Case report: Traumatic lingual hematoma

B. Massey, K. Juhasz, J. Licata, S. Schell, G. English

University of Pittsburgh Medical Center, Hamot, Erie, PA, United States of America
Ear Nose & Throat Specialists of Northwestern Pennsylvania, Erie, PA, United States of America
Western University of Health Sciences-COMP NW, United States of America

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We describe a case of traumatic lingual hematoma in a patient on dual antiplatelet therapy. After securing the airway, bilateral lingual artery embolization successfully halted expanding hematoma formation. Patient subsequently required tracheostomy for continued airway edema. Although patient course was wrought with postoperative complications, we review this case to illustrate how prompt hematoma evacuation and embolization can resolve significant vasovagal bradycardia and hypotension secondary to expansile lingual hematoma.

Introduction

Rapid enlargement of the tongue secondary to lingual hematoma can lead to life-threatening airway emergencies, necessitating prompt recognition and management of this condition. Due to its highly vascular nature and extensive anastomotic network, the tongue is at a high risk for hemorrhage and subsequent hematoma formation following trauma [1]. There are numerous reports in the literature of lingual hematoma, ranging from traumatic to spontaneous, with or without anticoagulation use. Common causes of traumatic lingual hematomas include: MVA's, assault, child abuse, and seizures. Spontaneous lingual hematomas are usually a result of an inherited coagulopathy or treatment with anticoagulants [2–6]. Some theorize that patients with vascular disease and atherosclerosis of the lingual arterial system may be at increased risk for lingual hematoma due to vessel tortuosity, fragility, and possible shearing [7]. Regardless of etiology, management of lingual hematomas is controversial and includes observation, airway control, steroids, antibiotics, reversal of any coagulopathy, IR embolization and surgical intervention [8]. Being able to recognize the initial presentation, underlying cause and relevant anatomy of lingual hematomas is critical to the proper management and treatment of this condition.

Case report

This case involves a 75-year-old female with PMH significant for left internal carotid artery (ICA) stent in 2009 and coronary stenting in 2014 on aspirin and clopidogrel who drove herself to the ER after falling down three stair steps at home. Upon arrival to triage, the patient's chief complaint was shortness of breath and bleeding from oral cavity. She was emergently taken to trauma bay and noted to have dysarthria, dysphonia, dyspnea, ventral tongue/floor of mouth (FOM) laceration, and significant airway compromise secondary to progressive tongue swelling. With the assistance of anesthesia, the patient was successfully intubated transorally via Glidescope video laryngoscopy on the first attempt.
Immediately thereafter, ENT was called to the ER for control of actively bleeding FOM laceration. On exam, a 5 cm transverse laceration at junction of ventral tongue base and FOM was identified with active brisk bleeding, as well as obvious hematoma within the intrinsic tongue musculature extending towards the base of tongue. Also noted was a small area of left lower lip buccal mucosa avulsion, diffuse submental swelling and ecchymoses. The lower lip and tongue were diffusely enlarged, with the tongue being displaced posterosuperiorly obstructing the oral cavity and oropharynx and protruding from the mouth. There was no clear identifiable left Wharton's duct. The genioglossus and FOM were found to be separated by a hematoma cavity extending ~4–5 cm posteriorly towards the root of tongue and vallecula. The proposed source of bleeding (FOM laceration) was sutured shut with simple interrupted 3-0 vicryl tamponade sutures. The anterior oral cavity and ventral tongue were packed with fibrillar and 4 × 4 gauze. The patient was observed for several minutes and there was no evidence of any persistent bleeding that would require urgent surgical intervention. Transfusion of functioning, uninhibited platelets was initiated given the patient’s coagulopathy secondary to dual anti-platelet therapy by administering a “superpack” of platelets. The patient was stabilized and taken to the radiology suite for CT imaging.

Fig. 1. CTA neck: axial left view showing significant extravasation within the central tongue.

Fig. 2. CTA neck: coronal view showing significant extravasation within the central tongue.
CTA neck showed a large focus of active extravasation within the central aspect of the oral tongue suggesting deep active hemorrhage (Figs. 1 and 2); marked enlargement of the oral tongue and lower lip consistent with hematoma. No acute facial fractures. CT head and C-spine were negative.

The patient was transferred to the ICU and Interventional Radiology (IR) was consulted. Hgb dropped from 12 to 8 and 2 units of pRBCs were given. ENT reevaluated the patient and noted her hematoma to be increasing in size and extending further into the anterior level 1 neck, without any significant lateral neck involvement. The patient abruptly became bradycardic with HR in the 20–30s and SBPs in the low 80s. The lingual hematoma was decompressed at bedside with expression of a large clot and hemostats were used to probe the cavity, which was noted to be remarkably larger than on initial exam now involving the entire length of the tongue to the preepiglottic space (Figs. 3 and 4). The patient's heart rate improved immediately thereafter and a Quick Clot gauze was placed in the hematoma cavity for temporary hemostasis. The decision was made to go to the IR suite urgently. Bilateral lingual artery Gel-foam embolization was performed for definitive hemostasis (Figs. 5–8).

Unfortunately, the patient's postoperative course was complicated by multiple issues. On day two, oral packing was removed. On day three the patient was started on a five-day course of IV decadron. Repeat CT was performed on day six which showed continued glossal edema. Tracheostomy was performed on day seven for continued airway obstruction. On day nine, the patient's tracheostomy tube became dislodged into a false passage requiring a trip to the OR for emergent trach change. The patient was transferred out of
the ICU on hospital day eleven. While on the floor she had a few episodes of oral cavity bleeding which were controlled with local measures including: ice-soaked gauze, pressure and aminocaproic acid solution. She was ultimately discharged to a rehab facility on day 17. Unfortunately, the patient went into cardiac arrest approximately one week after being discharged resulting in anoxic brain injury. The patient was made CMO and passed shortly thereafter.

**Discussion**

This case illustrates a rare but life-threatening complication of traumatic injury to the tongue in the setting of dual antiplatelet therapy. Although there are numerous reports of anti-coagulant therapy preceding spontaneous lingual hematoma or exacerbating traumatic lingual hematomas, we are unaware as yet of a similar association with anti-platelet therapy. Commonly reported medications include Warfarin, Streptokinase, Heparin, and various other agents [9].

Clinical signs of sublingual or lingual hematoma may include mass, swelling, bleeding, dyspnea, stridor, dysphagia and dysphonia. Because of its abundant vascularity, tongue enlargement can rapidly proceed to upper airway obstruction as it is displaced posterosuperiorly [10].

Management of sublingual hematoma must focus foremost on securing the airway. In non-emergent suspected lingual hematomas, the use of flexible fiberoptic laryngoscopy may allow clinicians to more accurately assess the degree of obstruction; thus,
preventing unnecessary intubations [11]. However, if impending airway compromise is suspected it is critical to quickly decide on an approach. Many authors recommend awake, fiberoptic nasal intubation for initial management followed by elective tracheostomy in the OR [10].

Once the airway is secured, attention should be focused at achieving hemostasis. Hemostatic management remains the most controversial and is partially decided based upon the underlying cause of the hemorrhage. In cases of spontaneous lingual hematoma, whether from an inherited or acquired coagulopathy, management is usually with observation and reversal of any coagulopathies if present [12]. Conversely, traumatic lingual hematoma management depends on the size and presence of active expansion. Treatment varies from local control and leech therapy to IR embolization or surgical ligation of culprit vessels [13]. As in our case, local control was attempted but ultimately unsuccessful thus we proceeded with IR embolization of the lingual arteries. Surgical intervention becomes necessary in instances where IR embolization is unavailable or unsuccessful. This most commonly entails extraoral ligation of the lingual artery and requires a detailed understanding of the involved anatomy [10].

To our knowledge, significant vasovagal bradycardia and hypotension secondary to expansile lingual hematoma that resolves

Fig. 7. Post-embolization left.

Fig. 8. Post-embolization right.
immediately after hematoma evacuation, as our patient experienced, has not been described in the literature. In our case, bilateral lingual artery particle embolization proved to be a successful method to stop the lingual hemorrhage. Unfortunately, given the patient’s mortality we were unable to provide any long term follow up and therefore were unable to determine the degree of tongue functionality and recovery she would have had.

Conflict of interest

Authors have no personal or financial disclosures.

Previous meeting presentation(s)

Poster presentation at UPMC Hamot Resident Research Days April 11–12, 2018.

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