CASE REPORT
trauma, facial;
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Genioglossus Hemorrhage After Blunt Facial Trauma

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We present the case of a 28-year-old man admitted to a Level I trauma center after a motorcycle crash in which he sustained blunt trauma to the face and neck, including a mandibular ramus fracture. The patient had no airway compromise on admission, but he acutely developed significant partial upper airway obstruction necessitating emergency endotracheal intubation and subsequent tracheostomy approximately 36 hours after admission. This obstruction was caused by an expanding hematoma of the genioglossus muscle. Potential causes of this complication are presented, and signs and symptoms of upper airway obstruction and airway management strategies are discussed. The etiology of traumatic upper airway obstruction is reviewed.
[Woodmansee VA, Rodriguez A, Mirvis S, Fitzgerald B: Genioglossus hemorrhage after blunt facial trauma. *Ann Emerg Med* April 1992;21:440-444.]

INTRODUCTION

The propensity for airway compromise in blunt neck and facial trauma is a primary concern for the acute care physician. Several reviews have discussed the signs and symptoms of airway compromise and the diagnostic modalities available to assist in arriving at the diagnosis of partial or complete upper airway obstruction.[1-3] An occluded upper airway is life-threatening and must be handled expediently using appropriate airway management techniques to establish a patent airway for the patient.[4]

CASE REPORT

A 28-year-old, fully immobilized man was admitted by helicopter transport to a Level I trauma center after a high-speed, head-on collision between a motorcycle and an automobile. He was ejected from the motorcycle on impact and landed under the rear of the motor vehicle. He was not wearing a helmet. There was no loss of consciousness at the scene or en route to the hospital. The patient presented complaining of oral bleeding, jaw pain, and left shoulder pain. He denied alcohol or illicit drug usage. His medical history was unremarkable, and he had no allergies to medications.

In the admitting area, the patient was awake, alert, and oriented to person only. The Glasgow Coma Score was 14/15, ▶

with one point subtracted for nonspontaneous and confused speech. The airway was intact, with a patent oropharynx on inspection. No stridor or hoarseness was noted. Frequent oral suctioning was necessary secondary to an avulsed right upper incisor, which was actively bleeding. His vital signs, including blood pressure of 120/70 mm, pulse of 104, and respirations of 20, were stable on admission. He was afebrile, and his cardiac monitor displayed sinus tachycardia without ectopy.

Physical examination revealed two abrasions over the right malar area. The right mandibular ramus was palpably tender and had an overlying 3-cm laceration that extended to the bone over the right mandibular angle. The right upper lateral incisor was completely avulsed, and the right upper central incisor was loose. There were two 5-mm hard-palate lacerations and two superficial lacerations to the tip of the tongue, measuring 5 mm and 1 cm. All of the lacerations were hemostatic.

Figure 1.
Lateral neck radiograph taken during initial evaluation of motorcycle crash victim. Note nasogastric tube in place with no obvious significant airway obstruction and vertical mandibular ramus fracture with slight distraction.

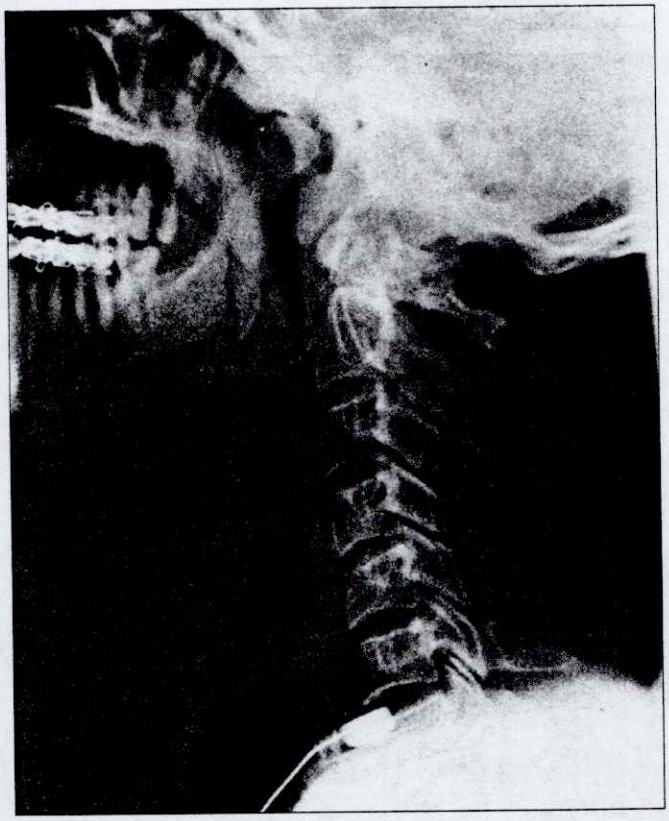
Cervical and neck examination revealed no bony tenderness or crepitance. No ecchymosis or edema was noted. A 4-cm superficial flap laceration not violating the platysma was found in zone 2 on the left.

The remainders of the head, eyes, ears, nose, and throat; cardiovascular; pulmonary; abdominal; extremity; and neurologic examinations were unremarkable.

Laboratory evaluation included arterial blood gases on room air with pH 7.39; Pa_{O_2}, 89 mm Hg; Pa_{CO_2}, 39 mm Hg; base excess, −0.5; bicarbonate, 24.0 mmol/L; and oxygen saturation, 99%. Admission hemoglobin and hematocrit were 13.0 mg/dL and 38.9%, respectively.

Lateral cervical radiographic evaluation was performed (Figure 1). This film was initially interpreted by the staff radiologist as showing a mandibular ramus fracture but no soft tissue abnormalities. The plain radiographs of the mandible showed a right vertical ramus fracture with mild distraction. Routine chest and pelvic plain radiographs as well as computed tomography (CT) scans of the head and abdomen were interpreted as negative by the staff radiologist. A facial CT scan showed only an isolated mandibular ramus fracture. ▶

Figure 2.
Soft-tissue lateral cervical radiograph taken approximately 35 hours after admission. Note marked anterior hypopharyngeal edema with inferior and posterior displacement into the airway, resulting in almost complete airway obstruction.



Consultations with the anesthesia and oral and maxillofacial surgery services were obtained. The anesthesia consult thought that the patient had a stable and patent airway and suggested no intervention. The oral and maxillofacial surgery consult placed arch bars and rubber bands and planned to perform mandibular-maxillary wiring the next day. The bleeding from the avulsed tooth socket was controlled with direct pressure. The neck, mandibular, and tongue lacerations were repaired primarily. The patient received prophylactic 1 g IV cefazolin and was admitted to the general surgical ward in stable condition.

Approximately 22 hours later, on rounds, the patient was reassessed and found to have a patent airway without respiratory compromise. He complained of a sore throat but was not stridorous and was able to swallow secretions. He was taking clear liquids without difficulty. Neck and facial examinations were unremarkable, and no edema was elucidated. Mandibular-maxillary wires and arch bars were placed by the oral and maxillofacial surgery service approximately 28 hours after the injury.

Approximately 30 hours after the injury the patient complained of dizziness and shortness of breath. The on-call surgical team evaluated the patient and found no tachypnea or stridor. The oral and maxillofacial surgery consult was called at that time to evaluate for upper airway obstruction and removed the mandibular-maxillary wires and arch bars. On physical examination, minimal posterior pharyngeal edema was noted. The uvula was midline. No neck edema was appreciated, and the arterial blood gases on 40% Fio_2 by face mask were Pao_2 of 77 mm Hg and oxygen saturation of 94%. It was documented at that time that the patient's distress was not thought to be secondary to oropharyngeal obstruction.

Approximately 35 hours after admission, the surgical team was called to see the patient again secondary to decreased mental status, agitation, inability to lie flat, and drooling. On physical examination, the patient was unable to swallow his secretions, stridorous, tachypneic, and using secondary muscles of respiration. He was also having difficulty phonating. Arterial blood gases on 100% Fio_2 by face-mask were Pao_2 of 159 mm Hg; $Paco_2$, 40 mm Hg; pH 7.36; and oxygen saturation, 99%. His partial thromboplastin time ▶

Figure 3.
Cervical CT image of upper airway of patient after intubation for upper airway obstruction. Note significant lateral and anterior impingement on the airway at the level of the hypopharynx and the large hematoma located in the body of the genioglossus muscle.

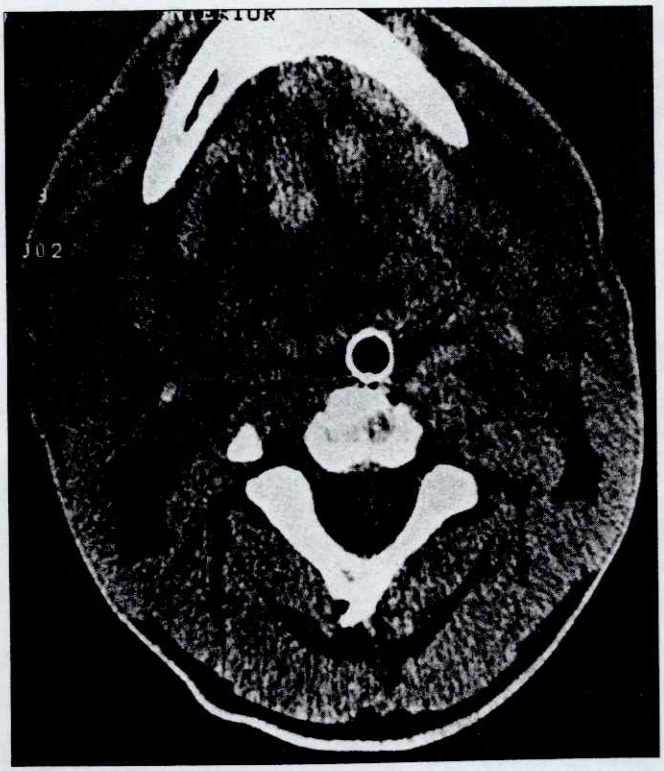
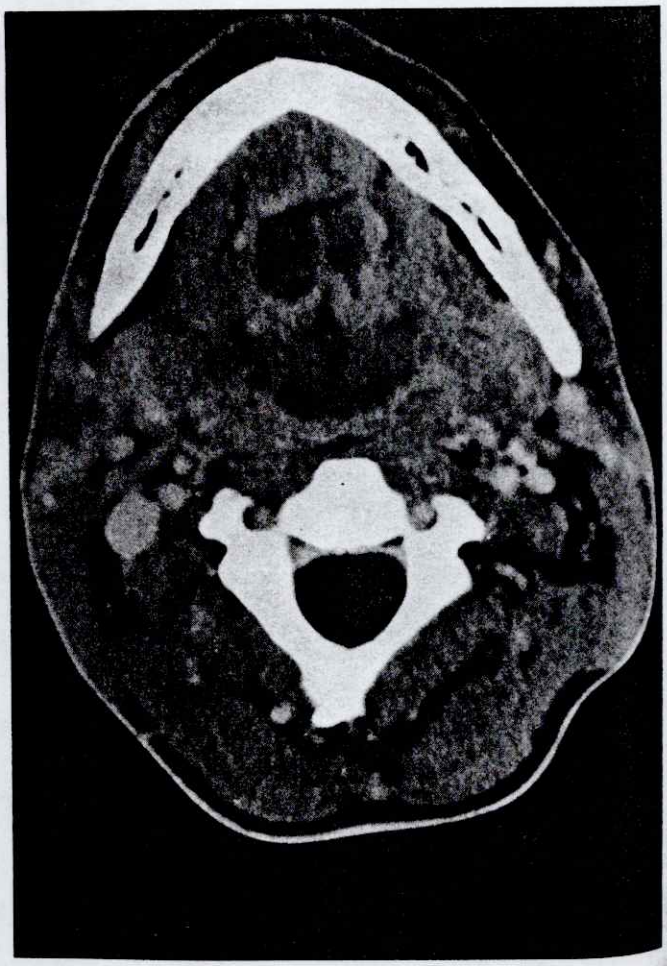


Figure 4.
Repeat cervical CT image of upper airway six days after acute upper airway obstruction. There is minimal resolution of either the size of the hematoma or the degree of airway compromise.



was 64 seconds (control, 26.6 seconds). A soft-tissue lateral neck radiograph revealed significant anterior upper airway obstruction in the hypopharynx with inferior displacement of the hyoid bone (Figure 2). The patient was nasotracheally intubated on the first attempt and transferred to the ICU. The partial thromboplastin time was rechecked immediately after intubation and found to be 28 seconds (control, 26.6 seconds).

The patient progressed well; the next morning, a CT scan of the cervical area demonstrated a large hematoma of the genioglossus muscle compressing the anterior airway (Figure 3). A tracheostomy was performed on the fourth hospital day. The patient remained stable and progressed well throughout his hospital stay. He was discharged home with a tracheostomy in place on the tenth hospital day after repeat CT scan of the cervical area revealed minimal resolution of the genioglossus hematoma or airway compromise (Figure 4). His hematoma gradually resolved, and he was decannulated on the 26th day after injury without complications.

DISCUSSION

The physician evaluating and treating facial trauma must always be cognizant of the potential for upper airway obstruction, possibly delayed as long as 36 to 48 hours after injury, as demonstrated by our patient. A high index of suspicion is necessary. Classically described signs and symptoms (Figure 5) are usually nonspecific in the early stages of obstruction[5] and may not be present until airway compromise becomes significant. Enlarging hematomas at the base of the tongue are particularly occult to physical examination until the soft tissues have expanded significantly. In retrospect, our patient displayed some nonspecific signs and symptoms[4] (eg, sore throat[6] and tachypnea) early in his hospitalization. Fortunately, his airway obstruction progressed over several hours, and adequate time was allowed for intervention when the classic signs and symptoms of airway obstruction developed.

Several diagnostic modalities are available for evaluation of a patient with a potential upper airway obstruction. Our patient received a lateral neck radiograph, arterial blood gas determinations, chest radiograph, and serial oropharyngeal and neck examinations on admission. These are all important adjuncts to the diagnosis of upper airway obstruction.[2] Neck extension for lateral neck radiographs better emphasizes airway compromise but is not feasible in the acute trauma evaluation due to the potential for cervical spine injury.

Other diagnostic adjuncts that may have been useful in this patient were digital palpation of the base of the tongue and an attempt to visualize the fauces of the submaxillary space through direct laryngoscopy or bronchoscopy. These were not done on admission secondary to the low index of suspicion for upper airway compromise. Cohen and Warman state that failure to visualize the submaxillary fauces precedes frank upper airway compromise and may predict the development of upper airway obstruction.[6]

Once a diagnosis of upper airway hematoma is made, the hematoma should be visualized, if possible. Blind intubation may lead to laryngospasm, hematoma rupture, and aspiration.[1] It is also suggested that serial hemoglobins be followed in patients with oropharyngeal and facial trauma.[7] A decreasing hemoglobin, although nonspecific, should alert the physician to the possibility of expanding hematoma with possible airway compromise.

Several potential causes for airway compromise existed in this patient. The most likely cause is the slow progression of an expanding hematoma of the genioglossus muscle. Our patient had a superficial zone 2 injury to his neck and a significant mechanism of injury. This implies significant blunt trauma to the genioglossus muscle. Blunt trauma as minor as whiplash injury can cause significant cervical hematoma formation and upper airway obstruction.[8]

Pooling of blood or saliva in the hypopharynx also can lead to upper airway occlusion.[9] However, this was noncontributory in our patient after initial stabilization secondary to suctioning and hemostatic control of his bleeding injuries.

In addition, mandibular fractures disrupt the normal support system to the tongue and can lead to compromise of the upper airway.[9] Our patient's mandibular fracture may have contributed to his airway compromise. The tongue sits with most of its bulk in the hypopharynx. Its origin is along the mandibular symphysis, and its attachment is to the hyoid bone.[3] When swelling occurs in the genioglossus muscle, there are inferior and posterior pressures on the epiglottis and hyoepiglottic ligaments. Genioglossus stability is usually lost with symphysis fractures.[6] It is postulated that the distraction of the mandibular fracture site in our patient compromised the stability of this muscle. The change in his airway status soon after mandibular wiring suggests that the realignment of the bony structure caused rebleeding and expansion of the genioglossus hematoma. Further worsening occurred after the arch bars and wire were removed. A direct relation between manipulation of the fracture site and hematoma expansion must be assumed. Strate and Boies advocate definitive alignment and repair of mandibular fractures at the time of initial assessment if the patient is stable.[7] ▶

Figure 5.
Signs and symptoms of partial upper airway obstruction

Nonspecific	Specific
Hyperventilation	Stridor
Tachypnea	Decreased phonation
Tachycardia	Inability to swallow
Dyspnea	Hoarseness
Mental status change	Drooling
Suprasternal or intercostal retraction	
Diaphoresis	
Cyanosis	

Our patient also may have had some qualitative coagulation defect antecedent to his injury that predisposed him to hematoma formation. Aspirin[4] and coumadin[6] have been implicated in expanding hematomas of the neck. Although our patient denied recent use of antiplatelet agents, the effects of these agents last throughout the life of the platelet (seven to ten days) and may have contributed to hematoma expansion. The patient may not have remembered aspirin or nonsteroid use occurring more than a few days before the injury. Laboratory error cannot be excluded, as repeat coagulation tests were normal.

Acute airway intervention in this case consisted of blind nasotracheal intubation, which was successful. Emergency cricothyroidotomy was indicated in this patient. As previously mentioned, there is a high risk for bleeding from hematoma disruption and subsequent aspiration with blind procedures through the upper airway. If this technique had failed, complete airway obstruction would probably have ensued.

SUMMARY

Our case emphasizes the need for a high index of suspicion for acute upper airway obstruction in patients with facial and neck injuries. Delayed hemorrhage into the parapharyngeal soft tissues may lead to airway compromise beyond the acute injury period. Bleeding into the region of the floor of the mouth may be particularly occult to inspection. Airway patency should be reassessed after surgical manipulation of the mandible, as manipulation may induce delayed hemorrhage or additional edema in injured tissues or change the orientation of the muscles supporting airway patency. ■

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