

Alveolar Osteitis: A Review of Current Concepts and Management protocols.

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Abstract: Alveolar osteitis or dry socket is one of the most common complication after tooth extraction especially in mandibular third molars. A lot of studies have been undertaken to investigate the etiology, pathogenesis, preventive methods and treatment of this condition but the results are inconclusive. The article aims to give an insight into the various facets of alveolar osteitis to help the dental practitioner in discerning and eliminating the risk factors as well as preventive and symptomatic management of this condition.

Keywords: Alveolar osteitis, Fibrinolysis, Alveolagia, Management.

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I. Introduction

Alveolar osteitis or Dry socket is a postoperative complication after tooth extraction which often impedes the healing process. It occurs in 0.5 to 5% of normal extractions and 1 to 37.5% of mandibular third molar extractions [1]. The first description of dry socket was given by Crawford in 1896. The term dry socket was used because the socket had a dry appearance after the clot and debris was washed away [2]. Subsequently various terminologies like alveolar osteitis, alveolitis sicca dolorosa, septic sockets and necrotic socket has been proposed [3]. Blum [4] defined alveolar osteitis as "postoperative pain inside and around extraction site which increases in severity at any time between the first and third day post extraction followed by a partial or total disintegrated blood clot within the alveolar socket with or without halitosis". Other features of alveolar osteitis include pain in ear and temporal region, low grade fever, bare bone, ipsilateral regional lymphadenopathy, inflamed gingival margin and greyish discharge [5].

Etiopathogenesis

The exact etiology of alveolar osteitis is still unknown even though several factors have been implicated. Poor oral hygiene, vasoconstrictors and reduced blood supply are the prime causes. Increased emphasis has been placed on difficult extraction causing fibrinolysis and release of pain producing chemicals. Birn [6] observed high concentration of plasma and increased fibrinolytic activity in alveolar bone lining dry socket lesion. Plasminogen, the precursor of plasmin circulates in blood and binds to clots at wound site. Tissue type and urokinase type plasminogen activators convert plasminogen to plasmin. Plasminogen induce fibrinolysis to dissolve blood vessel clots, increase capillary permeability and increase inflammation. The plasminogen tissue activation in post extraction socket induce fibrinolysis that dislodge the post extraction clot causing dry socket [7]. Mamoun [8] proposed that in a high stress extraction where high compressive forces are exerted on alveolar bone surrounding the tooth, events in a 24-96 hour period cause necrosis of osteoblasts surrounding the socket. The osteoblastic necrosis initiate fibrinolytic activity lysing the blood clot. At the same time socket bleeding stops causing a socket ischemia resulting in failure to resorb necrotic bone cells. Necrotic bone cells are exposed and uncovered for several days causing acute pain till the bone is covered by healing epithelium.

II. Risk Factors

1. Surgical Trauma

Surgical trauma and difficulty of surgery are key players in development of alveolar osteitis. Surgical extraction shows a ten fold increase of alveolar osteitis compared to nonsurgical extraction [3]. Open surgeries with flap reflection also showed a higher incidence of Alveolar osteitis [9]. Trauma from extraction and aggressive curettage caused inflammation of medulla of alveolar bone which release cell mediators that initiate fibrinolysis [6].

2. Site of Extraction

More in maxilla than in mandible because of increased bone density, decreased vascularity and reduced capacity of producing granulation tissue. Mandibular third molars are the most commonly affected site [10].

3. Gender of Patient

Most studies have shown that females have a higher predisposition for development of alveolar osteitis. In a study by Cohen et al [11], females were shown to have at least a two to threefold increase in osteitis compared to males probably due to increased oral contraceptive usage.

4. Experience of the Operator

Several studies have shown that operator experience plays a vital role in alveolar osteitis development especially with regard to mandibular third molar extractions. Larsen [12] in his study of 138 impacted third molars showed increased risk of alveolar osteitis in patients treated by inexperienced surgeons.

5. Smoking

The incidence of alveolar osteitis is significantly higher in smokers (12%) compared to non smokers (4%). There is a strong association between amount of smoking and incidence of dry socket [13]. Cryer et al [14] postulated that smoking is associated with release of endogenous catecholamines resulting in vasoconstriction and decreased tissue perfusion. Sweet et al [15] suggested that heat from burning tobacco together with suction applied by cigarette can cause clot dislodgement and affect healing.

6. Oral Contraceptives

The only medication that can cause alveolar osteitis is oral contraceptives. Catellani [16] et al found significant increase in frequency of alveolar osteitis after extraction of mandibular molars in patients with oral contraceptive use. They found that frequency is maximum in 1-22 days of 28 day cycle and is minimized from 23-28 days. Estrogen is believed to indirectly activate the fibrinolytic system and thereby lyse the blood clot [17].

7. Microbial Infections

Several studies have implicated the role of microorganisms in alveolar osteitis. Nitzan [18] assayed 13 species of anaerobic microorganisms in oral cavity for fibrinolytic activity and observed high fibrinolytic activity from cultures of *Treponema denticola* which was extracellular. Rocaniset al [19] highlighted the possible association of *Actinomyces viscosus* and *Streptococcus mutans* in alveolar osteitis. As bacteria increase in number in alveolar osteitis, and because certain species constantly secrete pyrogens at basal level, it has been postulated that bacterial pyrogens indirectly activate fibrinolysins in vivo [20].

8. Effect of Vasoconstrictors

Some studies have indicated that local anaesthetic vasoconstrictors are responsible for development of alveolar osteitis. However extractions done under general anaesthetic have also shown development of alveolar osteitis. Some studies showed that periodontal intraligamental injection showed higher incidence of alveolar osteitis compared to block injection [21]. Tsorlisset al [22] in his studies indicated that periodontal ligament anaesthesia did not result in high frequency of alveolar osteitis.

9. Excessive irrigation of socket

Al. Hindi [24] investigated the correlation between dry socket and excess saline irrigation in extraction of 574 teeth with half the group having no saline irrigation and the other half having their sockets irrigated with 20cc saline. Saline irrigation showed no positive correlation with dry socket development.

III. Prevention

Various researchers have tried to evolve methods for prevention of alveolar osteitis with little success. Several techniques have been proposed in the literature but several grey areas still exist.

1. Chlorhexidene

Chlorhexidene is a bisguamide antiseptic used as a mouthwash and preoperative irrigant. In a study by Field et al [24] where 0.2% (w/v) chlorhexidene gluconate solution was applied preoperatively on the gingival crevice of the patient after local anaesthetic administration, a significant reduction in dry socket compared to normal saline administration was observed. A similar study by Hermeschet al [25] using 0.12% chlorhexidene gluconate showed a significant reduction in incidence of alveolar osteitis. Bonine et al [26] in a study using 0.12% chlorhexidene gluconate rinse (Peridex) on extraction of 3rd molar sites post surgically for two weeks showed significant reduction in incidence of dry socket.

2. Antibiotics

Systemic antibiotics have shown significant reduction in the incidence of dry socket. Ramos et.al[27] in a systemic review and meta-analysis of systemic antibiotic use (Pencillins and Nitroimidazoles) after third molar extractions have shown significant reduction in risk of alveolar osteitis. Kupfer[28] in his study of 765 patients treated with Clindamycin has shown a remarkable reduction in incidence of alveolar osteitis. Sanchis et.al[29] used tetracycline compound placement in 200 extracted third molar sockets post removal and noted that there was no significant difference in dry socket incidence with its use.

3. Antifibrinolytics

Birn[30] investigated the antifibrinolytic activity of Aperiyl (3mg acetylsalicylic acid, 3 gm propyl-hydroxyl- benzoate, 20 mg tablet mass) has a pronounced inhibitory effect on fibrinolytic activity of dry socket. The inhibition of the plasmin and activator activity was complete. Anand et.al[31] studied the efficacy of tranexamic acid, an antifibrinolytic agent in comparison with a placebo with respect to reduction of incidence of alveolar osteitis after extraction of mandibular molars in 60 patients. The results supported the use of tranexamic acid both locally and systemically in reduction of alveolar osteitis in mandibular molars.

4. Antimicrobial Photo Dynamic Therapy (aPDT)

Low incidence of dry socket after using antimicrobial photo dynamic therapy seems to hold great promise in prevention of alveolar osteitis. Neugebauer et.al[32] used antimicrobial photo dynamic therapy (aPDT) with HELBO Blue and TheraLite laser on 100 patients in 130 jaws with one or more contralateral teeth removed at 1 week intervals. Results showed a significant lower incidence of alveolar osteitis in the aPDT group for the first and eighth post surgical days.

5. Low Level Laser Therapy (LLLT)

LLLT showed significant increase in wound healing when compared to Alveogyl group. Both Low power red laser (LPRL) 660nm and Low power infra red laser (LPIRC) 810 nm showed significant reduction on pain compared to subjects using Alveogyl. The 660 nm laser was most effective on second and third day and hence low level re laser should be investigated as an alternative to Alveogyl for treatment of alveolar osteitis[33].

IV. Management

1. Irrigation

Irrigation of dry socket post extraction has been traditionally followed using heated saline solution, gauze using iodoform, and prescription of codeine followed by irrigation of sodium perborate[34]. In a recent study by Motamedi[35] where the question arose whether to irrigate or not to irrigate an immediate post extraction socket, the conclusion was that post surgical irrigation of socket increases chance of development of alveolar osteitis especially in older patients. Socket bleeding and formation of blood clot was necessary for good osseous healing of the socket.

2. Dressings

The most useful socket healing medicaments include broad spectrum antibiotics specifically tetracyclins and clindamycin. Gelatin sponge, poly lactic acid and methyl cellulose are used as clot stabilizing socket implants. The record of such agents in preventing alveolar osteitis is mixed[36]. Other medicated dressings used include antibacterials, topical anaesthetics and obtundents like zinc oxide impregnated cotton pellets, Alvogyl (eugenol, iodoform and butamen), Dentalone, bismuth subnitrate and iodoform paste (BIPP) on ribbon gauze and metanidazole and iodoform paste[37].

3. Analgesics

NSAIDs are commonly recommended in treating alveolar osteitis. Ogunlewe[38] recommended pharmacologic treatment in combination with saline irrigation and curettage. They recommended 1mg acetaminophen hours post operatively and 1mg 8th hourly for next 24 hours which gave satisfactory results.

V. Conclusion

Lot of research has gone into the mechanism of alveolar osteitis but definitive etiology is still inconclusive. The preventive methods and treatment options are still highly ambiguous. Lot of studies need to be done to unravel the mystery of this annoying condition.

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