Modern concepts in understanding and management of the "dry socket" syndrome: comprehensive review of the literature

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Objectives. This paper provides a comprehensive review of the etiology, pathophysiology and current treatment of dry socket.
Study design. The Medline database (Ovid version) from 1966 to 2007 was searched for the term "dry socket" published in the English language, and 317 results were obtained. The articles were screened by abstract for relevance to etiology, pathophysiology, or treatment of dry socket. Treatment papers were ranked on the quality of evidence presented as assessed using the evidence-based systematic review worksheet of the University of Alberta. A total of 62 publications were included in the final review.
Results. Prevention methods remain the key to avoiding this complication. Prophylactic placement of topical antibiotics can be considered, whereas systemic antibiotics should be reserved for patients who are immunocompromised.
Conclusion. This paper provided a comprehensive review of the etiology, pathophysiology, and current treatment of dry socket in dental practice **(Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2009;107:30-35)**

Crawford first used the term "dry socket" in 1896.[1] Since that time, other terms have been used to describe dry socket: alveolar osteitis (AO), fibrinolytic alveolitis, alveolitis sicca dolorosa, localized osteomyelitis, and delayed extraction wound healing.[2] Dry socket can be debilitating, and 45% of the patients with dry socket may require up to 4 additional postoperative visits to provide care for the condition.[3] The present paper provides a comprehensive review of the etiology, pathophysiology and current treatment of dry socket.

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PATHOPHYSIOLOGY

Dry socket is the most common complication following a tooth extraction,[5] with a peak incidence in the 40–45-year-old age group.[6,7] Most studies state that the incidence of dry socket is 1%-4% for all routine dental extractions, and 5%-30% for impacted mandibular third molars.[8-12] The incidence of dry socket is higher in the mandible, occurring up to 10 times more often for mandibular molars compared with maxillary molars.[13] Typically, dry socket starts 1-3 days after tooth extraction and the duration usually ranges from 5 to 10 days.[8] Clinical features of dry socket are severe throbbing pain that starts 24-72 h after extraction, marked halitosis, and foul taste. One also sees an extraction socket devoid of clot with exposed bone that may be filled with food debris, edema of the surrounding gingiva, and regional lymphadentitis.[14] Fever is rare, and the condition is usually self-limiting unless the patient has had radiotherapy or is immunocompromised.[14] The pain that is experienced can be very debilitating, causing loss of sleep and affecting daily function. This pain poorly responds to over-the-counter and narcotic analgesics and can radiate to the temple, ear, and neck.[15] Dry socket is not characterized by redness, swelling, fever, or pus formation.[2] Histologic features include remnants of the blood clot and a massive inflammatory response characterized by neutrophils and lymphocytes which may extend into the surrounding alveolus.[16]

ETIOLOGY

Several theories have been presented on the etiology of dry socket. They include bacterial infection, trauma, and biochemical agents.[10] Birn in 1973 showed increased fibrinolytic activity and activation of plasminogen to plasmin in the presence of tissue activators in dry sockets.[16] This fibrinolytic activity is thought to affect the integrity of the postextraction blood clot.[2]

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Birn also stated that the increase in fibrinolysis was unlikely to dissolve the blood clot before the second day after surgery, because the clot contains antiplasmin, which must be neutralized before clot dissolution can occur.[17]

In a normal postextraction socket, thrombin and fibrinogen together form a fibrin clot over which epithelium migrates.[18] Then, during granulation tissue formation, new blood vessels grow into the clot and clot degradation occurs through the activity of fibroblasts and fibrinolysis via plasmin before the start of osteoproliferation.[18]

Birn stated that the increased fibrinolytic activity in dry socket is elicited by enhanced liberation of tissue activators from the alveolar bone consequent to trauma or infection.[19] In additional experimental work, he showed that these tissue activators release bradykinins and kininogenases, enzymes that take an active role in pain generation.[20] Furthermore, Birn found that the plasmin-like activity in dry sockets was not present at normal extraction sites.[17,21] This theory would explain how clot degradation occurs with accompanying pain when no redness, pus, or swelling is evident. However, use of transexamic acid, a tissue activator inhibitor, failed to reduce the incidence of dry socket,[22] whereas use of 2-(acetyloxy)benzoic acid mixed with propyl 4-hydroxybenzoate (Apernyl), a plasmin inhibitor, was successful.[23] Nitzan proposed that the plasmin described by Birn was not activated by tissue activators but was an independent product.[24] The use of local antibiotics, they argue, also does reduce dry socket, which is inconsistent with the concept of tissue activators. It was already known that bacterial products are used to treat thromboembolic disease by increasing fibrinolysis[8]; therefore, the implication of bacteria as the producers of plasmin-like products was made. In particular, Nitzan proposed that *Treponema denticola* was known to multiply and lyse blood clots without producing the clinical symptoms characteristic of infection, such as redness, swelling, or pus formation, and had been previously isolated from dry sockets.[24] *Treponema denticola* is also an anaerobic bacteria that was previously implicated in periodontal disease and is able to produce the fetid odor and bad taste characteristic of dry socket.[11] As will be discussed subsequently, antibiotics that are specific to anaerobes seem to reduce the incidence of dry socket.[10] Finally *T. denticola* exhibits plasmin-like fibrinolytic activity while other common oral bacteria have little such innate activity[25] and is a late colonizer of the mouth, which implicated it further because dry socket rarely occurs in childhood.[2] No papers, however, have been able to support a direct cause-effect relationship between bacteria and dry socket.[26]

RISK FACTORS

McGregor did a follow-up study of 10,000 extractions under local anesthesia and suggested gender, age, site of extraction, traumatic extractions, and smoking as predisposing factors.[27] These findings are collaborated by numerous articles.[14] Additional risk factors that have been presented include presence of pericoronitis,[2] high pre- and postoperative bacterial counts,[28,29] and inadequate irrigation.[3]

Gender

Sweet and Butler[30] observed that dry socket occurs in 4.1% of female patients versus 0.5% of men, and Tjernberg[31] reported a 5-fold increase in the incidence of the condition in females. Lilly[32] found that dry socket occurred 3 times more frequently in females taking oral contraceptives than in those who were not, and a recent study by Garcia et al.[33] supported this finding. Indeed it has been shown that estrogen in oral contraceptives can elevate plasma fibrinolytic activity,[34] which could in turn affect the postextraction clot stability. Catellani et al.[35] stated that the probability of dry socket increases with increased estrogen dosage in the oral contraceptive and that fibrinolytic activity appears to be lowest on days 23 to 28 of the menstrual cycle, because the first 21 days of the tablet cycle are active estrogen days whereas the next 7 days are free of estrogen. Women who had extraction during days 23 through 28 of the menstrual cycle had a reduced rate of dry socket.[5]

Age

Most literature supports that dry socket rarely occurs in childhood and that incidence increases with the patient's age, although the exact age bracket of highest incidence varies among different papers.[2,26,36] McGregor's follow-up study placed the highest incidence in third and fourth decade of life.[27] However, the prevalence of smoking in that age bracket might be a confounding factor.[37]

Trauma

Although some authors have not found a correlation between surgical trauma and dry socket,[38,39] the majority of the literature supports this relationship.[5,10,16,27,40,41] Trauma is postulated to result in compression of the bone lining the socket as well as possible thrombosis of the underlying vessels, reducing blood perfusion.[8] Some associate trauma with a reduction in tissue resistance and consequently wound infection by anaerobes.[8,42] Birn relates damage to cells and alveolar bone to release of tissue activators of fibrinolytic activity (factor XII or Hage-

man factor, urokinase from blood, tissue, and endothelial plasminogen activators).[16]

Smoking

Smoking has been shown to reduce neutrophil chemotaxis and phagocytosis, and impede production of immunoglobulin.[43] Meechan et al.[43] studied the outcome of 3,541 extractions and showed that smoking significantly reduced the immediate postextraction filling of sockets with blood and that there was a higher incidence of dry socket in heavy smokers (>20 cigarettes/day) compared with nonsmokers. Furthermore, sockets with reduced immediate postextraction blood filling showed a significantly greater incidence of dry socket.[41] Meechan et al.[43] stated that nicotine is known to be absorbed through the oral mucosa and to act as a vasoconstrictor. Sweet and Butler[44] found, in a study of 400 mandibular extractions, that the incidence of dry socket was substantially greater in smokers than in nonsmokers (6.4% vs. 1.4%, respectively), with patients who smoked >10 cigarettes/day having a 12% chance of developing the condition and those who smoked >1 pack/day having a 20% chance. The incidence of dry socket increased to 40% if the patient smoked either on the day of the surgery or within the first 24 h after surgery. Removal of the clot through suction and negative pressure during smoke inhalation has also been suggested.[44,45] "Shisha" or water pipe smoking has been shown to have similar effects as cigarette smoking on incidence of dry socket.[46]

Pericoronitis

In a study of 942 patients,[47] 14.1% of patients with pre-existing pericoronitis developed alveolar osteitis compared with 6.6% of patients without the condition, with a significant reduction in the incidence when prophylactic antibiotics were used. This was verified by subsequent studies.[7,39] Interestingly, Nitzan was able to isolate *T. denticola* from sites of pericoronitis.[24]

Inadequate irrigation

Butler at al.[10] conducted a study on bilateral impacted mandibular third molars in 211 patients where one side was irrigated with 175 mL of saline and the other with 25 mL. In that study, 12 instances of dry socket occurred in the high-volume lavage group and 23 in the low-lavage group, a statistically significant result. One postulated benefit of lavage is the reduced bacterial contamination of the socket.[18]

Anesthetic use

The literature is divided concerning the effects of local anesthetic on the incidence of dry socket. Dry socket is known to occur in cases of extractions under general anesthesia where no local anesthetic was used,[8] and Tsirlis et al.[48] showed that periodontal ligament (PDL) anesthesia did not result in an increase frequency of dry socket compared with block anesthesia. Similarly, Turner,[7] in a prospective study involving 1,274 extractions, found that forceful infiltration of an extra 2 mL of anesthetic resulted in a higher but statistically insignificant incidence of dry socket. Some investigators do not think that the vasoconstrictor in local anesthesia has a major outcome on dry socket.[49] However, a study by Meechan et al.[50] involving 1,533 single nonsurgical tooth extractions in male patients showed that use of repeated PDL anesthetic injections increased the likelihood of dry socket formation to 10.9% compared with 2.1% for single infiltration or block injection. Repeat use of conventional infiltration or block also increased the incidence of dry socket to 5.4%. Those authors also found that dry socket was more prevalent where prilocaine (Citanest) with 1:200,000 epinephrine was used versus lidocaine (Xylocaine) with 1:100,000 epinephrine. The authors argue that the epinephrine could reduce bleeding and might interfere with oxygen tension, thus reducing healing. They also state that epinephrine has been shown to increase fibrinolysis. Therefore it is the opinion of some investigators to exert caution with repeat PDL anesthetic injections.[4]

PREVENTIVE MEASURES

Antibiotics

The prevention of dry socket has in the past involved both pharmacologic and surgical approaches. Pharmacologic methods used in the prevention of dry socket have included use of antibiotic preparations placed into the socket after extraction and antiseptic rinses. Use of tetracycline-impregnated gelatin sponges or Gelfoam,[15,51] clindamycin-impregnated Gelfoam,[11] lincomycin in Gelfoam,[52] systemic use of metronidazole,[53] systemic penicillins,[54] and systemic use of erythromycin[55] have all shown a statistically significant ability to reduce the incidence of dry socket. Preoperative administration of these drugs also appears more effective than postoperative administration.[56] Placement of tetracycline in 200 postextraction sockets was recently shown to reduce postextraction pain and trismus, although there wase no significant effect on incidence of dry socket in that study.[57] Caution must be taken with dry tetracyclien powder, because it has been linked to a giant-cell reaction in 1 case, although aqueous tetracyclin has not shown such a reaction.[58]

In a recent review, Blum[8] stated that although penicillin, clindamycin, and erythromycin have had positive reports, of all systemic antibiotics metronidazole has shown the greatest promise in randomized double-

blind studies. Blum went further to state that metronidazole has a narrower spectrum and targets primarily anaerobes, therefore reducing the chance of bacterial resistance as well as being associated with fewer side effects than erythromycin, penicillin, or clindamycin. A study by Rood and Danford[6] showed significant pain relief within 24 h of use of metronidazole.

These findings support that anaerobic organisms are potentially involved in the pathogenesis of dry socket. However, a number of authors, including Fazakerley and Field,[14] recommend that the use of systemic antibiotics is not necessary in nonimmunocompromised patients unless there are symptoms of malaise and lymphadenopathy. They recommend that the use of antibiotics in the extraction socket be reserved for those with history of multiple dry sockets or for immunocompromised patients.

Chlorhexidine rinses

In view of the hazards of indiscriminate use of antibiotics, research was directed into looking at the effects of chlorhexidine rinses on dry socket. Larsen,[3] in a prospective randomized double-blind study evaluating the incidence of dry socket in 278 impacted third molars, found a significant 60% reduction in incidence of dry socket when a chlorhexidine rinse was given before the extraction (15 mL 2×/day 1 week before and 1 week after extraction), although concurrent use of intraoperative steroid injections in that study might have reduced its validity. Rango and Szkutnik[59] showed a 50% reduction in dry socket in patients who prerinsed for 30 s with a 0.12% chlorhexidine gluconate solution. Hermesch et al.[60] found a 38% reduction in the incidence of dry socket in patients who rinsed 30 s with 15 mL 0.12% chlorhexidine gluconate for 1 week before and 1 week after the extraction, with no side effects due to chlorhexidine use reported. Similarly Tjernberg[31] found that good plaque control and oral hygiene can reduce the incidence of dry socket after removal of mandibular third molars.

TREATMENT OF DRY SOCKET

On average, a time period of 7-10 days is required for exposed bone to become covered with new granulation tissue, and efforts must be made to relieve patient discomfort during this time period.[11] Various packing materials have been proposed, and Bloomer[61] recently showed that dry socket can be prevented by immediately packing sockets with a eugenol-containing dressing. However, such measures also are known to delay wound healing.[8] Similarly, Turner[9] used reflection of a flap, removal of bone particles, curettage, and removal of granulation tissue with irrigation and found that this method required fewer visits than zinc oxide–eugenol or iodoform gauze with eugenol techniques. Turner also stated that packing of the socket could delay socket healing and increase risk of an infection. Irrigation is known to remove debris, sequestra, and bacteria from the denuded bone.[62] If packing is to be used, Fazakerley and Field[14] recommended removal of sutures and gentle irrigation with warm saline under local anesthesia before application of a protective dressing composed of zinc oxide and eugenol mixed into a semisolid consistency applied to an iodoform ribbon gauze. The packing should be changed every 2 to 3 days and removed once pain is reduced. The use of petroleum-based carriers is discouraged to avoid myospherulosis, a complication of wound healing by action of lipids on extravasated erythrocytes.[18]

Choice of analgesics varies from a short course of nonsteroidal antiinflammatory drugs to narcotic-based preparations such as acetaminophen with codeine, hydroxycodone, or oxycodone. Colby[5] also recommended giving the patient a plastic syringe for home irrigation. Most authors presently do not recommend curetting the socket as a means of eliciting bleeding, because this procedure can increase pain.[5,8,62] Betts et al.[62] used 2% lidocaine jelly in a prospective double-blind study of 30 adult patients diagnosed with dry socket and found that the experimental group had significantly lower pain perception immediately and up to 60 min after irrigation than in those sockets that had been treated with placebo. No side effects due to topical lidocaine use were found.

SUMMARY

The occurrence of dry socket in an everyday oral surgery or dental practice is unavoidable. The risk factors for this temporary and debilitating condition are clearly identified. However, adherence to superb surgical technique in a young, healthy, and nonsmoking male patient still carries a 1%-4% incidence of dry socket. Surgeons must recognize additional risk factors in patients with particular medical conditions and include this information as a part of the informed consent.

Treatment options for this condition are generally limited and directed toward palliative care. The surgical site should be irrigated, avoiding curetting the extraction socket. Packing with a zinc oxide–eugenol paste on iodoform gauze can be considered to relieve acute pain episodes. Ultimately it is the host's healing potential which determines the severity and duration of the condition.

Prevention methods include avoiding smoking 24 h before and after surgery and atraumatic surgery with removal of bone and tooth fragments under copious saline irrigation. Placement of topical antibiotics, such as tetracycline, lincomycin, or clindamycin, on Gel-

foam can be considered, whereas systemic antibiotics should be reserved for patients who are immunocompromised. Other preventive measures, such as 1 week of preoperative chlorhexidine rinsing or performing surgery during the last week of the female menstrual cycle are impractical to be included in a surgical protocol for exodontias.

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