Case Report

How to break a mended heart☆

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ARTICLE INFO

Keywords:
Coronary artery bypass graft  
Internal mammary arteries  
Blunt chest trauma  
Blunt trauma-associated coronary injury

ABSTRACT

Thoracic trauma poses a risk of injury to the thoracic organs and great vessels, including the coronaries. We present an interesting case of occult, life-threatening coronary bypass graft injury resulting from thoracic trauma. In this case, the diagnosis and management were contingent on understanding the nature of the bypass graft, which was not apparent at the time of presentation in extremis. Ultimate hemostasis required cardiac catheterization and placement of an exclusionary stent. Though there are several case reports describing native coronary injury resulting from thoracic injury, we found a single case of thoracic trauma-associated coronary bypass graft injury, which was managed medically. The case we present here demonstrates that though coronary bypass graft injuries are life-threatening and rare, they can be managed with techniques utilizing cardiac catheterization if accompanied by a high index of suspicion. This case further demonstrates that additional cardiac studies for patients who present with high-impact thoracic injuries and a history of coronary bypass grafts may facilitate expeditious diagnosis and effective management.

Introduction

The internal mammary arteries (IMAs) are the gold standard conduit for coronary artery bypass graft (CABG), according to the Society of Thoracic Surgeons Clinical Practice Guidelines. These guidelines are based on consistent findings demonstrating improved survival and graft patency when compared to venous and other arterial conduits [1]. IMA bypass conduits can be placed with the proximal end intact with the artery's origin on the subclavian artery and the distal end bypassing the coronary lesion, or they can be placed as a free graft with end-to-end anastomoses — one connected to the aorta and the other distal to the coronary lesion. The question arises: Do IMA grafts and changes in their anatomic location have any consequences for blunt chest trauma?

Case description

A 75-year-old man fell from a horse and was taken to an outside hospital, where he was found to have polytrauma. This included subarachnoid and subdural hemorrhages as well as left hemopneumothorax, clavicle, rib, and sacral fractures. He was intubated and transferred to a Level 1 trauma center after undergoing placement of a chest tube with reported 2600 mL bloody output. He was resuscitated with five units of packed red blood cells, four units of fresh frozen plasma, and a unit of cryoprecipitate. His known past medical history included coronary artery disease, congestive heart failure, atrial fibrillation on rivaroxaban, prior CABG, and percutaneous coronary interventions.

☆ The authors declare no conflicts of interest.
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https://doi.org/10.1016/j.tcr.2018.04.005
Accepted 16 April 2018
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On presentation to our trauma bay, his blood pressure was 161/126 mm Hg, he was tachycardic with a heart rate of 119 beats per min, his respiratory rate was 12 breaths per min, and his oxygen saturation was 98%. He was hypothermic at 35.8 °C. His Focused Assessment with Sonography in Trauma was positive in the left upper quadrant because of the left hemothorax. Notably, he was neurologically intact and able to follow commands in the trauma bay.

Pan CT and CT angiogram confirmed his known injuries (clavicle, sacral, and left 1–7 rib fractures). Active arterial bleeding was identified from a branch of the left subclavian artery that was in proximity to the rib fractures, which were mainly anterolateral with no more than two adjacent ribs with anterior and posterior fractures or evidence of clinically significant flail chest. Head CT at this time, compared to the initial head CT, showed a stable “small volume” subdural hematoma without midline shift or herniation.

Though he responded to initial resuscitation, his recurrent hypotension persisted. Angiography with interventional radiology was implemented for planned embolization. Surprisingly, thoracic angiography revealed the patient had a CABG utilizing the left IMA and the active hemorrhage was from a small side branch of his IMA graft (Fig. 1). Cardiac surgery was consulted for possible repair under direct visualization, but this course was ruled out because of his many injuries. Interventional Cardiology was consulted and the patient was then transferred to the interventional cardiology suite, which was better able to perform coronary angiography due to proximity to specialty-specific instruments and freedom from limitations of the trauma hybrid OR suite. Since the perforated branch was a small caliber vessel, it was excluded with a covered stent.

The resuscitation of the patient was guided using thromboelastograms and hematocrits based on arterial blood gases and complete blood counts. He ultimately received 14 units of packed red blood cells, six units of fresh frozen plasma, one unit of cryoprecipitate, two units of platelets, 50 units/kg of prothrombin complex concentrate, and 10 mg/kg of vitamin K. The chest tube had a post-procedure output of 3600 mL, which eventually subsided. He was placed on dual antiplatelet therapy for the stent. The following day, transthoracic echocardiography demonstrated left and right ventricular dysfunction (ejection fraction < 35%, right ventricular systolic pressure > 40 mm Hg); there was no baseline echocardiogram for comparison. There was a mild decline in the patient’s neurologic exam on postoperative day 1, with loss of ability to follow commands but preserved spontaneous purposeful movements. He had stable subarachnoid and subdural intracranial hemorrhages.

Troponins obtained on postoperative day 2 were mildly elevated but down trending (from 0.49 to 0.33). Electrocardiogram demonstrated atrial fibrillation with a rapid rate and Q waves in anterolateral leads. Unfortunately, he also developed multiple pulmonary emboli requiring low-intensity heparin infusion, which, in combination with dual antiplatelet therapy, likely resulted in acute progression of intracranial hemorrhage. He developed a midline shift and uncal herniation on postoperative day 4 that was incompatible with meaningful neurologic recovery, resulting in withdrawal of care and subsequent death 5 days after presentation.

Discussion

This case presents the rare scenario of CABG injury resulting from thoracic trauma. The challenges we encountered involved the diagnosis and management. This patient’s prior history of CABG should prompt inclusion of coronary injury in the differential diagnoses, especially in the setting of persistent occult intrathoracic bleeding. There is no significant discussion in the literature
regarding differential susceptibility to injury in the setting of trauma between native and bypass grafts. We did not obtain any cardiac marker levels, electrocardiogram, or echocardiogram on presentation; these may have helped identify myocardial ischemia in an obtunded patient. In this case, the differential diagnosis considered with a CT angiogram changes when the patient’s history of a possible IMA bypass graft is added to the clinical scenario.

Coronary vessel injury resulting from thoracic trauma, though rare, has been described in several case reports. The most common mechanism is due to penetrating injury from fractured ribs causing coronary vessel laceration or rupture [2]. There are also case reports of coronary vessel dissection resulting from blunt thoracic trauma. The left anterior descending artery and right coronary artery are the most frequently implicated native coronary vessels. Injuries to coronary vessels often result from high-energy mechanisms of injury and are associated with thoracic cage injuries such as rib fractures, myocardial and pulmonary contusions, and aortic and thoracic vessel injuries, which may obfuscate signs of coronary vessel damage such as myocardial ischemia, hemopericardium, and hemothorax, and require a high index of suspicion. Abu-Hmeidan et al. compiled cases of coronary injury from blunt thoracic trauma that demonstrate a time lapse from injury/presentation to death/intervention ranging from 2 to 56 h [3]. This suggests that survivability of life-threatening injury may be optimized if coronary vessel injury is identified and managed effectively. Described effective interventions include vessel ligation, CABG, and stent placement [3–5].

Despite the many cases of CABG, there is a paucity of literature describing traumatic injury to bypass grafts. Considering the anatomical alterations that bypass grafts present to coronary anatomy, perhaps bypass grafts are more susceptible to injury in cases of thoracic trauma. However, only a single case was found during a search of the literature that presented with a vein graft occlusion 4 days after a motor vehicle collision that was managed medically [6].

In this case, the hemorrhagic shock was manageable with typical resuscitation and did not require damage control thoracotomy. Thoracotomy would have been more challenging due to prior sternotomy and would have placed additional risk of left IMA graft injury [7]. With injury to the graft, small lacerations and incomplete resolution on CT angiogram may decrease the likelihood of a thoracotomy successfully identifying the laceration [8,9]. This case highlights the role for interventional radiology, which has less differential susceptibility to injury in the setting of trauma between native and bypass grafts or among different types of bypass grafts. We did not obtain any cardiac marker levels, electrocardiogram, or echocardiogram on presentation; these may have helped identify myocardial ischemia in an obtunded patient. In this case, the differential diagnosis considered with a CT angiogram changes when the patient’s history of a possible IMA bypass graft is added to the clinical scenario.

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Conclusion

This case reminds us that blunt trauma-associated coronary injury, though life-threatening and rare, can be managed in the cardiac catheterization lab but requires a high index of suspicion. Additional cardiac studies for patients with high-impact thoracic injuries and a history of CABG, such as EKG, echocardiography, and coronary angiography, may identify injuries more quickly.

Conflict of interest statement

None.

Funding

None.

Acknowledgement

The authors would like to acknowledge Mark Edmiston, MD and Calvin Choi, MD, MS, FACC, FSCAI, who contributed intellectually to the content of this manuscript.

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