Alveolar Osteitis-A Review

Dr. R.Hariharan, Dr.N.Aravindha Babu, Dr.K.M.K.Masthan, Dr.R.Jayasri Krupaa,

Reader, Department of Oral pathology and Microbiology

Sree Balaji Dental College and Hospital

Bharath Institute of Higher Education and Research

ABSTRACT-

Alveolar osteitis or dry socket is one of the most common complication associated with the extraction of teeth. It is a common complication encountered by general dentist and specialists in day to day practice. A lot of research and literature has been attributed towards it. It is important to have a knowledge about this common post extraction complication. This review article discusses the etiology, risk factors, preventive measures and management of alveolar osteitis.

KEYWORDS:dry socket, complication, extraction, alveolalgia.

INTRODUCTION:

Alveolar Osteitis (ALVEOLAR OSTEITIS) is a notable complicationafter extraction or surgical removal of tooth. Generally known as "dry socket" this condition stays a typical postoperative concern that causes serious pain and frequent practice/clinic visits [1, 2].this adds up to increased treatment cause and metal agony to the patients. The specific pathogenesis of alveolar osteitis isn't surely known. Numerous scientists have contemplated alveolar osteitis, yet most ideas are still subject to noteworthy debate. The aim of this article is to discuss the various etiology, prevention and management of dry socket in clinical practice.

TERMINOLOGY:

"Dry socket" was first described in the literature in 1896 by Crawford [3]. Since then, other terms have been used to refer to this complications, such as "alveolar osteitis", "alveolitis", "localized osteitis", "alveolitis sicca dolorosa", "localized alveolar osteitis", "fibrinolytic alveolitis, "septic socket", "necrotic socket", and "alveolalgia", among others [4–6]. "Dry socket", which is the generic term, and "alveolar osteitis" are more commonly used terms.

DEFINITION:

The most recent defines alveolar osteitis as "postoperative pain inside and around the extraction site, which increases in severity at any time between the first and third day after the extraction, accompanied by a partial or total disintegrated blood clot within the alveolar socket with or without halitosis" [4]. The literature is abundant with diverse descriptive definitions for alveolar osteitis, which probably leads to the discrepancy in the diagnostic criteria. Several authors have agreed that pain and empty alveolus are found in all patients with alveolar osteitis [7-10]. Other defining factors that have been reported are radiating pain towards the ear and temporal region , rare maxillary involvement in ocular and frontal regions , halitosis , seldom low-grade fever , inflamed gingival margin , bare bone , ipsilateral regional lymphadenopathy , and greyish discharge [11-13].

INCIDENCE:

The incidence of alveolar osteitis for routine dental extractions is reported to be around 0.5%-5%[11]. The incidence of alveolar osteitis after surgical removal of third molars is higher. It is found to be around 1%-37.5%[12]. It has been well documented that the chances of developing a alveolar osteitis is approximately 10 times higher in cases of surgically removed teeth. The reported incidences are still controversial due to the lack of high quality evidence in respect alveolar osteitis.

ONSET:

the onset of alveolar osteitis is considered to occur 1–3 day after tooth extraction [14]. 95–100% of all cases of alveolar osteitis have been reported within a week [11].

ETIOLOGY:

The exact pathogenesis of alveolar osteitis is still not known. Birn published a series of articles between 1963-1977 for better understanding of the pathogenesis[7]. He suggested that there occurs a disintegration of the clot due to an increased local fibrinolytic activity. The fibrinolysis is the result of plasminogen pathway activation, which can be accomplished via direct (physiologic) or indirect (nonphysiologic) activator substances [7]. Direct activators are released after trauma to the alveolar bone cells. Indirect activators are elaborated by bacteria. The fibrinolytic activity is local because initial absorption of plasminogen into the clot limits the activity of plasmin[7]. In fact, it was found that active plasmin is inactivated in the general circulation by antiplasmins [15]. Birn and others have further reviewed the local differences in the fibrinolytic activity between body tissue and found higher fibrinolytic activity with bone and uterine tissues, in comparison to skeletal muscle, kidney, heart, brain, liver, spleen, lung, and thyroid tissues [16,17]. But the factors capable of triggering fibrinolysis are more ambiguous.

RISK FACTORS:

- ***Surgical Trauma and Difficulty of Surgery:*** Most authors agree that the trauma from surgery and difficulty of surgery play an important role in the development of alveolar osteitis [18]. The reason responsible might be due to the insult caused to the bone marrow during the traumatic procedure. This may lead the release of direct tissue activators which lead to bone marrow inflammation[19]. Surgical extractions, in comparison to nonsurgical extractions, result in a 10-fold increase incidence of alveolar osteitis [5]. Lilly et al. [8] found that surgical extractions involving reflection of a flap and removal of bone are more likely to cause alveolar osteitis.
- ***Lack of operator experience***. Larsen [2] concluded that surgeon's inexperience could be related to a bigger trauma during the extraction, especially surgical extraction of mandibular third molars. Alexander [6] et al reported a higher incidence of alveolar osteitis following extractions performed by the less experienced operators. Therefore the skill and experience of the operator should be taken into consideration as a risk factor.
- ***Mandibular third molars***. It has been shown that alveolar osteitis is more common following the extraction of mandibular third molars [5]. The reasons that are believed to be responsible for this include the increase in bone density, decrease in vascularity and reduced capacity tom produce granulation tissue [20]. However, there is no evidence suggesting a link between alveolar osteitis and insufficient blood supply. The area specificity is probably due to the large percentage of surgically extracted mandibular molars and may reflect the effect of surgical trauma rather than the anatomical site [19].
- ***Systemic Disease.*** Some researchers have suggested that systemic disease could be associated with alveolar osteitis[7, 10]. It is proposed that immunocompromised or diabetic patients are prone to development of alveolar osteitis due to altered healing . But no scientific evidence exists to prove a relationship between systemic diseases and alveolar osteitis.
- ***Oral Contraceptives***. Oral contraceptive is the only medication associated with developing alveolar osteitis. Oral contraceptives became popular in 1960s and studies conducted after 1970s show a significant higher incidence of alveolar osteitis in females. Sweet and butler [21] found that this increase in the use of oral contraceptives positively correlates with the incidence of alveolar osteitis. Oestrogen has been proposed to play a significant role in the fibrinolytic process. It is believed to indirectly activate the fibrinolytic system (increasing factors ii, vii, viii, x, and plasminogen) and therefore increase lysis of the blood clot [21]. Catellani et al. [22] further concluded that the probability of developing alveolar osteitis increases with increased Oestrogen dose in the oral contraceptives.
- ***Patient's gender***. Many authors claim that female gender, regardless of oral contraceptive use, is a predisposition for development of alveolar osteitis. Macgregor[23]reported a 50% greater incidence of alveolar osteitis in women than that in men.
- ***Smoking:*** Various researches have shown a strong corelation between smoking and alveolar osteitis. Among a sum of 4000 carefully eliminated mandibular third molars, patients who smoked a half-pack

of cigarettes daily had a four-to five-overlap increment in alveolar osteitis (12% versus 2.6%) when contrasted with non-smokers. The frequency of alveolar osteitis expanded to over 20% among patients who smoked a pack for each day and 40% among patients who smoked upon the arrival of medical procedure [21]. Blum guessed that this phenomenon could be because of the presentation of unfamiliar substance that could go about as a toxin in the careful site [4].

- ***Physical dislodgement of the clot***. Although a very commonly examined hypothesis, but no proof exists that physical dislodgement of the blood clot brought about by manipulation or negative pressure made by means of sucking on a straw would be a significant supporter of alveolar osteitis [4].
- ***Bacterial infection***. Most investigations uphold the case that bacterial contaminations are a significant danger for the improvement of alveolar osteitis. It has been indicated that the recurrence of alveolar osteitis increments in patients with poor oral hygiene and existing periodontal problems. A potential relationship of Actinomyces viscosusand Streptococcusmutans in alveolar osteitis was concentrated by rozantis et al. [24], where they showed deferred recuperating of extraction destinations after vaccination of these microorganisms in creature models. Nitzan et al. [25] observed high plasmin-like fibrinolytic exercises from colonies of treponema denticola, a microorganism present in periodontal infection. Catenalli[22] contemplated bacterial pyrogens in vivo and proposed that they are aberrant activators of fibrinolysis.
- ***Excessive irrigation or curettage of alveolus***. It has been proposed that unnecessary repetitive irrigation of alveolus might meddle with clotr arrangement and that rough curettage might harm the alveolar bone [7]. Nonetheless, the writing needs proof to affirm these charges in the improvement of ALVEOLAR OSTEITIS.
- ***Age of the Patient***. Little literature can be found concerning whether age is related with top occurrence of alveolar osteitis.
- ***Single extraction versus multiple extractions***. Limited proof exists showing higher prevalence of alveolar osteitis after single extractions versus different extractions [9,10].in addition, different extractions including periodontally unhealthy teeth might be less traumatic
- ***Local anaesthetic with vasoconstrictor***: it has been recommended that the utilization of local anaesthetics with vasoconstrictors builds the frequency of alveolar osteitis. Lehner [26] found that alveolar osteitis recurrence increments with invasion sedation on the grounds that the transitory ischemia prompts helpless blood gracefully. The investigations that followed demonstrated acknowledged that local ischemia because of vasoconstrictor in nearby sedation has no part in the advancement of alveolar osteitis.
- ***Saliva*** : a couple of investigators have contended that salivation is a danger factor in the improvement of alveolar osteitis but no firm logical proof exists to help this case. Birn found no proof that salivation assumes a part in alveolar osteitis [7].
- ***Bone/root fragments remaining in the wound.*** A few authors have recommended that bone/root parts leftovers could add to theimprovement of alveolar osteitis [4, 7].contrast to this, simpson, in his examination, observed that little bone/root sections are regularly present after extractions and these pieces don't really cause intricacies as they are frequently externalized by the oral epithelium [27].
- ***Flap design/use of sutures***. Some past literatures claim that design of a flap and the utilization of stitches influence the development of alveolar osteitis. However, later examinations discovered little proof to demonstrate such relationship [28]. In the nonappearance of any huge proof, it is sensible to accept that these are not major contributing components.

PREVENTION:

Since alveolar osteitis is the most common postoperative complication after extraction, many researchers have attempted to find a successful method for prevention. Numerous methods and techniques are proposed throughout the existing literature to assist with prevention of alveolar osteitis. However, this area remains a controversial topic as no single method has gained universal acceptance. The most popular of these techniques are discussed below.

- ***Systemic antibiotics***. Systemic antibiotics reported to be effective in the prevention of alveolar osteitis include penicillins , clindamycin , erythromycin and metronidazole [29]

- T***opical antibiotics***. A great number of studies have been performed in order to test the effectiveness of topical medicaments in preventing alveolar osteitis. The antibiotics studied have been used alone or in combination with differing doses and formulations.. Amongst the many antibiotics studied, topical tetracycline has shown promising results [30]. The reported method of delivery included powder, aqueous suspension, gauze drain, and gelfoam sponges (preferred). However, side-effects including foreign body reactions have been reported with the application of topical tetracycline [31].
- ***Chlorhexidine***. Several studies have reported that the pre- and perioperative use of 0.12% chlorhexidine decreases the frequency of alveolar osteitis after mandibular third molar removal .Ragno et al. [32] found as much as 50% reduction in the incidence of alveolar osteitis in patients who prerinsed with chlorhexidine solution. Caso et al. [33]after a meta-analysis of the available studies concluded that 0.12% chlorhexidine rinse on the day of surgery and for several days thereafter is beneficial.
- ***Para-hydroxybenzoic acid.*** Early literature reported that the topical use of para-hydroxybenzoic acid (phba), an antifibrinolytic agent, in extraction wounds decreased the incidence of alveolar osteitis. In addition, phba has been reported to have some antimicrobial properties [13].
- ***Tranexamic acid***. Tranexamic acid (tha), an antifibrinolytic agent, has been speculated to prevent alveolar osteitis when applied topically in the extraction socket. But a study by gersel-pedersen[34] did not show a significant reduction in the incidence of alveolar osteitis when compared to a placebo group. Local plasminogen inactivation alone was insufficient to cease the development of alveolar osteitis.
- ***Polylactic acid***. Polylactic acid (pla), a clot supporting agent, is a biodegradable ester that once was thought to be the ultimate solution for the prevention of alveolar osteitis. It was suggested that pla would provide a stable support for the blood clot and subsequent granulation and osteoid tissue. However, follow-up studies failed to support the success of pla. Complications were observed and the reported incidence of alveolar osteitis was actually higher when pla was used.
- ***Eugenol containing dressing***. Some authors have promoted the use of eugenol containing dressing to prevent development of alveolar osteitis [35]. However, irritant local effect of eugenol and the delay in wound healing due to prophylactic packing has been well documented in the literature and may be difficult to justify its use to prevent alveolar osteitis
- ***Sterile gloves.*** The use of sterile gloves instead of clean nonsterile gloves has not demonstrated a decrease in the incidence of alveolar osteitis and therefore not necessary [36]

MANAGEMENT:

The management of ALVEOLAR OSTEITIS is less controversial than its etiology and prevention. Most agree that the primary aim of dry socket management,is pain control until commencement of normal healing, and in the majority of cases local measures are satisfactory. In some instances, systemic analgesics or antibiotics may be necessary or indicated. The use of intra-alveolar dressing materials is widely suggested in the literature although it is generally acknowledged that dressings delay healing of the extraction socket. Different medicaments and carrier systems are commercially available with little scientific evidence to guide a selection process as demonstrated above. As the various formulations are reviewed, it becomes apparent that all of them are simply varying combinations of perhaps 18 different ingredients. Alvogyl (Septodent, Inc, Wilmington, DE) has been widely used in the management of ALVEOLAR OSTEITIS and is frequently mentioned in the literature. Alvogyl contains butamben (anesthetic), eugenol (analgesic), and iodophorm (antimicrobial). It is not recommended for use in extraction sockets.

CONCLUSION:

Despite of multiple years of research,little advancement has been made in tending to this regularly experienced postoperative complication in patients. The literature with respect to alveolar osteitis isn't reliable and frequently controversial. The full etiology of alveolar osteitis has not been established and also, varying definitions and management measures exist. The commonly prescribed medications are easily available but the current collection of writing doesn't offer enough help for the treating professional to manage any adverse effects that may occur. The formula for management begins from strict instruction and patients with

recognizable risk factors ought to be educated . Further examinations and well planned investigations are important to make firm inferences and to explain this complication.

REFERENCES:

1. T. P. Osborn, G. Frederickson Jr., I. A. Small, and T. S. Torgerson, "A prospective study of complications related to mandibular third molar surgery," Journal of Oral and Maxillofacial Surgery, vol. 43, no. 10, pp. 767–769, 1985.
2. P. E. Larsen, "Alveolar osteitis after surgical removal of impacted mandibular third molars: identification of the patient at risk," Oral Surgery Oral Medicine and Oral Pathology, vol. 73, no. 4, pp. 393–397, 1992.
3. J. Y. Crawford, "Dry socket," Dental Cosmos, vol. 38, pp. 929– 931, 1896.
4. I. R. Blum, "Contemporary views on dry socket (alveolar osteitis): a clinical appraisal of standardization, aetiopathogenesis and management: a critical review," International Journal of Oral and Maxillofacial Surgery, vol. 31, no. 3, pp. 309–317, 2002.
5. D. Torres-Lagares, M. A. Serrera-Figallo, M. M. Romero-Ru´ız, P. Infante-Coss´ıo, M. Garc´ıa-Calderon, and J. L. Guti ´ errez- ´ Perez, "Update on dry socket: a review of the literature," ´ Medicina Oral, Patologia Oral y CirugiaBucal, vol. 10, no. 1, pp. 77–85, 2005.
6. R. E. Alexander, "Dental extraction wound management: a case against medicating postextraction sockets," Journal of Oral and Maxillofacial Surgery, vol. 58, no. 5, pp. 538–551, 2000.
7. H. Birn, "Etiology and pathogenesis of fibrinolytic alveolitis ('dry socket')," International Journal of Oral Surgery, vol. 2, no. 5, pp. 211–263, 1973
8. G. E. Lilly, D. B. Osbon, E. M. Rael, H. S. Samuels, and J. C. Jones, "Alveolar osteitis associated with mandibular third 8 International Journal of Dentistry molar extractions," Journal of the American Dental Association, vol. 88, no. 4, pp. 802–806, 1974.
9. E. A. Field, J. A. Speechley, E. Rotter, and J. Scott, "Dry socket incidence compared after a 12 year interval," British Journal of Oral and Maxillofacial Surgery, vol. 23, no. 6, pp. 419–427, 1985.
10. A. J. MacGregor, "Aetiology of dry socket: a clinical investigation," British Journal of Oral Surgery, vol. 6, no. 1, pp. 49–58, 1968
11. A. E. Swanson, "Prevention of dry socket: an overview," Oral Surgery Oral Medicine and Oral Pathology, vol. 70, no. 2, pp. 131–136, 1990.
12. K. L. Fridrich and R. A. J. Olson, "Alveolar osteitis following surgical removal of mandibular third molars," Anesthesia Progress, vol. 37, no. 1, pp. 32–41, 1990.
13. P. J. Vezeau, "Dental extraction wound management: medicating postextraction sockets," Journal of Oral and Maxillofacial Surgery, vol. 58, no. 5, pp. 531–537, 2000.
14. W. E. Shafer, M. K. Hine, and B. M. Levy, Textbook of Oral Pathology, Saunders, Philadelphia, Pa, USA, 4th edition, 1993.
15. M. A. Lucas, L. J. Fretto, and P. A. McKee, "The binding of human plasminogen to fibrin and fibrinogen," Journal of Biological Chemistry, vol. 258, no. 7, pp. 4249–4256, 1983.
16. H. Birn, "Fibrinolytic activity of normal alveolar bone," Acta Odontologica Scandinavica, vol. 29, no. 2, pp. 141–153, 1971.
17. J. C. Southam and G. H. Moody, "The fibrinolytic activity of human and rat dental pulps," Archives of Oral Biology, vol. 20, no. 12, pp. 783–786, 1975
18. J. H. Brekke, M. Bresner, and M. J. Reitman, "Effect of surgical trauma and polylactate cubes and granules on the incidence of alveolar osteitis in mandibular third molar extraction wounds," Journal of the Canadian Dental Association, vol. 52, no. 4, pp. 315–319, 1986.
19. Y. M. Nusair and M. H. Abu Younis, "Prevalence, clinical picture, and risk factors of dry socket in a Jordanian Dental Teaching Center," Journal of Contemporary Dental Practice, vol. 8, no. 3, pp. 53–63, 2007.
20. N. A. Amaratunga and C. M. Senaratne, "A clinical study of dry socket in Sri Lanka," British Journal of Oral and Maxillofacial Surgery, vol. 26, no. 5, pp. 410–418, 1988.
21. J. B. Sweet and D. P. Butler, "Predisposing and operative factors: effect on the incidence of localized osteitis in mandibular third-molar surgery," Oral Surgery, Oral Medicine, Oral Pathology, vol. 46, no. 2, pp. 206–215, 1978

22. J. E. Catellani, S. Harvey, S. H. Erickson, and D. Cherkin, "Effect of oral contraceptive cycle on dry socket (localized alveolar osteitis)," Journal of the American Dental Association, vol. 101, no. 5, pp. 777–780, 1980
23. M. E. Cohen and J. W. Simecek, "Effects of gender-related factors on the incidence of localized alveolar osteitis," Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology and Endodontology, vol. 79, no. 4, pp. 416–422, 1995.
24. J. Rozanis, I. D. Schofield, and B. A. Warren, "Is dry socket preventable?" Dental Journal, vol. 43, no. 5, pp. 233–236, 1977.
25. D. Nitzan, J. F. Sperry, and T. D. Wilkins, "Fibrinolytic activity of oral anaerobic bacteria," Archives of Oral Biology, vol. 23, no. 6, pp. 465–470, 1978.
26. T. Lehner, "Analysis of one hundred cases of dry socket," Dental Practitioner and Dental Record, vol. 8, pp. 275–279, 1958.
27. H. E. Simpson, "The healing of extraction wounds," British Dental Journal, vol. 126, no. 12, pp. 550–557, 1969.
28. D. G. Kirk, P. N. Liston, D. C. Tong, and R. M. Love, "Influence of two different flap designs on incidence of pain, swelling, trismus, and alveolar osteitis in the week following third molar surgery," Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology and Endodontology, vol. 104, no. 1, pp. e1–e6, 2007.
29. W. R. Laird, D. Stenhouse, and T. W. Macfarlane, "Control of post-operative infection. A comparative evaluation of clindamycin and phenoxymethylpenicillin," British Dental Journal, vol. 133, no. 3, pp. 106–109, 1972.
30. D. C. Sorensen and J. W. Preisch, "The effect of tetracycline on the incidence of postextractionalveolar osteitis," Journal of Oral and Maxillofacial Surgery, vol. 45, no. 12, pp. 1029–1033, 1987.
31. J. R. Zuniga and J. C. Leist, "Topical tetracycline-induced neuritis: a case report," Journal of Oral and Maxillofacial Surgery, vol. 53, no. 2, pp. 196–199, 1995.
32. J. R. Ragno Jr. and A. J. Szkutnik, "Evaluation of 0.12% chlorhexidine rinse on the prevention of alveolar osteitis," Oral Surgery Oral Medicine and Oral Pathology, vol. 72, no. 5, pp. 524–526, 1991.
33. A. Caso, L.-K. Hung, and O. R. Beirne, "Prevention of alveolar osteitis with chlorhexidine: a meta-analytic review," Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology and Endodontology, vol. 99, no. 2, pp. 155–159, 2005.
34. N. Gersel-Pedersen, "Tranexamic acid in alveolar sockets in the prevention of alveolitis sicca dolorosa," International Journal of Oral Surgery, vol. 8, no. 6, pp. 421–429, 1979.
35. C. R. Bloomer, "Alveolar osteitis prevention by immediate placement of medicated packing," Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology, and Endodontics, vol. 90, no. 3, pp. 282–284, 2000.
36. L. K. Cheung, L. K. Chow, M. H. Tsang, and L. K. Tung, "An evaluation of complications following dental extractions using either sterile or clean gloves," International Journal of Oral and Maxillofacial Surgery, vol. 30, no. 6, pp. 550–554, 2001.