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Acute ST elevation myocardial infarction due to cricket ball injury

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Abstract
A 31-year-old previously healthy man presented to the Emergency Department due to Cricket Ball Injury. Cricket Ball Injury to the chest resulting in coronary thrombosis and ST elevation myocardial infarction (STEMI) is a rare case. An ECG revealed sinus tachyycardia at 112 beats/min with ST segment elevation in leads V1 to V4 and ST segment depressions in leads II, III, aVF and V6. An echocardiogram demonstrated severe hypokinesis of the mid and distal anteroseptal segments and the apex, with an estimated left ventricular ejection fraction of 47%. Angiography in such cases has generally disclosed complete epicardial coronary occlusion with thrombus, indistinguishable from the findings commonly found in spontaneous plaque rupture due to atherosclerotic disease. Coronary angiography via the right radial artery disclosed a total occlusion of the left anterior descending artery 2.5 mm from its origin. The treatment approach to STEMI following blunt chest trauma is immediate coronary angiography and revascularization with percutaneous intervention and continuation of dual antiplatelet therapy. Ongoing pain represents myocardial ischemia should be considered, and a screening ECG should be considered to identify rare cases of STEMI.

Keywords: cricket ball injury, STEMI, ECG

1. Introduction
Cardiac injury is an uncommon complication of blunt trauma; in most cases of cardiac dysfunction following blunt trauma injury, cardiac contusion is implicated. Arrhythmic death has been reported in children following blunt force injuries to the chest. In rare cases, coronary thrombosis and STEMI have been associated with blunt trauma to the chest in adults [1-3]. Angiography in such cases has generally disclosed complete epicardial coronary occlusion with thrombus, indistinguishable from the findings commonly found in spontaneous plaque rupture due to atherosclerotic disease [1-3]. In several reports, the presence of coronary artery dissection has been documented as the underlying pathophysiologic trigger for thrombosis [4-8]. We present the first case report of *Cricket Ball* trauma-associated coronary thrombosis without underlying atherosclerosis or coronary dissection.

2. Case Report
A 31-year-old previously healthy man presented to the Emergency Department after sudden hit of *Cricket Ball*. He initially complained of epigastric pain and nausea; she also reported substernal chest discomfort that became progressively more prominent. He had no coronary atherosclerotic risk factors and did not use oral contraceptives. He had stable vital signs; physical examination was notable only for abrasions of the chest wall. An ECG revealed sinus tachyycardia at 112 beats/min with ST segment elevation in leads V1 to V4 and ST segment depressions in leads II, III, aVF and V6(Figure 1A). CT imaging of the chest, disclosed a small left pneumothorax, focal myocardial hypoattenuation suggesting the possibility of cardiac contusion, and soft tissue attenuation of the subhepatic porta hepatis indicating hemorrhage. An echocardiogram demonstrated severe hypokinesis of the mid and distal anteroseptal segments and the apex, with an estimated left ventricular ejection fraction of 47%. The aortic root was without evidence of dissection; there was no pericardial fluid or aortic valvular insufficiency. Because of the ECG and echo findings, emergent cardiac catheterization was performed. Coronary angiography via the right radial artery disclosed a total occlusion of the left anterior descending artery 2.5 mm from its origin (Figure 1B).

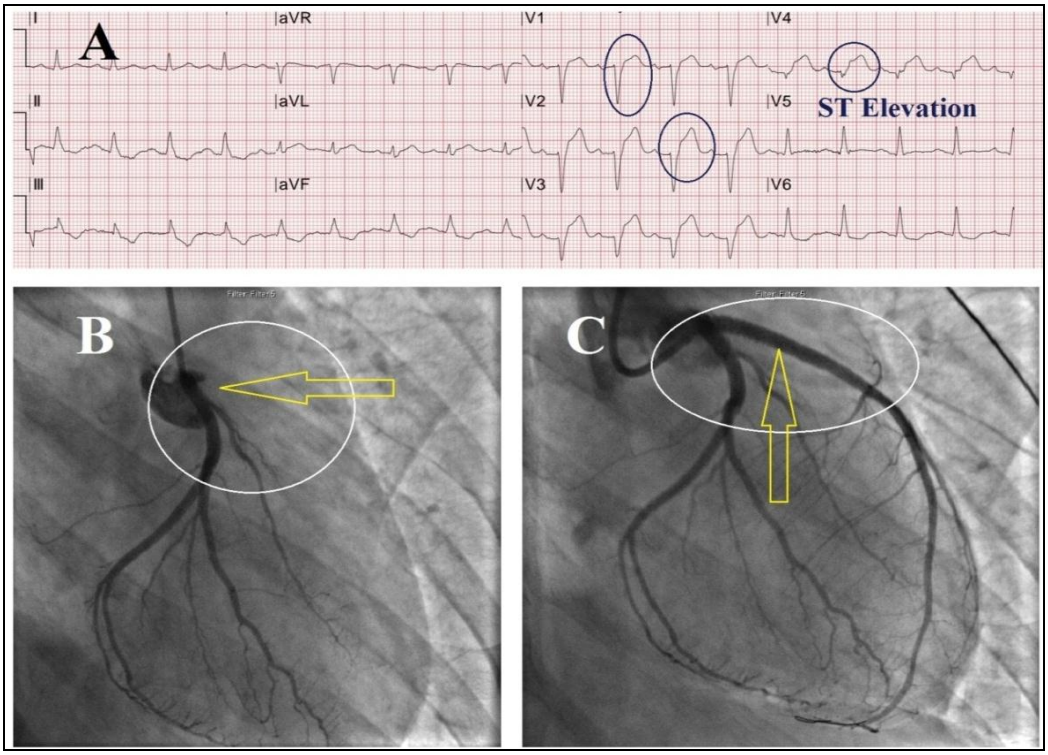

Fig 1(A): ECG showing ST segment elevation in leads V1 to V4 and ST segment depressions in leads II, III, aVF and V6. **(B):** (a) Coronary angiogram showing total occlusion of LAD. **(C):** Restoration of normal flow following balloon dilation and stent deployment.

All other epicardial vessels appeared to be normal. Antiplatelet and antithrombotic therapies with aspirin, clopidogrel, and bivalirudin were administered. A coronary wire was passed to the distal LAD, and balloon dilation with an undersized 2.5 mm balloon was performed, restoring normal antegrade flow (Figure 1C). Following PCI, antegrade flow was normal.

CT angiography disclosed no evidence of aortic dissection. The patient exhibited mild pulmonary congestion on the following day that responded to diuretic therapy. Maximum serum troponin I level was 69.1 ng/mL. Hypercoagulability evaluation, including homocysteine, anti-nuclear antibody screen, antithrombin III, protein C, free and total protein S, protein S activity, Factor V Leiden dmutation analysis, prothrombin 20210GA mutation, lupus anticoagulant profile, and anticardiolipin antibody, was negative. She was discharged on the 5th hospital day. Repeat echocardiography 3 weeks after her initial injury showed improvement in LV function, with residual mild to moderate hypokinesis of the mid to apical anterior wall and an ejection fraction of 54 %.

3. Discussion

A wide variety of cardiovascular pathologies have been associated with blunt trauma injury, including myocardial contusion, aortic transection or less often aortic dissection, trauma-induced ventricular arrhythmia or commotio cordis in young children, hemopericardium with tamponade, and aortic valve leaflet avulsion [9-12]. Acute myocardial infarction (AMI) is a rare but well-described complication of blunt trauma to the chest. Since the advent of primary percutaneous intervention, there have been several reports of AMI due to total epicardial thrombotic occlusion following blunt trauma and its successful treatment by percutaneous intervention [13-17]. Proposed mechanisms for AMI in this setting have included intimal injury due to shear forces imparted by the blunt trauma [18, 19], plaque rupture, coronary artery dissection, and coronary vasospasm [20]. In several reports, the presence of coronary artery dissection has been documented as the underlying pathophysiologic trigger for thrombosis [4-8]. Coronary dissection presumably occurs in association with deceleration trauma, in some cases involving the aorta. It is not known whether affected individuals carry a genetic predisposition to vascular dissection; however, spontaneous coronary dissection has been associated with fibromuscular dysplasia [21-22]. The mechanism of thrombosis in the case presented here remains obscure. Acute psychological stress has been implicated in AMI, following diverse stressors, including earthquakes [23], missile attacks [24], and international soccer matches [25], but these primarily involved patients having underlying atherosclerotic disease, whereas the patient in the current case was a 31-year-old male with no coronary risk factors and no atherosclerosis by angiography or by IVUS interrogation of the vessel wall. It appears most likely that thrombosis in this case was the result of endothelial injury, possibly caused by direct compression of the proximal left anterior descending artery by the force of the blow or via shock waves [26]. A mathematical model for blunt injury leading to hemodynamic shade zone formation with high and low shear stress and hyperviscosity has been developed by Ismailov [27].

The treatment approach to myocardial infarction (STEMI) following blunt chest trauma is immediate coronary angiography and revascularization with percutaneous intervention and continuation of dual antiplatelet therapy. We felt that use of GpIIb/IIIa inhibitor was contraindicated in our patient because of liver laceration and increased risk of bleeding.

4. Conclusion

Although significant chest pain would not be unexpected following severe blunt trauma injury to the chest, the possibility that ongoing pain represents myocardial ischemia should be considered, and a screening ECG should be considered to identify rare cases of STEMI.

5. Conflict of Interests

The authors report no financial relationships or conflict of interests regarding the content herein.

6. References

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