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Case Report

Traumatic Facial Artery Dissection from Chinstrap Injury: A Case Report

Alyssa V. Goodman, M.D.[1], Andrew P. Pirotte, M.D.[1], Derek R. Marlor, M.D.[2], Scott A. Turner, M.D.[3], Kirk A. Hance, M.D.[4]
The University of Kansas School of Medicine-Kansas City, Kansas City, Kansas
[1]Department of Emergency Medicine
[2]Department of Surgery
[3]Department of Surgical Critical Care and Trauma
[4]Department of Vascular Surgery

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INTRODUCTION

Traumatic dissection of the facial artery due to blunt trauma has not been previously reported. In contrast, blunt cerebrovascular injuries (BCVI) involving the carotid and vertebral arteries are well-documented and recognized as potentially devastating.[1,2] This case report presents a rare instance of facial artery dissection resulting from blunt trauma caused by a chinstrap injury. Clinicians should maintain a high index of suspicion for vascular injury in patients presenting after chinstrap-related trauma or other high-grade blunt injuries.

CASE REPORT

A 48-year-old male construction worker presented as a trauma activation after falling from scaffolding, sustaining blunt trauma to the head, face, and chest. His hard hat became caught on the scaffolding, and he was briefly suspended by his chinstrap before falling to the ground. He did not lose consciousness.

Upon arrival to the emergency department (ED), the patient reported pain in the right jaw and right chest. His primary survey was intact: airway was patent, bilateral breath sounds were present, central pulses were palpable, and his Glasgow Coma Scale (GCS) score was 15. The secondary survey revealed a 3 cm laceration over the angle of the right mandible. Vital signs remained stable.

Non-contrast computed tomography (CT) of the head, maxillofacial bones, and cervical spine revealed a small cortical chip fracture of the right mandible underlying the laceration (Figure 1). CT angiography (CTA) of the head and neck demonstrated segmental occlusion and/or high-grade stenosis of the right facial artery within the perimandibular laceration area, consistent with post-traumatic dissection (Figure 2). There was no evidence of hematoma, pseudoaneurysm, or active contrast extravasation. No additional injuries were identified on imaging.

The patient received cefazolin for the open fracture. Otolaryngology (ENT) and vascular surgery were consulted. ENT repaired the laceration and recommended nonoperative management of the mandibular chip fracture without need for follow-up. Vascular surgery advised against intervention for the isolated facial artery occlusion, as there was no involvement of the common, internal, or external carotid arteries. The patient was discharged home in stable condition with a prescription for amoxicillin-clavulanate.

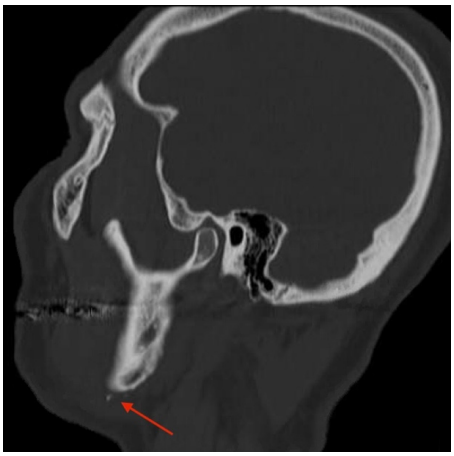

Figure 1. CT maxillofacial without contrast showing tiny right mandibular cortical chip fracture (arrow).

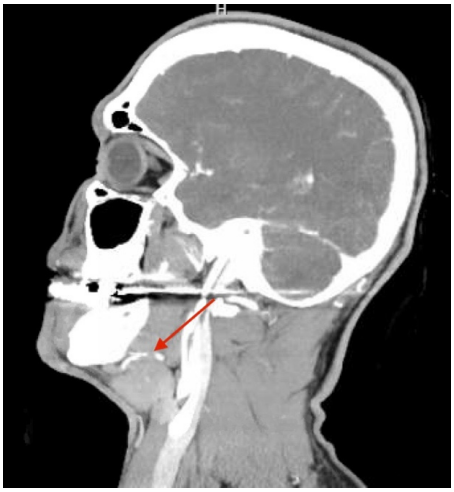

Figure 2. CTA head and neck showing traumatic occlusive dissection of the right facial artery (arrow).

DISCUSSION

The facial artery is a major branch of the external carotid artery (ECA). It originates from the anterior surface of the ECA within the carotid triangle of the neck and courses deep to the digastric and stylohyoid muscles before curving upward to cross over the body of the mandible. It follows a tortuous path toward the angle of the mouth and continues along the lateral aspect of the nose, terminating near the medial canthus of the eye.[3] The facial artery primarily supplies the superficial structures of the face.

There is limited literature describing blunt injury to the branch arteries of the internal and external carotids. To our knowledge, traumatic occlusive dissection of the facial artery due to blunt facial or neck trauma has not been previously reported. As a result, the appropriate treatment and surveillance of such injuries remain undefined and are largely guided by expert opinion. In such cases, it may be reasonable to extrapolate management strategies from existing guidelines for BCVIs.

BCVIs, non-penetrating injuries of the carotid and vertebral arteries, are well-described in the literature. They are identified in approximately 1-2% of blunt trauma patients and can result in severe complications

such as ischemic stroke and long-term neurologic impairment.[1,4] Current guidelines recommend early initiation of antithrombotic therapy in BCVI.[5]

In theory, blunt injury to a branch artery also may pose a risk of thrombotic or embolic complications, though with variable clinical significance. More likely sequelae may include acute bleeding or delayed pseudoaneurysm formation. In this case, management was guided by expert consultation. Vascular surgery reviewed the CTA and identified an isolated occlusion of the right facial artery, without injury to the common, internal, or external carotid arteries. Unlike carotid or vertebral artery injuries, which carry high risks for ischemic events, the extensive collateral circulation of the face significantly reduces the likelihood of serious ischemic outcomes from facial artery occlusion.

As with BCVI, the decision to initiate antithrombotic therapy for facial artery dissection should be based on patient-specific factors such as injury severity and bleeding risk. Our patient was deemed low risk for complications and did not require antithrombotic therapy or vascular follow-up. He was discharged from the ED in stable condition.

CONCLUSIONS

Traumatic dissection of the facial artery following blunt trauma is extremely rare and, to our knowledge, has not been previously described in the literature. More data are needed to better understand its incidence, potential complications, and appropriate management. In such rare presentations, emergency providers should consider consulting vascular surgery or other relevant specialists to guide care decisions.

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