Emergency surgery for penetrating neck trauma and common carotid artery laceration presenting with shock: a case report

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Abstract

Introduction: The neck is an important anatomical junction connecting the head and limbs to the trunk and several vital neurovascular and aerodigestive structures pass through it. Zone II is the commonest site of penetrating neck injuries (PNI) and is commonly associated with vascular injury. The mortality rate following PNI is approximately 10%, increasing to 20% with concomitant penetrating carotid injury.

Case description: A 43-year-old was brought to the emergency department in class III haemorrhagic shock with a 5mm, bleeding, penetrating neck injury in zone II of the neck. He had soft signs of vascular injury, bleeding was controlled with direct pressure, and resuscitation was commenced with intravenous crystalloids and blood products. Surgical exploration revealed a 5mm laceration in the distal common carotid artery which was repaired primarily. Postoperative computed tomography (CT) angiogram was normal and the patient had an uneventful recovery.

Discussion: Traumatic disruption of the platysma is described as PNI and accounts for 10% of all penetrating injuries. Vascular injuries are most commonly seen in zone II and are usually associated with hard and soft signs of vascular injury. Zone II injuries require operative exploration with preoperative CT angiography recommended in haemodynamically stable patients to identify the type of injury. Small defects in the carotid artery can be repaired primarily. Conclusions: Open surgical exploration is mandatory for all patients with penetrating neck injury and haemodynamic instability. CT angiogram is recommended prior to open exploration is stable patients with zone II PNI.

Keywords: Traumatic. Carotid artery injury. Penetrating neck injury.

Introduction

The neck is an important anatomical junction connecting the head and limbs to the trunk and several vital neurovascular and aerodigestive structures pass through it. The neck is divided into 3 zones to help classify and identify the possible injury pattern. Zone 1 includes the structures below the level of the sternal notch. Zone II includes the region between the sternal notch and the angle of the mandible and zone III is from the angle of the mandible to the base of the skull [1,2].

Penetrating neck injuries account for 10% of all penetrating injuries of which Zone II injury is the commonest. Of the vascular structures involved, penetrating carotid injury (PCI) is seen in 6-20%, vertebral artery injury in 0.5%, and jugular vein injury in 15% [1,3]. Significant injury to the carotid arteries can occur in 1-3% of patients in major blunt trauma as well. Both blunt and penetrating vascular injury can cause complete transection, pseudoaneurysm formation, dissection, arteriovenous fistula formation, and thrombosis from atherosclerotic plaque disruption [4].

Zone II injuries are commonly associated with hard or soft signs of vascular injury. The presence of a bruit or thrill, expanding pulsatile hematoma, pulsatile or severe hemorrhage, and pulse deficit are the hard signs of vascular injury. Hypotension or shock, non-pulsatile stable hematoma, central or peripheral nerve ischemia, and proximity to a major vascular structure are less predictive of vascular injury and hence considered to be soft signs [1,3,4].
Here it was looked at a 43-year-old with a combination of blunt and penetrating injury to zone II of the neck with soft signs of vascular injury who was later found to have a common carotid artery laceration.

Methods

Study design

Ethical Approval
Our institution does not require ethical approval for reporting individual cases or case series.

Case Report

A 43-year-old previously healthy Sri Lankan, ethnic Sinhalese man, a warehouse worker by profession was brought by ambulance to the accidents and emergency unit of a tertiary care hospital with a bleeding neck wound, 30 minutes after a stack of wooden crates containing glass bottles fallen onto his body. He was a smoker (5 pack years) and consumed alcohol but had no significant past medical, surgical, or family history.

On examination, there was a bleeding 5mm laceration in zone II of the neck, to the left of the midline, with a non-expansile stable hematoma without tracheal deviation. There was subcutaneous emphysema but no palpable bruit. Although there was evidence of profuse bleeding with extensive blood staining of clothes, there was only mild active bleeding which was controlled with direct pressure. He was pale and tachycardic with a low volume thready pulse with a pulse rate of 130/minute, hypotensive with a blood pressure of 76/40mmHg, and tachypnoeic with a respiratory rate of 36/minute. Fluid resuscitation with a 1L bolus of Hartmann’s solution was commenced, and intravenous tranexamic acid and antibiotics were administered along with tetanus prophylaxis.

Chest examination excluded life-threatening thoracic injuries. His Glasgow coma scale was 15/15 and had no focal neurological signs. Emergency-focused assessment with sonography for trauma was normal. The patient responded to fluid resuscitation and was transfused 1 unit of group-specific and 1 unit of crossmatched red cell concentrate. Since the bleeding was controlled with direct pressure, a laceration of a superficial neck vein was suspected.

Due to the unavailability of a CT angiogram at the hospital and the hemodynamic instability of the patient, the patient was taken to the operating theatre by the casualty surgeon for surgical exploration of the neck under general anesthesia. The patient was positioned supine with the head end elevated to 300. Upon induction of anesthesia, the patient started rebleeding profusely. Unsure of the source of bleeding, manual compression was applied both proximally and distally. The incision was extended along the anterior border of the sternocleidomastoid (SCM). Bleeding subcutaneous veins were not noted. Dissecting through tissue plains was complicated by soft tissue edema and hematoma.

The SCM was retracted laterally and the carotid sheath opened. A 5mm laceration was noted in the distal common carotid artery just proximal to the bifurcation. As a vascular surgeon was not available at the hospital, over the phone expert opinion was taken. The laceration was sutured with 7.0 polypropylene sutures. The internal jugular vein, internal and external carotid arteries, and vagus nerve appeared normal. The patient was transfused a further 2 units of crossmatched RCC and 6 units of fresh frozen plasma intraoperatively. The patient was transferred to the surgical intensive care unit for post-operative care. He was transferred to a specialist center for vascular surgical care on day 1 where a postoperative CT angiogram revealed good carotid flow without evidence of stenosis. He had an uncomplicated recovery and was discharged on day 5.

Figure 1 shows an intraoperative image showing the exposed common carotid artery (CCA) with the sutured laceration in the distal CCA.

Figure 1: Intraoperative image showing the exposed common carotid artery (CCA) with the sutured laceration in the distal CCA.
Discussion

The neck is a complex zone containing several vital structures that connect the head and limbs to the trunk and hence injuries to the neck are life-threatening. The mechanism of injury could be either blunt, penetrating, or a combination. Traumatic disruption of the platysma is described as a penetrating neck injury (PNI) and accounts for 10% of all penetrating injuries. The mortality rate following PNI is approximately 10%, increasing to 20% with concomitant penetrating carotid injury [3,5]. Injuries to the neck are described concerning three zones, zone 1 lies below the level of the sternal notch, zone II lies between the sternal notch and the angle of the mandible, and Zone III lies above the mandible to the base of the skull [1-3].

Vascular injury following PNI could either be arterial or venous. The rate of arterial injury varies between 6-25% with the carotid artery involved in 80% of cases and the vertebral artery in 43% of cases. Jugular vein injury is seen in 16% of PNI [1,3,5]. Vascular injuries are most commonly seen in zone II [3]. Such injuries are usually associated with hard and soft signs of vascular injury which raises clinical suspicion. The presence of a bruit or thrill, expanding pulsatile hematoma, pulsatile or severe hemorrhage, and pulse deficit are the hard signs of vascular injury. Hypotension or shock, non-pulsatile stable hematoma, central or peripheral nerve ischemia, and proximity to a major vascular structure are less predictive of vascular injury and hence considered to be soft signs [1,3,4]. Both blunt and penetrating injuries can lead to carotid artery laceration, transection, dissection, thrombosis, and fistula formation [1,5].

The management of patients with PNI depends on hemodynamic stability and the anatomical location involved. Hemodynamically unstable patients will require operative exploration while hemodynamically stable patients can be worked up with radiological imaging before decision-making [3].

Zone I and III injuries will require further workup with duplex ultrasonography, digital subtraction angiography (DSA), or CT or MR angiogram. While DSA has the advantage of the possibility of simultaneous intervention using endovascular techniques, it is an invasive procedure with a 1% risk of complications. Hence most clinicians prefer CT angiography as it is non-invasive and is more available. Most Zone I and III injuries can be managed by endovascular techniques. Endovascular management options available for the management of proximal CCA, and distal ICA injuries include stenting for intimal damage, and stenting or coil embolization for pseudoaneurysm [3,5].

Zone II injuries will generally require operative exploration. If the patient is hemodynamically stable, CT angiography is recommended before surgery to appreciate and identify the type of injury. Small defects in the carotid artery can be repaired primarily, while larger defects can be repaired using patch angioplasty, or grafts such as autologous interposition vein graft or polytetrafluoroethylene graft [3]. In the case of a life-threatening hemorrhage with shock, ligation remains the most feasible option. Collateral circulation between vertebra-carotid and vertebra-subclavian provides as an alternate source of blood and may prevent the development of signs of central or peripheral ischemia [5].

There is no consensus on the use of anticoagulants or antiplatelets in PCI [6]. In our case, the patient presented to the A&E in hemorrhagic shock with a penetrating zone II left neck laceration from a combination of blunt and penetrating trauma. Soft signs of vascular injury were present with a stable hematoma and bleeding controlled with direct pressure. Owing to hemodynamic instability and the lack of facility for a CT angiogram, the patient was taken to the theatre for exploration without a radiological workup. Although the venous injury was suspected clinically, exploration revealed a small tear in the common carotid artery which was repaired primarily. Antiplatelets or anticoagulants were not started by vascular surgical opinion due to the low risk of thrombosis in a high-flow vessel such as the carotids.

Conclusion

Open surgical exploration is mandatory for all patients with penetrative neck injury and haemodynamic instability. CT angiogram is recommended prior to open exploration is stable patients with zone II PNI.

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Ethical Approval

Our institution does not require ethical approval for reporting individual cases or case se.

Informed consent

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Similarity check
It was applied by Ithenticate®.

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