CASE REPORT

Coronary artery dissection after blunt chest trauma

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SUMMARY

Blunt thoracic trauma may result in cardiac injuries ranging from simple arrhythmias to fatal cardiac rupture. Coronary artery dissection culminating in acute myocardial infarction (AMI) is rare after blunt chest trauma. Here we report a case of a 37-year-old man who had an AMI secondary to coronary dissection resulting from blunt chest trauma after involvement in a physical fight.

BACKGROUND

Various cardiac injuries, ranging from arrhythmias to fatal cardiac rupture, have been reported following blunt chest trauma.¹ Mortality associated with blunt chest trauma is up to 15%.² Acute myocardial infarction (AMI) following blunt chest trauma is rare. It may result from myocardial contusion or injuries to coronary arteries. The true incidence is not known. The left anterior descending artery (LAD) is the most susceptible vessel in blunt thoracic injuries and the most common cause of blunt chest injuries are road traffic accidents.^{1 3} The reason for LAD involvement in most cases of blunt chest trauma is its relation to the anterior chest wall. Both direct trauma as well as deceleration are the mechanisms of injuries. Coronary injuries can include coronary artery dissection, plaque rupture or epicardial haematoma, which may result in AMI. Coronary artery dissection is uncommon. However, coronary arteries have been found normal in some cases of traumatic AMI. Spasm or spontaneous thrombus lysis might be possible mechanisms in these cases. It is interesting to note that mild trauma may also result in AMI. Only a few reports are available in the literature for coronary injuries proven by coronary angiography highlighting the role of therapeutic percutaneous or surgical revascularisation modalities. We report a case of blunt chest trauma during a physical fight causing dissection of the mid-LAD artery resulting in AMI, confirmed by intravascular ultrasound (IVUS) and treated by angioplasty.

CASE PRESENTATION

A 37-year-old man, smoker with no other comorbidities, sustained blunt chest trauma after a physical fight. He was presented to the emergency department with chest pain for 3 h after being hit on the chest. The pain was retrosternal, radiating to the throat and associated with shortness of breath, perspiration and an episode of vomiting. Physical examination was remarkable for bruises on the chest and shoulders. He was advised admission for further management but he left against medical advice (LAMA). Next day he returned with

intermittent chest pain. He was haemodynamically stable, first and second heart sounds were of normal intensity and there was no pericardial rub, murmur or added heart sound heard on physical examination. He was in Killip class II.

INVESTIGATIONS

ECG on initial presentation showed ST-segment elevation in leads V4–V6 ([figure 1](#)). Subsequent ECG on next day was comparable. First troponin I was 9 and second was 94. The level of myocardial band fraction of creatine kinase was also raised. Haemoglobin and serum creatinine were normal. Transthoracic echocardiography showed akinetic apex, hypokinetic basal to mid-anterior septal and mid-anterior segments with estimated ejection fraction of 35–40%. No pericardial effusion was seen. A CT of the chest performed to rule out possible injuries to the lungs and great vessels was unremarkable. The following day coronary angiography was performed revealing a large thrombus burden in proximal to mid-segment of the LAD with distal embolisation in D1 and apical LAD with thrombolysis in myocardial infarction (TIMI) III flow ([figure 2](#)). Other coronaries were normal.

DIFFERENTIAL DIAGNOSIS

- Acute anterior wall MI
- Aortic dissection
- Chest trauma

TREATMENT

At this point no intervention was performed. The patient was started on anticoagulation and antiplatelet therapy. Tirofiban (glycoprotein IIb/IIIa inhibitor) infusion was given for 24 h and then enoxaparin (low molecular weight heparin) was continued. After 4 days the patient was taken to the catheterisation laboratory for a relook angiogram. Coronary angiography revealed resolution of thrombus. IVUS showed a dissection flap in the mid-segment of the LAD ([figure 3](#)). It was treated with two bare metal stents, 4.0×18 and 3.5×15 mm, deployed in proximal to mid-LAD with overlapping of edges and postdilated with a non-compliant 4.0×12 balloon. The final angiogram showed good results with TIMI III flow ([figure 4](#)).

OUTCOME AND FOLLOW-UP

The patient did well and was discharged home the next day.

DISCUSSION

Blunt chest trauma has been associated with various cardiac injuries including arrhythmias, septal rupture, valve damage, coronary artery injuries,

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Figure 1 ECG showing ST segment elevation myocardial infarction.

aortic dissection and cardiac rupture.⁴ The mechanism may involve direct trauma, rapid deceleration injury and myocardial compression between spine and sternum.^{2 5} Coronary artery injuries can cause AMI. The mechanism for AMI after blunt chest trauma may include coronary artery dissection, intimal tear, plaque rupture, spasm, thrombus formation or external compression by an epicardial haematoma.² Shearing forces during the traumatic episode may produce a small intimal tear with activation of platelets and fibrin resulting in thrombus formation.⁵ AMI following coronary artery dissection is rare after blunt chest trauma.^{2 5} The LAD is the most commonly involved vessel, followed by the right coronary artery. The left circumflex is rarely involved.^{2 5} The susceptibility of the LAD to injury after blunt trauma is attributed to its relation to the anterior chest wall.⁴

Non-penetrating chest trauma usually results from some sport activities and motor vehicle accidents. Here we report a case of coronary artery dissection after blunt chest trauma during a physical fight.

Patients with coronary artery dissection usually present with AMI and/or sudden death. AMI has been reported to occur immediately and up to 5 weeks after trauma. It has also been noted that mild trauma may result in AMI. Patients with blunt chest trauma are often young and usually present with chest pain, although this may not always be considered as a symptom of AMI.² The differential diagnoses of chest pain in these patients usually considered are pneumothorax, aortic dissection and rib fracture.^{1 2} Therefore, strong suspicion and early recognition is very important for prompt management.

In our case the patient had blunt chest trauma as he sustained kicks and punches during a fight. Kicks and punches during a physical fight are high energy blows that can lead to acceleration-deceleration trauma. He was easily recognised within the first few hours of trauma because of chest pain and ECG changes but denial on the part of the patient resulted in delayed management. Our patient presented with AMI within a few hours after blunt chest trauma. He had a dissection of mid-LAD resulting in thrombus formation and subsequent AMI.

Figure 2 Coronary angiogram showing proximal left anterior descending artery dissection.

Figure 3 Intravascular ultrasound: dissection flap.

Figure 4 Final result with thrombolysis in myocardial infarction III flow.

Troponin is a sensitive marker of myocardial injury and may be elevated in traumatic coronary injury. In our case it was significantly elevated. Transthoracic echocardiography may reveal regional wall motion abnormality in case of ischaemia and AMI. CT angiography is a useful imaging modality for evaluation of coronary anatomy and possible mechanism of myocardial infarction. IVUS and optical coherence tomography provide better quantitative and qualitative assessment of coronary arteries than angiography.⁵ They can differentiate between plaque rupture, thrombus, dissection or external compression and thus help in determining the cause of occlusion of coronary arteries. They aid in selecting the best management strategy. We used IVUS for the confirmation of dissection of mid-LAD segment.

AMI following non-penetrating injury has been managed in several ways. Percutaneous coronary intervention with or without stent, coronary artery bypass grafting and thrombolysis in the setting of AMI associated with blunt trauma have been described. In our patient, deferred percutaneous coronary intervention with stent implantation was performed on the fifth day. Initially, our patient was treated conservatively with glycoprotein

IIb/IIIa inhibitor and low-molecular weight heparin as he had a large thrombus burden that could not be aspirated. Stent implantation also does not provide benefit in such situations. Before being put on an anticoagulant, the patient had CT of the chest to rule out injuries to great vessels and other thoracic viscera, which may occur after blunt chest trauma. The long-term treatment of these patients should consist of acetylsalicylic acid and an ACE inhibitor. Treatment with a statin is debatable.

Learning points

- ▶ Blunt chest trauma usually results from motor vehicle collision but it can also be caused by trauma during a physical fight.
- ▶ Acute myocardial infarction after blunt chest trauma is rare, but it carries high mortality and morbidity.
- ▶ A high suspicion is mandatory for recognising the condition early. In an acute setting, a timely intervention can salvage the myocardium.

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Competing interests None.

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