Natural History of Odontogenic Infection

The usual cause of odontogenic infections is necrosis of the pulp of the tooth, which is followed by bacterial invasion through the pulp chamber and into the deeper tissues. Necrosis of the pulp is the result of deep caries in the tooth, to which the pulp responds with a typical inflammatory reaction. Vasodilation and edema cause pressure in the tooth and severe pain as the rigid walls of the tooth prevent swelling. If left untreated the pressure leads to strangulation of the blood supply to the tooth through the apex and consequent necrosis. The necrotic pulp then provides a perfect setting for bacterial invasion into the bone tissue. Once the bacteria have invaded the bone, the infection spreads equally in all directions until a cortical plate is encountered. During the time of intrabony spread, the patient usually experiences sufficient pain to seek treatment. Extraction of the tooth (or removal of the necrotic pulp by an endodontic procedure) results in resolution of the infection.

Direction of Spread of Infection

The direction of the infection's spread from the tooth apex depends on the thickness of the overlying bone and the relationship of the bone's perforation site to the muscle attachments of the jaws.
If no treatment is provided for it, the infection erodes through the thinnest, nearest cortical plate of bone and into the overlying soft tissue. If the root apex is centrally located, the infection erodes through the thinnest bone first. In the maxilla the thinner bone is the labial-buccal side; the palatal cortex is thicker.

Once the bone has been perforated, local muscle attachments determine the specific location of its expression in the soft tissue. The most common "tooth abscess" erodes through the labial bone, occlusal to the muscle attachment, producing a vestibula abscess. The vestibular abscess is seen as a small pouch of pus in the soft tissue overlying the affected tooth . If no treatment is provided, rupture of the abscess occurs and a chronic sinus tract is established.

In the infection perforates the bone above the muscle attachment, fascial space involvement occurs. When space involvement happens, the potential for more severe infections with rapid spread becomes greater.

Fascial Space Involvement

Maxillary spaces

Erosion of maxillary tooth infection through the bone usually expresses itself in the labial-buccal surface of the maxilla. Most are seen as vestibular abscesses. Some, however, become fascial space infections. The two maxillary spaces that may be involved are the canine space and the buccal space.

The canine space becomes inected almost exclusively as a result of the maxillary canine tooth. The root of the tooth must be long enough so that the apex is superior to the insertion of the levator anguli oris muscle. The canine space is between the anterior surace of the maxilla and the levator labii

superioris. When infected, clinically evident swelling lateral to the nose exists, usually obliterating the nasolabial fold.

The buccal space becomes involved from the maxillary teeth when the infection erodes through the bone superior to the attachment of the buccinator muscle. The buccal space lies between the buccinator muscle and the skin and superficial fascia. All three maxillary molars may cause buccal space involvement, but the premolars rarely do. The buccal space swelling is ovoid, below the zygomatic arch and above the inferior border of the mandible. The buccal space may also be infected from the mandibular molar teeth. This involvement is not common, but it does occur.

In addition to these two maxillary space involvements, maxillary odontogenic infections may ascend to cause orbital cellulitis or cavernous sinus thrombosis. Orbita cellulitis is rarely the result of odontogenic infection but may occur. The clinical picture is similar regardless of the cause. Swelling and redness of the eyelids, chemosis, and exophthalmos occur. Involvement of orbital contents includes both vascular and neural components.

Cavernous sinus thrombosis may also occur as a result of the superior spread of an odontogenic infection. Spread to the cavernous sinus is hematogenous and may occur along an anterior or a posterior route. The orbital veins lack valves, permitting blood flow in either direction. This allows contaminated venous drainage to the cavernous sinus. The usual cause of cavernous sinus thrombosis is from nonodontogenic sources. On rare occasions it may be the result of an infected tooth.

Mandibular spaces

Infection may erode from mandibular teeth into a variety of spaces, in addition to the usual vestibular abscess. The three primary spaces are the submental, sublingual, and submandibular spaces; the three secondary spaces are the pterygomandibular, masseteric, and temporal spaces.

The primary spaces are those into which infection spreads directly from the teeth and bone.

The submental space lies between the anterior bellies of the digastric muscles and between the mylohyoid muscle and the skin. If the roots of the mandibular incisors are long enough to cause the infection to erode through apically to the attachment of the mentalit muscle, the infection may proceed under the inferior border of the mandible to the posterior aspect into the submental space. This is not, however, a common occurrence.

The sublingual and submandibular spaces exit on the medial aspect of the mandible. They are usually involved by lingual perforation of infection from the mandibular molars. The factor determining whether the infection is in the sublingual or submandibular space is the relationship between the area of the infection's perforation and the location of the mylohyoid muscle's attachment. If the location of the apex of the teeth is superior to that of the mylohyoid (premolars, first molar), the sublingual space is involved. If the apex of the tooth is inferior to the muscle (third molar), the submandibular space is involved. The second molar may involve either or both spaces, since its apex is typically at the mylohyoid line.

The subglingual space is between the oral mucosa and the mylohyoid muscle. Its posterior boundary is open, and it can thus communicate freely with the submandibular space. Clinically, when infection of the sublingual space occurs, little extraoral swelling occurs, but much intraoral swelling of the floor of the mouth develops on the affected side. If the infection becomes bilateral, the tongue may become markedly elevated.

The submandibular space lies between the mylohyoid muscle and the skin and superficial fascia. Like the sublingual space, it has an open posterior boundary and can communicate freely with the secondary spaces. When this space becomes infected, the swelling begins at the inferior lateral border of the mandible and extends medially to the digastric area and posteriorly to the hyoid bone.

The three secondary spaces of the mandible are posterior to the tooth-bearing portmon of the mandible in the angle-ramus area. They are called *secondary spaces* because they become infected by secondary spread of infection from other anterior spaces. The primary spaces feeding them are the buccal, sublingual, and submandibular spaces.

The masseteric space exists between the lateral aspect of the mandible and the masseter muscle. This space is involved most often by spread from the buchal space or from soft tissue infection around the third molar. When it is involved, the posteroinferior portion of the face swells. In addition to the swelling, the patient has mild to moderate trismus caused by inflammation of the masseter muscle.

The pterygomandibular space lies between the medial aspect of the mandible and the medial pterygoid muscle. This space becomes involved from spread from the sublingual and submandibular spaces and from soft tissue infection around the third molar. When this space is involved, little or no swelling is evident on either intraoral or extraoral examination. The patient almost always has significant trismus. Thus trismus without swelling is a valuable diagnostic clue for pterygomandibular space infection.

The temporal space is posterior and superior to the masseteric and pterygomandibular spaces. Bounded laterally by the temporalis fascia and medially by the skull, it is divided into two portions by the temporalis muscle. The two sections are known as the *deep* and *superficial temporal pouches.* Swelling is evident over the temporal area, posterior from the laterál aspect of the lateral orbital rim. Trismus is always a feature of this infection, caused by involvement of the temporalis muscle.

These three spaces are collectively known as the *masticator space,* since they are bounded by the muscles of mastication: masseter, medial pterygoid, and temporalis. The three individual spaces communicate freely with one another, so one rarely sees any single space involved alone. Thus the term *massticator space* does have some clinical usefulness, even if it lacks specific designation.

If all three of the primary mandibular spaces become involved with the infection, the infection is known as *Ludwig's angina.* Ludwig's angina, described in 1936, was a relatively common occurrence until the antibiotic era. It is a rapid, bilaterally spreading, gangrenous cellulitis of the submandibular, sublingual, and submental spaces. It usually spreads posteriorly to the secondary spaces as well. It produces gross swelling, elevation and displacement of the tongue, and tense, brawny induration of the

submandibular region superior to the hyoid bone. There is usually little or no fluctuance. The patient experiences severe trismus, drooling of saliva, tachypnea, and dyspnea. Impending compromise of the airway produces marked anxiety. The cellulitis can progress with alarming speed, producing an upper airway obstruction that may lead to death. The usual cause of Ludwig's angina is an odontogenic infection, usually from the mandibular second or third molar.

Cervical (deep neck) spaces

The deep neck spaces can become infected from a variety of sources. Odontogenic infections cause as much as 30% of all deep neck infections. The deep neck spaces have a variety of names and descriptions. Three are relatively consistent through the literature: the lateral pharyngeal space, the retropharyngeal space, and the prevertebral space, or danger space. The layers of deep cervical fascia form and bind these three spaces.

The lateral pharyngeal space is classically described as having the shape of an inverted pyramid or funnel. The base is the skull base at the sphenoid bone, and the apex is at the hyoid bone. It is located between the medial pterygoid muscle laterally and the superior pharyngeal constrictor medially. Anteriorly the boundary is the pterygomandibular raphe, around which it communicates with the spaces of the mandible. Posteromedially it extends to and is bounded by the prevertebral fascia and communicates freely with the retropharyngeal space. The styloid process and associated muscles and fascia divide the lateral pharyngeal space into an anterior compartment, which contains muscles, and a posterior compartment, which contains the carotid sheath and cranial nerves. When the lateral pharyngeal space is involved in an odontogenic infection, there are several typical findings. First and foremost is severe trismus. This is the result of involvement of the medial pterygoid muscle but may be caused by involvement of the other muscles of mastication as well. The severe trismus may interfere with accurate diagnosis and treatment. Lateral neck swelling, especially beyond the angle of the mandible, is usually seen. The lateral pharyngeal wall, if it can be visualized, usually bulges toward the midline. One can differentiate it from a primary peritonsillar abscess primarily because the latter rarely has significant trismus. Involvement of the lateral pharyngeal space creates complications. First is the fact that the odontogenic infection is severe and may be progressing at a rapid rate. Second is the direct effect of the infection on the contents of the space, particularly of the posterior compartment. This includes thrombosis of the internal jugular vein, erosion of the karotid artery or its branches, and interference with cranial nerves IX to XII or the sympathetic chain. Third is that the infection may progress from the lateral pharyngeal space to the retropharyngeal space.

The retropharyngeal space lies posteromedial to the lateral pharyngeal space. It is bounded anteriorly by the superior pharyngeal muscle and its investing fascia and posteriorly to the alar layer of prevertebral fascia. The space begins at the skull base at the pharyngeal tubercle and extends inferiorly to the level of C7 or T1, where the two layers of fascia fuse. This level is at the posterosuperior mediastinum. The retropharyngeal space has few contents, save for the retropharyngeal lymph nodes.

These nodes are more numerous in the child than in the adult, which may account for the more frequent involvement of this space in children. When the retropharyngeal space becomes involved secondary to odontogenic infection, the situation is almost always grave. Clinical signs and symptoms are those of a severe infection. Trismus is severe in essentially all patients at this stage. Evaluation of the retropharyngeal space is performed with the greatest success by evaluating a lateral radiograph of the neck. Involvement of the retropharyngeal space may also include the prevertebral space.

The prevertebral space is a potential space between the two layers of prevertebral fascia, the alar and prevertebral layers. It extends from the skull base inferiorly to the diaphragm. The space is also known as the *danger spare*. When the retropharyngeal space is involved as a result of an odontogenic infection, the patient is seriously ill and is in grave danger of death. Three great potential complications exist. First, the upper airway is in danger of obstruction as a result of anterior displacement of the posterior pharyngeal wall into the oropharynx. Narrowing of the upper airway as the retropharyngeal space swells is expected. Second, when the retropharyngeal spaces are filled with pus, a danger exists of spontaneous rupture of the abscess, resulting in aspiration, pneumonia, and asphyxiation. Rupture may also be caused by attempts at insertion of an endotracheal tube to secure the airway. Third, once the infection has gained access to the retropharyngeal spaces, the posterosuperior mediastinum or the entire posterior mediastinum may become infected also