ORAL SURGERY

LECTURE

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Odontogenic and Fascial Spaces Infections

Infection: is the invasion and the multiplication of pathogenic microorganisms into a bodily part or tissue which may produce subsequent tissue injury and progress to overt disease through a variety of cellular or toxic mechanisms.

The pathogenic organisms can be Bacteria, Viruses, Fungi or Protozoan.

The body's response to infectious agents is inflammatory response which is essentially protective, it consists of:

a. Hyperemia caused by vasodilatation of the arterioles and capillaries, and the release of permeability factors to allow for the passage of nutrients and leukocytes.
b. Precipitation of a network of fibrin to wall off the infected region.
c. Phagocytosis of bacteria and other organisms as well as dead cells by macrophages.

- Depending on the duration and severity of the infection, the inflammatory response is divided into:

  Acute inflammation; with rapid progression and typical signs and symptoms.
  Subacute inflammation; considered as transition between acute and chronic inflammation.
  Chronic inflammation; associated with longer duration and slight clinical symptoms. Infections could be local; confined to one body system, or generalized like Septicemia.

- **Signs and Symptoms of infection**

  These include systemic signs and symptoms like; fever, malaise and anorexia. There are also local signs and symptoms, which are present to some degree in all patients:

  a. **Rubor** (redness), it results from vasodilatation.
  b. **Tumor** (swelling), in infection it results from accumulation of tissue fluid or pus.
  c. **Calor** (heat), results from the inflow of warm blood from deeper tissues, increased velocity of blood and increased metabolic rate.
  d. **Dolor** (pain), results from pressure on the nerve endings, also from the action of liberated or activated factors such as Kinins, Histamine and Bradykinin on nerve endings.
  e. **Functio laesa** (loss of function), caused by mechanical factors and reflex inhibition of muscle movement associated with pain.

Odontogenic Infections

They are almost always bacterial in origin, they consist of Periapical, Periodontal or Pericoronal infections

1- **Periapical infections**: These infections arise when bacteria from infected necrotic pulp from a carious tooth or contaminated traumatic exposure of the pulp invade the periapical tissues through the apical foramen, sometimes the bacteria gain access through an accessory canal or through furcation area leading to infection that presents on the lateral surface of the tooth. These infections manifest themselves as a primary acute infection or as an exacerbation of a preexisting chronic periapical infection. The main clinical features include; a non-vital tooth, the tooth is tender to bite and to percussion, the periapical region is tender to pressure, the tooth is slightly raised in the socket due to the acute inflammation and swelling of the periodontal inflammation, the tooth may be mobile. There is an intense throbbing pain, if treatment is delayed bone resorption will allow the pus to drain under the periosteum with diminution of pain intensity. A periapical radiograph may show no significant changes except for widening of the periodontal space especially in acute infections or it may reveal the presence of periapical radiolucency.

2- **Periodontal infections:** it arises from preexisting periodontal pocket, in some cases food impaction or repeated occlusal trauma are the precipitating factors. The teeth involved are not necessarily non vital, an acute infection produces redness and swelling near the gingival margin, the pain is continuous, dull or throbbing but usually less than that experienced in acute periapical infections, pus may discharge from the gingival margin or may produce a sinus. The tooth may be mobile.

3- **Pericoronal infections:** Also called pericoronitis is an infection of the soft tissue covering the crown of a partially erupted tooth, it is almost always associated with partially erupted impacted lower third molars.

   Etiology involves:

   - Food impaction between the crown and the overlying gum (operculum) which is a favorable site for bacterial proliferation and inflammation.
   - Trauma to the overlying gum from the opposing tooth, but it is hard to determine whether the trauma occurs before or after the inflammatory edema of the gum.
   - Virulent microorganism.
   - Lowering of the host resistance.

   Pericoronitis appears to be the result of a combination of four mentioned etiological factors.

   The clinical presentation depends on the severity of infection:

   Chronic pericoronitis is either asymptomatic or there is mild discomfort. In Subacute pericoronitis there is a dull pain which is well localized, the gum pad may be tender and red and there may be expression of a white material from underneath the gum flap, this material is composed of desquamated epithelial cells, food stuff, dead and living bacteria, there may be slight trismus and lymphadenopathy (LAP). In Acute pericoronitis there is severe pain and limitation of mouth opening, intraoral swelling

and there may be extraoral swelling as well as fever, malaise and LAP. Pus may be expressed from underneath the flap, sometimes the pus may track submucosally and is expressed from a sinus in the molar or premolar region, this is termed migratory abscess.

In treating pericoronitis, it is essential to determine if the tooth is likely to achieve full eruption, this is done by full clinical and radiographic examination, consideration should be given to the age of the patient and history of previous attacks.

Treatment of pericoronitis consists of:

- Drainage of abscess if present.
- If there is no abscess, gentle irrigation of the pericoronal space with normal saline, sometimes an antiseptic solution can be instilled in this space.
- If the upper third molar irritates the swollen gum flap, the pressure should be relieved by extraction or grinding the offending cusps.
- Antibiotics are needed for cases of acute infections.
- Surgical extraction of the impacted teeth should be carried out after the inflammation subsides.

Bacteriology

Odontogenic infections are caused mostly by bacteria that live on or in the host, when such bacteria gain access to the deeper tissues, they cause infection. Most odontogenic infections are mixed infections, caused by multiple bacteria (aerobic and anaerobic), with an average of five species involved. The most common aerobic bacteria involved in odontogenic infections are Streptococci and Staphylococci (Aureus and epidermidis), other less common aerobic organisms include Neisseria species, Corynebacterium species and Haemophilus Influenzae. While the most common anaerobic bacteria are anaerobic Streptococci species, Peptostreptococcus, Bacteroids and Fusobacterium species.

Presentation of the odontogenic infection

Odontogenic infections may spread beyond the confines of the dentoalveolar bone into the soft tissue, they may be presented as:

Cellulitis; which results from spreading of infection into the loose connective tissues (C.T.), it presents as a warm, diffuse, erythematous, indurated and painful mucosal or cutaneous swelling. It does not result in the formation of large amount of pus, Streptococci are more often associated with cellulitis, these organisms produce enzymes such as Streptokinase and Hyaluronidase that break down fibrin and C.T. ground substance, facilitating the rapid spread of infection. Antibiotics and removal of the cause of infection are usually sufficient. Incision and drainage are indicated if there is no improvement or if evidence of purulent collection is identified.

Suppurative infections; characterized by abscess formation, which can be defined as a thick-walled pocket of tissue containing pus. Pus consists of necrotic tissue, dead and

living bacteria and dead white cells, it is often associated with Staphylococci and anaerobes such as Bacteroides.
S. aureus produce the enzyme Coagulase that coats the bacteria with fibrin and reduces the ability of the host cells to phagocytize it. The area of infection may or may not be fluctuant, treatment is by incision and drainage with antibiotics, followed by the treatment of the cause of infection.

Routes of Spread of infection: it can occur through the following routes
1. By direct continuity through the tissue.
2. By the lymphatics to the regional lymph nodes which may result in secondary infection. In acute infections the lymph nodes are enlarged, soft and tender, the surrounding skin may be red and edematous. In chronic infections the enlarged nodes are firmer, not tender with no redness or edema of the surrounding skin.
3. By blood stream, this is uncommon, it occurs along the veins, it can result in serious complications like cavernous sinus thrombosis and septicemia.

The factors that influence the spread of odontogenic infections:
1. Virulence of the organism, it is the disease producing ability of the organisms.
2. Status of patient's immune system, several conditions affect the general health of the patient adversely such as diabetes, malnutrition, alcoholism, HIV infections or patients taking immunosuppressive drugs.
3. Anatomical factors influencing the direction of spread of infection such as:
- The site of the source of infection.
- The point at which the pus escapes.
- The natural barriers to the spread of pus such as fascia, muscles or bone.

Physical examination:
Clinical examination: It includes inspection, palpation and percussion and it aims to identify the signs and symptoms of infection like the presence of swelling, redness or draining fistula. Palpation is used to examine the size, note the tenderness, assess local temperature and to determine the presence of fluctuation. Trismus should be noted with measurement of the interincisal distance.
Intraorally, the teeth and gingiva should be examined for the presence of caries, restorations or localized swelling. Percussion determines the areas of tenderness. Pulp testing may be needed to assess the vitality of the teeth. The intraoral examination should include the ducts of the parotid and submandibular salivary glands, tongue, soft palate, tonsillar fossa and oropharynx. Manifestations of serious odontogenic infections may include; airway compromise, septicemia, fever, lethargy, fatigue, dehydration, rapid progression, dysphagia, odynophagia and drooling.
Radiological examination: radiographs to identify the cause of infection, periapical, occlusal and OPG radiographic views are used. Ultrasound, CT scan and MRI can also be used in deep infections.

Laboratory studies: are also indicated to evaluate the immune system, white blood cells (WBC) and differential WBC count.

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Infection of the fascial spaces

Fascial spaces are potential spaces, they are surrounded by muscles, loose C.T. and bone, they contain different anatomical structures and they are separated by collection of pus, blood or by surgeon's finger. They are contiguous and infection spreads readily from one space to another. A thorough knowledge of the anatomy of the face and neck is necessary to predict the pathways of spread of infections.

Fascial spaces are sometimes classified as **primary**; which are directly adjacent to the origin of the odontogenic infections, and **secondary**; that become involved following the spread of infection to the primary spaces.

Infection of spaces in relation to the lower jaw

1. **Submental space infection :**

   Anatomic boundaries: this space lies between the Mylohyoid muscle above, skin, subcutaneous tissue, Platysma muscle and deep cervical fascia below, laterally by lower border of the mandible and anterior bellies of Digastric muscle. It contains submental lymph nodes embedded in adipose tissues.

   Source of infection : direct source from infected lower incisors and canines, lower lip, skin overlying the chin or from the tip of the tongue and the anterior part of the floor of the mouth. An indirect source of infection from submandibular spaces. The site of the swelling is mostly extraoral including the chin and submental areas which are firmly swollen.

   The site of incision and drainage: is extraoral horizontal incision through the skin posterior to the crease behind the chin, providing dependent drainage and most esthetically acceptable scar. It may be drained intraorally through the Mentalis muscle via the labial vestibule, but the dependent drainage can not be established

2. **Submandibular space infection**

   Anatomic boundaries it is bounded by Mylohyoid muscle superiorly, anterior and posterior bellies of Digastric muscle inferiorly, hyoid bone posteriorly, Mylohyoid, Hyoglossus and Styloglossus muscles medially, laterally the space is bounded by the skin, superficial fascia, Platysma, deep fascia and the lower border of the mandible. This space contains the submandibular salivary gland and lymph nodes in addition to facial artery and vein, lingual and Hypoglossal nerve as they course deep to the submandibular salivary gland.

   Source of infection from the lower molar teeth especially second and third molars, as the infection perforates the lingual cortex of the mandible below the Mylohyoid muscle attachment. Infection can also spread from the tongue, posterior part of the

floor of the mouth, upper posterior teeth, cheek, palate, the maxillary sinus and the submandibular salivary gland. Indirectly the infection may spread from infected sublingual and submental spaces. Submandibular space infections can spread posteriorly to the pharyngeal space. Submandibular space infection presents as a firm or fluctuant erythematous swelling of the submandibular region, the swelling bulges over and obliterates the inferior border of the mandible, there may be trismus, other signs and symptoms of infection may or may not be present.

Site of incision and drainage it is extraoral incision made parallel and about 2 cm. below the inferior border of the mandible to avoid injury to the marginal mandibular branch of the facial nerve, the incision extends through the skin and subcutaneous tissue only while the space is entered bluntly to avoid structures within the space.

3. **Sublingual space infection:**

Anatomic boundaries this is a V-shaped space, it is bounded anteriorly and laterally by the mandible, superiorly by sublingual mucosa, inferiorly by the Mylohyoid muscle and medially by Genioglossus, Geniohyoid and Styloglossus muscles.

Source of infection it is usually from the premolar and less commonly from molar teeth when the infection perforates the lingual cortex of the mandible above the attachment of the Mylohyoid muscle. Indirectly the infection may spread from submental and submandibular spaces. Infection from sublingual space may invade the submandibular and pharyngeal spaces. Clinically there is erythematous swelling of the floor of the mouth that may extend through the midline since the barrier between the two sublingual spaces is weak, usually there is elevation of the tongue.

Site of incision and drainage intraorally by an incision through the mucosa only parallel to Wharton's duct and lingual cortex in anteroposterior direction and away from the sublingual fold. This space may be drained extraorally through submandibular and submental incisions through the Mylohyoid muscle if the infection of these latter spaces is also evident.

Ludwig's Angina

It is a massive firm cellulitis, affecting simultaneously the submandibular, submental and sublingual spaces bilaterally. It is a very serious conditions that require prompt treatment, it was described by Wilhelm Friedrich von Ludwig in 1836.

Causes

- Dental infections in 90% of the cases.
- Submandibular salivary gland infections.
- Mandibulr fractures.
- Soft tissue lacerations and wounds of the floor of the mouth. The term angina is related to the sensation of suffocation. If untreated this condition is almost fatal mainly due to posterior extension of the infection into the epiglottis causing epiglottic edema and respiratory obstruction.

Signs and Symptoms:

there is a firm extensive bilateral submandibular swelling, intraorally there is swelling of the floor of the mouth that raises the tongue which may protrude from the mouth in extreme cases. The patient is toxic, feverish and there is dyspnea and difficulty in swallowing.

Treatment:

- securing the airway, endotracheal intubation is very difficult in this situation, tracheostomy may be needed, but it is also difficult to perform due to the massive neck edema.
- General anesthesia should be avoided.
- Early surgical drainage of all the infected spaces bilaterally under local anesthesia, little pus is obtained since the infection is usually cellulitis.
- Intravenous antibiotic, using a combination of Penicillin and Metronidazole. Some authorities advocate high dose of antibiotics without surgery until fluctuation develops.

4. **Buccal space infection**

   Anatomic boundaries bounded by the Buccinator muscle and buccopharyngeal fascia medially, skin of the cheek laterally, labial musculature anteriorly, zygomatic arch superiorly, the inferior border of the mandible inferiorly and the pterygomandibular raphe posteriorly. It contains the buccal pad of fat, facial artery and the parotid duct.

   Source of infection of this space can be related to both jaws. The relationship of the origin of the Buccinator muscle from the alveolar bone and the apices of the upper and lower premolars and molars determines the direction of the spread of infection from these teeth. If the infection exits the alveolar bone above the attachment of the muscle in the upper alveolus or below the attachment in the lower alveolus, the infection spreads to the buccal space. Otherwise the infection spreads intraorally into the vestibule where it can be drained easily. Usually the swelling appears in the cheek, the inferior border of the mandible can still be palpated.

   Site of incision and drainage intraorally by a horizontal incision in the buccal mucosa below the parotid duct, the incision should be through the mucosa only, the space should be entered bluntly using artery or sinus forceps through the Buccinator muscle to avoid damage to the facial artery and nerve. The incision can be placed extra orally if the pus points cutaneously.

5. **Masticator spaces infection** These are well differentiated spaces but they communicate with each other as well as with the buccal, submandibular and pharyngeal spaces.

   They are:

   - Masseteric space.

- Pterygomandibular space.
- Temporal space.

Masseteric space infection (also called sub masseteric space)

Anatomic boundaries this space lies between the outer surface of the ascending ramus of the mandible medially, the Masseter muscle laterally and the parotid gland posteriorly.
Source of infection usually from molar teeth especially lower third molars, it can also occur after fracture of the angle of the mandible or it can also spread from buccal space. The swelling is moderate in size over the ascending ramus and the angle of the mandible region. This infection is characterized by a marked trismus. Chronic abscess can run a protracted course and can spread to the muscle itself or it can cause osteomyelitis of the ramus of the mandible.
Site of incision and drainage extra orally below and behind the angle of the mandible, the incision is carried through the skin and the subcutaneous tissue then by blunt dissection through the Platysma muscle and the deep fascia, after incising the attachment of the muscle at the angle the periosteal elevator is inserted beneath the muscle and in close contact with the outer surface of the ramus of the mandible to drain all the pus.
Intraorally drainage can be carried out through an incision along the anterior border of the ramus of the mandible, but in this case the drainage can be insufficient as it is not in a dependent point, also intraoral drainage may prove to be very difficult due to the presence of trismus.

Pterygomandibular space infection
anatomic boundaries it is bounded medially by the Medial Pterygoid muscle, laterally by the medial surface of the ramus of the mandible, Lateral Pterygoid muscle superiorly, parotid gland posteriorly and the pterygomandibular raphe and the Superior Constrictor muscle of the pharynx anteriorly.
Source of infection usually from molar teeth especially lower third molars, it can also result after inferior dental nerve block with contaminated needle or solution. Infection can spread from submandibular, sublingual and infratemporal spaces. Swelling is minimal near the angle of the mandible or sometimes there is no swelling at all, but there is a marked trismus.
Site of incision and drainage extraorally, the same as that described in the masseteric space infections but directed to the inner surface of the ramus. Intraorally can be drained through an incision made just medial to the pterygomandibular raphe and dissecting along the inner surface of the ramus., but the presence of trismus can prevent efficient drainage.

Temporal space infection

Anatomic boundaries the Temporalis muscle divides this space into two spaces:

- Superficial temporal space; between the muscle and temporal fascia.
- Deep temporal space; between the muscle and the temporal bone.

The temporal space is contiguous with the pterygomandibular and masseteric spaces.

Source of infection upper and lower molars, or by extension from the other masticator spaces. The swelling is behind the lateral orbital rim and above the zygomatic arch, it is almost always associated with trismus.

Site of incision and drainage extraoral, through an incision superior and parallel to the zygomatic arch between the lateral orbital rim and the hair line. Intraorally this space can also be drained through an incision along the anterior border of the ascending ramus with the artery forceps directed upwards on the outer aspect of the ramus, but the presence of trismus makes this approach difficult.

Lateral pharyngeal space infection (parapharyngeal space)

Anatomic boundaries this space extends from the base of skull to the hyoid bone, it is conical in shape, the lateral boundaries include the medial surface of the Medial Pterygoid muscle, the medial wall is the Superior Constrictor muscle, Styloglossus muscle, Stylopharyngeus muscle and the Middle Constrictor muscle of the pharynx. Posteriorly by the parotid gland and anterirorly by pterygomandibular raphe. This space can be divided into two compartments; anterior and posterior, the latter contains the carotid sheath.

Source of infection spread of infection from upper and lower molar teeth, most commonly from lower third molar infections by the way of submandibular, sublingual and pterygomandibular spaces. A non-odontogenic infection can spread to this space like tonsillar infections. Infections of this space are serious, the patient exhibits pain, fever, chills, medial bulge of the lateral pharyngeal wall, extraoral swelling below the angle of the mandible and trismus. It may lead to respiratory obstruction, septic thrombosis of the internal jugular vein and carotid artery hemorrhage.

Site of incision and drainage intraoral incision medial to the pterygomandibular raphe with the dissection medial to the Medial Pterygoid muscle. Extraoral incision at the level of the hyoid bone anterior to the Sternocleidomastoid muscle (SCM) and the dissection continued superiorly and medially between the submandibular gland and the posterior belly of Digastric muscle. Through and through drainage can also be applied.

Retropharyngeal space infection

Anatomic boundaries extend from the base of the skull to the upper mediastinum (C6- T1), it is bounded anteriorly by posterior wall of the pharynx and posteriorly by the Alar fascia.

Source of infection upper and lower molar teeth by lateral pharyngeal space by the way of pterygomandibular, submandibular, sublingual spaces. It can also result from nasal and pharyngeal infections. The swelling causes bulge of the posterior pharyngeal wall, there is dysphagia, dyspnea, and fever. Lateral neck radiograph may reveal widening of the retropharyngeal space.
Site of incision and drainage extraorally by an incision anterior to the SCM below the hyoid bone, SCM and the carotid sheath are retracted laterally and blunt dissection is carried out deeply to enter the space. Some authors advocated intraoral drainage by an incision along the posterior pharyngeal wall in extreme Trendlenburg position and suction. Most anesthesiologists prefer tracheostomy to secure the airway.

Peritonsillar abscess (Quinsy)
Anatomical boundaries it is localized between the C.T. bed of the faucial tonsil and the Superior Constrictor muscle of the pharynx.
Source of infection it arises from tonsillitis, but it is occasionally a complication of pericoronitis of the lower third molar. It causes swelling of the anterior pillar of the fauces and a bulge of the soft palate of the affected side which may reach the midline and push the uvula. Also there is acute pain, dysphagia, the voice becomes muffled, odynophagia, drooling and anorexia.
Site of incision and drainage the incision is placed in the point of maximum fluctuation, this can be done under local anesthesia, if general anesthesia is used the anesthetist should be experienced and good suction be available to prevent aspiration and the patient should be in head down position.

Infections of spaces in relation to the upper jaw

1. **Upper lip infection**
   Infections of the upper incisors and canines can spread to the upper lip usually on the oral side of Orbicularis Oris muscle and points in the vestibule. Infection of the upper lip can lead to serious complications like orbital cellulitis or cavernous sinus thrombosis by extension of infection through the superior labial vein to anterior facial vein to ophthalmic vein to cavernous sinus. Incision for drainage is made near the vestibule intraorally.
2. **Canine fossa infections**
   Anatomic boundaries it lies between the canine fossa and the muscles of the facial expression.
   Source of infection mostly is the canine and first premolar but the infection can spread from upper incisor teeth. Infection occurs when it spreads in the area above the origin of the Levator Anguli Oris and is directed toward the medial edge of the Levator Labii Superioris. The swelling is lateral to the nose leading to obliteration of the nasolabial fold and may lead to periorbital cellulitis, there is risk of cavernous sinus thrombosis.

Site of incision and drainage intraoral horizontal incision in the buccal vestibule.

3. **Buccal space infections**
   Infections spread from infected upper molar teeth where it spreads buccally above the attachment of Buccinator muscle. This space is already discussed
4. **Subperiosteal abscess in the palate**
   This potential space lies between the palatal mucoperiosteum and the underlying bone, the mucoperiosteum is strongly attached in the midline and at the gingival margin, pus may accumulate beneath the mucoperiosteum leading to its separation from the underlying bone.
   Source of infection it may spread from the apex of the lateral incisor which is close to the palatal bone. Also, infection can spread from the palatal root of multirooted upper molars. It can also originate from palatal periodontal pocket. the swelling causes palatal bulge between the gingival margin and the midline, confined to one side.
   Site of incision and drainage anteroposterior incision parallel to greater palatine vessels.

5. **Maxillary antrum Infection**
   Source of infection from upper molars and less frequently premolars may spread to the maxillary antrum, this depends on the size of the maxillary antrum and the length of the root.
   Signs and symptoms: It causes acute sinusitis with facial pain that worsens on bending or leaning forward, the infection may lead a chronic course leading to mucosal thickening and polyps. Occipitomental radiograph shows opaque maxillary sinus or fluid level.
   Site of drainage Pus may drain partially through the sinus osteum, extraction of the causative tooth leads to drainage of pus but it may leave a defect in the floor of the sinus and cause oroantral fistula. If the defect is small and with antibiotic treatment the socket may heal uneventfully, but larger defects may require further management.

6. **Infratemporal space infection**
   Anatomic boundaries this space is bounded laterally by the ramus of the mandible and the Temporalis muscle, medially by lateral pterygoid plate, superiorly by infratemporal surface of the greater wing of the sphenoid. It is traversed by the maxillary artery and contains pterygoid venous plexus. It represents the upper extremity of the pterygomandibular space.
   Source of infection directly from upper molar teeth or through contaminated needle from the pterygomandibular space. Infection may spread to the temporal space. There could be moderate swelling in the temporal region with trismus, usually the patient is toxic with high temperature. These infections are serious since they can spread through the pterygoid venous plexus to the cavernous sinus through

emissary vein or it can spread to the middle cranial fossa with headache, photophobia, irritability, vomiting and drowsiness.

Site of incision and drainage intraorally through an incision buccal to the upper third molar following the medial surface of the coronoid upward and backward, but with the presence of trismus this approach is difficult. Extra orally through an incision in the upper and posterior edges of the Temporalis muscle within the hair line passing downward, forward and medially. Infection related to the maxillary teeth can spread to the masticator spaces and pharyngeal spaces and these were already discussed.

Cavernous sinus thrombosis

It is a very serious ascending infection, although not a fascial space infection but it can be caused by odontogenic infections especially of upper teeth. It can also result from upper lip, nasal and orbital infections.

Infection can spread to the cavernous sinus through two routes:

- Anterior route; through the valve-less angular vein and inferior ophthalmic vein.
- Posterior route; through the pterygoid venous plexus and transverse facial vein.

This infection has a high mortality rate.

Clinical features:

- Marked edema and congestion of the eyelids and conjunctiva which can be bilateral due to the spread of infection to the other side.
- Proptosis (exophthalmos) and ptosis.
- Ophthalmoplegia and dilated pupil.
- Papilloedema with multiple retinal hemorrhage.
- Fever.
- Depressed level of consciousness.

Treatment:

It is an emergency that requires a neurosurgical consultation, the lines of treatment include:

- Antibiotic treatment
- Heparinization to prevent extension of thrombosis.
- Treatment of the odontogenic cause.

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Principles of treatment of odontogenic infections

In treating odontogenic infections, the clinicians need to identify the presence of infection through the presence of the local and systemic signs and symptoms of the infection and whether it is cellulitis or abscess, also to determine the state of the host defenses, as these can be depressed by many factors such as; physiologic factors, disease related factors, immune-system related factors and drug suppression related factors.

Treatment of odontogenic infections requires medical, surgical or dental therapy or a combination.

Dental treatment; aims to eliminate the source of infection through endodontic treatment, periodontal treatment or extraction of the offending tooth. These factors should be taken in consideration:

- The extent of infection.
- Patient general health status.
- Degree of trismus.
- Biomechanical necessity of retaining the tooth.

Surgical treatment; aims to drain the accumulated pus to rid the body of the toxic purulent material and to relieve and decompress the tissues allowing better perfusion of blood to the infected area.

Methods of drainage

1. Through the root canal after access opening.
2. Through the socket by extraction of the offending tooth.
3. Through fenestration of alveolar bone using surgical handpiece and bur, made at the level of the root apex, to drain periapical abscess, after reflection of a semilunar mucoperiosteal flap.
4. Through incision and drainage of an abscess.

Incision and drainage

Is one of the oldest surgical procedures, it requires a thorough knowledge of facial and neck anatomy which is necessary particularly in draining deep tissue abscesses, on the other hand drainage of vestibular and dentoalveolar abscess is easily carried out.

Indications of incision and drainage:

1- When there are signs of accumulation of pus .
2- When the involved compartment is inaccessible; like pterygomandibular or pharyngeal spaces ,
3- Where is no improvement with adequate doses of antibiotic
4- Recurrence of pyrexia or a sudden increase in temperature and severe trismus .
5- Serious and rapidly evolving infections of the neck and floor of the mouth, like Ludwig's angina.

Principles of incision and drainage:

1. Incise in healthy skin and mucosa as possible,
2. incisions should be placed in an area of maximum fluctuation in a dependent position to encourage drainage by gravity.
3. Incisions made in areas where the tissue is necrotic or beginning to perforate may leave unaesthetic scars.
4. It should be placed in an esthetically acceptable area, in a natural skin crease or fold.
5. Incision in the skin of face and neck should include only the skin and subcutaneous tissues and the dissection through deeper tissues is continued bluntly using closed sinus or artery forceps which are advanced by controlled pressure to the pus containing tissue space and opened inside the cavity, then the forceps are drawn open then closed again and reinserted. The process is repeated until all the pus is evacuated. This is termed Hilton's method (after john Hilton, an English surgeon 1804-1878).

The forceps should not be closed inside the tissue to avoid damage to vital structures. Intraorally, in vestibular abscess, the pus accumulates under the mucosa with no intervening vital structures, so incision and drainage is made by the scalpel through the abscess cavity, here scalpel blade no. 11 is preferably used. Generally, all portions of the abscess cavity should be explored to ensure evacuation of all compartments, sometimes through and through drainage is necessary.

6. After incision and evacuation of pus a drain is inserted into the abscess cavity and is stabilized with suture. Corrugated drains or Iodoform ribbon gauze can be used for this purpose. Drains should not be left for long periods; they should be removed when the drainage is minimal.
7. Wound margins should be cleaned daily to remove clots and debris.

Note: Needless to say, that a sample of pus should be obtained and sent to the laboratory for culture and sensitivity, sometimes this can be achieved by aspiration with a syringe and needle prior to incision and drainage.

Medical treatment; it consists of supportive care which include hydration, soft diet, analgesics and good oral hygiene, and antibiotic therapy. It is essential to say that medical treatment is not a substitute for surgical treatment if indicated. In treatment of odontogenic infections,antibiotics are indicated therapeutically in the following cases:

1. Acute cellulitis.
2. Acute pericoronitis with elevated temperature and trismus.
3. Deep fascial space infections.
4. Dental infections in the compromised host.

Principles for the use of appropriate antibiotics

Once the decision has been made to use antibiotics as an adjunct to treat an infection, antibiotics should be properly selected, the following guidelines are useful:

1. Identification of the causative organisms, this is determined either in the laboratory where the organism can be isolated from pus, or empirically based on the knowledge of the pathogenesis and the clinical presentation of specific infections.
2. Determination of the antibiotic sensitivity.
3. The use of specific, narrow spectrum antibiotic, for example; if the causative organism is Streptococcus, sensitive to Penicillin, Cephalosporin and Tetracycline, then Penicillin should be selected because it has the narrowest spectrum. The main advantages of using narrow spectrum antibiotic are; less opportunity to develop resistance and minimize the risk of superinfection.
4. The use of least toxic antibiotic.
5. Patient's drug history should be known, especially drug allergy and various drug reactions.
6. It is imperative to know that the combat of the infection is the result of the host defense mechanism and that antibiotics are used as an adjunct to this process. Bacteriostatic antibiotics inhibit the growth and the reproduction of the bacteria usually by inhibiting protein synthesis, whereas bactericidal antibiotics kill the microorganisms by interfering with cell wall synthesis or nucleic acid synthesis. The main advantages of bactericidal antibiotics are:
   - Less reliance on the host resistance.
   - Kill the bacteria.
   - Work faster.
   - Greater flexibility with dosage interval.
7. The use of antibiotic with proven history of success.
8. Cost of the antibiotic should be taken into consideration.
9. Antibiotics should be administered in proper dose and proper dose interval.

10. Proper dose of administration either; oral (most commonly used), intramuscular or intravenous.
11. The antibiotic administration should be continued for an adequate length of time.
12. A combination of antibiotic therapy is indicated in:

- When it is necessary to increase the antibiotic spectrum.
- To increase bactericidal effect against specific organisms.
- To prevent the rapid emergence of resistant bacteria.
- Severe, rapidly progressing infections.

Most odontogenic infections respond to a combination of Penicillin and Metronidazole. Monitoring the patient in the follow up appointments the clinicians should look for the following:

- Response to treatment.
- Recurrence of infection.
- Presence of allergic reaction.
- Toxicity reaction.
- Secondary infections.

Failure of antibiotic therapy may be attributed to:

- Inadequate surgical treatment.
- Depressed host defense.
- Presence of foreign body.
- Problems associated with the patient and antibiotic, like lack of patient's compliance and inadequate dose...etc.

Sinus formation

When the abscess is not drained properly and neglected for a sufficient period of time, it will burst and drain spontaneously leading to sinus formation in an unfavorable site, the sinus is puckered, thickened and depressed.
If the source of infection is not treated the sinus will become chronic and it will be subject to exacerbations and remissions. During the active phase the sinus will exhibit signs of inflammation and may be tender while in the quiescence phase it heals over.
Sinus excision, after treatment of the source of infection, is carried out through an elliptical incision around the external orifice of the sinus, the tract of the sinus is followed bluntly to its source on the surface of the bone of the mandible. The resulting defect is closed in layers to eliminate dead space, the skin is closed with careful eversion to ensure an acceptable scar.

Necrotizing Fasciitis (flesh-eating bacterial infection)

Necrotizing fasciitis, known colloquially as flesh-eating bacterial infection due to the unique characteristic of the infection, does not obey the typical organization of the fascial planes of the head and neck.

First described by Pearse in 1938, who reported a 49% mortality rate, the progression of cervical necrotizing fasciitis does not follow the normal fascia planes of the head and neck. This is due to the unusually aggressive nature of the disease process. Often patients are immunocompromised, leading to this unusual and complex clinical course and extensive progression of an odontogenic head and neck infection.

Cervical necrotizing fasciitis is often polymicrobial in nature, is strikingly destructive, and is often fatal with a mortality rate of 7% to 20%. When the infection progresses to the thoracic region as a descending necrotizing mediastinitis, the mortality rate rises dramatically.

Management includes:

1- Removal of the offending source of the infection
2- Prompt, very aggressive debridement and removal of all affected soft tissues.
3- Creation of a surgical airway and continued intensive care unit monitoring and management.
4- Broad-spectrum empiric bactericidal intravenous (IV) antibiotics are generally always indicated in these cases because all involved tissues cannot be completely eradicated until specific culture and sensitivity results are available to guide specific antibiotic regimens.
5- Medical optimization of the patient

The disease process is characterized by the rapid spread of the infection on the superficial surface of the anterior (investing) layer of the deep cervical fascia deep to the platysma muscle.

Note: the necrosis of the platysma muscle and overlying skin due to thrombosis of the underlying muscles and soft tissues, as well as the dermal blood supply. The extensive tissue necrosis, including the skin, must be debrided thoroughly to halt the continued spread of the disease process.