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Dry Socket (Alveolar Osteitis): Incidence, Pathogenesis, Prevention and Management

Girish G Gowda, Deepak Viswanath, Mahesh Kumar, DN Umashankar

ABSTRACT

Alveolar osteitis (AO) is the most common postoperative complication after tooth extraction. The pathophysiology, etiology, prevention and treatment of the alveolar osteitis are very essential in oral surgery. The aim of this article is to provide a better basis for clinical management of the condition. In addition, the need for identification and elimination of the risk factors as well as preventive and symptomatic management of the condition are discussed.

Keywords: Alveolar osteitis, Localised osteitis, Septic socket, Halitosis, Pain.

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INTRODUCTION

Dry socket is the most common postoperative complications following the extraction of teeth. This term was first described by CRAWFORD in 1986.¹ Birn labeled this complication as 'fibrinolytic alveolitis'.²⁻⁴ Several other terms have been used in referring to this condition like alveolar osteitis (AO), localized osteitis, postoperative alveolitis, alveolgia, alveolitis sicca dolorosa, septic socket, necrotic socket, localized alveolitis and fibrinolytic alveolitis.^{5,6} The clinical features of AO present disintegration of formed blood clot, halitosis and pain with varying intensity from the extraction socket, which usually occurs 2 to 4 days after extraction.^{7,8}

INCIDENCE

The incidence of AO is 10 times more in mandible when compared to maxilla ranging from 1 to 4% of extractions, reaching 45% for mandibular third molars.^{6,9} AO may affect women in ratio of 5:1 with respect to males.^{8,10} Due to changes in endogenous estrogens during the menstrual cycle since estrogens activate the fibrinolytic system in an indirect way in females.¹¹

ONSET AND DURATION

AO occurs 1 to 3 days after tooth extraction and within a week between 95 and 100% of all cases of AO have been

registered.¹²⁻¹⁵ The duration varies from 5 to 10 days depending on the severity of the condition.

ETIOLOGY

The exact etiology of AO is not well understood. Birn suggested that the etiology of AO is an increased local fibrinolysis leading to disintegration of the clot. However, several local and systemic factors are known to be contributing to the etiology of AO.

CONTRIBUTING/RISK FACTORS

1. *Surgical trauma and difficulty of surgery:* Most authors agree that surgical trauma and difficulty of surgery play a significant role in the development of AO.⁴ This could be due to more liberation of direct tissue activators secondary to bone marrow inflammation following more traumatic extractions.¹⁶
2. *Lack of operator experience:* Many studies claim that operator's experience is a risk factor for the development of AO. Larsen concluded that surgeon's inexperience could be related to trauma during the extraction, especially surgical extraction of mandibular third molars.¹⁷
3. *Mandibular third molars:* It has been shown that AO is more common following the extraction of mandibular third molars. Some authors believe that increased bone density, decreased vascularity, and reduced capacity of producing granulation tissue are responsible for the site specificity.¹⁸
4. *Systemic disease:* Studies suggested that systemic disease could be associated with AO.^{4,19} Immuno-compromised or diabetic patients being prone to development of AO due to altered healing.⁸
5. *Oral contraceptives:* Increase in use of oral contraceptives positively correlates with incidence of AO. Estrogen has been proposed to play significant role in fibrinolytic process. It is believed to indirectly activate the fibrinolytic system and therefore increase lysis of the blood clot.²⁰
6. *Smoking:* Studies reported that among patients with total of 400 surgically removed mandibular third molars, those who smoked half-pack of cigarettes per day had four- to five-fold increase in AO compared to nonsmoking patients.²⁰

7. *Physical dislodgement of the clot:* Physical dislodgement of the blood clot caused by manipulation or negative pressure created via sucking on a straw would be a major contributor to AO.⁷
8. *Bacterial infection:* Most studies support that bacterial infections are major risk for development of AO. The frequency of AO increases in patients with poor oral hygiene and pre-existing local infection like pericoronitis and advanced periodontal disease.²¹ Nitzan et al observed high plasmin-like fibrinolytic activities from cultures of *Treponema denticola*, a microorganism present in periodontal disease.²²
9. *Excessive irrigation or curettage of alveolus:* Excessive repeated irrigation of alveolus might interfere with clot formation and violent curettage might injure the alveolar bone.⁴ However, the literature lacks evidence to confirm these allegations in the development of AO.
10. *Age of the patient:* Little agreement can be found as to whether age is associated with peak incidence of AO. Blondeau et al²³ concluded that surgical removal of impacted mandibular third molars should be carried out well before age of 24 years, since older patients are at greater risk of postoperative complications in general.
11. *Bone/root fragments remaining in the wound:* Studies suggested that bone/root fragments and debris remnants could lead to disturbed healing and contribute to development of AO.^{4,7} Simpson showed that small bone/root fragments are commonly present after extractions and these fragments do not cause complications as they are often externalized by the oral epithelium.²⁴
12. *Local anesthetic with vasoconstrictor:* Studies suggested that use of local anesthesia with vasoconstrictors increases the incidence of AO. Lehner²⁵ found that AO frequency increases with infiltration anesthesia because of temporary ischemia. However, some studies showed that ischemia lasts for 1 to 2 hours and is followed by reactive hyperemia, which makes it irrelevant in the disintegration of blood clot.^{4,26} It is currently accepted that local ischemia due to vasoconstrictor in local anesthesia has no role in development of AO.

PATHOGENESIS

In AO there is increased local fibrinolysis which leads to disintegration of the clot by conversion of plasminogen to plasmin. Fibrinolysis is the result of plasminogen pathway activation, which can be via direct (physiologic) or indirect

(nonphysiologic) activator substances.⁴ Direct activators are released after trauma to the alveolar bone cells. Indirect activators are released by bacteria. Fibrinolytic activity is local because initial absorption of plasminogen into the clot limits the activity of plasmin (Fig. 1).^{8,27}

SIGNS AND SYMPTOMS

Severe, debilitating, constant pain that continues through the night, becoming most intense at 72 hours postextraction. It can be associated with foul taste and halitosis. The pain responds poorly to over-the-counter analgesic medication. Clinically, an empty socket (lacking a blood clot) with exposed bone is seen. Other symptoms include low grade fever and regional lymphadenopathy.^{15,28}

PREVENTION

Since AO is the most common postoperative complication after extraction, many researchers have attempted to find a successful method for prevention. However, this area remains a controversial topic as no single method has gained universal acceptance. The most popular of these techniques are discussed below.

1. *Antibiotics:* Systemic antibiotics like penicillin's, clindamycin, erythromycin and metronidazole are effective in preventing AO. Development of resistant bacterial strains and hypersensitivity is possible on routine use of systemic antibiotics pre or postoperative.⁸ Local application of tetracycline in the form of powder, aqueous suspension, gauze drain and gel foam sponges show promising results in reducing incidence of AO when compared to other antibiotics.^{10,29}
2. *Chlorhexidine:* Pre or postoperative use of CHX mouth rinse significantly reduces the incidence of AO after the extraction of mandibular third molars. A 50% reduction in the incidence of AO was observed in patients who prerinsed for 30 seconds with 0.12% CHX solution.⁷ Use of 0.2% bioadhesive CHX gel reduced incidence of AO.³⁰

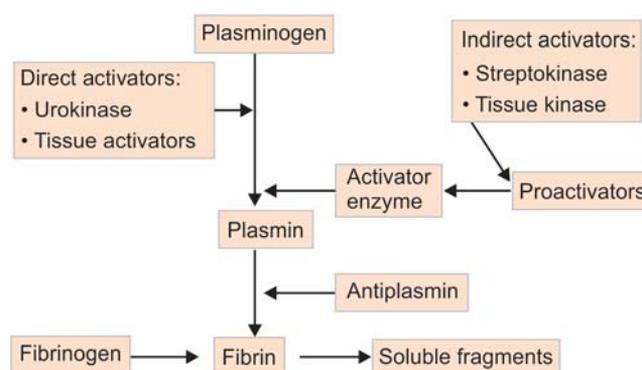


Fig. 1: Pathogenesis of AO⁴

3. *Eugenol containing dressing*: Eugenol acts as an obtundent. Commercially available dressing Alvogyl® (contains eugenol, butamben and iodoform) should be replaced every 2 days. The incidence of AO was seen 8% in sockets which were immediately packed with medicated dressing and 26% in sockets which were not immediately packed.^{31,32}
4. *Steroids*: The topical application of hydrocortisone and oxytetracycline mixture has shown decreased incidence of AO after removal of impacted mandibular third molars.⁷
5. *Antifibrinolytics*: Tranexamic acids have been reported to be used to prevent incidence of alveolar osteitis.²⁶
6. *Low level laser therapy (LLLT)*: It was found that low level laser therapy (LLLT) increases speed of wound healing and reduces inflammation when compared to Alvogyl and SaliCept. LLLT is applied after irrigation of socket with continuous-mode diode laser irradiation (808 nm, 100 mW, 60 seconds, 7.64 J/cm²).³³
7. *Biodegradable polymers, topical hemostatics, oxidized cellulose foam (OCF)*: Use of polylactic acid granules, ActCel®, (topical hemostatic agent) and oxidized cellulose foam, showed reduced incidence of AO.^{27,34, 35}
8. *PRP and PRF IN AO*: Studies reported substantial reduction in the incidence of AO following treatment of the extraction site with PRP and or combination of PRF and gelatin sponge.^{36,37}
9. *Dextranomer granule*: Dextranomer showed a significantly faster pain relief and decrease in the incidence of AO.³⁸

SYMPTOMATIC MANAGEMENT

On average, a time period of 7 to 10 days is required for exposed bone to become covered with new granulation tissue, and efforts must be made to relieve patient discomfort during this period. Turner³⁹ used reflection of flap, removal of bone particles, curettage and removal of granulation tissue with irrigation and found that this method required fewer visits than ZOE pack. Fazakerley and Field⁴⁰ recommended gentle irrigation with warm saline under local anesthesia before application of ZOE dressing with iodoform ribbon gauze. The packing should be changed every 2 to 3 days and removed once pain is reduced. Choice of analgesics varies from short course of NSAID'S drugs to narcotic-based preparations such as acetaminophen with codeine, hydroxycodone or oxycodone.

CONCLUSION

The etiology of AO is multifactorial and ultimately host's healing potential determines the severity and duration of the condition. AO is a self-limiting condition, the cause of

which remains elusive. Management is aimed at relieving the patient's pain until healing of the socket occurs. Healing is facilitated and accelerated through reducing the insult to the wound by food debris and microorganisms, by irrigation of the socket with chlorhexidine, followed by placement of medicated dressing and prescription of analgesics. The patient should be kept under review to check the socket is healing, especially if a dressing is placed. Ultimately, it is the host's healing potential which determines the severity and duration of the condition.

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ABOUT THE AUTHORS

Girish G Gowda (Corresponding Author)

Senior Lecturer, Department of Oral and Maxillofacial Surgery
Krishnadevaraya College of Dental Sciences, Bengaluru, Karnataka
India, Phone: 9880822544, e-mail: drgirishggowda@gmail.com

Deepak Viswanath

Professor and Head, Department of Pedodontics and Preventive
Dentistry, Krishnadevaraya College of Dental Sciences, Bengaluru
Karnataka, India

Mahesh Kumar

Reader, Department of Oral and Maxillofacial Surgery, Krishnadevaraya
College of Dental Sciences, Bengaluru, Karnataka, India

DN Umashankar

Reader, Department of Oral and Maxillofacial Surgery, Krishnadevaraya
College of Dental Sciences, Bengaluru, Karnataka, India