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Occlusive Coronary Artery Dissection Following a Motor Vehicle Collision

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Background	Motor vehicle collisions are the most common cause of blunt chest wall trauma. Nonpenetrating chest trauma may result in cardiac injuries that can vary from asymptomatic to fatal. Although rare, coronary artery dissection is a potential life-threatening complication of blunt chest trauma.
Summary	A 22-year-old male with no previous history of cardiovascular disease presented after a high-speed rollover motor vehicle collision. In the trauma bay, he complained of chest pain and dyspnea. His troponin was elevated at 0.094ng/mL. Electrocardiogram findings were significant for ST elevation in the anterolateral leads, and echocardiogram demonstrated markedly reduced left ventricular ejection fraction (LVEF). Anterior myocardial infarction was suspected; therefore the patient underwent coronary angiography. He was found to have a proximal left anterior descending (LAD) artery dissection causing a complete occlusion of the mid-LAD artery. He underwent percutaneous transluminal coronary angioplasty and bare metal stent placement with successful restoration of blood flow to the distal LAD artery; however LVEF remained low on follow-up echocardiograms.
Conclusion	The manifestations of traumatic cardiac injury may present hours, days, or weeks after the inciting event. Patients complaining of chest pain after sustaining blunt chest trauma should undergo prompt cardiovascular workup. The management of traumatic coronary artery dissection remains disputed due to its rare occurrence. Currently accepted treatments include conservative management, angiography with stenting, and surgical revascularization. Patient prognosis is variable and dependent on the extent of vessel occlusion.
Keywords	LVEF: left ventricular ejection fraction; LAD: left anterior descending; ECG: electrocardiogram

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Case Description

A quarter of all trauma-related mortalities can be attributed to thoracic injuries.[1] Blunt trauma of the chest wall may be the result of extrinsic thoracic compression or may be caused by a rapid acceleration-deceleration force. Motor vehicle collisions account for 70 to 80 percent of blunt chest trauma.[2] When a patient presents after having sustained a known or suspected nonpenetrating chest trauma, trauma surgeons should have a high index of suspicion that a cardiac injury has occurred.[3] Due to its positioning between the vertebral column and the sternum, the heart is vulnerable to injury from blunt chest trauma.[4] The spectrum of pathology associated with blunt cardiac injury ranges from asymptomatic to fatal. The various cardiac injuries that may result from nonpenetrating chest trauma include myocardial contusion, simple or complex arrhythmias, hemopericardium with or without cardiac tamponade, valvular damage, septal wall rupture, free wall rupture, ventricular aneurysm, and pericardial laceration or rupture. An uncommon sequelae of blunt chest trauma is myocardial infarction caused by either myocardial contusion, or more rarely, due to coronary artery dissection.[5-9] Because of its infrequent occurrence, the current understanding of traumatic coronary artery dissection originates primarily from case reports.[10] This report describes the case of a healthy young male presenting with a coronary artery dissection that resulted in a myocardial infarction after a motor vehicle collision.

The patient was a 22-year-old male involved in a rollover motor vehicle collision. He was the unrestrained driver of a vehicle estimated to have been traveling at 65 miles per hour. The vehicle was found thirty-five feet from the road and the airbags did not deploy. The patient had to be extricated from the vehicle by emergency personnel.

On arrival to the trauma bay, the patient was complaining of significant chest pain with associated shortness of breath. His heart rate was averaging 120 beats/minute with blood pressures of 150/90 mm Hg. The primary survey revealed diffuse chest wall tenderness, especially over the sternum, and a right lower extremity that was shortened and internally rotated. He was noted to have ST changes on telemetry, and a twelve-lead electrocardiogram (ECG) demonstrated sinus tachycardia with ST elevation in the anterolateral leads and reciprocal depression in the inferior leads (Figure 1). His troponin T level was 0.094 ng/mL.

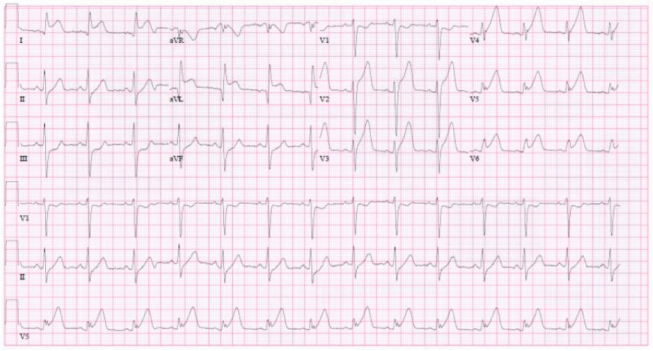

Figure 1. Twelve-lead electrocardiogram demonstrating sinus tachycardia with ST elevation in the anterolateral leads and reciprocal depression in the inferior leads

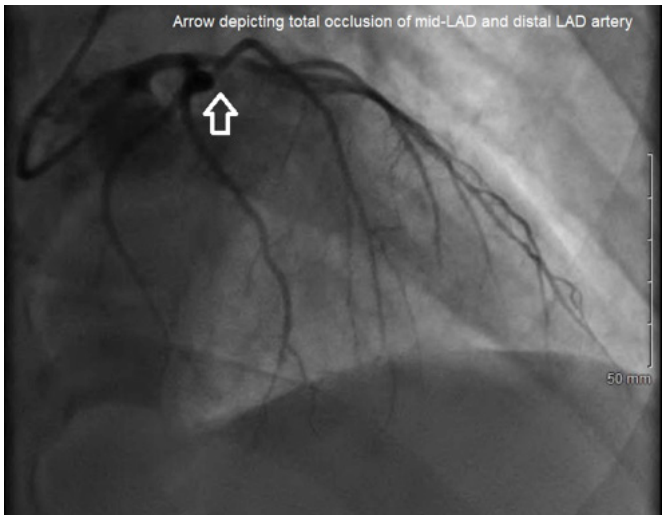

Figure 2. Initial coronary angiography demonstrating total occlusion of the mid-LAD artery as the result of a dissection in the proximal portion of the LAD artery

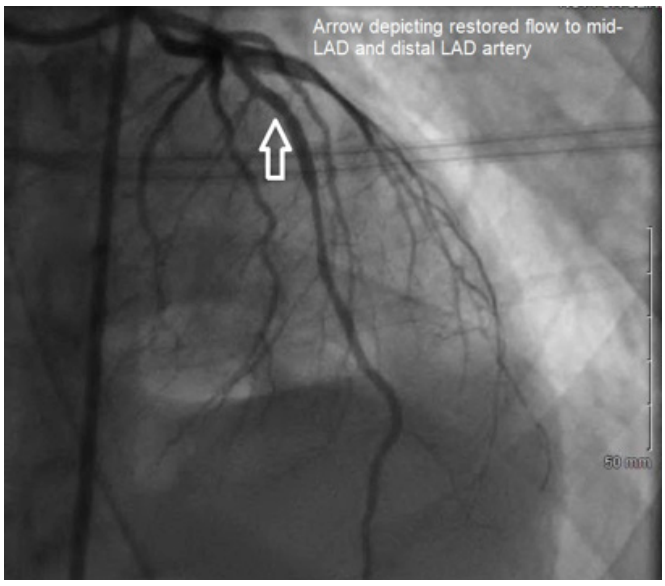

Figure 3. Coronary angiography after angioplasty and stent placement demonstrating restored blood flow to the distal LAD artery

There were no indications of cardiovascular disease or other serious health problems in the patient's past medical history. He denied prior cardiac symptoms. He endorsed a four-year pack-per-day smoking history. Family history was significant for a mother with hypertension and diabetes mellitus; however there was no family history of myocardial infarction or stroke.

The focused assessment with sonography for trauma exam did not demonstrate any evidence of pericardial effusion or free fluid. Computed tomography demonstrated a small soft tissue density in the anterior mediastinum, a displaced right acetabular fracture, a nondisplaced left acetabular fracture, a right-sided retroperitoneal hematoma, and a C5 corner fracture. Orthopedic surgery performed a right hip reduction with right femur traction pin placement in the trauma bay. The patient was then transferred to the surgical intensive care unit.

Despite fluid resuscitation and analgesic administration, the patient became increasingly more tachycardic, with heart rates reaching 140 beats/minute. A repeat ECG demonstrated worsening of the ST elevation in the anterolateral leads. A bedside echocardiogram revealed a left ventricular ejection fraction (LVEF) of 35 percent with hyperdynamic function of the basal segments, hypokinesis of the mid segments, and akinesis of the apical segments.

The patient was transported to the cardiac catheterization suite, where he underwent coronary angiography and cardiac catheterization. The patient was found to have a dissection in the proximal portion of the left anterior descending (LAD) coronary artery resulting in a complete occlusion of the mid-LAD artery (figure 2). Balloon angioplasty was performed with restoration of normal flow (figure 3). A bare metal stent, measuring 3mm x 24mm, was placed from the proximal LAD to the proximal first diagonal artery. Angiography did not reveal abnormalities of any of the other coronary vessels.

Despite percutaneous transluminal coronary angioplasty and bare metal stent placement, the patient's LVEF consistently remained around 25 percent on repeat echocardiograms. The patient's troponin level gradually trended down; however, he demonstrated persistent ST elevation on telemetry suggestive of microvascular obstruction. In addition to dual antiplatelet therapy with aspirin and clopidogrel, he was prescribed a daily beta blocker and angiotensin converting enzyme inhibitor for mortality benefits secondary to his ischemic cardiomyopathy. Given the patient's precarious cardiac status, orthopedic surgery postponed all operative interventions until a later date. The patient was eventually discharged to an acute rehabilitation facility. At the time of discharge, the patient was asymptomatic from a cardiac standpoint. Even though congestive heart failure symptoms were not observed, and follow-up myocardial perfusion imaging did not demonstrate thrombosis of the stent, surveillance echocardiograms demonstrated gradual decline in the patient's LVEF despite pharmacologic optimization. An implantable cardioverter defibrillator was placed for primary preventative measures; however, there are no plans for left ventricular assist device placement or cardiac transplantation unless the patient becomes symptomatic.

Discussion

It is presumed that extrinsic compression of the chest wall or sudden deceleration forces can produce a shearing of the coronary vessel wall, which then produces intimal tearing and subintimal hemorrhage. The tear activates platelet aggregation, fibrin, and other clotting cascade components precipitating intraluminal thrombus formation, with or without coronary spasm.[10-12] The intraluminal thrombus, coronary spasm, or combination of the two can eventually lead to coronary artery occlusion. It is hypothesized that the anatomic location of the LAD artery, specifically the junction of the proximal and mid-portions, place it at greater risk of extrinsic compression from the anterior chest wall.[10,11]

The manifestations of traumatic coronary artery dissection are variable. Depending on the expansion of the false lumen, symptoms may not present for several days, or even weeks, following the inciting event.[13,14] In certain situations, the dissection may be observed and treated conservatively, whereas in other cases it has the potential to generate malignant ventricular arrhythmias and could result in sudden cardiac death.[15] As seen in this patient, coronary artery dissection can lead to coronary ischemia and myocardial infarction. Acute EKG changes in the setting of blunt chest trauma should trigger prompt cardiovascular evaluation, including serial cardiac enzymes and echocardiography.[15] In the setting of coronary artery dissection, early recognition and diagnosis with transesophageal echocardiogram or coronary angiography may limit the impact of the infarction.[10,16,17] For this particular patient, there are several possibilities as to why the myocardium did not

regain function. One potential explanation could be the presence of direct myocardial contusion. While the presence of direct myocardial contusion could have conceivably exacerbated the myocardial ischemia caused by the coronary artery dissection, it ultimately would not have changed patient's treatment strategy. An additional possibility may have been persistent microvascular occlusion and lack of microvascular integrity. Alternatively, a study by Ito et al suggested that abrupt restoration of myocardial blood flow by angioplasty could worsen the damage of the postischemic coronary microvasculature.[18]

Due to its rare occurrence, the optimal treatment of traumatic coronary artery dissection is currently not well-established, therefore management must be individualized.[6,8,16] Based on how a patient presents, one may pursue conservative management, angiography with stenting, surgical revascularization, or thombolytic therapy.[11] In the presence of occlusion or stenosis, reperfusion may be achieved by means of direct angioplasty.[17] The use of thrombolytics in polytrauma patients remains controversial.[16,17] Furthermore, there is concern that thrombolytic therapy could worsen dissections in the acute phase.[8] Although the patient's myocardium ultimately did not regain function following angioplasty and stenting, it is difficult to know whether an alternative management strategy would have been more advantageous. Surgical revascularization in the form of coronary artery bypass is a maximally invasive procedure compared to cardiac catheterization, and the associated morbidity and mortality risks did not outweigh the potential benefits in this particular patient at initial presentation. For these reasons, we would likely implement the same management strategy for similar cases in the future. Had there been evidence of stent thrombosis on repeat myocardial perfusion imaging, the option of surgical revascularization would have been reconsidered.

There are a variety of factors that can affect a patient's prognosis, with severity and duration of vessel occlusion being of particular importance.[8] It is thought that in the absence of ongoing ischemia, complete healing of traumatic coronary occlusion occurs within six months of initial injury; however, these patients are still at risk of developing aneurysmal disease.[10] In addition, there are concerns that a persistent intimal flap could act as a nidus for rethrombosis, ultimately leading to restenosis.[12] In regards to prognosis, our patient's three-year mortality risk is less than ten percent based on the Meta-Analysis Global Group in Chronic Heart Failure risk calculator. This case illustrates the complexities of the decision-making process involved with caring for polytrauma patients. Although the patient suffered permanent cardiac damage related to his traumatic injuries, he was still able to achieve an acceptable quality of life in the postrecovery period.

Conclusion

Albeit rare, coronary artery dissections are a potentially life-threatening complication of blunt chest trauma. This report describes the case of a healthy young male presenting after a motor vehicle collision, and subsequently found to have a proximal dissection of the left anterior descending artery leading to an anterior wall myocardial infarction. The report also highlights the importance of prompt cardiovascular workup initiation by trauma surgeons for patients complaining of chest pain after having sustained blunt chest trauma.

Lessons Learned

The manifestations of traumatic cardiac injury may present hours, days, or weeks after the inciting event. The management of traumatic coronary artery dissection remains disputed due to its rare occurrence. Patient prognosis is variable and dependent on the extent of vessel occlusion.

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