Review Article

Current Recommendations for Treatment of Dry Socket-A Review

Savant Kiran[1], Vinayak G. Naik[1], Rakshit Vijay Sinai Khandeparker[1], Hunny Jain[2], Vikas Berwal[3]

Department of Oral and Maxillofacial Surgery, [1]Bapuji Dental College & Hospital, Davangere, Karnataka, [2]Yogita Dental College Khed, Ratnagiri, [3] PGI Rohtak, Haryana

ABSTRACT:
Alveolar osteitis (AO) is a well known complication which occurs on tooth extraction. It is commonly known as Dry Socket. Its incidence is approximately 3% for all routine extractions and can reach over 30% for impacted mandibular third molars. It is one of the most studied complications in dentistry, and a great number of studies have searched for an effective and safe method for its prevention and treatment. One of the great clinical challenges since the first case was reported has been the in consistence and differences in the various definitions of dry socket and the criteria used for diagnosis. The treatment of AO includes conventional methods like use of antibiotics, medicated gauze, gel, rinse. It is a very common condition arising on extraction of mandibular molars it is associated with postoperative pain in and around the extraction site, accompanied by a partially or totally disintegrated blood clot within the alveolar socket, with or without halitosis. This paper is a review of the literature on dry socket, presented a suggestion is made as to how best this painful condition may be managed.
Key word: Alveolar osteitis, alveolalgia, alveolitis sicca dolorosa

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Corresponding Author: Dr. Savant Kiran, Department Of Oral &Maxillofacial Surgery, Bapuji Dental College & Hospital, Davangere, Karnataka, Email: kiransawant@gmail.com

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Introduction

Dry socket is the most common postoperative complication after tooth extraction, with an onset at 2 to 4 days after surgery.[1-5] It was first described by Crawford in 1876. It has also been referred to as alveolar osteitis, localized osteitis, alveolalgia, alveolitis sicca dolorosa, septic socket, necrotic socket, localized osteomyelitis, fibrinolytic alveolitis, and others.[6] Blum (2002) described alveolar osteitis as being the presence of "postoperative pain in and around the extraction site, which increases in severity at any time between one and three days after the extraction, accompanied by a partially or totally disintegrated blood clot within the alveolar socket, with or without halitosis".[1] A localised fibrinolysis (resulting from conversion of plasminogen to plasmin, which acts to dissolve fibrin crosslinks) occurring within the socket and subsequently leading to loss of the blood clot is believed to underlie the pathogenesis of alveolar osteitis.[7] Hansen in 1960 described alveolitis simplex, featured by accidental loss of the clot and the absence

of pain, in addition to alveolitis sicca dolorosa and granulomatous alveolitis.[8] Hermesch et al classified this complication into 3 types: superficial alveolitis marginal, suppurative alveolitis, and dry socket. In marginal alveolitis, the perialveolar mucosa becomes inflamed and partially covered by granulomatous tissue and is painful during mastication. In suppurative alveolitis, the clot becomes infected and is covered by a green-grayish membrane and can contain dental fragments or osseous sequestrum. It causes medium in- tensity pain, and fever can also be present. In dry socket, the alveolar osseous walls are exposed, with total or partial clot loss, dark coloration, and a fetid odor. Continuous, intense, and frequently radiating pain is present that is not relieved by analgesics. Local hyperthermia and lymphadenopathy can also occur with this type of alveolitis.[9] Microscopically, dry socket is characterized by the presence of inflammatory cellular infiltrate, including numerous phagocytes and giant cells in the remaining blood clot, associated with the presence of bacteria and necrosis of the lamina dura.[10] In 1973, Birn reported that the inflammatory process can extend to the medullar spaces and sometimes the periosteum, resulting in connective tissue inflammation of the contiguous mucosa, with microscopic features typical of osteomyelitis. Degradation of the blood clot in association with dissolution of erythrocytes and fibrinolysis, deposits of hemosiderin, and the absence of organized granulation tissue have also been described in histopathologic investigation of dry socket.[11] Many denominations, classifications, and descriptions of dry socket have been reported. However, despite the controversies, in general, dry socket has been characterized as an inflammation in the alveolus of recently extracted teeth, for which pain and the period of onset are specific clinical signs indicative of proper diagnosis.

Etiology: Real dry socket is characterized by the partial or total premature loss of the blood clot that forms in the interior of the alveolus after extraction. This must be distinguished from other conditions, such as hypovascularization of the alveolar bone, caused by vascular and hematologic impairment; osteonecrosis induced by radiotherapy; osteopetrosis; Paget's disease; cement- osseous dysplasia, and so forth, in which the clot forms in the interior of the alveolus.[1]The exact etiology of dry socket has not yet been defined. However, several local and systemic factors are known to contribute and have been described in published studies. It was observed that occurrence of dry socket in female patients is up to 4.1% versus 0.5% of men, a 5-fold increase in the incidence compared to males. Due to changes in endogenous estrogens during the menstrual cycle the chances of dry socket in females is increased since Estrogens activate the fibrinolytic system in an indirect way. Hence menstrual cycle should be takes into consideration before scheduling extraction. Dry socket may affect women in ratio of 5:1 with respect to males. Incidence of dry socket increases if the extraction is performed by a less experienced operator. Therefore the skill and experience of the operator should be taken into consideration.[12,13] Micro-organisms like Enterococcus, Streptococcus viridians, Streptococcus,Bacillus coryneform, Proteus vulgaris, Pseudomonas aeruginosa, Citrobacter freundii, and Escherichia coli were found to be present in the alveolus. It was observed that there is a possible association of Actinomyces viscosus and Streptococcus mutans at the extraction site which further leads to delayed healing of the extraction socket. It is also observed that anaerobic micro-organisms like Treponema denticola have Plasmin like fibrynolytic activity which can be one of the risk factors to increase incidence of dry socket.[14]

Smoking has been shown to reduce neutrophil chemotaxis and phagocytosis, and impede production of immunoglobulin. Nicotine is absorbed through oral mucosa and hence acts as a vasoconstrictor. In a study of 400 mandibular extractions it was observed that , the incidence of dry socket was substantially greater in smokers than in non-smokers (6.4% vs. 1.4%, respectively), with patients who smoked 10 cigarettes/day had a 12% chance of developing the condition and those who smoked 1 pack/day had a 20% chance. Incidence of dry socket also increases to 40% if the patient smokes either on the day of the surgery or within the first 24 h after surgery. Also there are chances of removal of the clot through suction and negative pressure during smoke inhalation leading to dry socket.[5]Smoking water pipes or cigarettes may increase the risk for dry socket following extraction of mandibular molars.[15] Increase in the use of oral contraceptives can increase the incidence of AO. Estrogen plays a significant role in the fibrinolytic process. It is believed that they indirectly activate the fibrinolytic system (increasing factors II, VII, VIII, X, and plasminogen) and therefore increase lysis of the blood clot.[12] The risk of post extraction complications for mandibular third molars increased with females who are on oral contraceptives.[16] Incidence of alveolar osteitis increases with excessive trauma during extraction, especially in procedures that involve reflection of flap and excessive removal of bone. Mandibular third molar surgery is a relatively difficult and long procedure involving flap reflection, grinding into dense bone and tooth splitting. Hence, the third molar area is the most common site of dry socket occurrence. Excessive trauma causes compression of the bone lining the socket impairing vascular penetration. Subsequently excessive trauma can lead to the thrombosis of the underlying vessel.[1] Systemic disease could be associated with alveolar osteitis. Diabetic patients can be more prone to development of alveolar osteitis due to altered healing. But no such scientific evidence exists to prove a relationship between systemic diseases and alveolar osteitis.[12] Age Alveolar osteitis commonly occurs in age group of 20 – 40 years of age with a peak value in the age range 30 – 34 years and rarely after 50 years of age. Adult patients with age group above 21 years of age requires more time for recovery after third molar surgery. Excessive irrigation can interfere with clot formation and delay the healing of sockets. It also leads to increase in bacterial infection. However there is lack of evidence to confirm this assertion. Previous literature study claims that design of flaps and sutures affect the development of Alveolar osteitis but it is also reported that use of flap designs has no significant effect on incidence of Alveolar osteitis and there is no reduction in postoperative complications.[17]

Current recommendations to prevent and manage the dry socket

Dry socket prevention is determined by the medical and dental history of the patient, physical examination findings, patient laboratory examination results, and the presence of contributing factors. The dentist should ask preoperatively whether or not the patient has had a dry socket previously as some patients appear to be more susceptible than others. The patient should also be advised not to smoke for at least 48 hours post extraction. To void complications, strict guidelines for maintaining an aseptic field during the procedure and the correct indication and use of the surgical technique must be followed. In 2002, Blum suggested that factors inherent to the patient must also be considered as risk factors for dry socket. These included a history of dry socket, deep osseous impaction of mandibular third molars, poor oral hygiene, a recent history of pericoronitis, ulcerative gingivitis or active

illness associated with the tooth to be extracted, smoking (in particular _20 cigarettes daily), oral contraceptive use, and immunocompromised patients.[1] Dry socket is a self-limiting condition. However, due to the severity of pain experienced by the patient, it usually requires some symptomatic treatment. The range of treatments for a dry socket include treatments directed locally to the socket, including: irrigation of the socket with a 0.12-0.2% chlorhexidine rinse and instructing in home use of a syringe for irrigation; placement of a self-eliminating dressing such as Alvogyl (containing eugenol, butamben and iodoform); placement of an obtundant dressing such as zinc oxide, eugenol and lidocaine gel; or, a combination of these therapies and, where appropriate, the prescription of systemic antibiotics. Development of resistant bacterial strains and hypersensitivity is possible on routine use of systemic antibiotics pre and/ or ostoperative [13]. Alveolus irrigation after extraction with varying amounts of physiologic saline revealed that increasing the amount of physiologic saline (25, 175, and 350 mL) progressively decreased the incidence of dry socket (10.9%, 5.7%, and 3.2%, respectively). Analgesic dressings have also been used to prevent dry socket; however, most of such agents contain eugenol, a component that delays the healing process.[18] Mouthwash with chlorhexidine digluconate at 0.12% has been an efficient antiseptic for the prevention of dry socket. Some studies have shown important reductions in the incidence of dry socket after extraction of mandibular third molars.[19,20] Although some studies reported that this antiseptic eliminates almost 95% of all saliva bacteria, it was demonstrated that the remaining 5% are capable of causing infection.[21] The use of both systemic and topical antibiotics has been shown to reduce the incidence of dry socket.[1] Systemic penicillins, clindamycin and metronidazole, and topical tetracycline powder have all been shown to be effective. Ren and Malmsrom (2007) showed in a meta-analysis of 2,932 patients that antibiotics reduce the risk of alveolar osteitis and wound infection only when the first dose was given before surgery. The reason for the reduction in incidence of dry socket following preoperative administration of antibiotics is unclear as infection is not believed to be of significance in the pathogenesis of a dry socket, although a reduction in bacterial count does decrease the incidence. Use of Tetracycline has showed side effects along with foreign body reactions. The reported method of delivery included powder, aqueous suspension, gauze drain and Gel foam sponges [12]. The topical application of a Hydrocortisone and Oxytetracycline mixture however, has been shown to significantly decrease the incidence of AO after the removal of impacted mandibular third molars.[1]

It was found that Bupivacaine Hydrochloride reduced post-operative pain after extraction of mandibular third molars by irrigating the socket with Bupivacaine Hydrochloride, a local anesthetic .[24] Low Level Laser Therapy (LLLT) increases speed of wound healing and reduces inflammation 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).[25] Biodegradable ester acts as a clot supporting agent by providing a stable support for blood clot[12] Used to control hemorrhage and for wound protection. ActCel® is a topical hemostatic made from treated and sterilized cellulose. ActCel® enhances coagulation process and also acts as bacteriostatic.[26]

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