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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.

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 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

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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 $7.2 \times 10^3/mm^3$, hematocrit of 44% and platelet count of $257 \times 10^9/L$. Coagulation profile showed an international normalized ratio of 1.1. Electrolytes and renal function studies were within normal limits. Electrocardiogram (ECG) 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 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 ECG (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.

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

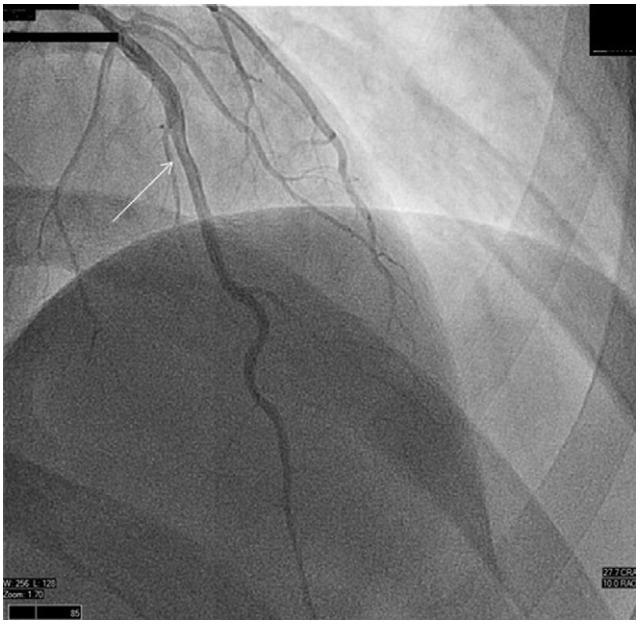

Fig. 1. Cardiac catheterization, right anterior oblique cranial view showing mid-LAD 30% focal, non-obstructive lesion.

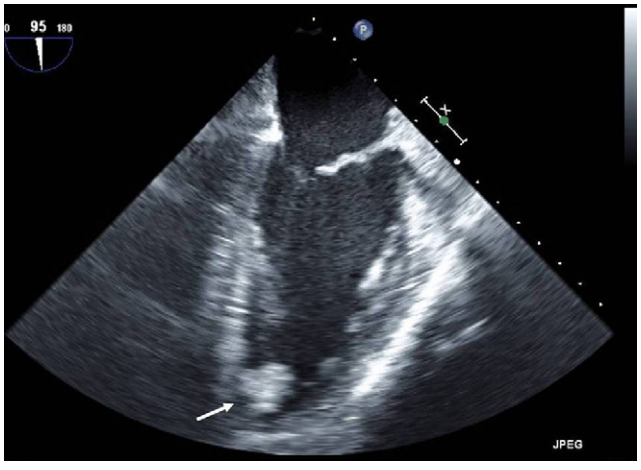

Fig. 2. Mid-esophageal view of TEE showing apical thrombus.