Trauma Induced ST-Elevated Myocardial Infarction

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Introduction

Acute coronary syndrome related to blunted chest trauma is a rare event. Traumatic cardiac injuries can vary between arrhythmias, coronary rupture or occlusion, leaflet avulsion, chamber rupture, or aortic dissection[1,2]. We present a case of trauma induced ST-elevation myocardial infarction (STEMI) involving the right coronary artery following a motorcycle accident with multiple traumatic injuries.

Clinical Course

33-year-old male with history of tobacco use presented to our hospital after a motor vehicle accident. He had been riding his motorcycle that morning and had missed a turn falling off his bike sustaining multiple injuries. He denied any anginal symptoms prior to his fall. A bystander was able to call 911 and paramedics brought him to our emergency department. On presentation, the patient was reporting substantial left sided lower back and flank pain, but no chest pain. He was requiring high amounts of supplemental O2 via non-rebreather facemask at 15 L/min to maintain oxygen saturation above 90%. EKG obtained showed ST elevation in leads II, III, and aVF with reciprocal changes in leads I and aVL (Figure 1). His vital signs on presentation were HR 126 bpm, Resp 38 breaths/min, BP 105/44, SpO2 95% on 15 L/min. He was given aspirin 325 mg by ER provider.

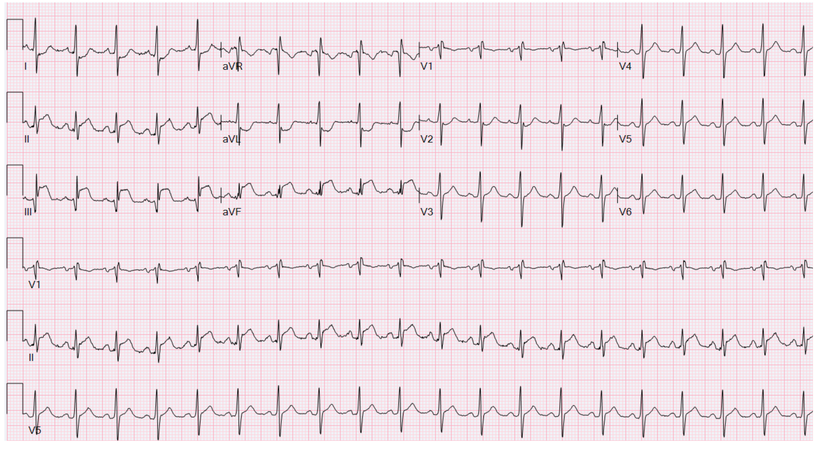

Figure 1. EKG: sinus tachycardia with incomplete RBBB and inferior ST-elevation concerning for acute MI.

CTA of chest abdomen pelvis was performed which showed multiple left-sided rib fractures involving 5th – 10th ribs, bilateral pulmonary contusions, 10% left-sided pneumothorax, and a grade III splenic laceration with sub-diaphragmatic hematoma. Bedside echocardiogram was attempted but given subcutaneous air visualization was very poor. His troponin returned at 0.47 ng/mL. Interdisciplinary discussion was held with general surgery, critical care, and the patient discussing his multiple traumatic injuries and risk of bleeding with PCI. Despite the patient's lack of chest pain it was felt that with his ST-elevation on EKG and elevated troponin warranted urgent intervention and the patient was take for left heart catheterization.

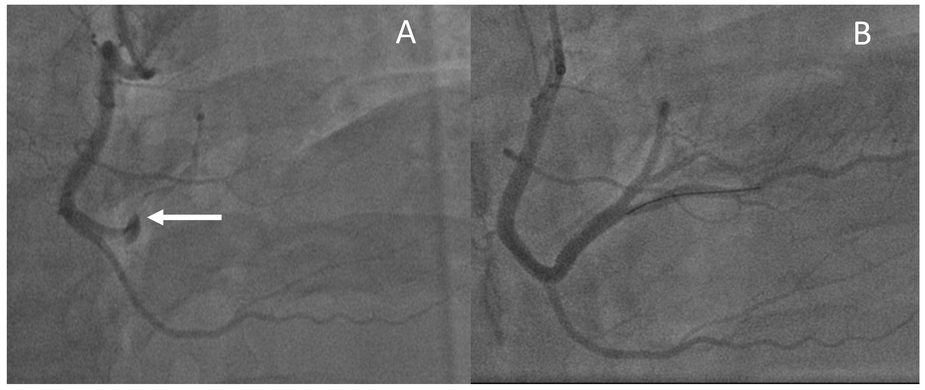

Figure 2. Coronary angiography A) RCA with 100% occlusion of distal RCA due to thrombus (white arrow). B) RCA post-PCI to distal RCA with 4.0 x 28 Promus ELITE DES. RCA – Right coronary artery, DES – Drug-eluting stent.

Left heart catheterization revealed a 100% occluded distal RCA due to thrombus. Flow was able to be restored following angioplasty and received a 4.0 x 28 Promus ELITE DES to his distal RCA and loaded with ticagrelor 180 mg (Figure 2). Patient was transferred to the ICU following the procedure given concerns for bleeding related to his multiple traumatic injuries and started on dual antiplatelet therapy with aspirin 81 mg daily and ticagrelor 90 mg BID. His troponin peaked at 41.81 ng/mL. His acute kidney injury and rhadomyolysis appreciated on presentation resolved with fluid resuscitation and he did not require transfusion during his hospitalization. Echocardiogram performed 4 days following is STEMI showed EF 57% with poorly visualized wall motion. His traumatic injuries were managed medically and did not require surgical intervention during his hospitalization. He was able to be discharged home 6 days following his presentation. On follow up 10 days post-discharge he reported doing well with only rib pain and no bleeding events on his ticagrelor and aspirin.

Discussion

Myocardial infarction related to blunt chest trauma remains a very uncommon occurrence. Proposed mechanisms of injury included vessel rupture, intimal dissection with thrombosis, fissuring of pre-existing atherosclerotic plaque, embolism to coronary arteries, and vessel spasm[3,4]. Several mechanisms are possible in our patient including vasospasm related to direct chest trauma or fissuring/dissection of intima with thrombosis given thrombus on left heart catheterization with no other identifiable CAD. STEMI related to blunt chest trauma remain rare occurrences and the clinician needs to maintain a high index of suspicion and utilize as many tools available to them including EKG, echocardiogram, and cardiac enzymes. Our diagnosis of STEMI relied heavily on his EKG findings and cardiac enzymes as bedside echocardiogram was not able to clearly delineate wall motion even with an echo-enhancing agent. Proceeding directly to coronary angiography may be necessary, but should be directed by a multidisciplinary approach given these patients' numerous injuries and potential for bleeding.

References

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