Coronary atherosclerotic plaque rupture following thoracic trauma: an uncommon cause of angina and ventricular tachycardia ("torsade de pointes")

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CASE REPORT

Coronary atherosclerotic plaque rupture following thoracic trauma – an uncommon cause of angina and ventricular tachycardia ("torsade de pointes")

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The patient underwent an off-pump coronary artery bypass graft with arterial grafts to the LAD and circumflex arteries four weeks after the initial presentation. It should be noted that, during surgery and before the graft was placed, as the surgeon inspected the heart looking for evidence of cardiac trauma, ST-T elevation was observed on the heart monitor. This observation was confirmed by ECG and persisted for 24 h, evolving with new Q waves in leads II and III and poor progression of R waves in leads V1 through V3, with a striking elevation of CKMB-mass (which peaked at 25 times the normal level 17 h after surgery). An echocardiogram performed on the second day postoperatively revealed a very small area of hypokinesia in the apex. The patient had an uneventful recovery. An echocardiogram performed at the one-month follow-up was completely normal.

Another treadmill test was performed forty days after surgery; the patient was able to tolerate maximal exercise, and no ECG changes suggestive of myocardial ischemia or arrhythmias were observed. A coronary computed tomography angiogram (16×0.5-MDCTA, Aquilion16TM, Toshiba Medical Systems Corporation, Otawara, Japan) with calcium score evaluation revealed a non-calcified atherosclerotic plaque on the proximal segment of the LAD. Inside the plaque, an area of very low density (<40 Hounsfield Units) was observed (Figure 2), a finding compatible with a coronary thrombus. Both of the grafts were patent.
Although acute myocardial infarction (AMI) caused by a trauma with laceration of the coronary arteries has been previously described in many case reports, especially in the vicinity of the LAD, most reported cases involved ST-elevation MI or ventricular aneurysm preceded by a history of trauma, as recently reviewed. In the present case, myocardial necrosis was detected by serum markers one week after chest trauma, even though the ECG remained normal. Coronary intravascular ultrasound was not recommended to further investigate the extension of the stenotic lesions due to the risk of disrupting the left main plaque. Thus, the initial CK-MB and elevation of troponin might have been caused by direct trauma, whereas the ruptured plaque and thrombus might be responsible for the delayed clinical symptoms.

Notably, there was a striking moment in time relating the thoracic trauma and the onset of anginal symptoms. We believe that a more careful interpretation of the patient’s presenting symptoms would have been helpful to identify the cause. Non-invasive imaging revealed a coronary thrombus inside an atherosclerotic plaque, most likely resulting from the chest trauma and leading to exercise-induced ischemia and malignant ventricular arrhythmia. However, the possibility of post-CABG thrombus formation.

Figure 1 - A treadmill test (Bruce protocol) indicating a non-sustained polymorphic ventricular tachycardia (“torsade de pointes”) (Panel A), followed by ST-T ischemic changes (Panel B).

Figure 2 - A coronary-computed tomography angiogram indicating a low-density image in the proximal segment of the LAD that is compatible with a coronary thrombus (white arrows).
cannot be excluded, as the CT was only performed after surgery.

Another important finding in this case report was the intraoperative myocardial infarction. The most probable etiology of this event was the dislodgement and embolization from the thrombus, which previously rested on the ruptured plaque. Other possible causes of intraoperative myocardial infarction, including hemodynamic instability or surgical technique, are improbable in the present case.

At a follow-up visit one year after the procedure, the patient was doing well and had not experienced the recurrence of any of his symptoms.

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REFERENCES