Infections and Edema

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KEYWORDS

• Deep neck infection • Retropharyngeal abscess • Ludwig angina
• Peritonsillar abscess • Supraglottitis • Hereditary angioedema
• ACE inhibitor angioedema • Allergic angioedema

KEY POINTS

- Infection and angioedema of the head and neck are common causes of impaired airways.
- History, physical examination, and fiberoptic laryngoscopy are critical in evaluation.
- Preintervention discussion and planning by anesthesia and otolaryngology are essential.
- A decision regarding the primary modality and contingency plans to secure the airway is made.
- Regular practice by the entire team in simulation laboratories will maintain and hone skills.

INTRODUCTION

Infectious and inflammatory conditions of the head and neck often present with impaired and challenging airways. Careful evaluation and accurate diagnosis will allow for prompt and safe management of the airway and the treatment of the underlying condition. Development of primary and contingency plans for securing the airway is critical and requires discussion and coordination between the anesthesiologist and otolaryngologist. This article focuses on the evaluation and management of patients with infections involving the upper aerodigestive tract and neck as well as angioedema.

DEEP NECK INFECTIONS

The occurrence of deep neck infections has been declining since the introduction of antibiotics. Deep neck infections are generally polymicrobial. *Streptococci*, *Peptostreptococcus*, *Staphylococcus*, and anaerobes are the organisms most commonly cultured. These bacteria are generally part of the normal oral flora but become virulent when mucosal barriers are violated.[1]

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Successful management of deep neck infections often involves control of the airway, effective antibiotic therapy, and timely surgical intervention. A focused history and physical examination should be obtained if possible, followed by either control of the airway if compromised, imaging to assess extent of infection, and surgical drainage if needed. Patients may present with generalized symptoms of fever and malaise or with more localizing symptoms of shortness of breath, odynophagia, dysphagia, sore throat, neck pain, stiffness, or voice changes. The most common finding on examination is neck swelling.[2,3] The examiner may also note dyspnea, stridor, elevation, or firmness in the floor of the mouth, fever, and pharyngeal wall or soft palate swelling. Although rare, bleeding may be related to a sentinel bleed from infectious involvement of the carotid artery. Tachypnea or stridor may indicate impending airway obstruction and mandate immediate attention.[4]

The parapharyngeal (lateral pharyngeal) space is the most commonly involved space (38.4%) in several large studies.[5] Odontogenic and upper airway infections are the 2 most common causes of deep neck infections (53% and 31%, respectively).[1,6] *Streptococcus viridans* and *Klebsiella pneumoniae* were the most common organisms (34% each). In another large retrospective review of 234 patients, deep neck infections were the first manifestation of a malignant tumor in 6% of cases, which should raise awareness that an infected tumor may present as a deep neck space infection.[7]

Submandibular Space

Submandibular space infections can involve either of the 2 subcompartments, the sublingual or the submylohyoid, or both. These 2 subcompartments communicate posteriorly around the mylohyoid muscle. Ludwig angina is the name for the prototypical infection of the submandibular space infection and requires that both compartments be involved. An odontogenic source of infection is identified in 70% to 85% of cases. Other causes may include a floor-of-mouth laceration, mandibular trauma, tumor, sialadenitis, or lymphadenitis. The most frequent bacterial isolate is the α-hemolytic *Streptococcus*. The distensible tissue in the floor of the mouth can be pushed superiorly, displacing the tongue posteriorly, and decreasing its range of motion (**Fig. 1**A). On examination, the suprahyoid neck is often tender with woody induration with rare fluctuance (see **Fig. 1**B). Computed tomography (CT) may show a collection (**Fig. 2**). Infection may spread from the submandibular space to the lateral pharyngeal space by way of the buccopharyngeal gap created by the styloglossus muscle, further complicating the situation.[4]

Airway management in this group can be quite challenging. Standard direct laryngoscopy with a Mac or Miller blade will often fail because of the floor-of-mouth firmness and retro-displacement of the tongue. Awake nasal fiberoptic intubation is usually the best option, with the patient sitting up and their head in the sniffing position. This position maximally opens the pharyngeal airway to allow for passage of the endotracheal tube (ETT). It allows assessment of the airway, which is helpful in planning for extubation. Wire-reinforced anode ETT are ideal for this purpose because of their flexibility; smaller sizes[6,7] are used because their outer diameter is slightly larger than the standard polyvinyl chloride ETTs. The nostrils are first decongested and anesthetized with a mixture of 0.05% oxymetazoline and 4% lidocaine on 1/2 $\times$ 3-inch Cottonoids followed by dilation using soft silicone nasal airways lubricated with 4% lidocaine jelly (**Figs. 3–6**). Pretreatment with intravenous dexmedetomidine makes this process considerably easier because it allows for sedation without significant suppression of the respiratory drive. Oxygen is usually delivered via a nasal cannula placed between the teeth during the intubation. The otolaryngologist should be prepared for tracheotomy if fiberoptic intubation is not successful (**Boxes 1** and **2**, **Table 1**).