

Fatal blunt trauma with rare and undetected superior vena cava rupture: a case report and literature review

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Abstract: Laceration of the superior vena cava is an uncommon clinical entity almost always related to penetrating trauma. Rarely, however, it may occur after blunt thoracic trauma.

The authors describe an unusual case of rupture of the vena cava and tear of the aortic arch in a 51-year-old man struck on the chest by a falling tree he was felling. The man arrived at the emergency ward still conscious, but after about 4 hours from the accident was pronounced dead.

Autopsy findings consisted in a rupture at the lower third of the superior vena cava, just above the cavo-atrial junction, and an aortic laceration at the level of the isthmus, measuring approximately 3 cm in length. The mechanism of death was attributed to cardiac arrest secondary to hypovolemic shock from massive hemorrhage resulting from rupture of the superior vena cava and aorta laceration.

Key Words: blunt trauma, hypovolemic shock, superior vena cava injury.

INTRODUCTION

The term thoracic vascular trauma comprises both blunt and penetrating traumatic injury to the thoracic vasculature, i.e. the aorta and its brachiocephalic branches, the pulmonary arteries and veins, the superior vena cava and intrathoracic inferior vena cava and the innominate and thoracic veins [1].

The reported incidence of vascular trauma is as high as 9% of all injuries treated in emergency department [2], yet thoracic vascular trauma requires a high index of suspicion along with timely recognition/resuscitation to be dealt with effectively [1, 3].

Penetrating trauma accounts for the majority of thoracic great vessel injuries and is usually due to weapon-inflicted wounds produced by gunshot, shrapnel or stabbing, but iatrogenic interventions are not infrequently to blame [1].

With an estimated 7,500-8,000 cases per year in the United States, blunt thoracic aortic trauma is second

only to head injury as a cause of trauma-related death [4].

The most frequent cause of vascular lesions to the thorax is represented by sudden forceful deceleration typical of motor vehicle accidents [5]. In the latter, 10-15% of deaths are due to rupture of the great thoracic vessels [6]. Somewhat less common causes include blast injuries, falls from heights and crushing injuries [7].

The mechanism of death is often hemorrhagic shock resulting from the vessel laceration.

The pulmonary veins, the innominate artery, and most commonly, the thoracic aorta are the most susceptible vessels to this type of lesion [8].

Thoracic aortic lesions primarily involve the descending aorta in 54-65% of cases, followed by the ascending aorta or transverse arch (12%) or multiple vessels (13-18%) [9].

Given its short length (i.e. 7 cm), injury to the superior vena cava is quite infrequent and rarely described in the literature.

The authors report a case of blunt trauma to the

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chest causing rupture of the superior vena cava and aorta arch in a 51-year-old man. The ensuing hemorrhagic shock led to cardiopulmonary arrest a few hours after the traumatic event.

While the intrathoracic aorta is particularly susceptible to injury after blunt trauma [10-12], rupture of the superior vena cava is an extremely rare event.

CASE REPORT

The case reported herein concerns a 51-year-old, blue-collar trade worker. While engaged in tree-felling activities in a wooded area, a falling tree accidentally kicked back, striking the man in the chest and flinging him to the ground. Immediately after the incident, the victim remained conscious and complained of dyspnea and chest pain. Emergency services were called and upon their arrival the man was found sitting up, conscious and pale, but was responsive only to questions, with difficulty, and painful stimuli. His other vital signs were a heart rate of 92 beats per minute, oxygen saturation of 92 % breathing air and a blood pressure of 140/70 mmHg. During transport to the nearest hospital his vital signs worsened, i.e. saturation falling to 70 % in air, blood pressure dropping to 60/40 mm Hg and heart rate increasing to 125 bpm, with an ominous GCS of 3.

Upon arrival at the emergency department, the patient showed signs of shock and was hypotensive, with a hemoglobin value of 10.6 mg/dl, however there was no abdominal guarding or rigidity. Total body CT revealed traumatic injury to the thoracic aorta, characterized by aortic wall thickening and the presence of a mural hematoma with a multilayered appearance and appreciable dissection at the isthmus level (Fig. 1). Further, imaging also revealed signs of mural hematoma



Figure 1. CT scan. CT revealed traumatic injury to the thoracic aorta, characterized by aortic wall thickening and the presence of a hematoma.

extending to the supra-aortic branches and modest haemopericardium.

Treatment consisted in transfusing two units of blood, infusion with Ringer's lactate and placement of a dry suction chest drain.

While being transferred to the operating table the subject underwent cardiac arrest and, after standard, yet unsuccessful, attempts at CPR, was pronounced legally dead.

An autopsy was performed on the decedent 24 hours later and external examination revealed a large bruised and abraded area of skin in the thoraco-abdominal region comprised between the mamillary lines and the transumbilical line, made up of multiple parallel linear lesions, from 1 cm to 3 cm in width (Fig. 2 A).

Hypostasis was scarce and fixed, extending over dorsal regions. Gross internal examination showed bilateral rib fractures and a fracture of the body of the sternum with intramuscular hematoma of the parasternal and intercostal muscles. As for the intrathoracic organs, the pericardial sac presented a tear with a bruise on the external surface of the heart. The thoracic aorta showed a vast mural hematoma with a multilayered appearance, especially evident at the aortic arch, extending to the supra-aortic branches, although also present in the descending aorta. A tear measuring about 3 cm in length was detected at the level of the aortic isthmus (Fig. 2 B). Lastly, there was a rupture measuring about 2 cm at the lower third of the superior vena cava, just above the cavo-atrial junction (Fig. 2 C-D).

Based on the above mentioned findings the mechanism of death can be attributed to cardiopulmonary arrest secondary to hypovolemic shock from massive bleeding caused by rupture of the superior vena cava and aortic laceration incurred through blunt thoracic trauma.

DISCUSSION

Blunt thoracic great vessel trauma is a relatively sporadic event, representing less than 5% of traumatic vascular injuries, whereas penetrating lesions remain the overriding mechanism [13]. To estimate the actual incidence and mortality rates, one should take into account the vast number of decedents who died without confirmation before reaching the hospital [14]. Nevertheless, traumatic rupture of the great thoracic vessels is associated with extremely high mortality rates. Approximately 80-90 % of cases are indeed fatal within a few minutes. In less severe cases that are not immediately fatal, patients frequently succumb, nonetheless, within hours due to secondary sites of hemorrhage from multisystem trauma, often involving the head and abdominal organs [13].

In victims of blunt thoracic trauma the great vessel most susceptible to lesions is the aorta [15]. The

laceration is generally transverse and often appears as a sharp incision-like cut.

Whether by sudden forceful deceleration/acceleration or other non-deceleration mechanisms such as trauma by blunt object [16], all entailing compression of the thorax and mediastinal organs and structures, the mechanism of injury, in the case of the aortic tears, is postulated to be due to shear mechanical forces onto a relatively mobile segment of the vessel adjacent to a fixed portion, i.e. the aortic arch and the proximal descending aorta just below the isthmus. Accordingly, vascular injury tends to occur at this level [17,18]. When blunt thoracic trauma produces an aortic intimal tear a secondary dissection may ensue, expanding longitudinally by separating the layers within the aortic wall. In the majority of cases, the lesion consists in a laceration of all 3 layers, i.e. aortic transection or rupture, whereas in a portion the adventitial wall and adjacent mediastinal structures contain the lesion, thus representing a barrier to free hemorrhage, at least initially. The massive bleeding resulting in the rupture of the wall is exceedingly difficult to control and often not even resolvable surgically [19].

In contrast, lesions of the superior vena cava usually result from penetrating trauma. Lacerations from blunt chest trauma are extremely rare [20] due to the reduced length of the vessel in the anterior right superior mediastinum, its course and relative fixity and resistance to external stress [1]. In 45 % of cases with superior vena cava injury, death befalls the victim prior to receiving emergency care [21].

The diagnosis of superior vena cava rupture, whether intrapericardial or extrapericardial, is typically made in retrospect.

In cases where death is not abrupt, the clinical picture of patients may range from asymptomatic to irreversible shock due to internal hemorrhage. The hemodynamic instability that ensues is characterized by a persistent hypotension and tachycardia. Also, traumatized patients may present with thoracic pain, dyspnea, unconsciousness or coma. Sometimes pericaval hematoma may clot, thus limiting further bleeding.

In stable patients with suspected thoracic great vessel injury, contrast material-enhanced computed tomography (CT), has a 97 to 99.3% sensitivity and 87.1

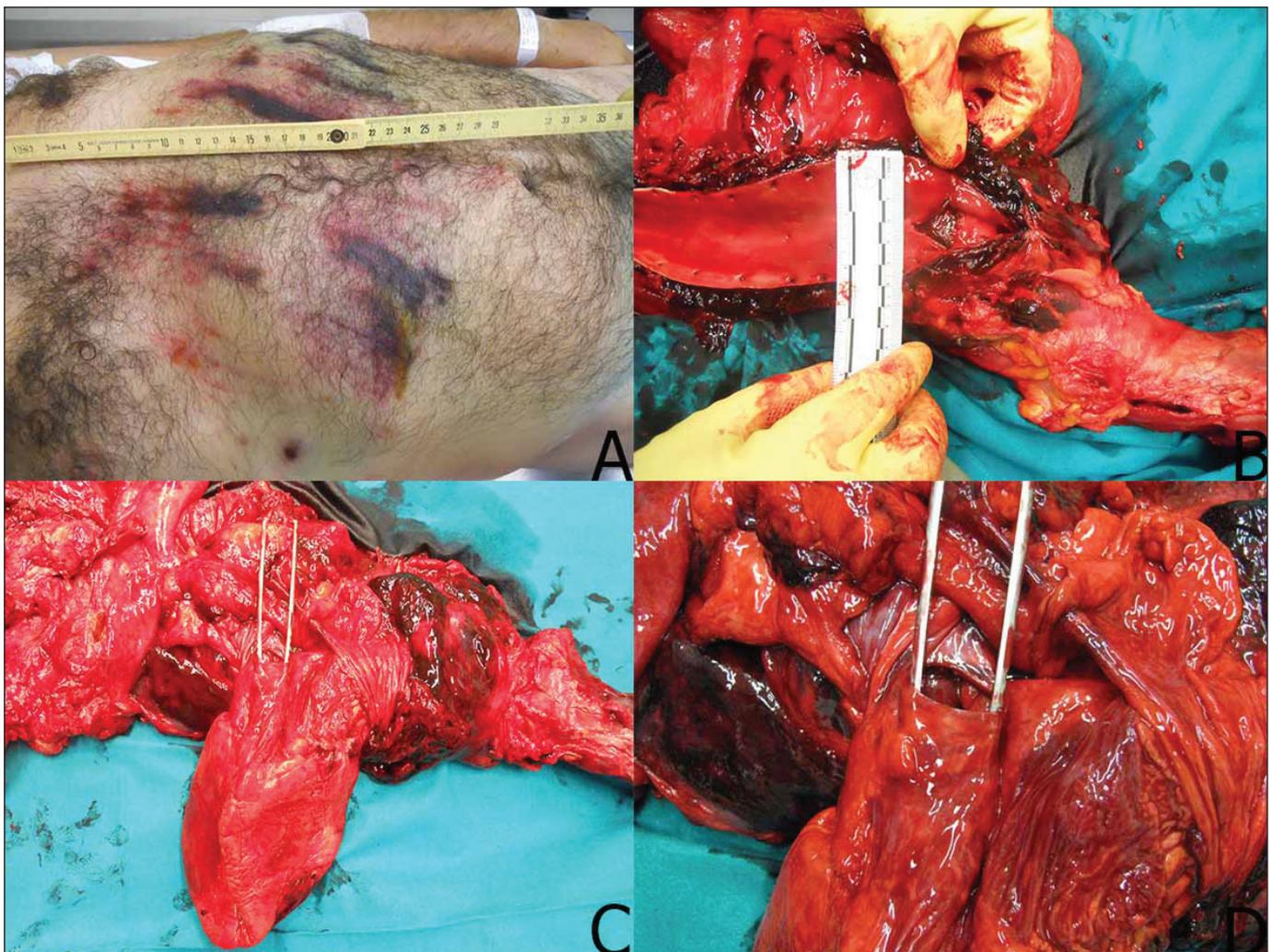


Figure 2. Autopsy findings. **A.** A large bruised and abraded area of skin in the thoraco-abdominal region comprised between the mamillary lines and the transumbilical line; **B.** A tear measuring about 3 cm in length was detected at the level of the aortic isthmus; **C-D.** A rupture at the lower third of the superior vena cava, just above the cavo-atrial junction.

to 99.8 % specificity, although dynamic spiral, contrast-enhanced, computed tomography (CT) angiography is progressively turning into the imaging modality of choice [1, 22, 23]. Transthoracic ultrasound, comprised in the Focused Assessment Sonography for Trauma (FAST) scan, should be routinely performed both in blunt and penetrating trauma to assess the pericardial space. In effect, FAST is increasingly becoming the foremost imaging procedure in the initial trauma-patient workup, even to evaluate the pleural space following blunt and penetrating injury [1].

However, in the setting of multiple vascular lesions of thoracic vessels, imaging techniques for rupture of the superior vena cava often prove far from ideal, thus requiring surgical exploration. All the more, some patients undergo delayed ruptures that occur a while after the trauma. The therapeutic evaluation is usually dictated by the clinical presentation. A patient with massive hemothorax should promptly undergo surgery [24]. However, in hemodynamically stable patients with multisystem injuries surgery may be delayed.

The recommended surgical approach is through a median sternotomy. Superior vena cava injuries are usually amenable to repair via lateral venorrhaphy. Nevertheless, repair of caval injuries may present difficulties, mainly consisting in inadequate exposure because of hemorrhage. Although vascular isolation techniques (e.g. atrio-caval shunting) have been proposed to limit bleeding so as to facilitate repair of these venous injuries, the moderately high mortality rates raise doubts about their reliability. Total cardiopulmonary bypass, however, can be used to decrease the blood return to the surgical field, while maintaining perfusion. With circulatory assistance, an abdominal inferior vena caval to right atrial cannulation circuit is recommended. Finally, the vena cava is repaired from within the lumen via access through a right atriotomy [1, 25].

As previously mentioned, superior vena cava tear due to blunt trauma is an extremely rare event having been described only sporadically in the literature.

Couves *et al.* [20] reported a case of superior vena cava laceration following blunt chest trauma in a 21-year-old subject. Surgical repair via median sternotomy was immediately successful in controlling hemorrhage.

Chaer *et al.* [26] reported a case of the superior vena cava and right atrium injury occurring in a 20-year-old patient involved in a high-speed motor vehicle collision.

Pascual *et al.* [25] describe another case of rupture of the intrapericardial superior vena cava and innominate artery injury in a patient of 49 years. The latter had hemodynamic instability and then underwent surgical intervention for vascular repair through median sternotomy. Postoperatively, a left subclavian

artery dissection was detected by CT and managed conservatively with anti-platelet therapy.

In the case reported herein, the stump of the falling tree trunk violently impacting the man's chest caused laceration of both the superior vena cava and of the proximal descending thoracic aorta, which led to internal hemorrhage and extensive bruising of surrounding tissues.

The massive retrosternal hemorrhage obscured the superior vena cava injury, which failed to be detected initially by CT scans, remaining unrecognized until subsequent autopsy investigation.

The victim's death occurred about 4 hours from the thoracic trauma in that, at the onset, the vascular lesions were partially buffered by surrounding tissues, thus initially staving off internal hemorrhaging. In spite of this, after an initial window of opportunity wherein the clinical picture remained stable, the massive hemorrhage following the double-vessel laceration relentlessly progressed to hemorrhagic shock and, ultimately, the fatal outcome.

The case illustrated highlights the challenge for emergency departments to expeditiously recognize, treat and repair great thoracic vessel injuries, while assessing the patient's clinical status carefully. Also, due consideration should be given to the possibility that an initial stability of the clinical picture might result from the buffering effect of surrounding tissues that curbs hemorrhage to some extent. Moreover, at the operating table, it is also necessary to assess the integrity of all major epi-aortic vessels as well as the SVC. Inasmuch as laceration of the latter vessel is rare, initial assessment via CT may not suffice for thorough preoperative classification.

CONCLUSION

Rupture of the superior vena cava is an infrequent event, almost always occurring in relation to penetrating thoracic trauma. Although rarely, it does ensue from blunt thoracic trauma. The case report herein illustrated might strike one as being a peculiar phenomenon, yet similar cases have sporadically been described in the literature. Lesions of the great thoracic vessels require a careful evaluation of the patient's clinical status, a high index of suspicion and accurate evaluation by appropriate imaging modalities. Timely surgical repair is the sole therapeutic intervention capable of effectively restoring the clinical picture. To date, the mortality rate is, nevertheless, extremely high and death most often occurs before arriving at the emergency ward.

Conflict of interest. The authors declare that there is no conflict of interest.

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