Case Report

Coronary artery dissection in a patient with traumatic femoral shaft fracture

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Abstract

We report a 24-year-old man who developed postoperative pulmonary edema and desaturation, after open reduction with internal fixation for left femoral shaft fracture sustained in a motorcycle accident. Cardiac catheterization revealed a left anterior descending coronary artery dissection. Review of his present history, showed that he neither had chest discomfort nor suffered from hemodynamic decompensation preoperatively. Only the abnormal 12-lead ECG with moderate tachycardia was suggestive of myocardial ischemia. Coronary artery dissection, although uncommon, is a disastrous complication following blunt chest trauma, and needs thorough preoperative evaluation to exclude its occurrence.

1. Introduction

Coronary artery dissection secondary to blunt chest trauma is a rare complication. Traumatic coronary artery dissection associated with femoral shaft fracture in the absence of chest trauma is even rarer. We report a case of femoral shaft fracture who sustained O2 desaturation and pulmonary edema postoperatively following an open reduction and internal fixation with an interlocking nail. Subsequently, a left anterior descending artery dissection was confirmed by cardiac catheterization. Because the patient denied any chest discomfort and manifested no hemodynamic decompensation preoperatively, the establishment of correct diagnosis and management were delayed.

2. Case report

A previously healthy 24-year-old male (86 kg, 175 cm) sustained left femoral shaft and bilateral mandibular fractures in a traffic accident whilst motocycling. In our emergency department, his vital signs were stable, except for a rapider pulse of 110/min. He complained of difficulty in talking and painful mouth and left leg. Multiple abrasions were noted over the four limbs and face, but there was no ecchymosis or abrasion over the anterior chest. Laboratory studies were unremarkable except for mild anemia, elevated liver enzymes and total bilirubin (hemoglobin 11.8 g/dL, aspartate transaminase 231 U/L, total bilirubin 2.75 mg/dL). The plain chest X-ray revealed no rib fracture, and no pneumothorax. Plain films of the head and legs showed bilateral mandibular fractures and a complete fracture of the left femoral shaft, respectively. The initial ECG showed ST segment elevation and Q wave in leads V1–V5, (Fig. 1), highly suggestive of myocardial ischemia or infarction. Because he denied suffering chest discomfort, and manifested no chest trauma or hemodynamic decompensation, the possibility of myocardial infarction was not considered. After preliminary treatment, the patient was transferred to the orthopedic ward and put on the waiting list for emergent operation for the fractures. Intravenous morphine 5 mg was given intermittently for pain relief. Forty-five hours after the accident, the patient was sent to the operating room for open reduction of the femoral shaft fracture under general anesthesia. The anesthesia was induced with thiopental (5 mg/kg), fentanyl (2 µg/kg), and cisatracurium (0.15 mg/kg). After tracheal intubation with a 7.0-mm nasal RAE tube, anesthesia was maintained with sevoflurane in O2.

After induction, the heart rate increased from 124 beats/min to 135 beats/min. Fluid supplement with crystalloid 1500-ml was administered but the tachycardia did not improve. The systolic and diastolic blood pressures were around 110 mmHg and 60 mmHg, respectively. We increased the depth of anesthesia by increasing sevoflurane concentration and administering additional doses of fentanyl (2 µg/kg). However, the tachycardia persisted and sometimes rose to 160 beats/min whilst the mean blood pressure was around 80 mmHg. Packed erythrocytes were transfused for suspected occult blood loss due to the femoral shaft fracture. The
Operation lasted for 3.5 h and intraoperative blood loss and urine output were 500 mL and 1200 mL, respectively. Total fluid replacement was 4000 mL of balanced crystalloids and 4 U packed erythrocytes. The partial pressure of arterial oxygen was 225.2 mmHg at 70% FiO2 15 min after induction. The intraoperative oxygen saturation by pulse oximeter was 100% at 70% FiO2. We extubated the patient at the end of the operation, however, severe O2 desaturation was immediately noted so the patient was reintubated. The arterial blood gas analysis and then performed showed that PaO2 was 70 mmHg at 100% FiO2. Therefore, he was sent to the traumatic intensive care unit (ICU) for further postoperative care.

On arrival at the ICU pinkish frothy sputum was sucked out from the endotracheal tube. Chest X-ray findings were compatible with pulmonary edema. Further, a 12-lead ECG showed a pattern of wide complex tachycardia (HR 158 beats/min) and right bundle branch block. After consultation with the cardiologist, chest computer tomography was done on suspicion of pulmonary embolism. Nevertheless it showed patent main pulmonary arteries but possible with partial thrombosis in the LLL branch of the left pulmonary artery. The echocardiogram undertaken on postoperative day one showed hypokinesis of the anterior wall of the left ventricle and impaired left ventricular contractility with an ejection fraction of 38%. Cardiac enzymes were very high (creatinine kinase 6510 U/L, creatinine kinase myocardial band 95 U/L, and troponin I >40 ng/ml) in a subsequent laboratory study. Myocardial infarction was suspected for the first time. The large amount of fluid given intraoperatively might possibly heavily burden the impending failing heart and ultimately led to the development of acute pulmonary edema. After diuresis with intravenous furosemide, the signs of pulmonary edema on plain chest X-ray were cleared within 3 days, and the patient was successfully extubated 4 days post-operation. This rendered the diagnosis of fat embolism syndrome or pulmonary embolism improbable.

Afterwards, coronary angiography revealed dissection of the distal left main coronary artery with involvement of the proximal left anterior descending artery (LAD) (Fig. 2). A stent implant was inserted without difficulty and his recovery was uneventful. The patient was discharged on postoperative day 35.

3. Discussion

Although reports do exist describing coronary artery dissection following blunt chest trauma, to our knowledge, this is the first account which reports inadvertent (unintentional) delay in diagnosing a coronary artery dissection until after a surgical procedure for an unrelated disease entity. Traumatic coronary artery dissection is often accompanied by rib fracture, hemo- or pneumothorax. Although the initial ECG of the patient was suggestive of myocardial injury, in the absence of chest trauma, chest discomfort and dyspnea, cardiac injury was not seriously considered during the preoperative evaluation. When his tachycardia heightened during the operation, our first assumption was volume depletion, but it persisted after crystalloid challenge. We then thought it was due to inadequate depth of anesthesia; however, the tachycardia did not improve by deepening the anesthesia with sevoflurane or additional administration of fentanyl. After transfusion of packed erythrocytes was futile to improve the tachycardia, cardiac dysfunction was highly suspected. Since acute pulmonary edema occurred in consequence of intraoperative fluid overload, failure to maintain normal oxygen saturation with spontaneous respiration after extubation was inevitable. During surgery he could maintain normal oxygen saturation and did not manifest the signs of pulmonary edema, possibly due to application of positive pressure ventilation.

Fig. 1. Preoperative electrocardiogram shows ST–T change at leads V1–V5.

Fig. 2. Coronary angiography demonstrated dissection flap from distal left main coronary artery to proximal left anterior descending coronary artery with 90% stenosis (arrow) (LAD – left anterior descending coronary artery, Cx – circumflex coronary artery).
Postoperative echocardiography confirmed the cardiac dysfunction and cardiac catheterization uncovered the etiology.

Coronary artery dissection, coronary artery occlusion, and acute myocardial infarction after blunt chest trauma are rare. The possible mechanism is that, the generation of shear forces in the arterial wall after sudden deceleration from speedy motion resulting from an impact compression of the anterior chest wall, could result in coronary intimal tears. In addition, thrombosis formation or rupture of a pre-existing atherosclerotic plaque will lead to coronary artery occlusion. The vessel most frequently subjected to injury is the left anterior descending coronary artery, followed by ST segment elevation and the development of hyperacute T waves in the affected area, such a supposition. The earliest feature of coronary occlusion is Q waves. ECG and cardiac enzyme abnormalities provide early proximal left anterior descending coronary artery involvement, or general, the treatment modality depends upon the clinical management of post-traumatic coronary artery dissection is assessed by intracoronary ultrasound: a case report. J Korean Med Sci 1998; 13:325–32.

Before an emergent operation, echocardiography and ECG are useful noninvasive tools for screening of cardiac complications after traffic accident. If there are abnormal cardiac signs that cannot be explained preoperatively, coronary artery angiography should be considered. Cardiac dysfunction should be guarded perioperatively, and if it is evident, then transesophageal echocardiography can be applied to evaluate cardiac status during surgery.

For evaluating the location and extent of coronary artery injury, angiography remains the unique procedure of choice for accurately depicting intimal tear or thrombosis. It has been suggested that intravascular ultrasound is a useful alternative. Smayra et al. have reported the use of multi-detector row computed tomography to assess a coronary dissection. Traumatic intimal dissections will generally heal within 6 months. Although conservative management of post-traumatic coronary artery dissection is favored, the risk of hemorrhage from coexisting injuries should be pondered over when thrombolytic agents are to be used. In general, the treatment modality depends upon the clinical scenario. In cases who have ongoing ischemia with left main or proximal left anterior descending coronary artery involvement, or with a large area of myocardial damage, aggressive treatment strategies are favored, such as performing endovascular stenting or bypass surgery.

In this case, we came across a traumatic coronary artery dissection without superficial clues of a possible cardiac injury such as chest discomfort or bruising over the anterior chest wall. The patient’s young age and lack of chest discomfort were factors that led to a delayed diagnosis of coronary artery dissection. With an abnormal preoperative ECG, further investigation such as cardiac enzymes study and/or echocardiography should be arranged before the operation. Assessing the severity of trauma based only on the patient’s complaint is insufficient. Since surgical intervention for this patient although urgent but was not necessarily emergent, delaying or cancelling the operation for further work-up of the cardiac function was justifiable based on the exceedingly abnormal ECG finding. The importance of thorough preoperative evaluation cannot be underemphasized. Notably, transesophageal echocardiography is another valuable tool for ruling out possible cardiac pathology in the event of perioperative hemodynamic instability. We hope to increase awareness that trauma may involve late as and produce immediate sequel, leading to remedial treatment and medicolegal events.

References