A CASE OF TRACHEOBRONCHIAL OBSTRUCTION ORIGINATING FROM A POSTERIOR PHARYNGEAL THROMBUS RELATED TO MAXILLO-MANDIBULAR RECONSTRUCTION OF A PREVIOUS AMELOBLASTOMA RESECTION

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Abstract

Airway obstruction during initiation of general anesthesia is an obstacle faced by anesthesia providers that typically is overcome with conventional maneuvers or instruments. The present case report describes a pharyngeal thrombus aspirated into the trachea, while attempting an awake fiber optic intubation on a patient with a known previous maxillofacial bleed several days prior to surgery. The thrombus likely formed from the pooling of blood originating from a branch of the maxillary vessels, which can be seen postoperatively after extensive maxillary or mandibular surgery, as in this case. This thrombus was dislodged with either mechanical manipulation of the patient's airway or stimulation of the patient's airway reflexes, resulting in aspiration of the clot. Inability to adequately ventilate the patient despite proper endotracheal tube placement resulted in emergent tracheotomy and cardiovascular collapse of the patient. Though common and having multiple etiologies, tracheobronchial obstruction is potentially a catastrophic event requiring methodical intervention and when needed, excellent communication with surgical colleagues. A presumptive diagnosis can be reached by utilizing patient history, physical exam findings and a retrospective look at radiographic studies.

Keywords: Tracheal obstruction, pharyngeal thrombus, maxillofacial bleeding, mandibular ameloblastoma.

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Introduction

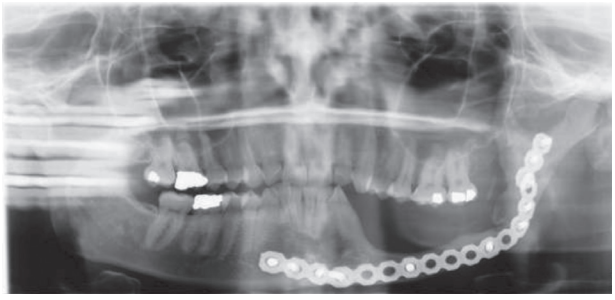
Tracheobronchial obstruction is a common yet perilous obstacle encountered by anesthesia providers. Obstruction can be classified broadly as either chronic or acute with each having different management strategies. Onset of acute tracheal obstruction often occurs suddenly and without warning; thus, requiring the clinician to quickly determine the etiology of obstruction and best treatment modality. The etiology of acute tracheobronchial obstruction is multi faceted with the most common causes being bronchospasm, foreign body aspiration and mucosal edema[1]. This case report describes the management and diagnosis of a tracheobronchial obstruction resulting from a posterior pharyngeal thrombus. The patient has been de-identified by removing obvious demographic information meeting IRB exemption standards, and therefore, no patient consent was needed.

Case Report

This case involves a 29-year-old female, 170 cm and 55 kg, with a history of previous mandibular ameloblastoma status post resection and mandibular reconstruction with a rib graft approximately ten years prior. She presented to the LSU Oral & Maxillofacial Surgery service, where she was found to have hardware failure (Figure 1). A secondary reconstruction was than performed by the OMFS service, which included free fibula flap reconstruction of the mandible and repositioning of the left supra-erupted maxillary teeth with a Hemi-Lefort I osteotomy. The secondary reconstructive procedure was lengthy due to its complexity, need for hardware removal, and additional maxillary repositioning.

Fig. 1

Hardware failure of previously reconstructed mandible with rib s/p ameloblastoma resection. Also, note supraeruption of left maxillary teeth for which Hemi-Lefort would be done.



The patient was then admitted to the Intensive Care Unit (ICU) to be monitored for airway and graft patency. The patient progressed reasonably well over the next several days and was transferred to the floor. Six days post operatively from the second graft repair, the patient developed a new onset cough. Subsequently, that night the patient had a prolonged coughing spell, which was followed by three episodes of hematemesis and an estimated blood volume of 700 cc. The patient was then transferred to the ICU for close monitoring. A maxillofacial computerized tomography (CT) was ordered and described significant soft tissue swelling displacing the left parapharyngeal soft tissue into the hypopharyngeal airway and displacing the glottis (Figure 2, 3). Despite the noted swelling, the airway was sufficiently patent to allow aspiration according to the radiology report. With the observed clinical findings, the patient was scheduled to go to the operating room (OR) that evening for exploration and evacuation of hematoma. The anesthetic plan for the patient consisted of an awake fiber optic nasal intubation due to limited mouth opening, airway deviation and the surgical site.

After transport to the OR and placement of standard American Society of Anesthesia (ASA) monitors, the patient's airway was topically anesthetized with 2% lidocaine. The patient was sedated with 5 mg of midazolam i.v. and 100 mg of ketamine i.v. titrated

Fig. 2

Maxillofacial computerized tomography demonstrating probable extramucosal thrombus in the left nasopharynx extending to the posterior nasopharynx.

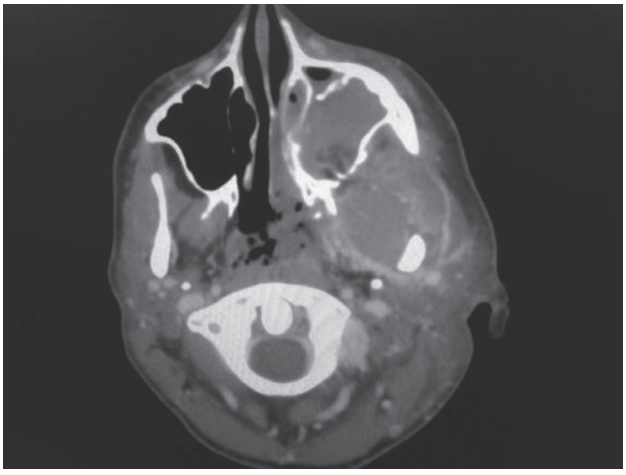


Fig. 3
Saggital image depicts the thrombus completely obliterating the the patentcy of the pharynx at the level of the soft pallet and extending to the level of the glottis.

over 5-15 minutes, and an awake nasal fiber optic intubation was attempted. On the initial attempt at fiber optic intubation, there was difficulty visualizing the glottis despite pharyngeal suction. Also, soon after inserting the fiberoptic scope into the nasopharynx and advancing to the posterior pharynx, the patient quickly began to desaturate. The initial attempt was aborted and mask ventilation was initiated. At this time, the patient had significant independent inspiratory effort, however, it was difficult to ventilate by mask, requiring excessive airway pressures to obtain a very modest tidal volume of approximately 80-90 ml. Despite the apparent obstruction, oxygen saturations of greater than 90% were achieved and a second attempt at tracheal intubation was attempted orally. This attempt was quickly successful as confirmed by visualization of tracheal rings, detection of end tidal CO2, equal breath sounds bilaterally and condensation in the endotracheal tube (ETT). When attempting to inflate the cuff of the ETT, it became apparent that the cuff had a leak and sufficient pressure was not achievable to adequately ventilate the patient and mask ventilation was resumed over the ETT. Ventilation remained difficult but was facilitated to a significant degree by the defective tube in place. The initial consideration to exchange the defective ETT was dismissed due to difficulty maintaining adequate ventilation with an appropriate mask seal and a known conduit into the trachea. Also, the patient developed bradycardia with a rate of 20 beats per minute, despite SpO2 readings in the 80's. After multiple doses of atropine and epinephrine in response to the bradycardia resulting in a heart rate of 40 beats per minute, the patient further continued to decompensate with continued difficult ventilation, prompting the anesthesia team to request an emergent tracheotomy by the surgeons. During placement of the tracheotomy, the patient went into ventricular tachycardia and advanced cardiac life support (ACLS) was initiated. Initial defibrillation resulted in return of spontaneous circulation with vasopressor and inotropic support. Upon gaining surgical access to the trachea a large 6 x 3 cm thrombus was removed from the trachea with the surgical suction distal to the previously inserted ETT (Figure 4). The previously inserted ETT was withdrawn to allow a 6.0 ETT tube to be passed into the trachea. It was secured with sutures after confirmation of bilateral breath sounds. The patients SpO_2 immediately began to rise and ventilation returned to baseline. The patient was transferred to the intensive care unit (ICU) on an epinephrine infusion for cardiovascular support with apparent global neurological deficit. The patient was

Fig. 4
Large thrombus evacuated from trachea distal to the tip of the ETT after emergent tracheostomy. The thrombus dimensions are remarkably similar to those in the axial image above.

unresponsive although some of the findings were clouded by intraoperative sedation. Subsequent review of the maxillofacial CT scan the patient received the day of surgery revealed occlusion of the left nasal passage and narrowing of the posterior nasopharynx with extra mucosal material (Figure 2), which was described on a reinterpretation by a radiologist as extramucosal fluid/soft tissue. Postoperatively, the patient made good improvements and regained both speech and motor function to baseline.

Discussion

Given the retrospective findings on the CT scan, it is likely that the thrombus in the airway originated from the nasopharynx. Presumably the thrombus formed from the pooling of blood originated from the maxillary bleed. It was then dislodged with either mechanical manipulation of the patient's airway or stimulation of the patient's airway reflexes, resulting in aspiration of the clot. If the CT images were not actually of a pharyngeal thrombus, it is plausible that blood was aspirated during the preoperative episodes of hematemesis when the patient's mouth was wired closed. Thus, the thrombus was already present in the trachea; but, was not totally occluding the passage and became dislodged either prior to or during airway manipulation. No foreign material was detected in the tracheal lumen to a level just superior to the carina on maxillofacial CT two hours prior to arrival to the operating room, but there was a dense extra mucosal material adherent to the pharyngeal wall just superior to the glottic opening.

Etiology of Tracheobronchial Obstruction

As mentioned above, the etiology of tracheobronchial obstruction can be broadly categorized as either chronic or acute. Chronic causes include emphysema, pulmonary fibrosis, and cancers of the larynx, trachea, or mediastinal organs[2]. Acute causes of tracheobronchial obstruction include infection, bronchospasm, mucous impaction, and foreign body aspiration[1]. The first report of endobronchial obstruction from a blood clot was described in 1929[3]. While there have subsequently been limited case reports detailing obstruction secondary to blood clot, other documented etiologies include bronchiectasis, tuberculosis, pulmonary arteriovenous malformation, pulmonary infarction, trauma, and mitral stenosis[1,4-9]. A tracheobronchial blood clot should be considered in the differential diagnosis when substantial hemoptysis is present. However, it is important to note that approximately 30% of endobronchial blood clots can occur without evidence of gross hemoptysis[1].

Diagnosis, Management and Treatment

While a definitive diagnosis of a tracheobronchial clot includes direct visualization of the clot, a presumptive diagnosis can be made using clinical and physical exam findings. These include decreased or absent breath sounds accompanied by occasional inspiratory or expiratory wheezes. Other physical exam findings are synonymous with atelectasis and include decreased vocal fremitus, restricted chest wall motion, and flatness to percussion[1]. Tracheobronchial clots in ventilated patients manifest with a sharp rise in peak inspiratory pressures and a concomitant decrease in tidal volumes[1]. An unique presentation of tracheobronchial obstruction is a ball-valve obstruction[6,8,10-11]. In this situation, the blood clot acts as a one-way valve that allows air into the respiratory tract, but blocks expiratory flow. This can result in ipsilateral hyperexpansion with increased risk of tension pneumothorax and subsequent hemodynamic compromise[11]. One such case of a thrombus acting as a ball-valve occurred after tracheostomy. Ultimately in this case, the clot was removed by using an ET tube as a suction catheter after unsuccessfull removal with a conventional suction catheter[16].

Management of the clot is primarily dictated by the site of obstruction. In mechanically ventilated patients, this differentiation can be made by using a suction catheter to assess the patency of the endotracheal tube[1]. A "cuff deflation test" can also be used to identify if the obstruction is proximal or distal[12]. Further decisions on management and treatment should include careful consideration of the patients clinical status as any attempts to remove the clot carries the risk of causing further damage or bleeding. A patient who is hemodynamically stable with no signs

of airway compromise may be best managed by close monitoring in the ICU. Of note, there have been several case reports that document the spontaneous resolution of clots over the course of 3 days[1].

If further treatment is needed, initial modalities include saline lavage, suctioning, and forceps extraction via flexible bronchoscope. Additional management techniques include use of rigid bronchoscopy, topical thrombolytics, and Fogarty catheter dislodgement. Fogarty catheter manipulation has been documented to be particularly useful if the clot is found to be firmly adherent to the surrounding tissue[4]. The direct application of thrombolytics to the clot has also been found to be effective in dissolving the clot. This facilitates the removal of the clot in a piece meal fashion under direct visualization. Both streptokinase and urokinase have been used successfully in previous case reports[13-15]. For large clots, such as the one presented in this report, a novel approach may be required. Similar to the case previously mentioned where an ET tube was used as a suction catheter[16].

Conclusion

Tracheobronchial obstruction is a multi faceted complication that every anesthesia provider should be familiar with. The onset of acute obstruction requires the clinician to form a quick and definitive management plan. A presumptive diagnosis can be reached by synergistically utilizing physical exam findings, radiographic studies, and contextual clues. This case reiterates the importance of the anesthesia provider to maintain vigilance during a crisis situation.

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