



Traumatic Rupture of Isthmic Aorta: a Case Report

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ABSTRACT

We report the case of a 24-year-old male patient admitted in our service with the diagnosis of rupture of isthmic aorta, after a motor vehicle accident. The particularity of the case was the approximately 24 hours delay in recognition of the aortic lesion, mainly due to the presence of multiple associated thoracic injuries and the necessity of intensive care manoeuvres imposed by the haemodynamic and respiratory status of the patient, at his presentation in the Emergency Department. We performed the replacement of the affected aortic segment by tubular graft interposition, with good short-term results.

Keywords : isthmic aorta, rupture, traumatic, thoracic CT-angiography

INTRODUCTION

Trauma is the leading cause of death under the age of 35 years worldwide. Traumatic aortic rupture is responsible for 18% of all road accident mortality. Eighty percent of these patients die at the scene of the accident. Of the survivors, 50% die within 24 hours if left untreated. Rapid transport and resuscitation, awareness of the injury, availability of multi-slice computed tomography (MSCT), and timely intervention can significantly improve survival in aortic injury⁽¹⁾. The era of high speed transportation has brought with it a changing pattern of traumatic injuries. Rupture of the thoracic aorta, which is a distinct and not infrequent entity, is a striking example of this phenomenon. The most frequent sites of rupture of the aorta are the ascending aorta, just proximal to the origin of the innominate artery, and the descending aorta, just distal to the origin of the left subclavian artery⁽²⁾.

The clinical presentation of patients with blunt chest trauma varies widely and ranges from minor reports of pain to florid shock. The presentation depends on the mechanism of injury and the organ systems injured. The time of injury, mechanism of injury, estimates of MVA velocity and deceleration, and evidence of associated injury to other systems (eg, loss of consciousness) are all salient features of an adequate clinical history. Information should be obtained directly from the patient whenever possible and from other witnesses to the accident if available⁽³⁾. Although good results are reported by those who advocate delaying repair by a few days, no evidence currently validates delaying the repair of aortic rupture beyond the time required for the evaluation and treatment of other emergency conditions⁽⁴⁾.

CASE REPORT

A 24-year-old man, without significant past medical history, was injured in an automobile accident resulting in blunt chest trauma in february 2012. He was admitted in the intensive care unit of an outside hospital approximately 2 hours after the accident, for multiple chest lesions, and suspicion of cervical spine trauma. At the referring institution the patient was relatively hemodynamically stable with

inotrope support and orotracheal intubation was accomplished for assisted ventilation; a left tube thoracostomy was also performed, with the evacuation of approximately 500 cc of hemorrhagic liquid. The initial chest X-ray and CT scan revealed multiple rib fractures bilaterally (with comminuted fractures of the IInd, IIIrd and IVth ribs on the right, and Vth through VIIIth ribs on the left), sternal fracture, hemothorax on the left, and severe pulmonary contusion, but showed no clear evidence of thoracic aorta or arch vessels injuries. The suspicion for such lesions raised from the patient's unfavorable course in the first 24 hours following the trauma, despite the intensive care management. Consequently, the decision to repeat the chest CT with intravenous contrast was taken; this time, there was evidence for an aneurysmal dilation of the isthmic aorta with an intimal flap pleading for dissection; also the CT showed the persistence of areas of pulmonary contusion bilaterally, the drain in left lateral thoracic position and a minimum left pneumothorax (Figures 1 and 2).

Figure 1 – Axial slice of CT-angiography showing post-traumatic aneurysmal dilation of isthmic aorta (arrow), associated with mediastinal hemorrhage, left pleural effusion, pulmonary contusion, and multiple rib fractures on the left.

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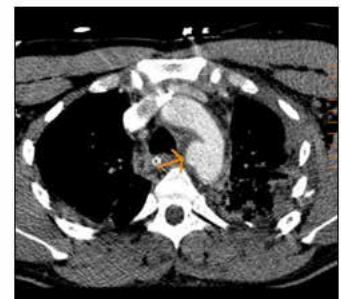
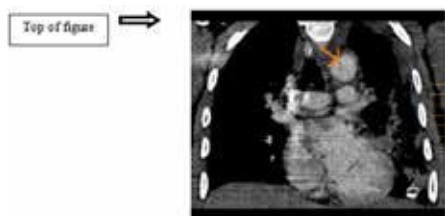


Figure 2 – Coronal reconstructed CT-angiography showing post-traumatic flap of intimal dissection at the level of

isthmic aorta (arrow).

After establishing the correct diagnosis, the patient was transferred in our service. At admission, the patient was sedated, with altered general status, with assisted ventilation in BIPAP mode with a iFO_2 of 0.6 and an aSO_2 of 95-96%; the clinical exam revealed pale teguments, rhythmic cardiac sounds, with a frequency of 110-120/min but no pathologic murmurs, and a blood pressure of 105 / 70 mmHg in both arms; the peripheral pulses were present bilaterally. Also an epidural thoracic catheter was inserted for continuous regional anesthesia with Bupivacaine 0.125%. The laboratory exam showed an alteration in liver function, with elevated ALT (176 U/l) and AST (143 U/l) and LDH (465 U/l), and rhabdomyolysis with a CK of 1613 U/l. The transthoracic echocardiography performed at admission revealed a dissection flap at the level of terminal ascending aorta and aortic arch, which ended before the emergence of great vessels on the left. There was also a small area of akinetic myocardium (possible myocardial contusion) on the anterior wall, but the aortic valve and the LVEF were normal. The chest x-ray reconfirmed the multiple rib fractures and the left pleural effusion partially drained.

After a short preparation, the patient was operated upon. Through left thoracotomy in the 4th intercostal space we isolated the the aortic arch just distal to the origin of the left carotid artery and the descending aorta. We found a large hematoma over the aortic isthmus. Femoral vein to femoral artery partial cardiopulmonary bypass was instituted, and the aorta was cross-clamped proximal and distal to the hematoma. Evacuation of the hematoma revealed the rupture of the posterior wall of the isthmic aorta, on approximately half of circumference. We resected the affected segment and reestablished the continuity of the aorta with the use of an end-to-end Dacron tubular graft. The time of the aortic cross-clamping was 150 minutes. Postoperative course was relatively uncomplicated. We maintained the assisted ventilation in order to prevent the respiratory distress caused by the associated thoracic injuries; hemodynamically the patient was stable without inotrope support, but he developed a marked rhabdomyolysis syndrome, with a maximum value of CK of 27224 U/l in the 3rd postoperative day. On the fourth postoperative day the patient was transferred to the referring institution, for monitoring and further treatment.

On subsequent follow-up 6 months after injury, the patient was asymptomatic, and the thoracic CT-angiography showed the complete permeability of the aortic graft, with a segmentary stenosis just below the distal anastomosis (Figures 3 and 4).

Figure 3 – Coronal reconstruction of chest CT-angiography showing the stenosis at the level of distal anastomosis (arrow).

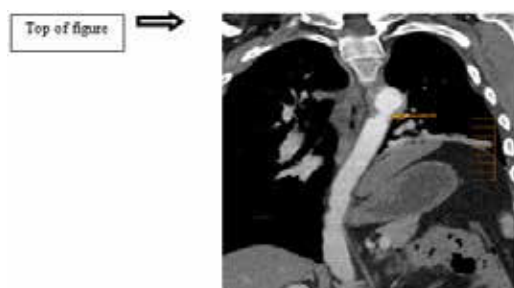


Figure 4 – 3D Reconstruction MIP of CT-angiography showing the tubular graft (P) at the level of isthmic aorta, with segmentary stenosis of approximately 35% on proximal anastomosis, and of approximately 50% on distal anastomosis (arrows).



High-speed MVAs are the most common cause of blunt thoracic aortic injuries and blunt injuries of the major thoracic arteries. The mechanisms of injury are rapid deceleration, production of shearing forces, and direct luminal compression against points of fixation (especially at the ligamentum arteriosum). Many of these patients die from vessel rupture and rapid exsanguination at the scene of the injury or before reaching definitive care. Blunt aortic injuries follow closely behind head injury as a cause of death after blunt trauma⁽³⁾. The presented case met the conditions for the described types of injuries, and there should have been a high grade of suspicion for an aortic rupture.

Physical clues include signs of significant chest wall trauma (eg, scapular fractures, first or second rib fractures, sternal fractures, steering wheel imprint), hypotension, upper extremity blood pressure differential, loss of upper or lower extremity pulses, and thoracic spine fractures. Signs of cardiac tamponade may be present. Decreased breath sounds and dullness to percussion due to massive hemothorax can also be found. Up to 50% of patients with these devastating, life-threatening injuries have no overt external signs of injury. Therefore, a high index of suspicion is warranted for earlier intervention⁽³⁾. In our case, the lack of evident signs and symptoms, associated with the multiple thoracic wall and pulmonary lesions, made the diagnosis relatively challenging.

Radiology plays an important role in evaluation of a trauma patient. Although chest radiography is recommended for initial evaluation of the trauma patient by the Advanced Trauma Life Support course, we hypothesized that precise physical examinations and history taking accurately identify those blunt trauma patients at low risk for chest injury, making routine radiographs unnecessary⁽⁵⁾. At presentation, both the chest x-ray and the CT-angiography were inconclusive for an aortic lesion. Some studies have established that the absence of widened mediastinum is encountered in 18 percent of patients with aortic rupture, leading to unavoidable delay in diagnosis. Consequently, transesophageal echocardiography could be the method of choice for the initial examination of all patients with severe thoracic trauma, quickly revealing intrathoracic bleeding, myocardial contusion, and thoracic aortic injury⁽⁶⁾. At admission in our service, due to technical reasons, we performed the transthoracic echocardiography, which found clear evidence for the dissection of the aortic isthmus, thus sustaining the correct diagnosis.

In patients with severe blunt chest trauma, TEE and CT have similar diagnostic accuracy for the identification of surgical acute traumatic aortic injury. TEE also allows the diagnosis of associated cardiac injuries and is more sensitive than CT for the identification of intimal or medial lesions of the thoracic aorta⁽⁷⁾. Despite all the "characteristic" clinical and roentgenographic findings, the recognition of aortic injury is often delayed or missed. Commonly, coexisting injuries to other

organs may either mask the signs of aortic injury or divert the physician's attention away from the diagnosis of rupture of the aorta. For these reasons, the possibility of this injury must always be entertained, and careful evaluation of the patient with multiple traumatic injuries should include all body systems, not just those overtly damaged ⁽²⁾.

The management of these injuries, especially those of the thoracic aorta, is evolving. Many patients have delayed repair of contained descending thoracic aortic ruptures. This approach is most frequently used when severe associated injuries are present that require urgent correction. Temporizing medical therapy includes the administration of short-acting beta-blocking agents (eg, labetalol, esmolol) to control the heart rate and to decrease the mean arterial pressure to approximately 60 mm Hg. Because repair of thoracic aortic injuries using cardiopulmonary bypass is associated with fewer major neurologic complications, some authors advocate stabilization of the victim plus beta-blocker administration until transfer is feasible to a facility where the injury can be repaired using cardiopulmonary bypass or centrifugal pump techniques. These techniques maintain distal aortic perfusion. Results have been excellent, and postoperative paraplegia rates have been significantly reduced ⁽³⁾.

The danger of exsanguination is constantly present in patients who survive the initial injury. For this reason, repair of the aortic rupture should be performed as soon as the diagnosis is confirmed ⁽²⁾. Endovascular stent grafts are being developed to repair thoracic aortic injuries. While several authors have reported success in treating such injuries with endo stents, the long-term durability of the stents is yet unknown. Further experience with this technique will allow more victims with concomitant severe injuries to become operative candidates ⁽³⁾. In our case, for repair of the aortic lesion we chose the classic open surgery with femoral vein to femoral artery partial cardiopulmonary bypass, taking into consideration the risk of sudden free aortic rupture at the onset of operation, and also the experience of the surgical team.

Future directions for improving the diagnosis and management of blunt thoracic trauma involve diagnostic testing, endovascular techniques, and patient selection. Endovascular techniques for the repair of great vessel injuries will be developed further and applied more frequently. Also, patient selection and nonsurgical therapies for delayed operative management of thoracic aortic rupture will be refined ⁽³⁾.

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