Severe Odontogenic Infections in Children That Require Emergency Hospitalization: An Update

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Abstract

Odontogenic infections are the most common infections inflicting mankind. The morbidity associated with this could be local complications like cellulitis, abscess or systemic disseminated infection. These complications can prove fatal if stringent management is not taken which includes prompt diagnosis, management and closely monitored hospital care. This review paper highlights severe infections of odontogenic origin affecting the children that demand emergency hospital care.

Keywords

- severe odontogenic infections
- dental emergencies
- odontogenic infections in children
- Ludwig's angina
- cavernous sinus thrombosis

Introduction

Odontogenic infections (OIs) are one of the most widespread illnesses in the oral and maxillofacial areas, with a mortality rate of 10 to 40%.[1] OIs in children may involve more than one tooth and usually arise from carious lesions, periodontal problems, pathology (e.g., dens invaginatus), or a history of trauma.[2,3] OIs have a tendency to affect children in the mixed dentition stage.[4] These infections are mostly localized but in the acute infection stage, infection propagates very rapidly, ranging from mild buccal space infection to severe life-threatening multispace infection.[4,5] Due to the wide marrow spaces in a child's jaws as well as the fact that growing children's bones are less dense than adult bones, infections in the child's jaws can progress rapidly.[6] Due to the rapid systemic involvement among children, detection as well as treatment of acute orofacial infections is essential.[5,7] Untreated OIs can result in pain, difficulty in consuming food or beverages, abscess, cellulitis, septicemia, airway compromise, and life-threatening infections.[2,4,5]

Etiopathogenesis of Odontogenic Infection

Orofacial space infections of odontogenic origin are highly prevalent among children, and are mostly caused by necrotic pulp, partially erupted teeth, or traumatized teeth (►**Fig. 1**).[5,6] Seow reported that developmental abnormalities like dens evaginatus, dentin dysplasia, dentinogenesis imperfecta, and familial hypophosphatasia also cause space infections.[8] Pediatric orofacial space infections of odontogenic origin are polymicrobial and mixed (combination of both aerobic and anaerobic) in nature with a predominance of anaerobes.[5,6] Several conditions leading to odontogenic infections are enumerated in ►**Table 1**. Dodson et al

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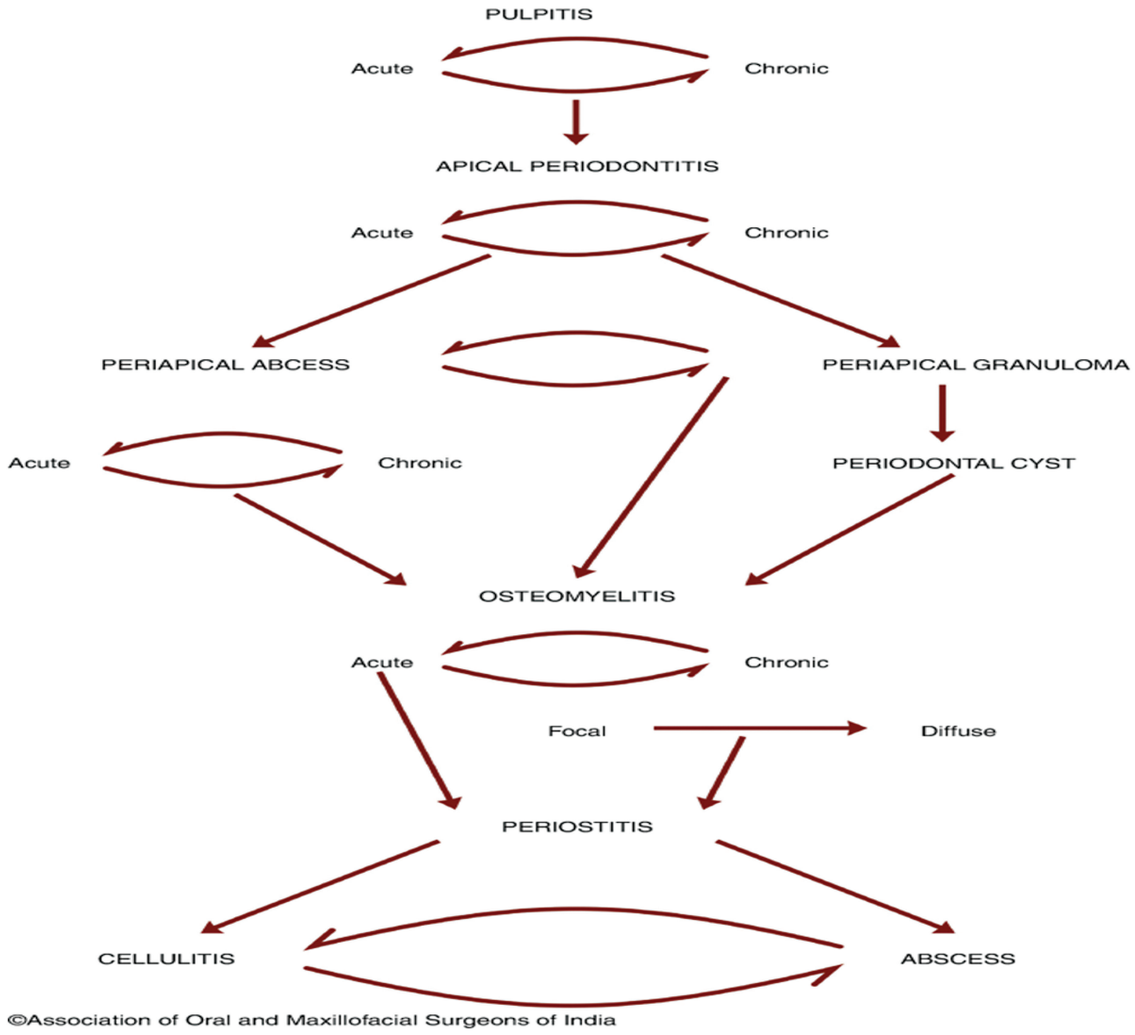

Fig. 1 Etiopathogenesis of odontogenic infection (OI). (Adapted Shukla and Mehrotra.[13])

discovered that upper face infections particularly preseptal (periorbital) cellulitis, were more common in children younger than 5 years, whereas lower face infections were more common in children aged 6 to 12 years.[9] The most frequently reported space infection was submandibular space infection followed by buccal space infection.[6,9–12] This might happen because both primary and permanent mandibular molars are more susceptible to dental caries and as a result become infected more frequently leading to buccal space infection.[6,8] The main difference among adults and children on the level

Table 1 Local and systemic conditions leading to odontogenic infection[1,13–22]

Local condition	Systemic condition			
	Physiological condition	Disease related	Defective immune system	Drug suppression related
• Hematoma • Traumatic surgery • Pre existing • Infection • Compromised vascular supply • Necrosis	• Patient's inability to deliver defending agent, i.e., WBCs, Abs complement – Shock – Fluid imbalance – Advanced age – Obesity	• Metabolic disease, diabetes mellitus • Malnutrition • Alcoholism • Leukemia • Lymphoma • Agranulocytosis	• Congenital agammaglobulinemia • Multiple myeloma • Splenectomy • Radiation • Immunosuppressive infectious disease, AIDS	• Cytotoxic drugs (chemotherapy) • Glucocorticoids • Azathioprine • Cyclosporine

Abbreviations: Abs, antibodies; WBC, white blood cell.

Table 2 Characteristics of the 3 stages of infection[4,5,13,23]

Characteristic	Inoculation	Cellulitis	Abscess
Duration (days)	0–3	2–5	4–10
Discomfort	Mild	Severe, diffused	Mild, localized
Palpation	Soft, doughy	Firm, indurated	Fluctuant, tender
Pus	None	None	Present
Skin	Normal	Red	Red periphery
Bacterial species	Aerobic	Mixed	Anaerobic

Table 3 Severity scores for severe odontogenic infections according to anatomical space involvement[13,24]

Severity	Spaces involved
Low	• Vestibular • Subperiosteal • Body of the mandible • Infraorbital • Buccal
Moderate	• Submandibular • Submental • Sublingual • Pterygomandibular • Submasseteric • Superficial temporal • Deep temporal (or infratemporal)
Severe	• Lateral pharyngeal or pterygopharyngeal • Retropharyngeal • Pretracheal • Prevertebral • Mediastinum • Intracranial infection

of infection spread is the relationship of tooth apices to the attachment of facial muscles on the maxilla and mandible.[6,8]

Inoculation, cellulitis, and abscess are the three stages of progression for OIs (►**Table 2**).[3–6,13,23,24] Bacteria penetrate the surrounding facial spaces through direct extension from the periapical region of the involved tooth which can result in severe infections involving the adjacent anatomical spaces (►**Table 3**).[4,5,23]

Clinical Symptoms:

Clinical signs and symptoms of odontogenic infections have been described in ►**Table 4**.

Diagnosis

Adequate history of the onset and duration of symptoms and current and prior illnesses is necessary for the diagnosis of OI. In addition to drug sensitivity, previous surgical and medical procedures for the same condition, their success,

Table 4 Clinical symptoms of odontogenic infection[4,5,13,25]

Local symptoms	Clinical symptoms
Pain	Fever
Swelling	Malaise
Surface erythema	Toxic appearance
Pus formation	Lymphadenopathy
Trismus	Increased white blood cell (WBC) count
Dysphasia	Increased Arneth count
Dyspnea	Increased erythrocyte sedimentation rate (ESR)

Table 5 Commonly used antibiotics for treating odontogenic infection[5,7,23,26,27]

Antibiotic	Adult dosage	Pediatric dosage
Penicillin V	600 mg every 6 h	25–50 mg/kg/d divided into 4 doses
Amoxicillin	500 mg every 8 h	25–50 mg/kg/d divided into 4 doses
Cephalexin	500 mg every 6 h	25–50 mg/kg/d divided into 4 doses
Metronidazole	500 mg twice daily	15–30 mg/kg/d divided into 3 doses
Clindamycin	300–450 mg every 6 h	10–30 mg/kg/d divided into 3–4 doses
Moxifloxacin	400 mg daily	–
Erythromycin	500 mg every 8 h	30–50 mg/kg/d divided into 2–4 doses

and the use of immunosuppressant medications are important. In nutshell a comprehensive evaluation is required. Local examination must include visual inspection to identify the cause and palpation to assess the tissue consistency. Ophthalmic evaluation is also necessary while examining maxillary infections. Intraoral and panoramic radiographs are the initial diagnostic tools that definitely aid in determining the underlying cause of the condition.[4,5,13]

Treatment

Treatment of OIs generally encompasses the prescription of appropriate antibiotics as well as diagnosis and management of the root cause of the infection (►**Fig. 2**).[4,5,7,23,24] Amoxicillin is the first choice for treating children, according to the literature and clinical evidence.[4,5,7,23,25–28] The use of amoxicillin in combination with sulbactam pivoxil or clavulanic acid is advised for patients who have previously received insufficient systemic treatment (►**Tables 5**,**6**). Hospitalization of the patient must be done wherever indicated (►**Table 7**). Clindamycin or clarithromycin as are suitable antibiotics for patients with penicillin hypersensitivity.[4,5,7,23–28] According to the

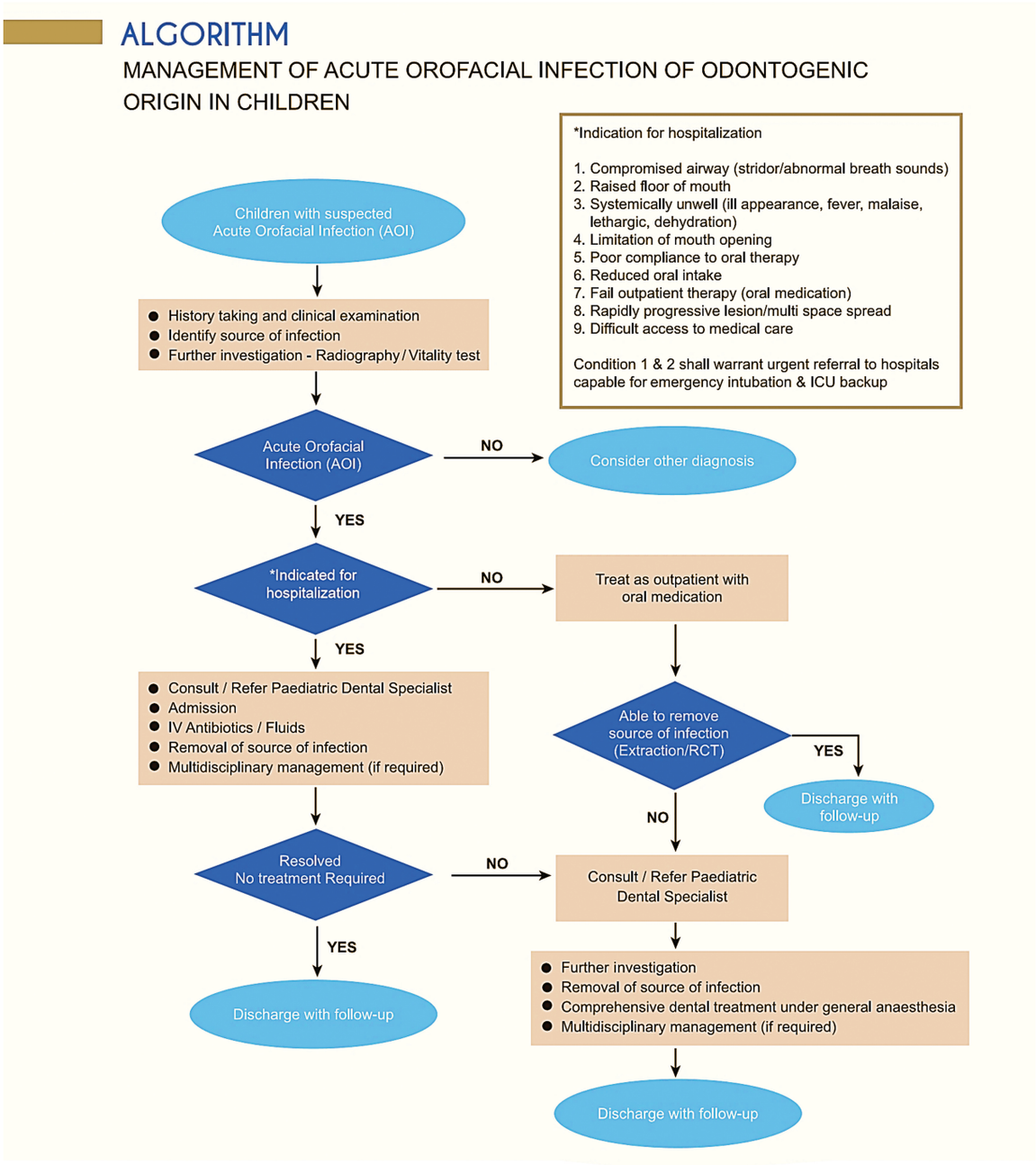

Fig. 2 Management of orofacial infection of odontogenic origin in children. (Adapted from Oral Health Division, Ministry of Health Malaysia.[5])

literature and our clinical experience, it is preferable to avoid radical procedures such as extractions during the acute stage of infection considering the patient's anatomo-physiological and psychological aspects.[26–28]

Malaysian clinical practice guidelines recommend that the antibiotics be administered immediately followed by immediate surgical intervention in persistent/rapid progression cases. The removal of infection source either through extraction or pulp therapy depends on restorability, cooperativeness of the child, and orthodontic considerations. Comprehensive dental treatment should be done under general anesthesia for an uncooperative child.[5]

Complications

Complications of OIs lead to odontogenic sinusitis, periorbital infections, cavernous sinus thrombosis, bacterial endocarditis, Ludwig's angina, cervicofacial necrotizing fasciitis, brain abscess, meningitis, mediastinitis, septicemia, gangrenous encephalitis, gangrenous pneumonia, thrombophlebitis of the jugular veins, and edema of glottis.[1,6,28,29]

A. Ludwig's Angina

Ludwig's angina is a rapidly spreading bilateral cellulitis of the sublingual and submaxillary spaces, which can compromise

Table 6 Antimicrobials commonly used for treating odontogenic infections in children[5,7,26,27]

	Antimicrobial	Dose
First choice in recent infections	Amoxicillin	• 20–50 mg/kg/d thrice a day • 500 mg thrice a day
Late untreated infection after treatment with first-choice antimicrobial	Amoxicillin and clavulanic acid	• 40–80 mg/kg/d thrice a day 400 mg amoxicillin + 57 mg clavulanic acid thrice a day
	Amoxicillin and Sulbactam Pivoxil	• 100–200 mg/kg/d thrice a day • 875 mg amoxicillin + 125 mg pivoxil sulbactam thrice a day
Beta-lactam hypersensitivity	Clarithromycin	• 7.5–15 mg/kg/d twice a day (<1 g/d) • 250 mg twice a day • 500 mg twice a day
	Clindamycin	• 10–30 mg/kg/d, twice a day • 300 mg four times a day • 600 mg four times a day

Note: Monitor response to treatment and patient outcome within 24 hours.
After evaluating the patient with a complete medical history, treatment of affected tooth can be conservative (restorative and endodontic approach) or radical (extraction).

Table 7 Criteria for hospitalization in children with odontogenic infections[5,27]

Hospitalization criteria for children
Immunocompromised patient
Cellulitis
Raised floor of mouth
Compromised airway dyspnoea and dysphagia
Involvement of deep facial spaces
Fever >38°C
Increased trismus <10 mm
Reduced oral intake
Poor compliance to oral therapy
Initial treatment failure

the airway. It was first introduced by Wilhelm Friedrich von Ludwig in 1836. Etiology, diagnosis, and microbiological features are listed in **Box 1**.[1,28–34]

Box 1: Etiology, Diagnosis, Microbiology and Management of Ludwig's Angina

Etiology
- 90% OI of the lower teeth.
- **Risk factors:** poor dental hygiene, tooth extraction, trauma to the floor of the mouth, peritonsillar abscess, and intravenous (IV) drug abuse

Diagnosis
Clinical signs and symptoms
- Inability to close the mouth.
- Inability to swallow and drooling.
- Dysphonia.
- Dyspnea and stridor.
- Fever and chills.
- Tachycardia.
- Severe pain and tenderness around the submandibular region.
- Laryngeal edema.
- Tongue elevation.
- "Bull neck," associated with an increased submental fullness and a lack of mandibular angle definition.

Microbiology
- Throat swabs to identify the involved microorganism.
- Blood cultures are usually negative but useful in the immunocompromised patients.
- Samples should be sent for Gram stain, culture, and sensitivity after aspiration or drainage.

Management of Ludwig's angina
- Maintenance of airway (emergency treatment) in case of respiratory stridor by emergency tracheostomy.
- Prolonged antibiotic therapy.
- Early surgical intervention to relieve tension.
- Treat the cause.

In case of massive swelling tissue is under tension, with respiratory distress and steps to be taken are shown in **Box 2**.

Box 2: Measures to be taken in event of respiratory distress

Incision and Drainage (I and D)
⬇
Decompress the tissues
⬇
Release tissue under tension
⬇
Release pus (if present obtain it for culture and sensitivity)
⬇
Reduce the pressure of edematous tissue on the airway
⬇
Allow prompt drainage

B. Cavernous Sinus Thrombosis

Septic Cavernous Sinus Thrombosis (CST) was first described in 1778 as a rare condition that may lead to significant morbidity and mortality if not diagnosed and treated precociously.[1,29,35]

Etiology

- Facial infections and paranasal sinusitis are the most common causes, followed by otogenic, odontogenic, pharyngeal, and distant sepsis.
- About 7% of CST is of dental origin.
- Infection from paranasal sinuses and infection of the middle third of the face, dental abscess, tonsils, soft palate, and ears reach the cavernous sinus through tributaries.[1,29,35]

Eagleton's Criteria for Diagnosis

- Evidence of bloodstream infection.
- Early signs of venous obstruction in the retina, conjunctiva, or eyelid.
- Paresis of the third, fourth, and sixth cranial nerves resulting from inflammatory edema.
- Abscess extending to the adjacent tissues.
- Evidence of meningeal irritation.[1,29,35]

Clinical features and management of CST have been described in **Box 3**.[1,29,35]

Box 3 Clinical features of CST

• The most common signs of CST are related to direct injury to cranial nerves III and VI and impaired venous drainage from the orbit and eye. • Patients are often gravely ill. • Headache, fever, vomiting, nausea, and chill initially—alarming severity. • Occlusion of the ophthalmic veins causes photophobia, increased lacrimation, proptosis, chemosis and edema of the eyelids, etc. • Paralysis of the external ocular muscles, exophthalmos, intraocular hemorrhage, and even blindness can occur. • Complete paralysis of the cranial nerves III, IV, and VI are common. • The condition is initially unilateral but often becomes bilateral within 2 to 3 days. • Death may occur due to brain abscess, meningitis, septicemia, or pyemia.
Treatment
• Drug of choice: chloramphenicol every 6 hours intravenously. • Dehydration: fluid replacement. • Anticoagulants to prevent venous thrombosis.

C. Meningitis

Meningitis is the most common neurologic complication resulting from infections in the oral and maxillofacial region. Headache, fever, neck stiffness, and vomiting are some of its hallmark symptoms. The patient is often confused and may become comatose.[1,29,36]

Treatment

- Massive antibiotics for long duration.
- Combinations of chloramphenicol and penicillin G.
- Maintain electrolyte balance by infusing IV fluids.
- Avoiding vascular collapse and shock by controlling cerebral oedema.
- Massive doses of steroids and mannitol.
- Antibiotics to be continued a week after the symptoms subside and cerebrospinal fluid (CSF) returns to normal.
- Septic shock may need fresh blood transfusion.[1,29,36]

D. Brain Abscess

This can occur from a bacteremia accompanying an OI. Infections of the brain cause inflammation, localized edema, and septic thrombosis resulting in the development of single or multiple abscesses.[1,28,29,37,38]

Signs and Symptoms

- Headache most consistently seen associated with nausea and vomiting.
- The majority of patients are afebrile.
- Hemiplegia, papilledema, aphasia, convulsions, hemisensory deficit, hemianopia, and abducens palsy.
- Abscess of the frontal lobe may cause stupor, confusion, and subtle changes in personality.[1,28,29,37,38]

Diagnosis: The final diagnosis is based on the clinical findings, radionuclide scanning, and computed tomography (CT) scan.[1,28,29,37,38]

Treatment: Drug therapy (antibiotics, steroids, and mannitol to reduce cerebral edema) and surgical drainage. IV chloramphenicol is the initial antibiotic of choice until the causative organism has been identified and its antibiotic sensitivity determined.[1,28,29,37,38]

E. Mediastinitis

Chest pain, severe dyspnoea, recurrent fever, and radiographic signs of mediastinal widening are indications of infection extending from the deep neck spaces into the mediastinum. Rarely mediastinitis may also be caused by OI that spreads readily along the great vessels in the perivascular space of the carotid sheath.[1,29,39]

It may appear very late and should be suspected in patients with exacerbation of fever associated with substernal pain. Progressive septicemia, mediastinal abscesses, pleural effusion, emphysema, and pericarditis may occur with death as the final outcome. Necrotizing mediastinitis of dental origin is associated with the combined effect of aerobic and anaerobic bacteria invading the surrounding tissue away from their normal oral environment.[1,29,39]

Treatment of suppurative mediastinitis consists of extrinsic long-term antibiotic therapy and surgical drainage of the mediastinum. Specimen for culture should be obtained regularly for long-term therapy.[1,29,39]

F. Necrotizing Fasciitis

It is called as a "killer or flesh-eating bug" in layman's terms. Acute dermal gangrene, hospital gangrene, suppurative

fasciitis, and synergistic necrotizing cellulitis are other names for it in addition to hemolytic streptococcal gangrene. It is more common in immune-compromised patients with an incidence of 1 to 10%. It is an inflammatory illness that starts in the deep fascia and spreads quickly, causing secondary necrosis of the subcutaneous tissues.[1,29,40]

Etiology

- Local tissue injury induced by surgical intervention of intraperitoneal infections, ischiorectal and perianal abscesses, and bacterial invasion surgery.
- Intramuscular injections and intravenous infusions.
- Minor bug bites introducing streptococci into the wounds, but the bacteriologic pattern changes due to hypoxia-induced anaerobe proliferation.
- Patients with systemic diseases may have local ischemia and hypoxia.
- Immunocompromised patients.[1,29,40]

Clinical Features

- It presents as sudden onset of pain and swelling at the site of trauma or recent surgery. Infection begins with an area of erythema that quickly spreads to normal skin without being raised or sharply demarcated giving a dusky or purplish skin discoloration near the site of insult over a course of hours to days.
- The first symptom of necrosis is a significant undermining of the epidermis and subcutaneous tissue. Anesthesia can be observed in the affected area, and it is usually caused by thrombosis of the subcutaneous blood arteries, which leads to nerve fiber destruction.
- When left untreated, deeper muscle layers may become involved, resulting in myositis or myonecrosis.
- Tissue necrosis, putrid discharge, bullae, acute pain, gas generation, quick burrowing across fascial planes, and a lack of classical inflammation are the most essential indications.[1,29,40]

Management

- Airway management and oxygen supply are important.
- Obtain IV access.
- Normal saline or lactated Ringer's solution is more often used to resuscitate fluids.
- Begin antibiotics as soon as possible (IV penicillin is the initial drug of choice.)
- Aggressive surgical debridement may be required.[1,29,40]

Conclusion

Patients inflicted with OI requiring hospital stay are those patients who have underlying systemic disease, resistance to certain antibiotics, and bacteremia due to dental procedures. Strategies to improve oral health through reduction in the incidence of untreated dental caries are likely to decrease the incidence of severe OIs. Antibiotics are an essential adjunct in surgical management and should be used discretely. Early diagnosis and definitive intervention of OI portends a better prognosis.

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Conflict of Interest
None declared.

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