GCS 15
• Self extricated
• Stable vitals
• Amnestic of events
• ?LOC
Events – first 24 hours

**Primary and Secondary Survey -> STU**
- Rib fractures, sternal fracture, head laceration
- ECG – Normal Sinus
- CT Head/C-Spine - NAD
- Cardiac Troponins at admission, 4 hours, 6 hours NAD (serial 3’s peaked at 4)
- Admitted for analgesia and observation

**MET Call**
- Sudden onset worsening chest pain
- HR 110 RR 24 BP 150/100 Sats 92%RA T36.4
- ECG – new RBBB
- Type 1 Respiratory Failure
Blunt Cardiac Injury
Blunt Cardiac Trauma: A Review of the Current Knowledge and Management
Raid Yousef, MD, and John Alfred Carr, MD
Department of Trauma Surgery, Hadley Medical and Level 1 Trauma Center, Flint, Michigan

Blunt cardiac injuries are highly lethal. A review of the world’s English literature on the topic reveals a lack of Level 1 evidence and few cohesive guidelines for the management of these patients. An online database query was performed using the PubMed medical database.

The most common injury is transection rupture of a cardiac chamber, seen in 39% to 64%, or multiple chamber ruptures in 26% [3, 5].

In contrast to motor vehicle crashes, up to 54% of people who fall from a height exceeding 6 meters (20 ft) have some type of BCI from small endocardial tears to transectional rupture [6]. The chance of cardiac injury increases as the height increases, such that the surrogates should be concerned about some type of possible endocardial tear or intimal hematoma in any patient who falls more than 6 meters (20 ft) [7]. For those with documented cardiac injury by autopsy after death from a fall, sternal fractures were present in 75% and multiple sternal fractures in 56% [7]. Thus, the combination of a fall greater than 20 feet with a sternal fracture should prompt a thorough cardiac evaluation.

Death at the Scene

When death from blunt trauma is proven by autopsy to be from cardiac injury, the most common lethal cardiac injuries were transectional rupture of 1 or more cardiac chambers [44%], tears occurring at the venous-atrial confluence (33%), or a blunt coronary artery dissection or tear [22.9, 9, 0.4%]. Given the anterior location, the right ventricle is the most commonly injured chamber in 44% of the victims [6, 8, 9, Table 3.1]. The right atrium and left ventricle are the second and third, with injuries in 38% to 30% [6, 9, Table 3.1].

Years occurring at the venous-atrial confluence are believed to be due to rapid desiccation that results in the other injuries associated with the mass of the heart continuing to move forward or laterally while the posterior ventricle walls do not move, resulting in a distinction-arrestion injury at the venous-atrial confluence.
**Epidemiology**
Identified in 10% of trauma admissions
- 50% MVA related
- 35% Pedestrian
- 9% Motorcycle
- The rest from falls / assaults / misc

**Pathophysiology**
Majority of BCI’s that make it to hospital alive are low severity.
- Distraction-avulsion tearing in rapid deceleration of mobile ventricles
  - = Transmural cardiac chamber rupture (34-69%)
- Majority are right ventricle

**Presentations**
*Structural Injury*
- Intramural haematoma
- Valvular injury
- VS rupture
- Coronary / MI

*Electrical*
- Conduction disturbances
- Dysrhythmias
Screening for BCI

• Mechanism

• ECG – All patients should get an ECG
  • ’Classic’ teaching is that BCI cases RBBB
  • Tachyarrhythmias (Sinus and SVT)
  • PVCs may occur, but difficult in isolation to interpret
  • Most ECG changes are transient, intermittent, evolving, and clinically irrelevant
  • New abnormalities require admission and monitoring
  • Poor sensitivity for right ventricle, and limited specificity if there is pre-existing comorbidities.

• Cardiac Troponins
  • Controversial
  • Together with normal ECG has near 100% NPV
  • Limited PPV

• Echo
  • Persistent haemodynamic instability
Troponins

- TnI TnC TnT - 3 types all present in skeletal muscle
- cTnI Represents cardiac muscle injury
- Overlap between chest wall damage and cardiac muscle damage
- 30% cross reactivity of monoclonal antibody assays
- No clear level of cTnI that indicates injury to myocardium
- cTnI > 1ng/mL 60%-70% of those patients actually had a documented cardiac injury - should be then evaluated with ECHO
- Can be used to rule out if cTnI is less than 0.4ng/ml 4 to six 6 hours after injury
NPV of Serial Troponins

2014 Systematic Review (Guild et al)
- 123 Journal Articles, 10 included, 1691 Patients
- Serial Troponins at Admission, 4, 6, 12, 24 hours

Table. Studies examining NPV of normal cardiac Tn concentrations in patients with BCT

<table>
<thead>
<tr>
<th>Report</th>
<th>N</th>
<th>Tn sampling</th>
<th>Abnormal Tn cutoff, ng/mL</th>
<th>Duration of assessment</th>
<th>NPV of normal Tn for study-defined adverse outcome, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mair et al(^8)</td>
<td>32</td>
<td>Adm, 12 h, 24 h, daily</td>
<td>TnT ≥0.5</td>
<td>5 d(^a)</td>
<td>50</td>
</tr>
<tr>
<td>Fulda et al(^9)</td>
<td>71</td>
<td>Adm, 6 h, 12 h, 18 h, 24 h, 48 h</td>
<td>TnT ≥0.2</td>
<td>Hospital stay(^b)</td>
<td>68</td>
</tr>
<tr>
<td>Ferjani et al(^10)</td>
<td>128</td>
<td>Adm, 4 h, 24 h</td>
<td>TnT ≥0.5</td>
<td>24 h(^a)</td>
<td>82</td>
</tr>
<tr>
<td>Adams et al(^11)</td>
<td>44</td>
<td>Adm, 6 h, 12 h, 18 h, 24 h, daily for 3 d</td>
<td>TnI &gt;3.1</td>
<td>3 d(^a)</td>
<td>100</td>
</tr>
<tr>
<td>Mori et al(^12)</td>
<td>32</td>
<td>6, 12, 24, 48, and 96 h</td>
<td>TnI ≥0.4</td>
<td>Hospital stay(^b)</td>
<td>100</td>
</tr>
<tr>
<td>Rajan and Zellweger(^13)</td>
<td>187</td>
<td>Adm, 6 h, 12 h, 18 h, 24 h, daily up to 7 d</td>
<td>TnI ≥0.35</td>
<td>3.3 mo</td>
<td>100</td>
</tr>
<tr>
<td>Bertiachant et al(^14)</td>
<td>94</td>
<td>Adm, 8 h, 16 h, 24 h, 48 h</td>
<td>TnI ≥0.1, TnT ≥0.1</td>
<td>17 mo</td>
<td>77 (TnI), 74 (TnT)</td>
</tr>
<tr>
<td>Edouard et al(^15)</td>
<td>728</td>
<td>Adm, 6 h, 12 h</td>
<td>TnI ≥2</td>
<td>Hospital stay(^b)</td>
<td>98</td>
</tr>
<tr>
<td>Velhamos et al(^16)</td>
<td>333</td>
<td>Adm, 4 h, 8 h</td>
<td>TnI &gt;1.5</td>
<td>Hospital stay(^b)</td>
<td>94</td>
</tr>
<tr>
<td>Kouritas et al(^17)</td>
<td>42</td>
<td>Adm, 24 h</td>
<td>Not specified</td>
<td>6 mo</td>
<td>100(^c)</td>
</tr>
</tbody>
</table>

Adm, sampled on admission; BCT, blunt chest trauma; NPV, negative predictive value; TnI, cardiac troponin I; TnT, cardiac troponin T.

\(^a\)Follow-up based on Tn measurement only.

\(^b\)No out-of-hospital follow-up, mean range 1–11.5 d.

\(^c\)With normal electrocardiogram and echocardiogram.
Echo

- ECG changes with troponin rise
- Persistent haemodynamic instability

Table 2

Echocardiographic findings in acute cardiac contusion

<table>
<thead>
<tr>
<th>Transthoracic echocardiography</th>
</tr>
</thead>
<tbody>
<tr>
<td>Regional wall motion abnormalities</td>
</tr>
<tr>
<td>Pericardial effusion</td>
</tr>
<tr>
<td>Valvar lesions</td>
</tr>
<tr>
<td>Right and left ventricular enlargement</td>
</tr>
<tr>
<td>Ventricular septum rupture</td>
</tr>
<tr>
<td>Intracardiac thrombus</td>
</tr>
<tr>
<td>Transoesophageal echocardiography</td>
</tr>
<tr>
<td>Aortic endothelial laceration or aortic dissection</td>
</tr>
<tr>
<td>Aortic rupture</td>
</tr>
</tbody>
</table>

Heart 2003 May; 89(5): 485–489
Commotio Cordis

- Sudden Cardiac Death due to blow to chest wall
- Triggers Ventricular Fibrillation
- Mechanism – 50kmh impact with small dense object, directly over cardiac silhouette left of sternum, 10-30 msec prior to the peak of the T wave, from depolarization primarily caused by mechano-sensitive ion channels
- 15% survival rate
- Young patients, males
Applying to our patient

ECG Changes – new RBBB
• Admitted for observation with holter monitoring
• May not have been new RBBB?

Serial Troponins Negative
• BCI unlikely – if taken alone evidence would suggest an NPV of 98-100%
• Unclear how to interpret in conjunction with abnormal ECG.
Current Management Guidelines

**Level 1 Evidence**
- ECG performed on patients with suspected BCI

**Level 2 Evidence**
- If ECG has abnormality -> patient should be admitted for observation
- Normal ECG + Normal Troponin = rule out (optimal timing yet to be determined)
- Echo for patients with haemodynamic instability or persistent new arrhythmia
- Sternal fracture alone does not predict cardiac injury
- Creatinine phosphokinase not useful
- Nuclear medicine studies add little over echo

Multidetector CT / CTA with ECG-gate capability evolving to be gold standard.

*Clancy et al 2012*
Case Closure

- Patient was admitted to ICU for epidural to prevent development of type II respiratory failure.
- Positive Pressure Ventilation
- Serial ECGs – NAD
- Discharged back to ward
- No BCI
- Discharged home with analgesia.
“any trauma patient with a likely mechanism who has chest wall pain and a new arrhythmia or cardiac pump failure has a cardiac contusion”

- Michael McGonigal
References


