Late onset traumatic aortic isthmus transection after acetabular fracture surgery

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ABSTRACT

Background: Traumatic acetabulum fractures are common injuries and are often associated with cardiopulmonary injuries such as hemothorax or pneumothorax. Aortic transection is a well-described cause of death, particularly after a deceleration injury. However, late dissection on arcus aorta level is rare.

Case Presentation: We describe the late presentation of an aortic transection case with acetabular fracture caused by a motor vehicle accident treated with thoracic endovascular aortic repair procedure and discuss the possible mechanism of injury.

Conclusion: In high-energy traumas, such as acetabular fractures, one should be alert for traumatic aortic injuries, especially if the patient has a chest trauma.

Keywords: Acetabular fracture, aorta transection, TEVAR procedure, late onset.

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Background

Traumatic acetabular fractures are common and are often associated with other injuries like thoracic trauma, abdominal injury, etc. Thoracic trauma is present in 10%-15% of all trauma cases and is responsible for approximately 25% of the trauma-related deaths [1]. Aortic transection due to thoracic trauma - especially rib fractures - is a well-described cause of death, particularly after a deceleration injury. However, late transection on arcus aorta level is quite rare. We describe the late presentation of such a case and discuss the possible mechanism of injury.

Traumatic injury of the aorta is a life-threatening condition in which early diagnosis and treatment is crucial for survival. In those patients with blunt trauma and an associated aortic transection, only 15% survive to hospital admission. An overwhelming majority would die without surgical intervention [2]. Blunt aortic transection is the most common mechanism of traumatic injury [3,4]. We describe the case of an aortic transection determined at the postoperative 6th day.

Case Presentation

In October 2019, a 43-year-old male patient was admitted to the emergency clinic for polytrauma due to motor vehicle accident. At first, the patient was admitted to the nearest state hospital then transferred to the university hospital. After physical examination, roentgenograms, and computerized tomography (CT) scans, subarachnoid hemorrhage was found on falx cerebri which was assessed by a neurosurgeon and did not require surgical intervention. Left-sided hemothorax was determined and after assessment by the thoracic surgeon, no surgery was needed. There was pelvic injury on the left acetabulum and dislocation of the hip. Closed reduction was carried out and checked by x-ray imaging (Figure 1). There was no abdominal organ or major vessel injury reported on CT scans. The results of the patient's blood work were as follows: hemoglobin was 11.9 gr/dl, hematocrit was 44%, platelet count was 270/ml, creatine kinase was 5130 U/l, creatinine was 1.65 mg/dl, and INR was 1.19. The patient was admitted to the intensive care unit (ICU) due to the major injuries.

In the first 24-hour period, hemoglobin counts reduced to 8.1gr/dl, which probably occurred due to hemodilution and creatine kinase increased to 5,981 U/l. The patient started to receive 250 cc/hours crystalloid fluids for crush syndrome. Enoxaparin sodium 4,000 IU per day was given by subcutaneous injection. The patient's chest x-rays and brain CT imaging showed no progression for hemothorax or subarachnoid hemorrhage; therefore, surgery was not required. After 4 days in the ICU, the patient's condition was stable, hemoglobin level was 9.6 g/dl, no progression was seen concerning the hemothorax or subarachnoid

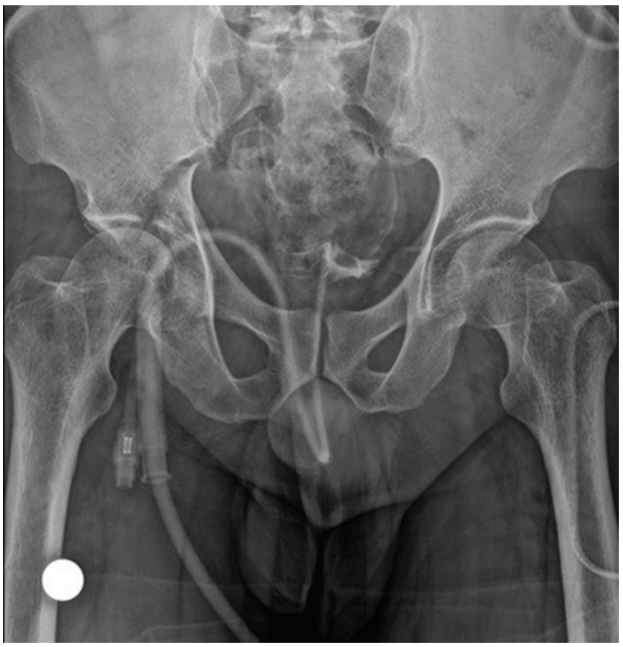

Figure 1. *Preoperative pelvis AP X-ray of the patient.*

hemorrhage, and he was ready for surgery for acetabular fracture.

The patient was operated 4 days after the admission. For venous thrombosis prophylaxis, 1 g of cefazolin was given 30 minutes prior to surgery. The patient was positioned in supine position and under general anesthesia, using the modified Stoppa approach, the anterior column was restored and fixated with two reconstruction plates. No complication occurred. Then, the patient was positioned in lateral decubitus and by using Kocher-Langenbeck approach the posterior wall fixation was achieved with one reconstruction plate. Total surgery duration was 150 minutes and blood loss was approximately 550 ml. Two units of erythrocyte suspension were administered during the operation. Neurovascular examination was normal after the surgery. Hemovac drains contained 300 ml blood in total and was removed after 48 hours. Prophylactic antibiotic (cefazolin 2 g per day) was given for 2 days.

The patient stayed in the ICU for 2 days after the operation. Hemoglobin levels were higher than 10 mg/dl, blood pressure was stable and normal, and creatine kinase levels were down to 3,292 U/l; therefore, the patient was transferred to orthopedics and traumatology ward. The patient showed no distress or discomfort besides occasional pain on the wound site for 4 days. On the postoperative 6th day, the patient had chest pain mostly on the left thoracic region and shortness of breath. Blood pressure was 130/80 mmHg; electrocardiography (ECG) showed sinus tachycardia with 110 beats per minute; sPO2 was 97%. Troponin-T was 32.86 (14-52) pg/ml and CK-MB was 2.69 (0-4.94) ng/ml. After 4 hours, hypoesthesia, numbness, and loss of motor function developed on lower extremities. 1/5 motor function on flexor and extensor

Figure 2. *TEVAR procedure carried out by the cardiovascular surgery department.*

muscles had been determined in the physical examination. Femoral and popliteal arterial pulses were weak by palpation. Troponin-T was elevated to 53.49 pg/ml; CK-MB was 5.07%; Pro-BNP was 487.3 pg/ml; ECG and sPO2 showed no particular difference. The patient was referred to cardiology and cardiovascular surgery departments and thoracoabdominal CT angiography scans were carried out. 16 hours after the initial symptoms had started and the patient developed anuria. Hypoesthesia level rose to the umbilical region. Neurosurgery consultation was carried out; brain CT scans were carried out; and no pathological findings were detected. However, thoracoabdominal CT angiography showed dissection on arcus aorta level. It was revealed that paraplegia occurred due to spinal ischemia and anuria due to loss of renal perfusion. The patient had been transferred to the ICU. Initial diagnosis was traumatic aortic isthmus transection and the patient had been transferred to another hospital and thoracic endovascular aortic repair (TEVAR) procedure was carried out by the cardiovascular surgery department (Figure 2).

Discussion

Thoracic traumas are common injuries encountered after high-energy trauma and are present in 10%-15% of all trauma cases [1]. Acetabular fractures are also common injuries associated with high-energy trauma and co-morbid injuries. Patients exposed to high-energy trauma are particularly susceptible to complications, and a larger number of rib and acetabulum fractures are associated with a higher risk [1]. The most frequent mechanical complications are hemothorax or pneumothorax, resulting from intercostal vascular damage or lung laceration [5]. Injuries to the liver, kidneys, and spleen are also recognized [5].

A major vascular injury as a direct consequence of a fractured acetabulum has been described. In our patient, transection on arcus aorta was not caused by direct trauma of the bone fragments in the acetabulum fracture and also was not related to acetabulum fracture surgery. However, it is possible that the aortic injury occurred simultaneously with the original chest trauma. Transection did not manifest itself until 6 days after the traumatic event. The mechanism of the delay is not entirely clear, as the patient was in a stable condition with no signs of transection and there was no reason to suggest any significant underlying injury. Also, transection of the aorta is seen with sudden deceleration and stress at aortic isthmus and transition zone of the aorta. A major vascular cause of hemothorax is rupture of thoracic aortic pseudoaneurysm or aortic dissection. Its diagnosis and treatment are always challenging [6]. Approximately 80% of the cases with thoracic aorta injury are lost suddenly at the scene of the traumas [6]. Coexisting devastating injuries are frequent and clinical picture is often nonspecific and obscured [6]. Our other theory is that there may be a pseudoaneurysm in the transected area which might be the cause of the transaction.

Clinical symptoms suggesting aortic injury include systolic intrascapular murmur, tachypnea, hemothorax, hoarseness, superior vena cava obstruction, paralysis, paraplegia, history of unconsciousness, external evidence of chest trauma, and others [7-10]. The eventual diagnosis was only discovered at the time when the patient had chest pain mostly on the left side and shortness of breath. After 4 hours, hypoesthesia, numbness, and loss of motor function developed on lower extremities, 1/5 motor function on flexor and extensor muscles had been determined in physical examination. 16 hours after the initial symptoms had started, the patient developed anuria. Hypoesthesia level had rose to the umbilical region.

Crass et al. [11] presented an experimental model of thoracic compression which showed that compression of the articulated skeleton in an antero-posterior direction resulted in postero-inferior displacement of the manubrium, first rib, and medial clavicle, which impinged on and caused near-transection of the aortic model. The level of injury was just distal to the subclavian artery. Cohen et al. [12] conducted a study using calculations made from the cross-sectional anatomy on CT scans, demonstrated the point of aortic impingement and provided further evidence in support of the osseous pinch mechanism. In our case, thoracoabdominal CT angiography showed the traumatic aortic isthmus transection and TEVAR procedure was carried out by the cardiovascular surgery department. The transected aorta in our patient is unlikely to have resulted from any mechanism other than the impinging bony structures. However, it is not possible to conclude that "osseous pinch" is the mechanism of injury in all cases of aortic transection.

Conclusion

Aortic transection may emerge long after the initial trauma, as it was 10 days in our case. We assume that the aortic isthmus injury was not related to acetabulum fracture surgery and developed at the time of the motor vehicle accident and did not manifest itself until 10 days after the traumatic event. In high-energy traumas such as acetabular fractures, one should be alert for traumatic aortic injuries, especially if the patient has chest trauma.

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List of Abbreviations

CT	Computed tomography
ECG	Electrocardiography
TEVAR	Thoracic endovascular aortic repair

Conflict of interest

The authors declare that there is no conflict of interest regarding the publication of this article.

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Consent for publication

Written consent was obtained from the patient.

Ethical approval

Ethical approval is not required at our institution to publish an anonymous case report.

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Summary of the case

1	**Patient (gender, age)**	M, 43-year-old
2	**Final diagnosis**	Left acetabular fracture, hemothorax, subarachnoid hemorrhage, and aorta transection
3	**Symptoms**	Left hip pain, chest pain, hypoesthesia, numbness, and loss of motor function developed on lower extremities, anuria
4	**Medications**	Enoxaparin sodium 4,000 IU per day, 250 cc/hours crystalloid fluids for crush syndrome, 1 g of cefazolin
5	**Clinical procedure**	TEVAR procedure was carried out
6	**Specialty**	Rare case