**Odontogenic infections of the mouth and face**

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**PERIAPICAL (DENTOALVEOLAR) ABSCESS:**

Usually a non-vital pulp produces no more consequence than an asymptomatic and sterile periapical granuloma. Untreated, these may produce intermittent pain in periods of acute inflammation and persist as chronic inflammation with periods of exacerbation of symptoms. However, infection may eventually develop into an acute periapical infection with abscess formation.

 In an abscess, bacteria cause localised tissue necrosis, and pus forms by the action of neutrophil proteolytic enzymes. The process is localised by granulation tissue forming the abscess wall. The surrounding soft tissues become oedematous with inflammatory exudate. Once an apical abscess is established, it is unlikely to resolve spontaneously.

**Pathology:**

Apical abscesses are polymicrobial infections and frequent cultivable isolates include *Veillonella*, *Porphyromonas*, *Streptococcus*, *Fusobacterium* and *Actinomyces* species. Despite the mixed nature of the infection, penicillins remain the most effective antibiotics, with metronidazole reserved for those allergic to penicillin. However, an apical abscess cannot be treated by antibiotics alone; the causative tooth

or its pulp must be dealt with because bacteria in the pulp chamber are inaccessible to the drug. Local dental treatment is usually effective without the addition of antibiotics.

 **‘FASCIAL’ OR TISSUE SPACE INFECTIONS**

When pus from an apical abscess or pericoronitis breaks out into soft tissue, its path is guided by muscle attachments and fascia. These can divert the path of drainage away from the mouth into the tissues of the face, where pus and spreading infection can localise in the ‘fascial spaces Anatomical descriptions of these spaces imply that fascia is a well-organised fibrous sheet dividing the face and neck into defined compartments and spaces. In reality, there are no spaces, but the inflammation and infection tend to localize reproducibly in tissue planes bounded by subcutaneous tissue, the masticatory muscles and muscles of the neck and the carotid sheath.

The fascial spaces are only potential spaces enlarged by accumulation of exudate or pus. Because the spaces have a large volume, pressure in the

exudate is reduced, and it tends to accumulate rather than burrow onward to the surface. When the space is distended, its blood supply is disrupted, and the environment becomes avascular and anaerobic, favouring infection and inhibiting host defences.

The fascial spaces extend from the base of the skull to the mediastinum, and the inflammatory exudate acts as a vehicle to spread the infection into potentially life-threatening sites. The large volume of exudate and bacterial load produce pyrexia, toxaemia and symptoms of pain and trismus.

**Sign and symptoms:**

* Severe pain.

 • Swelling.

 • Discharge.

• Swinging pyrexia.

• Tachycardia.

• raised white cell count.

Later:

• drooling;

• difficulty breathing or speaking;

• severe trismus;

• stridor (inspiratory wheeze

• Death by sepsis/ respiratory arrest/ intracranial or intrathoracic complications.

**FACIAL CELLULITIS**

The great majority of fascial space infections are in the form of cellulitis in which, unlike a localised abscess, bacteria spread through the soft tissues .Cellulitis causes gross inflammatory exudate and tissue oedema, associated with fever and toxaemia. The characteristic features are diffuse swelling, pain, fever and malaise. The swelling is tense and tender, with a characteristic board-like firmness. The overlying skin is taut and shiny. Pain and oedema limit opening the mouth and often cause dysphagia. Systemic upset is severe with worsening fever, toxaemia and leucocytosis. The regional lymph nodes are swollen and tender.

**Pathology**

The bacterial flora is similar to the dentoalveolar abscess from which it is derived, but with a greater proportion of anaerobes such as *Porphyromonas*, *Prevotella* and *Fusobacterium* and anaerobic streptococcal species.

***Ludwig’s angina***

Ludwig’s angina is a severe form of cellulitis that usually arises from the lower second or third molars. It involves the sublingual and submandibular spaces bilaterally, almost simultaneously, and readily spreads into the lateral pharyngeal and pterygoid spaces and can extend into the mediastinum. The main features are rapidly spreading sublingual and submandibular cellulitis with painful, brawny swelling of the upper part of the neck and the floor of the mouth on both sides With involvement of the parapharyngeal space, the swelling tracks down the neck and oedema can quickly spread to the glottis Swallowing and opening the mouth become difficult, and the tongue may be pushed up against the soft palate. Oral obstruction or oedema of the glottis causes worsening respiratory obstruction. The patient soon becomes desperately ill, with fever, respiratory distress, headache and malaise.

***Management***

1-The principles of treatment for cellulitis are to provide immediate aggressive antibiotic treatment to prevent further spread of infection and to remove the causative tooth or deal with pericoronitis as soon as possible.

2-Drainage plays little role in treatment of pure cellulitis because there is no collection of pus. However, when there is potential compromise of the airway or a suggestion that pus may be localizing, then drains may be placed to relieve tissue tension. A microbiological sample can be obtained at the same time.

3-In Ludwig’s angina, or when the airway is compromised by any infection, the main requirements are immediate admission to hospital, securing the airway by tracheostomy if necessary.

**FACIAL ABSCESS**

Depending on the micro-organisms and effectiveness of host defences, pus from an apical abscess or pericoronitis may localise in the tissues (facsial space-tissue spaces) to form a discrete abscess rather than spreading. Systemic signs are less marked and inflammation and swelling less extensive in abscess than in cellulitis. Eventually an abscess will point to a surface and drain spontaneously, but this is best prevented by early intervention because formation of a sinus to the skin is usually followed by disfiguring scarring. When pus starts to collect in the tissues, the brawny diffuse swelling of oedema and cellulitis can still be present, but a localised zone of softening develops over the pus, with a darker red zone of inflammation. Pyrexia increases. If left too long before drainage, the overlying skin becomes fluctuant just before the abscess drains spontaneously.

**Management of abscess:**

The principles of management of abscess are the same as for cellulitis, except that early surgical drainage of pus is essential (incision & drainage/S with triple Abs.). Small abscesses may resolve with high-dose antibiotics alone, but better and more rapid resolution will follow surgical drainage in most cases.

**Tissue spaces commonly involved by dental infection**

1-*Submental space*

 between mylohyoid and the skin, platysma and investing layer of fascia. Contains the submental lymph nodes drained from Lower incisors.

2*-Submandibular space*

 Between mylohyoid, and the skin, platysma and investing layer of fascia and between the hyoglossus and body of mandible. It contains the submandibular gland and submandibular lymph nodes and communicates anteriorly with the submental and posteriorly and upward into the sublingual space drained from Lower canine, premolar and molar teeth, when their apices lie below the mylohyoid attachment.

3-*Sublingual space*

 Between hyoglossus and the tongue muscles medially and mylohyoid and the body of mandible laterally. Contains the sublingual gland. Communicates posteriorly with the submandibular space around the posterior free edge of the mylohyoid around the submandibular gland and duct the source of infection are Lower incisor and canine teeth. Molars less frequently when apices are above the mylohyoid attachment.

4-*Buccal space*

 Between buccinator muscle and the overlying skin, platysma and fascia. Posteriorly limited by ramus of mandible and masseter. Contains the buccal pad of fat. Communicates posteriorly with the pterygomandibular space the source of infection are Usually upper molar and premolar teeth, sometimes lower molars when their apices lie below the buccinators attachment.

5-*Canine fossa space*

 bounded by the muscles of lips and face, the source of infection Upper lateral incisors, canines or first premolars, including periodontal abscesses.

6-*Submasseteric space*

 Between the lateral surface of the ramus of the mandible and the periosteum with masseter muscle, the source of infection rarely involved, usually from pericoronitis around the lower third molar.

**ANTIBIOTIC ABSCESS**

The antibiotic abscess or ‘antibioma’ is an abscess that has been controlled but not eliminated by antibiotic treatment. This may arise after inadequate, often prolonged intermittent antibiotic treatment, particularly at insufficient dose. It may also arise from effective antibiotic treatment

Provided without ensuring that a collection of pus has been drained. The pus can be rendered sterile or nearly so, and the surrounding granulation tissue matures to dense scar tissue, producing a thick zone of fibrosis around the pus. The patient has a hard mass, with puckering of the skin

if superficially located, and either mild symptoms of intermittent pain and swelling or no symptoms at all. Treatment may be conservative, but resolution takes many months, and it is usually better to excise the whole mass. Drainage alone removes any residual infection, but the main signs

arise from the fibrosis. Antibioma is commoner in countries where antibiotics are available without prescription.

**NECROTISING FASCIITIS**

Necrotising fasciitis is an uncommon, rapidly spreading, potentially lethal infection by a poly microbial such as Staph,/Strep/bacteroides& clostridia that causing necrosis and rapid dissolution of subcutaneous tissues and fascia with loss of attachment of the overlying skin. Muscles are relatively spared. Rarely, the infection can have a dental source and may threaten the airway. The treatment is wide surgical debridement & I.V abs.