



Clinical case report based study

Cardio-embolic stroke following remote blunt chest trauma

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ABSTRACT

A cardio-embolic stroke as a sequela of remote blunt chest trauma is a rare clinical presentation. Blunt chest trauma can cause various acute cardiac complications like arrhythmias, cardiac contusion etc. However, delayed consequences such as left ventricular thrombus resulting in thromboembolic phenomena are reported infrequently.

A 30-year-old healthy man presented to an outside facility with transient neurological deficits. An MRI brain showed lesions suggestive of embolic etiology. A trans-thoracic echocardiogram (TTE) showed a 1.5 × 1.5 cm mass present in the left ventricular (LV) apex. Patient was transferred to our institution for cardiac surgery evaluation. On detailed questioning, he reported an incident of blunt chest trauma during a martial arts exhibition fight that took place 2 years back. Given this history, a cardiac catheterization was done, which showed 30% stenosis in mid-left anterior descending artery (LAD) without any other significant obstructive lesion. A trans-esophageal echocardiogram (TEE) showed akinesis of the LV apex and confirmed TTE finding of a mass, consistent with an apical thrombus. Surgery was deferred and patient was started on anticoagulation. A cardiac MRI done 2 weeks later showed evidence of apical infarction in the LAD territory.

LAD is the most commonly affected coronary vessel by blunt traumatic injuries, likely due to its vulnerable anatomical position on the anterior aspect of the heart. A variety of mechanisms including intimal tear, rupture and spasm have been implicated in the pathogenesis of myocardial infarction after blunt chest trauma.

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1. Introduction

Acute cardiovascular complications of blunt chest trauma such as, arrhythmias, valvular avulsions, myocardial contusion and rupture have been reported.¹ However, a delayed presentation with cardio-embolic stroke as a complication is unanticipated and has rarely been reported. Our review of literature did not find any case reports of an embolic stroke as a delayed complication of blunt chest trauma.

The present case highlights the importance of recognizing potential complications and also the importance of detailed history taking in the accurate diagnosis of this condition in order to prevent unnecessary and potentially dangerous inter-ventions.

2. Case report

A 30-year-old previously healthy Caucasian male presented to an outside facility with word finding difficulty and right sided neglect lasting for few minutes during a wrestling match. At presentation, his symptoms had resolved completely.

Past medical history was unremarkable. He did not smoke or consume alcohol and did not have a family history of hypercoagulable disorders or coronary artery disease. He was not on any home medications and led an otherwise healthy lifestyle with vigorous sporting activities.

Neurological examination at admission was within normal limits. A CT scan of the brain was unrevealing. A subsequent MRI of the brain showed 3 punctate lesions in the left cerebral hemisphere, concerning for an embolic phenomenon. A trans-thoracic echocardiogram (TTE) was performed, which showed a 1.5 × 1.5 cm hyperechoic mass present in the left ventricular apex. In the absence of a history of cardiomyopathy or coronary artery disease and the absence of obvious regional wall motion abnormalities, a concern for tumor was raised and the patient was

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transferred to our institution for further evaluation by cardiac surgery regarding possible excision of mass.

On presentation to our institution, he was stable without further neurological symptoms. On detailed questioning, he reported an incident of blunt chest trauma with a kick to the chest during a martial arts exhibition fight that took place 2 years ago. He had experienced severe crushing chest pain radiating to arms bilaterally, with diaphoresis and dyspnea following the episode. He visited his local emergency room and was sent home after a period of observation. His chest pain resolved by itself and he had no recurrence of symptoms. The patient did not recall being given a specific diagnosis and details of the investigations done were unavailable.

On examination, he appeared well without distress. Vital signs revealed a blood pressure of 100/70 mmHg with a heart rate of 85 beats per minute. The respiratory rate was 18 breaths per minute. He had no carotid bruits. Cardiac exam revealed normal heart sounds, with no audible murmurs or rubs. The lungs were clear to auscultation and abdominal examination was unremarkable. There was no peripheral edema. Neurological examination was normal without any focal neurological deficits.

Initial laboratory studies revealed a white blood cell count of $7.2 \times 10^3/\text{mm}^3$, hematocrit of 44% and platelet count of $257 \times 10^9/\text{L}$. Coagulation profile showed an international normalized ratio of 1.1. Electrolytes and renal function studies were within normal limits. Electrocardiogram (EKG) showed normal sinus rhythm and non-specific T wave abnormalities. Chest X-ray was within normal limits.

TTE was reviewed and was concerning for possible small apical infarct. Given these findings and the past history of chest pain in the setting of blunt chest trauma, potential traumatic injury to the coronary arteries was suspected and a cardiac catheterization was performed. Patient was noted to have a right dominant coronary circulation without obstructive coronary disease. Of note, the mid segment of his left anterior descending artery (LAD) had a focal, 30% stenosis (Fig. 1). Proximal LAD was normal. Given these findings, the initially planned surgical intervention was held and given the poor image quality of the TTE, a trans-esophageal

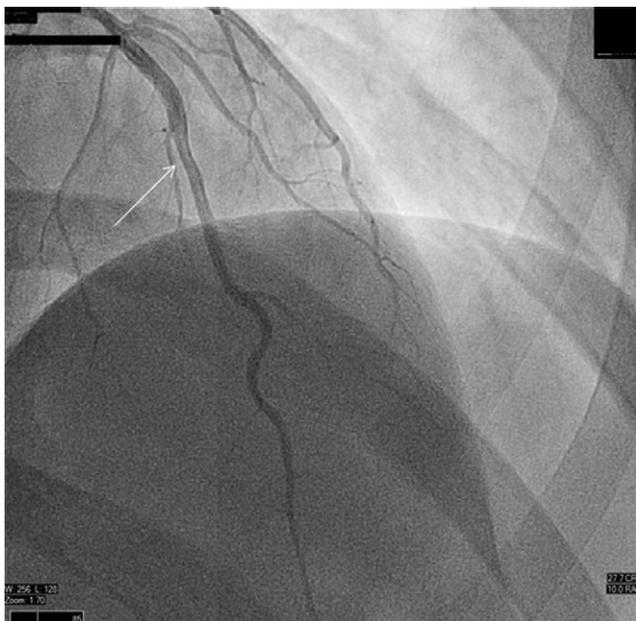


Fig. 1. Cardiac catheterization, right anterior oblique cranial view showing mid-LAD 30% focal, non-obstructive lesion.

echocardiogram (TEE) was performed (Fig. 2). This confirmed the presence of a mass in the left ventricular apex, the appearance of which was consistent with a thrombus rather than a tumor. The TEE also showed akinesis of the apical wall. The patient was started on therapeutic anticoagulation with intravenous heparin and warfarin.

A cardiac magnetic resonance imaging (MRI) was done 2 weeks after initiating anticoagulation. Delayed hyperenhancement sequence demonstrated areas of sub-endocardial and transmural infarction associated with moderate to extensive akinesis of the apical segment, consistent with LAD territory infarction (Fig. 3). The previously noted LV thrombus had completely resolved.

It was concluded that the LV thrombus was a delayed sequela of his apical infarction, probably secondary to a dissection of his LAD resulting from blunt chest trauma.

3. Discussion

Cardio-embolic phenomenon as a sequela of remote blunt chest trauma is an unexpected and exceedingly rare presentation. Blunt chest trauma has been reported to cause direct cardiac damage in 5–15% of the cases.² The following complications have been reported; arrhythmias, valvular avulsions, myocardial contusion, rupture, and rarely coronary artery dissection.³

Traumatic coronary artery injury and/or dissection is a potentially life threatening complication to consider in patients presenting with prolonged chest pain following chest trauma. The most common mechanisms of injury include direct, bidirectional, decelerative, blast, and concussive forces.⁴ The most frequently affected vessel is the LAD (71–76% of cases) followed by the right coronary (12–19.0%) and the circumflex coronary artery (3.2–6%). The anatomic proximity of the LAD to the anterior chest wall probably makes it more vulnerable to damage from direct trauma.^{5,6}

Sudden cardiac death is a common mode of presentation and early mortality is high.^{7,8} While dissections leading to occlusion of the vessel might present with obvious ischemic changes on EKG (ST-segment elevations), prompting emergent intervention, non-occluding dissections might remain sub-clinical and undiagnosed. Patients who initially develop non-occlusive dissections, may often have delayed onset or worsening of symptoms as the dissection propagates resulting in clot expansion and compromise of the true lumen.⁹ In those who survive the initial trauma, failure to recognize this underlying condition might result in a missed opportunity to intervene and prevent disastrous consequences both in the short and long term.

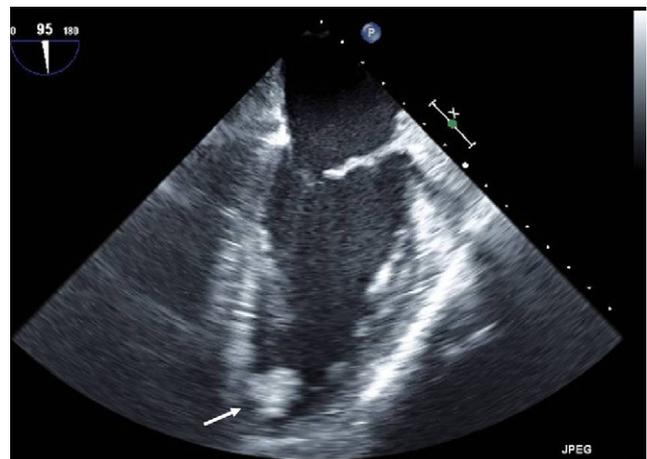


Fig. 2. Mid-esophageal view of TEE showing apical thrombus.

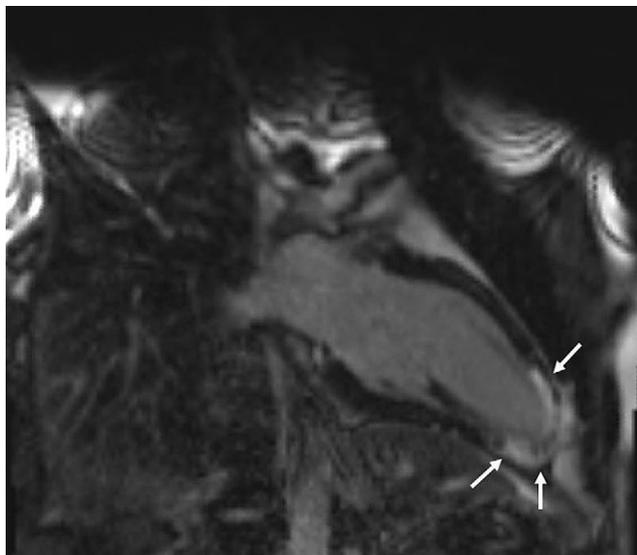


Fig. 3. Vertical long axis view of cardiac MRI, delayed hyperenhancement sequence showing areas of sub-endocardial and transmural infarction (white areas) in the LAD territory. Apical thrombus is not seen.

Ventricular thrombus formation following blunt chest trauma and subsequent peripheral embolic phenomena has been reported previously.^{1,10}

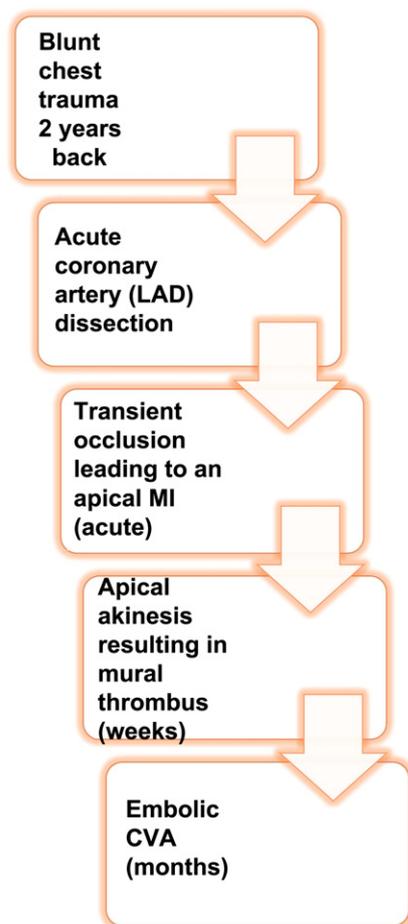


Fig. 4. Likely sequence of events leading to cardio-embolic stroke.

4. Likely mechanism of LV thrombus formation and embolization in our patient

The most likely sequence of events was that a missed anterior myocardial infarction due to LAD dissection led to the formation of a LV thrombus in the setting of an akinetic anterior myocardial segment, with subsequent embolization of the clot (Fig. 4). It has been reported in prospective studies in the 1980's (pre-reperfusion era) that the incidence of LV thrombi could be as high as 40% in patients with untreated anterior infarction.^{11,12} In this patient with an effectively untreated or non-reperused myocardial infarction, his risk for thrombus formation was much higher than in a patient with some form of reperfusion (thrombolysis or percutaneous coronary intervention). Most thrombi have been known to develop within 2 weeks of the myocardial infarction. This still leaves the question of delay in the occurrence of the embolic event. We believe that the patient had the undetected LV thrombus for more than a year prior to embolization. Evidence to support this hypothesis is limited; however, in a series of 85 patients with LV thrombus, an embolic event occurred in 11 (13%) of those with documented LV thrombi.¹³ The patients in this study were followed for up to 2 years and although cases of delayed embolization were uncommon, they were reported. Embolization delayed for up to 96 months after myocardial infarction was reported in that study.¹³

5. What might lead to a missed diagnosis of this condition?

Firstly, patients might be young and have no risk factors for coronary artery disease (CAD) and their symptoms can be attributed to musculoskeletal chest pain from the trauma and hence missing this diagnosis is easy.¹⁴ In one published series, 82% of the patients with acute myocardial infarction after blunt chest trauma were less than 45 years old. In this series, the most common trauma was motor vehicle accidents, followed by sporting accidents involving kicks or shoulder impact to the chest during wrestling matches, soccer, rugby and basketball.⁶

Secondly, various conventional investigations that are available to routinely diagnose acute myocardial infarction might have significant limitations in this setting. New or presumably new ischemic EKG changes (ST-segment elevation and diffuse ST-segment depressions) might alert the emergency room physicians to this dangerous condition. However, open chest wounds or skin damage might hinder obtaining an EKG in a timely fashion and patients with non-occlusive dissections may not have ischemic changes on their EKG. Elevated cardiac biomarkers may not be diagnostic as they could be elevated due to other reasons such as cardiac contusion, which is not uncommon in these patients.

Finally, imaging modalities such as echocardiography to look for wall motion abnormalities and resting perfusion imaging looking for perfusion defects might aid in diagnosis but are also fraught with many of the above limitations.

In our patient, many of the above mentioned reasons could have led to a missed diagnosis. The extent of myocardial injury was small and did not compromise his overall cardiac function and he did not have significant CAD, which explains his asymptomatic clinical course for the two years following his trauma. The tell tale signs of a probable traumatic dissection, in the form of a focal 30% lesion (after undergoing remodeling) in his distal LAD helped us to come to a diagnosis.

There are no established guidelines recommending routine screening for coronary artery dissection in patients presenting with blunt chest trauma. Despite all their limitations, it is important to get an EKG and cardiac biomarkers at presentation and have a low threshold to repeat these tests if patient's symptoms change.¹⁵ Imaging modalities such as TTE, TEE, ECG-gated CT scan and cardiac MRI can be used to aid in the diagnosis and quantify the extent

of cardiac damage.¹⁶ It is reasonable to have a low threshold to perform urgent coronary angiography if acute ischemia due to coronary artery dissection is suspected.⁹ According to the review by Christensen et al, 90% of patients who survived myocardial infarction had angiography at some point. In these patients, angiography was employed immediately on presentation or after several months following the trauma.⁶ When coronary angiography was delayed for several months, often no evidence of disease was found. The likely explanation is spontaneous clot lysis within the false lumen.²

Treatment options for traumatic acute coronary artery dissection include medical therapy (thrombolytics, antithrombin and antiplatelet agents), angioplasty with or without stenting, bypass surgery and in extreme cases, cardiac transplantation.⁹ A case series that reviewed 24 patients revealed that 8 patients were treated with coronary artery bypass grafting, 7 were treated with stent placement, and 9 were treated conservatively.¹⁷ Given that the mechanism of coronary artery occlusion in coronary artery dissection is not thrombosis, it is perhaps prudent to avoid thrombolytics and glycoprotein IIb/IIIa antagonists. There exists the theoretical risk of propagation of the dissection with these agents.^{18–20}

Once delayed sequela have occurred, as in our patient, the treatment focuses on treatment of thrombus, secondary prevention of LV thrombus formation and cardio-embolic phenomena with anticoagulation. Patients with an anterior myocardial infarction and an LV ejection fraction less than 30% are at particularly high risk of systemic embolization. Although no randomized control trial data exist, in a meta-analysis that included 270 patients with anterior myocardial infarction and documented LV thrombus, anticoagulation (compared to no anticoagulation) was associated with an 86 percent reduction in the rate of embolization.²¹ Based on these findings, the 2012 American College of Chest Physicians Guidelines on Antithrombotic Therapy and Prevention of Thrombosis Guidelines recommends the use of warfarin for at least 3 months with a goal international normalized ratio of 2.0–3.0, followed by patient-selective decisions to continue anticoagulation.²²

In conclusion, this case of unrecognized myocardial infarction in the setting of blunt chest trauma highlights the importance of careful consideration of cardiac injury in the emergency setting. It is essential to include less likely but considerably ominous conditions in the differential. Additionally, it is equally important to obtain a detailed history regarding past traumatic events when young and otherwise healthy individuals present with evidence of cardiac damage.

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Conflicts of interest

All authors have none to declare.

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