Journal of Dental Reports
ISSN: 2583-2255
Malik AM, 2022-J Dent Rep
Research Article

Alveolar Osteitis: A Latest Review

Arshad Mahmood Malik*

Dean, Professor Department of Oral and Maxillo-facial Surgery, HBS Dental College, Islamabad, Pakistan.

***Corresponding Author:** Arshad Mahmood Malik, Dean, Professor Department of Oral and Maxillo-facial Surgery, HBS Dental College, Islamabad, Pakistan.

Received Date: 07-09-2022

Accepted Date: 07-25-2022

Published Date: 08-10-2022

Abstract

Dry socket or Alveolar Osteitis (AO) is amongst the most common complications following extraction of teeth in dentistry. A great number of research literature is available to alveolar osteitis with reference to its etiology and pathophysiology. Many studies are available for techniques to prevent AO but controversy still exists regarding the actual etiology, pathophysiology, methods of prevention and treatment. Review of the concepts and controversies surrounding AO is an aim of this article.

Keywords: Dry socket; Alveolar osteitis; Postoperative pain; Radiating pain.

Introduction

"Dry socket" was first described in 1896 by Crawford [1]. Many other terms also been referred to these complications, such as septic socket", "localized osteitis "alveolalgia", alveolar osteitis", necrotic socket", "alveolitis sicca dolorosa", "alveolitis", "localized alveolar osteitis" and "fibrinolytic alveolitis" [2,3]. In spite of acceptance of Birn's theory by many authors, the term fibrinolytic osteitis is not commonly used [2,3]. The "alveolar osteitis" is more commonly used while "Dry socket" is referred as general public term. Approximately there are more than eighteen definitions of AO [2]. The most accepted defines AO 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"[2] Sever postoperative pain results in excessive use of medications, repeated hospital visits hence increase financial, psychological and physical burden to the patient while delayed recovery period increased cost to the surgeon as well [1,2]. The previous studies regarding pathogenesis of dry socket are yet not well understood [1-3]. The studied-on AO are still subject to controversies regarding pathophysiology of risk factors and contributing factors. Birn, labeled it as fibrinolytic alveolitis with reference to understanding of the pathophysiology [4,5].

Citation: Malik AM. Alveolar Osteitis: A Latest Review. J Dent Rep. 2022;3(1):1-11.
DOI: https://doi.org/10.37191/Mapsci-2583-2255-3(1)-023

The pain in empty alveolus is commonly present in all patients with AO [4,6,7]. The other signs and symptoms some time may exaggerate the intensity of AO like radiating pain towards the ear and temporal region [8,9] maxilla, frontal, and ocular regions [4] seldom low-g-rade fever [8,9], inflamed gingival margin [10] greyish discharge [11], bare alveolar bone [10] ipsilateral regional lymphadenopathy [8,9], and halitosis [8,12]. Simple dental extractions reported the incidence of AO has been in the range 0.5% to 5% [13-16]. In mandibular third molars extraction it varies from 1% to 37.5% [17,18]. While surgical extractions about 10 times higher incidence are reported [2]. About 95–100% of all cases of AO appear within a week [13] generally AO onset is considered to occur 1–3 day after tooth extraction [8,19,20].

Etiology

Birn suggested that the etiology of AO is an increased local fibrinolysis leading to disintegration of the clot [4]. The fibrinolysis is the result of plasminogen pathway activation. The activator substances in AO are direct (physiologic) or indirect (non-physiologic) [5]. Due to trauma to the alveolar bone cells the direct activators are released while bacterial streptokinase release indirect activators. The fibrinolytic activity is limited to local area because initial absorption of plasminogen into the clot limits the activity of plasmin. The active plasmin is inactivated in the general circulation by antiplasmins [21]. Birn and many researchers revealed the local differences in the fibrinolytic activity between different body tissues. Higher fibrinolytic activity was observed with bone and uterine tissues, in comparison to, thyroid tissues, heart, kidney, brain, spleen, liver, lung, and skeletal muscle [22,23]. The factors responsible of triggering fibrinolysis are found to be more ambiguous. The risk factors and contributing factors for development of dry socket has been reviewed by many researchers [4,5,21-23].

Risk factors

The risk factors reported are, Systemic Disease, Oral Contraceptives, Smoking, Bacterial Infection, Excessive Irrigation or Curettage of Alveolus, Local Anesthetic with Vasoconstrictors, Bone/Root Fragments Remaining in the Wound and different Flap Design/Use of Suture.

Systemic disease

Many systemic diseases reported to be associated with AO [4,6]. The most commonly immunocompromised and especially diabetic patients being prone to development of alveolar osteitis due to altered healing [24]. Conditions in Which Pre-existing Alveolar Bone Hypovascularity, such as Vascular or Hematological Disorders, Radiotherapy-induced Osteonecrosis, Cemento-osseous Dysplasia, Osteopetrosis, Paget's Disease the occurrence of AO is controversial.

Oral contraceptives

Oral contraceptive is well known drug associated with dry socket. Oral contraceptives show a significant higher incidence of AO in females [25-27]. Estrogen plays a significant role in the fibrinolytic process, Sweet and Butler [28] suggested that increase in the use of oral contraceptives directly correlates with the AO incidence. Studies suggested that oral contraceptives indirectly activate the fibrinolytic activity by increasing factors II, VII, VIII, X, plasminogen hence increase the lysis of blood clot in AO [29]. It has been

also found that occurrence of AO enhanced with the increase of estrogen dose in oral contraceptives by Catellani, et. al [30]. To reduce the risk of hormonal cycle's involvement in AO it is suggested for scheduling the elective surgical exodontia [27].

Smoking

A dose dependent relationship between smoking and the occurrence of alveolar osteitis has been studied. 4000 surgically removed mandibular third molars, patients who smoked a half-pack of cigarettes a day had developed AO a four- to five-fold increase (12% versus 2.6%) as compared to non-smokers. AO increased to more than 20% who smoked a pack per day, but it increased to 40% who smoked on the surgery day [31]. However, the exact mechanism like direct local affect including heat or suction for the increase of AO incidence is not very clear [32]. The introduction of foreign substance by means of smoke fumes could act as a contaminant in the extraction wound [33].

Microbial manifestation

It has been documented in most studies that bacterial infections are a major risk factor for the generation and growth of AO. The recurrence of AO increases in patients with risk factors such as poor OH [34] preexisting local infections such as pericoronitis [35] as well as periodontitis. The isolation of the causative organisms has been made via cultures.

Rozantis, et. al [36] demonstrated delayed healing of extraction sites after the inoculation of specific microorganisms such as Actinomyces viscosus and Streptococcus mutans, in animal models. Cultures of Treponema denticola, a periodontal disease microorganism, exhibited high plasmin like fibrinolytic projections, according to Nitzan et. al [37]. Catenalli [38] studied bacterial pyrogens in vivo and proposed that they are indirect activators of fibrinolysis.

Excessive cleansing or curettage of socket

Studies have hypothesized that repeated and excessive irrigation of alveolus might hinder the clot formation and that aggressive curettage might also be injurious to the alveolar bone, both of which may lead to the formation of dry socket [4]. However, the literature lacks evidence to certify these claims put forth in the development of AO.

Vasoconstrictors with local infiltration

The use of local anesthesia with vasoconstrictors increases the risk of AO. Lehner [39] studied that because of the poor blood supply due to infiltration anesthesia, a temporary ischemia is induced which increases the frequency of AO. However, followed studies suggested that ischemia lasts for one to two hours and is followed by reactive hyperemia, which is a negligible factor in the disintegration of blood clot [4,40].

Moreover, in a study it was documented that there is no significant difference in the AO of a tooth extracted with infiltration anesthesia versus regional block anesthesia with vasoconstrictor [32]. Therefore, it is currently accepted that local anesthesia with vasoconstrictor has no role in the development of local ischemia which can lead to the formation of AO.

Bony fragment remnants in the wound site

Chow O and H Birn have suggested in their studies that bone/root fragments and debris can lead to delayed wound healing, and

consequently aid in the development of AO [2,4]. Simpson, on the other hand, in his study, showed that small bone/root fragments are commonly present after extractions and these fragments do not necessarily cause complications as the epithelium is able to form an external barrier [41].

Design of the flap and suture usage

Some previous literature claims that flap design and the use of sutures affect the development of AO [25]. However, little evidence is found to authenticate such relationship in studies that have recently been conducted [42]. In the absence of any significant evidence, it is practical to assume that these are not major contributing factors [3].

Contributing factors

The contributing factors in pathophysiology of AO are, Lack of Operator Experience, Mandibular Third Molars, Patient's Gender, Age, and Physical Dislodgement of the Clot, Single Extraction versus Multiple Extractions, Saliva, Surgical Trauma and Difficulty of Surgery

Operator's inexperience

Operator's inexperience is also considered as a major risk factor for the development of AO. A study carried out by Larsen [43] stated that surgeon's lack of experience during the surgical extraction of third molars can lead to deleterious consequences.
Alexander and Oginni, et. al [44]. have reported an increased incidence in AO carried out by inexperienced surgeons. Henceforth operator's skills and experience should be considered [3,43,44].

Extraction of mandibular wisdom teeth

Many studies have shown the same pattern of increased AO after extraction of third molar [45,46]. It is a common belief among some authors that increased density of the bone, decreased vascularity, and reduced capacity of formation of granulation tissue can lead to formation of AO [45]. However, no evidence could provide a nexus between AO and reduced blood supply. The reason why surgically extracted third molar are prone for the development of AO is due to surgical trauma, and not for their anatomic location [32].

Patient's gender

In many studies, it has been stated that female gender is more susceptible for the development of AO, despite the use of oral contraceptive pills. MacGregor [14] reported an increase of 50% occurrence of AO in females as compared to men in his study of 4000 extractions, whereas Colby had no difference to show in the same scenario and stated that there is no gender association with the development of AO [14].

Patient's age

There is little evidence to prove the association of incidence of AO with age. The literature supports the general principle of greater risk associated with old age [3]. Blondeau, et. al [47] concluded that surgical removal of impacted mandibular third molars should be carried out well before age of 24 years.

Clot dislodgement

In a contemporary opinion, there is no corroboration that dislodgement of blood clot caused by manipulation or negative

Citation: Malik AM. Alveolar Osteitis: A Latest Review. J Dent Rep. 2022;3(1):1-11.
DOI: https://doi.org/10.37191/Mapsci-2583-2255-3(1)-023

pressure is created by sucking a straw as a major contributor AO [32].

Single extraction compared to multiple extractions

Limited data exists indicating a higher prevalence of AO after single extractions [9,12] as compared to multiple extractions. In one study, AO prevalence was 7.3% following single extractions and 3.4% following multiple extractions [32]. This difference could be a possibility due to the fact that patients with single extractions have less pain as compared to the patients with multiple extractions, whose teeth are damaged drastically [48]. Moreover, multiple extractions involving periodontal disease teeth may be less traumatic extraction gave very promising results in prevention of AO [49].

Saliva

A few authors have argued that saliva is a risk factor in the development of AO [50,51]. However, no firm scientific evidence exists to support this claim. Birn found no evidence that saliva plays a role in AO [4].

Trauma due to surgery and difficulty of surgery

Many authors agree on the same point that trauma and difficulty while performing surgery can play a significant role in the development of AO [34,52,53]. This is because traumatic extractions lead to the production of direct tissue activators following bone marrow inflammation [32].
An increase in incidence of AO is seen by 10-folds in surgical extraction as compared to non-surgical extractions [24] Lilly, et. al [6] stated that surgical extraction inclusive of a flap design and bone removal are more prone towards the development of AO.

Prevention

Numerous techniques are proposed in existing literature for its prevention. However, no single method has gained solo acceptance. Most common techniques are discussed as under.

Systemic antibiotics

Systemic antibiotics including penicillins [54,55] clindamycin [54,56] erythromycin [56] and metronidazole [19,57] are used systemically pre/postoperative, however the prophylactical use is disputed due to development of resistant bacterial strains, hypersensitivity, and mainly killing of normal host commensals [2,58].

Topical antibiotics

The use of topical tetracycline has shown to be an auspicious drug amongst other local antibiotics [59-62]. Foreign body reactions such as Myospehrulosis have been reported with the application of from petroleum-based tetracycline-hydrocortisone combination [63-65].
Study reported a nerve dysesthesia six months after mandibular third molar extraction by the use of medications in the socket [64]. The method of delivery included powder, gauze drain, suspensions and Gelfoam sponges. It has been also suggested that virtually anything into the alveolus, including plain Gelfoam, will result in the improvement in AO symptoms [8].

Para-hydroxybenzoic acid

AO prevention by para-hydroxybenzoic acid (PHBA), an antifibrinolytic agent has been reported [66,67]. PHBA is available in alveolar cone that consists of acetylsalicylic acid and PHBA. A pernyl success in AO

proved good, but it is found that it inhibited the bone healing. PHBA has been reported to have good antimicrobial effects [9,68,69]. Aspirin cause local irritation and acute inflammation of the socket [70].

Chlorhexidine

Pre- and perioperative use of 0.12% chlorhexidine decreases appearance of AO after third molar extraction [29,40,71,72]. It has been reported that rinsing with chlorhexidine solution before extraction there is 50% reduction of AO [73]. 0.12% chlorhexidine rinse on the day of extraction gave very promising results in prevention of AO [49].

Polylactic acid

The polylactic acid (PLA), is a biodegradable ester (a clot supporting agent), has been advocated in prevention of AO. PLA provide a stable support for the blood clot, granulation, and osteoid tissue but few follow-up studies failed to support the PLA role in AO Complications and incidence of AO was higher when PLA was used [74,75].

Tranexamic acid

Tranexamic acid (transamin), an antifibrinolytic agent, has been recommended to prevent AO when applied topically or IV after in the extraction [74]. But when compared to a placebo group it did not show a significant reduction in the incidence of AO and local plasminogen inactivation found to be insufficient to cease the appearance of AO [75].

Steroids

Corticosteroid remained in use to decrease postoperative complications but to prevent development of dry socket no promising results found [76]. Topical application of hydrocortisone and antibiotics significantly lessens AO especially after wisdom molar removal [8,77]. It is observed the use of steroids alone without any antibiotic combination is not promising [78].

Eugenol containing dressing

The eugenol containing dressing to prevent development of AO has been very popular [79]. The local irritant effect and the delay in wound healing by eugenol made it be difficult to justify its use in prevention of dry socket [3,80,81].

Lavage

Butler and Sweet [42] reported that tht copious preoperative and intraoperative lavage reduce the incidence of dry socket reported significant reduction in AO, however they found that increase or decrease in the lavage volume do not affect its efficacy.

Aminoacrinide

9-aminoacridine is an antiseptic agent; its effectiveness in reducing the incidence of AO is found to be less effective [82].

Use of sterile gloves

The effect of sterile gloves uses instead of clean nonsterile gloves to decrease in the incidence of AO found to be very insignificant [83,84].

Management

The primary aim of dry socket management is pain control and promoting normal healing process. The majority of cases local measures are satisfactory. The systemic analgesics or antibiotics may be necessary or indicated in some cases. The intra-

alveolar dressing materials are widly use but may result in delayed healing of extraction socket [81]. Different medicaments combinations of perhaps 18 different ingredients are in use worldwide [4]. Alvogyl in the management of AO contains butamben (anesthetic), eugenol (analgesic), and iodophorm (antimicrobial) is frequently mentioned in the literature. The retardation of healing and inflammation is reported when the sockets were packed with Alvogyl. The use of ZnO eugenol, fibrin substitutes, whitehead varnish and BIPP has also been reported to be effective in management of AO.

Conclusion

The literature postoperative condition of alveolar osteitis is not consistent and often conflicting. The most previous studies are poorly designed, lack of ideal analysis, statistically biased, or consist of very individual opinions. Non established etiology of alveolar osteitis resulted in varying controversial descriptive definitions and diagnostic criterias. The initiation of fibrinolytic process appears to be interfacing multiple independent factors in wound healing of AO [3]. To prevent this complication no single universally acceptable method. Multitudes of intra-alveolar medicaments are available on the market however their complications/severe reactions from placed in the socket are rare. The management of this complication should begin with patient education and identifiable risk factors should be informed in detail about this anticipated complication of exodontia. Further well-designed studies based on latest investigations methodologies are necessary to draw firm conclusions of AO treatments.

References

1. JY Crawford. Dry socket, Dental Cosmos. 1896;(38):929-31.
2. Chow O, Wang R, Ku D, Huang WJ. Alveolar Osteitis: A Review of Current Concepts. Oral Maxillofac Surg. 2020;78(8):1288-96. PubMed | CrossRef
3. Alexander RE. Dental extraction wound management: a case against medicating postextraction sockets. J Oral Maxillofac Surg. 2000;58(5):538-51. PubMed | CrossRef
4. Birn H. Bacteria and fibrinolytic activity in “dry socket”. Acta Odontol Scand. 1970;28(6):773-83. PubMed | CrossRef
5. H Birn. Etiology and pathogenesis of fib7rinolytic alveolitis (“dry socket”). Int J Oral Maxillofac Surg. 1973;2(5):211-63. CrossRef
6. Lilly GE, Osbon DB, Rael EM, Samuels HS, Jones JC. Alveolar osteitis associated with mandibular third molar extractions. J Am Dent Assoc. 1974;88(4):802-6. PubMed | CrossRef
7. Fernandes GJ, Hatton MN. Prevention of Alveolar Osteitis. A Case Report and Review of Literature. N Y State Dent J. 2016;82(1):21-5. PubMed
8. Vezeau PJ. Dental extraction wound management: medicating postextraction sockets. J Oral Maxillofac Surg. 2000;58(5):531-7. PubMed | CrossRef
9. Vezeau PJ. Dental extraction wound management: medicating postextraction sockets. J Oral Maxillofac Surg. 2000;58(5):531-7. PubMed | CrossRef
10. Rakhshan V. Common risk factors of dry socket (alveolitis osteitis) following dental extraction: A brief narrative review. J Stomatol Oral Maxillofac Surg. 2018;119(5):407-11. PubMed | CrossRef
11. Laraki M, Chbicheb S, El Wady W. Alveolitis: review of the literature. Odontostomatol Trop. 2012;35(139):19-25. PubMed
12. Field EA, Speechley JA, Rotter E, Scott J. Dry socket incidence compared after a 12-year interval. Br J Oral Maxillofac Surg.1985;23(6):419-27. PubMed | CrossRef
13. MacGregor AJ. Aetiology of dry socket: a clinical investigation. Br J Oral Surg. 1968;6(1):49-58. PubMed | CrossRef

Citation: Malik AM. Alveolar Osteitis: A Latest Review. J Dent Rep. 2022;3(1):1-11.
DOI: https://doi.org/10.37191/Mapsci-2583-2255-3(1)-023

14. MacGregor AJ. Aetiology of dry socket: a clinical investigation. Br J Oral Surg. 1968;6(1):49-58. PubMed | CrossRef
15. Krogh HW. Incidence of dry socket. J Am Dent Assoc. 1937;24(11):1829-36.
16. Heasman PA, Jacobs DJ. A clinical investigation into the incidence of dry socket. Br J Oral Maxillofac Surg. 1984;22(2):115-22. PubMed | CrossRef
17. Cardoso CL, Rodrigues MT, Júnior OF, Garlet GP, de Carvalho PS. Clinical concepts of dry socket. J Oral Maxillofac Surg. 2010;68(8):1922-32. PubMed | CrossRef
18. Isiordia-Espinoza MA, Aragon-Martinez OH, Bollogna-Molina RE, Alonso-Castro ÁJ. Infection, alveolar osteitis, and adverse effects using metronidazole in healthy patients undergoing third molar surgery: a meta-analysis. J Maxillofac Oral Surg. 2018;17(2):142-9. PubMed | CrossRef
19. DW Nitzan. On the genesis of "dry socket". J Oral Maxillofac Surg. 1983;41(11):706-10. PubMed | CrossRef
20. Lucas MA, Fretto LJ, McKee PA. The binding of human plasminogen to fibrin and fibrinogen. J Biol Chem. 1983;258(7):4249-56. PubMed
21. Birn H. Fibrinolytic activity of normal alveolar bone. Acta Odontol Scand. 1971;29(2):141-53. PubMed | CrossRef
22. Southam JC, Moody GH. The fibrinolytic activity of human and rat dental pulps. Arch Oral Biol. 1975;20(12):783-6. PubMed | CrossRef
23. Torres-Lagares D, Serrera-Figallo MA, Romero-Ruíz MM, Infante-Cossío P, García-Calderón M, Gutiérrez-Pérez JL. Update on dry socket: a review of the literature. Med Oral Patol Oral Cir Bucal. 2005;10(1):81-5. PubMed
24. Schow SR. Evaluation of postoperative localized osteitis in mandibular third molar surgery. Oral Surg Oral Med Oral Pathol. 1974;38(3):352-8. PubMed
25. Sweet JB, Butler DP. Increased incidence of postoperative localized osteitis in mandibular third molar surgery associated with patients using oral contraceptives. Am J Obstet Gynecol. 1977;127(5):518-9. PubMed | CrossRef
26. Rakhshan V. Common risk factors of dry socket (alveolitis osteitis) following dental extraction: A brief narrative review. J Stomatol Oral Maxillofac Surg. 2018;119(5):407-11. PubMed | CrossRef
27. Ogata Y, Hur Y. A higher incidence of dry socket may be related to the use of oral contraceptives after impacted mandibular third-molar extraction. J Am Dent Assoc. 2016;147(10):840-2. PubMed | CrossRef
28. Sweet JB, Butler DP. Predisposing and operative factors: effect on the incidence of localized osteitis in mandibular third-molar surgery. Oral Surg Oral Med Oral Pathol. 1978;46(2):206-15. PubMed | CrossRef
29. Sweet JB, Butler DP. The relationship of smoking to localized osteitis. J Oral Surg. 1979;37(10):732-5. PubMed
30. Catellani JE, Harvey S, Erickson SH, Cherkin D. Effect of oral contraceptive cycle on dry socket (localized alveolar osteitis). J Am Dent Assoc. 1980;101(5):777-80. PubMed | CrossRef
31. Nusair YM, Younis MH. Prevalence, clinical picture, and risk factors of dry socket in a Jordanian dental teaching center. J Contemp Dent Pract. 2007;8(3):53-63. PubMed
32. Blondeau F, Daniel NG Extraction of impacted mandibular third molars: postoperative complications and their risk factors. J Can Dent Assoc. 2007;73(4):325. PubMed
33. Penarrocha M, Sanchis JM, Sáez U, Gay C, Bagán JV. Oral hygiene and postoperative pain after mandibular third molar surgery. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2001;92(3):260-4. PubMed | CrossRef
34. Kaposvári I, Körmöczi K, Horváth F, Buglyó A, Turai AR, Joób-Fancsaly Á. Incidence and case-control study of delayed-onset infection after lower third molar surgery. Orv Hetil. 2018;159(31):1278-83. PubMed | CrossRef
35. Haraji A, Motamedi MH, Rezvani F. Can flap design influence the incidence of alveolar osteitis following removal of impacted mandibular third molars?. Gen Dent. 2010;58(5):187-9. PubMed
36. J Rozanis, ID Schofield, BA Warren. "Is dry socket preventable?". Dent J. 1977;43(5):233-6. PubMed
37. Nitzan D, Sperry JF, Wilkins TD. Fibrinolytic activity of oral anaerobic bacteria. Arch Oral Biol. 1978;23(6):465-70. PubMed | CrossRef
38. Catellani JE. Review of factors contributing to dry socket through enhanced fibrinolysis. J Oral Surg. 1979;37(1):42-6. PubMed
39. Lehner T. Analysis of one hundred cases of dry socket. Dent Pract Dent Rec. 1958;8:275-9.

Citation: Malik AM. Alveolar Osteitis: A Latest Review. J Dent Rep. 2022;3(1):1-11.
DOI: https://doi.org/10.37191/Mapsci-2583-2255-3(1)-023

40. de França AJB, Costa RTF, Monteiro JLGC, Rocha NS, do Egito Vasconcelos BC, de Moraes SLD. Comparison of three anesthetic techniques for the removal of posterior mandibular teeth with 2% lidocaine: a systematic review. Clin Oral Investig. 2020;24(12):4143-52. PubMed | CrossRef
41. Simpson HE. The healing of extraction wounds. Br Dent J. 1969;126(12):550-7. PubMed
42. Butler DP, Sweet JB. Effect of lavage on the incidence of localized osteitis in mandibular third molar extraction sites. Oral Surg Oral Med Oral Pathol. 1977;44(1):14-20. PubMed | CrossRef
43. Canellas JVDS, Fraga SRG, Santoro MF, Netto JNS, Tinoco EMB. Intrasocket interventions to prevent alveolar osteitis after mandibular third molar surgery: A systematic review and network meta-analysis. J Craniomaxillofac Surg. 2020;48(9):902-13. PubMed | CrossRef
44. Oginni FO, Fatusi OA, Alagbe AO. A clinical evaluation of dry socket in a Nigerian teaching hospital. J Oral Maxillofac Surg. 2003;61(8):871-6. PubMed | CrossRef
45. Alsaleh MK, Alajlan SS, Alateeq NF, Alamer NS, Alshammary F, Alhobeira HA etal., Patient's Compliance with Post-extraction Instructions Following Permanent Teeth Extraction. J Contemp Dent Pract. 2018 1;19(12):1517-1524. PubMed | CrossRef
46. Jaafar N, Nor GM. The prevalence of post-extraction complications in an outpatient dental clinic in Kuala Lumpur Malaysia--a retrospective survey. Singapore Dent J. 2000;23(1):24-8. PubMed
47. Blondeau F, Daniel NG. Extraction of impacted mandibular third molars: postoperative complications and their risk factors. J Can Dent Assoc. 2007;73(4):325. PubMed
48. Birn H. Fibrinolytic activity of alveolar bone in "dry socket". Acta Odontol Scand. 1972;30(1):23-32. PubMed | CrossRef
49. Abu-Mostafa N, Al-Daghamin S, Al-Anazi A, Al-Jumaah N, Alnesafi A. The influence of intra-alveolar application of honey versus Chlorhexidine rinse on the incidence of Alveolar Osteitis following molar teeth extraction. A randomized clinical parallel trial. J Clin Exp Dent. 2019 1;11(10):871-e876. PubMed | CrossRef
50. Riba-Terés N, Jorba-García A, Toledano-Serrabona J, Aguilar-Durán L, Figueiredo R, Valmaseda-Castellón E. Microbiota of alveolar osteitis after permanent tooth extractions: A systematic review. J Stomatol Oral Maxillofac Surg. 2021;122(2):173-181. PubMed | CrossRef
51. Krakowiak PA. Alveolar osteitis and osteomyelitis of the jaws. Oral Maxillofac Surg Clin North Am. 2011;23(3):401-13. PubMed | CrossRef
52. Brekke JH, Bresner M, Reitman MJ. Effect of surgical trauma and polylactate cubes and granules on the incidence of alveolar osteitis in mandibular third molar extraction wounds. J Can Dent Assoc. 1986;52(4):315-9. PubMed
53. Colby RC. The general practitioner's perspective of the etiology, prevention, and treatment of dry socket. Gen Dent. 1997;45(5):461-7;471-2. PubMed
54. Çebi AT. Evaluation of the effects of intra-alveolar irrigation with clindamycin, rifampicin and sterile saline in alveolar osteitis treatment. J Stomatol Oral Maxillofac Surg. 2020;121(6):680-683. PubMed | CrossRef
55. Krekmanov L, Nordenram A. Postoperative complications after surgical removal of mandibular third molars. Effects of penicillin V and chlorhexidine. Int J Oral Maxillofac Surg. 1986;15(1):25-9. PubMed | CrossRef
56. Bystedt H, Nord CE, Nordenram A. Effect of azidocillin, erythromycin, clindamycin and doxycycline on postoperative complications after surgical removal of impacted mandibular third molars. Int J Oral Surg. 1980;9(3):157-65. PubMed | CrossRef
57. Isiordia-Espinoza MA, Aragon-Martinez OH, Bollogna-Molina RE, Alonso-Castro ÁJ. Infection, Alveolar Osteitis, and Adverse Effects Using Metronidazole in Healthy Patients Undergoing Third Molar Surgery: A Meta-analysis. J Maxillofac Oral Surg. 2018;17(2):142-149. PubMed | CrossRef
58. Ataoğlu H, Oz GY, Candirli C, Kiziloğlu D. Routine antibiotic prophylaxis is not necessary during operations to remove third molars. Br J Oral Maxillofac Surg. 2008;46(2):133-5. PubMed | CrossRef
59. Øyri H, Jonsdottir O, Jensen JL, Bjørnland T. The use of a tetracycline drain reduces alveolar osteitis: a randomized prospective trial of third molar surgery under local anesthetics and without the use of systemic antibiotics. Oral Surg Oral Med Oral Pathol Oral Radiol. 2019;128(3):205-212. PubMed | CrossRef
60. Davis WM Jr, Buchs AU, Davis WM. The use of granular gelatin-tetracycline compound after third molar removal. J Oral Surg. 1981;39(6):466-7. PubMed
61. Gelesko S, Long L, Faulk J, Phillips C, Dicus C, White RP Jr. Cryotherapy and topical minocycline as adjunctive measures to control pain after third molar surgery: an exploratory study. J Oral Maxillofac Surg. 2011;69(11):324-32. PubMed | CrossRef

62. Akota I, Alvsaker B, Bjørnland T. The effect of locally applied gauze drain impregnated with chlortetracycline ointment in mandibular third-molar surgery. Acta Odontol Scand. 1998;56(1):25-9. PubMed | CrossRef
63. Sanchis JM, Sáez U, Peñarrocha M, Gay C. Tetracycline compound placement to prevent dry socket: a postoperative study of 200 impacted mandibular third molars. J Oral Maxillofac Surg. 2004;62(5):587-91. PubMed
64. Zuniga JR, Leist JC. Topical tetracycline-induced neuritis: a case report. J Oral Maxillofac Surg. 1995;53(2):196-9. PubMed | CrossRef
65. Fisher SC, Horning GM, Hellstein JW. Myospherulosis complicating cortical block grafting: a case report. J Periodontol. 2001;72(12):1755-9. PubMed | CrossRef
66. Birn H. Antifibrinolytic effect of Apernyl in "dry socket". Int J Oral Surg. 1972;1(4):190-4. PubMed | CrossRef
67. Puidokas T, Kubilius M, Nomeika D, Januzis G, Skrodeniene E. Comparative Analysis of Blood Clot, Plasma Rich in Growth Factors and Platelet-Rich Fibrin Resistance to Bacteria-Induced Fibrinolysis. Microorganisms. 2019;7(9):328. PubMed | CrossRef
68. Ritzau M, Swangsilpa K. The prophylactic use of propylic ester of p-hydrobenzoic acid on alveolitis sicca dolorosa. A preliminary report. Oral Surg Oral Med Oral Pathol. 1977;43(1):327. PubMed | CrossRef
69. Kjellman O. Apernyl as alveolar inlay in connection with the removal of impacted third molars of the lower jaw. A clinical double blind investigation of 100 patients. Sven Tandlak Tidskr. 1973;66(2):197-200. PubMed
70. Pankhurst CL, Lewis DA, Clark DT. Prophylactic application of an intra-alveolar socket medicament to reduce postextraction complications in HIV-seropositive patients. Oral Surg Oral Med Oral Pathol. 1994;77(4):331-4. PubMed | CrossRef
71. Tjernberg A. Influence of oral hygiene measures on the development of alveolitis sicca dolorosa after surgical removal of mandibular third molars. Int J Oral Surg. 1979;8(6):430-4. PubMed | CrossRef
72. Cho H, David MC, Lynham AJ, Hsu E. Effectiveness of irrigation with chlorhexidine after removal of mandibular third molars: a randomised controlled trial. Br J Oral Maxillofac Surg. 2018;56(1):54-59. PubMed | CrossRef
73. Abu-Mostafa N, Al-Daghamin S, Al-Anazi A, Al-Jumaah N, Alnesafi A. The influence of intra-alveolar application of honey versus Chlorhexidine rinse on the incidence of Alveolar Osteitis following molar teeth extraction. A randomized clinical parallel trial. J Clin Exp Dent. 2019;11(10):e871-e876. PubMed | CrossRef
74. Hooley JR, Golden DP. The effect of polylactic acid granules on the incidence of alveolar osteitis after mandibular third molar surgery. A prospective randomized study. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 1995;80(3):279-83. PubMed | CrossRef
75. Moore JW, Brekke JH. Foreign body giant cell reaction related to placement of tetracycline-treated polylactic acid: report of 18 cases. J Oral Maxillofac Surg. 1990;48(8):808-12. PubMed | CrossRef
76. Chen YW, Chi LY, Lee OK. Revisit incidence of complications after impacted mandibular third molar extraction: A nationwide population-based cohort study. PLoS One. 2021-22;16(2):0246625. PubMed | CrossRef
77. Rutledge JL, Marcoot RM. Terra-Cortril/Gelfoam for reduction of the incidence of localized osteitis following mandibular third molar removal. J Oral Med. 1984;39(1):51-3. PubMed | CrossRef
78. Saghiri MA, Asatourian A, Sheibani N. Angiogenesis and the prevention of alveolar osteitis: a review study. J Korean Assoc Oral Maxillofac Surg. 2018;44(3):93-102. PubMed | CrossRef
79. Bloomer CR. Alveolar osteitis prevention by immediate placement of medicated packing. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2000;90(3):282-4. PubMed | CrossRef
80. Riba-Terés N, Jorba-García A, Toledano-Serrabona J, Aguilar-Durán L, Figueiredo R, Valmaseda-Castellón E. Microbiota of alveolar osteitis after permanent tooth extractions: A systematic review. J Stomatol Oral Maxillofac Surg. 2021;122(2):173-181. PubMed | CrossRef
81. Schatz JP, Fiore-Donno G, Henning G. Fibrinolytic alveolitis and its prevention. Int J Oral Maxillofac Surg. 1987;16(2):175-83. PubMed | CrossRef
82. Johnson WS, Blanton EE. An evaluation of 9-aminoacridine/Gelfoam to reduce dry socket formation. Oral Surg Oral Med Oral Pathol. 1988;66(2):167-70. PubMed | CrossRef
83. Cheung LK, Chow LK, Tsang MH, Tung LK. An evaluation of complications following dental extractions using either sterile or clean gloves. Int J Oral Maxillofac Surg. 2001;30(6):550-4. PubMed | CrossRef
84. Adeyemo WL, Ogunlewe MO, Ladeinde AL, Bamgbose BO. Are sterile gloves necessary in nonsurgical dental extractions? J Oral Maxillofac Surg. 2005;63(7):936-40. PubMed | CrossRef

Citation: Malik AM. Alveolar Osteitis: A Latest Review. J Dent Rep. 2022;3(1):1-11.
DOI: https://doi.org/10.37191/Mapsci-2583-2255-3(1)-023