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CASE REPORT

Early Anticoagulation may prevent Stroke in Blunt Carotid Artery Injury

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Abstract

Blunt carotid artery injury (BCAI) is a rare sequelae of blunt neck trauma. It may lead to stroke and major neurological deficits. Therefore, early diagnosis and management are crucial to prevent or reduce associated morbidity. We report a 22-year-old male who sustained an accidental left BCAI with an air-gun bullet. The injury was suspected on clinical assessment and confirmed by a CT/angiogram of the neck. The patient was promptly started on therapeutic anticoagulation and did not develop any neurological deficits. Our case emphasizes the importance of non-invasive management of BCAI, which requires a high index of clinical suspicion for diagnosis. Once confirmed, starting therapeutic anticoagulation early is vital to avoid fatal consequences, namely strokes, in such patients.

Keywords: Blunt injury; Carotid artery; Stroke; Anticoagulation; Vascular Trauma

Introduction

Blunt carotid artery injury (BCAI) is a rare event following blunt trauma with an incidence of 1-2%.[1] Its clinical manifestations can range from being asymptomatic to causing catastrophic consequences, such as cerebral ischemia. The mortality rate ranges from 20-40%, but it is significantly increased with a delayed diagnosis.[2] Several factors might contribute to this high variability: a) time between injury and presentation to the emergency room, b) level of trauma care at the hospital where the patient first presented, c) the grade of BCAI, d) timing of diagnosis & management. In addition, 50% of survivors may have significant permanent neurological deficits.[2] Another associated challenge, from a critical care perspective, is having specialized trauma facilities capable of managing severe arterial injuries.

A high index of clinical suspicion based on the mechanism of injury and associated injuries should lead to early screening and intervention to mitigate a potentially devastating outcome.

Here, we present a case of BCAI, emphasizing that prompt early management may result in a good outcome for this group of injuries.

Case Presentation

A 22-year-old soldier was transferred from the military clinic to our hospital's emergency department with a history of an accidental air-gun bullet injury to the left side of his neck during military training, which occurred two hours before presentation. He had sustained a laceration on the left side of his neck and was only complaining of left-sided neck pain. At the scene, the patient bled about 300 ml from the wound, according to the paramedics' report. There was no history of dizziness, loss of consciousness, blurred vision, palpitations, or dyspnea. There was also no history of injury to the head or back. The patient is not known to have had any previous surgeries, medical illnesses, or allergies.

In the trauma room, the patient was managed initially according to the Advanced Trauma Life Support (ATLS) protocol. The vital signs were: blood pressure 100/50 mmHg, heart rate 78 beats/minute, respiratory rate 20 breaths/minute, oxygen saturation 98 %, temperature 36.5 °C. During the primary survey, all parameters appeared intact, and there was no focal neurological deficit. In the secondary survey, a left-sided neck wound was observed in the lateral part of zone III, with an inverted U-shape measuring roughly 3x5 cm, associated with surrounding subcutaneous edema. The neurological examination revealed a Glasgow Coma Scale (GCS) score of 15/15, intact higher mental functions, and power of 5/5 in both upper and lower limbs.

Routine blood workup was normal. A CT/angiography of the neck vessels was performed per the carotid protocol (Figure 1). The findings were as follows:

1. Left internal carotid artery thrombus at the C3-C4 level, measuring 2.2 cm long and occupying 80% of the vessel lumen.
2. Intimal flap in the distal part of the left common carotid artery, just proximal to its bifurcation
3. Left-sided, deeply located hematoma measuring 11x17 mm at the site of the previously described vascular injury
4. Surgical emphysema
5. No evidence of active extravasation or pseudoaneurysm formation.

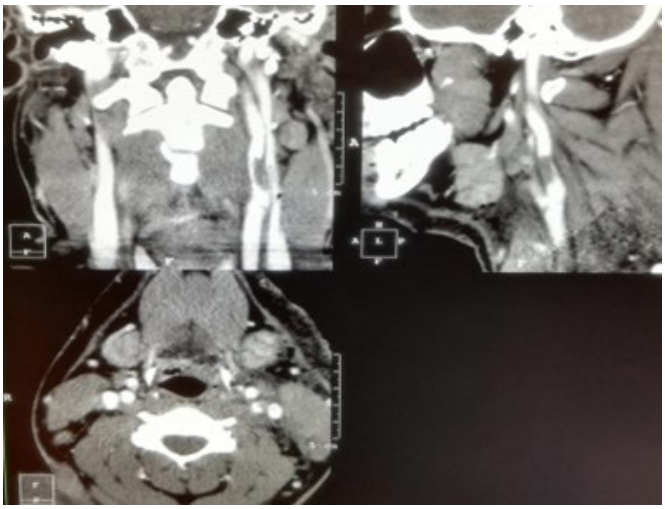

Figure 1: CT angiogram of the neck at presentation in coronal, sagittal, and axial views showing traumatic thrombosis involving the left internal carotid artery at the C3-C4 level, occupying 80% of the lumen circumference along a 2.2 cm length; intimal flap in the distal part of the left common carotid artery just behind its bifurcation; left-sided, deeply located hematoma measuring 11x17 mm at the site of the previously described vascular injury; surgical emphysema; and no evidence of active extravasation or pseudoaneurysm formation.

Although the patient was asymptomatic, the CT findings were significant. At this point, a therapeutic intervention in the form of open surgical or endovascular intervention was strongly considered. However, the risk of procedure-related morbidity and/or mortality is high in BCAI patients. Therefore, we decided to proceed immediately with conservative management.

Accordingly, based on his weight, the patient was started on a therapeutic dose of intravenous heparin (bolus followed by continuous infusion). He was then admitted to the intensive care unit for close monitoring and observation. The coagulation profile was repeated every 6 hours, and the heparin infusion dose was adjusted accordingly to maintain the Activated Partial Thromboplastin Time (APTT) between 60-90 seconds. The patient stayed in the ICU for 24 hours before being

transferred to the vascular surgery ward. On the first day post-injury, aspirin 81 mg once daily was started. On the second day post-injury, warfarin 5 mg once daily was added with a target INR of 2-3. On the third day post-injury, the heparin infusion was stopped, and an enoxaparin sodium 80 mg subcutaneous injection every 12 hours was initiated.

A week later, a CTA of the neck was repeated with the following findings (Figure 2):

1. The previously noted thrombosis involving the left internal carotid artery has receded at the C3-C4 level, with luminal patency regaining (currently 33%).
2. Near-total disappearance of the intimal flap at the left common carotid artery.
3. No evidence of extravasation.
4. Resolution of the previously noted hematoma and surgical emphysema.

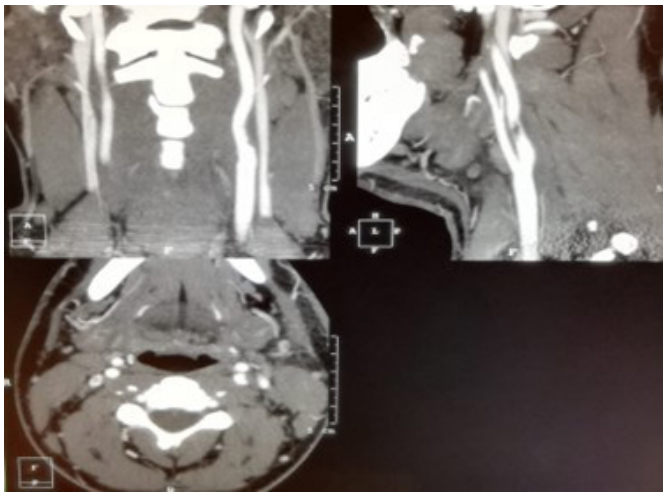

Figure 2: CT angiogram of the neck one week after the initial presentation in coronal, sagittal, and axial views showed regression in the previously noted thrombosis involving the left internal carotid artery at the level of C3-C4, with regaining of luminal patency (currently 33%); near-total vanishing of the intimal flap at the left common carotid artery; no evidence of extravasation; and resolution of the previously noted hematoma and surgical emphysema.

During his hospital stay, the patient did not complain of dizziness or blurred vision, nor did he develop any neurological deficits. He was discharged home on enoxaparin sodium 80 mg SD BD for one week, warfarin 5 mg, and aspirin 81 mg.

The patient had no complaints or neurological deficits at the one-month clinic follow-up. The warfarin was subsequently stopped, and he remained on antiplatelet therapy.

One year later, the patient remained asymptomatic and free of neurological deficits at the clinic follow-up. A repeated CTA of the neck (Figure 3) showed complete healing of the arterial injury with resolution of the thrombus.

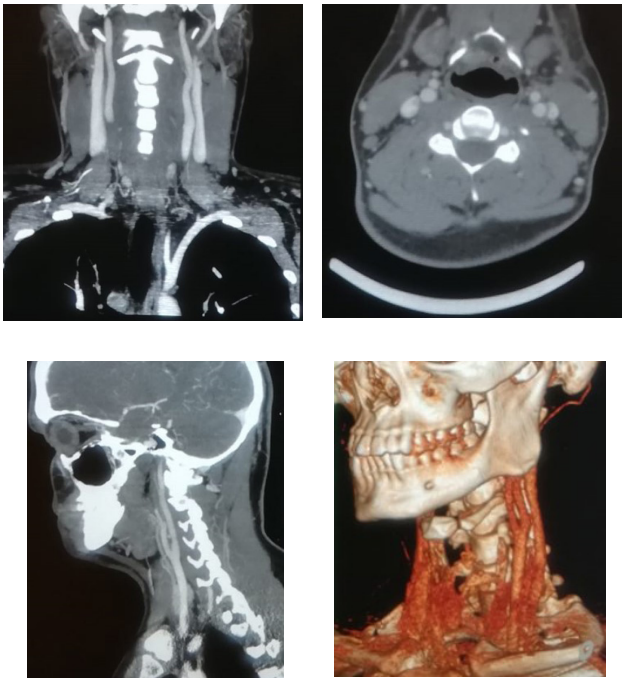

Figure 3: CT angiogram of the neck one year after the initial presentation in coronal, sagittal, and axial views, along with CT 3D reconstruction, showing complete resolution of the previously noted thrombus.

Discussion

The aim of this case report is to guide vascular and trauma surgeons on management of BCAI.

The first case report of BCAI was published in 1872 by Verniuel.[3] From that era until now, BCAI is still considered to be a rare post-trauma condition, with an incidence of 1-2% following blunt traumatic injury.[1] A study has revealed that BCAI has adverse outcomes, and without immediate diagnosis, they can result in cerebral ischemia, in 40-80% of cases and mortality rates ranging from 25-60%.[4] Another retrospective studied on patients whose diagnoses were delayed for over 48 hours following their condition had found that those patients had a mortality rate of 80%.[5]

Regarding BCAI management, there are two primary modalities: conservative and interventional. The Biffl *et al.* classification helps manage patients and is a prognostic indication for follow-up. The Denver grading scale has five grades:

- Grade I: Arteriographic appearance of irregularity of the vessel wall or a dissection/intramural hematoma with less than 25% luminal stenosis.
- Grade II: Intraluminal thrombus or raised intimal flap is visualized, or dissection/intramural hematoma with 25% or more luminal narrowing.
- Grade III: Pseudo-aneurysm formation.
- Grade IV: Vessel occlusion.
- Grade V: Transection with free extravasation[6]

Based on this classification, the management of patients is as follows: For grades I-IV, conservative therapy in the form of anticoagulant or antiplatelet therapy for 7-10 days is recommended, followed by repeat imaging, and if there is an improvement, the patient is continued on antiplatelet therapy for 3-6 months, after which they are re-evaluated. On the other hand, for grade V injuries, endovascular intervention is preferred over conventional surgical therapy. Other indications for endovascular intervention include: 1) in grade I-IV injuries, if there is a contraindication to anticoagulant therapy, and 2) signs of progressive injury.

Li et al. compared the outcomes between conservative and surgical management groups. They found that the mortality rate was 28% in the conservative group and 19% in the surgical group. There was no significant difference in the length of ICU stay between the conservative and operative groups.[2]

In a separate study, Fabian et al. found that the mortality rate was 20% in patients treated with heparin, in contrast to 100% in those who did not receive heparin ($p<0.01$).[7] In their study group, death was always due to stroke. Therefore, early anticoagulation can significantly lessen the outcomes of BCAI.

In the case we presented here, the patient initially was asymptomatic with no neurological deficits. A high index of clinical suspicion led us to diagnose a significant ipsilateral carotid injury. Based on the Denver grading scale, it was classified as a grade II injury. Therefore, we decided to treat the patient conservatively, with immediate anticoagulation followed by antiplatelet agents. Potential complications associated with anticoagulant use, such as gastrointestinal bleeding and intracranial hemorrhage, were carefully considered & monitored. Despite these risks, the patient had no neurological deficits during admission or the follow-up period. In the first repeated CTA of the neck, performed one week after the injury, luminal narrowing was significantly improved from 80 to 33%. Finally, in the second repeated CTA of the neck, which was performed one year after the injury, there was a complete resolution of the injuries.

A high index of suspicion is required for the diagnosis of BCAI. Once the diagnosis is confirmed, anticoagulation therapy should be promptly started to avoid fatal consequences. To ensure optimal patient outcomes, surgeons should follow the current scientific evidence regarding appropriate indications for intervention versus conservative treatment in such injuries.

Conflict of Interest

None

Ethical approval

Approved by the Research and Ethics Committee, Bahrain Defense Force Hospital, Bahrain.

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