

PATHOGENESIS AND MANAGEMENT OF DRY SOCKET (ALVEOLAR OSTEITIS)

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SUMMARY

Alveolar osteitis is a common post extraction complication developing within 1-4 days following surgery.¹⁻³ It is characterized by intense pain that is not relieved by analgesics.¹ The most frequent site of occurrence is the mandibular third molar region. Females are known to show a higher incidence of occurrence.⁴ Studies indicate smoking, traumatic extractions, leaving tooth and bone debris in extraction sockets, excessive irrigation of socket, compromised blood supply and use of contraceptives, as possible predisposing factors.⁵⁻²⁵ Inflammation of the socket is believed to be a result of dislodgment or disintegration of the blood clot that forms within the socket immediately after extraction.^{11,26-27} Dry socket can be prevented by ensuring sterile surgery and by the use of numerous non-pharmacological measures; good history, identification and elimination of risk factors, and pharmacological agents; systemic antibiotics, antiseptics, antifibrinolytics, obtundant dressings and photodynamic therapy.^{8,28-34} Once the condition develops it is treated symptomatically and by use of obtundant dressings. Initial results of a study carried out in Khyber College of Dentistry, Peshawar are showing promising results with surgical management. This literature review summarizes the current understanding of etiology, pathogenesis, prevention and management of alveolar osteitis.

Key words: Alveolar osteitis, Dry socket, pathogenesis, management

INTRODUCTION

Alveolar Osteitis (dry socket) can be defined as the inflammation of the extraction socket occurring 1-4 days post operatively, characterized by intense throbbing pain, accumulation of disintegrated clot and food debris in the socket and malodor.^{1,2} 95-100% patients report within 7 days of surgery with pain.³

The term dry socket was first coined by Crawford in the year 1896.¹ Local osteitis, post operative osteitis, alveoalgia, alveolitis sicca dolorosa, septic osteitis, necrotic socket, localized osteomyelitis and fibrinolytic alveolitis have also been used in reference to this condition.

Females are more prone to developing alveolar osteitis due to the use of contraceptives (discussed later).⁴ Age groups above 30 also show a higher rate of onset.⁴

ETIOLOGICAL AND PREDISPOSING FACTORS

Alveolar osteitis is known to have a multifactorial origin.⁵ Some of the more common etiological and predisposing factors are discussed below:

The role of bacteria in developing symptoms of alveolar osteitis has been long recognized, predisposing patients with poor oral hygiene and preexisting infections to developing dry socket.^{6,7} Taking proper antibacterial measures during surgery can reduce the incidence of dry socket.⁸ Rozanis et al⁹ narrated the association of *Actinomyces viscosus* and *Streptococcus mutans* with the condition by showing the delayed healing of socket after placement of organisms in animal models. Nitzan et al¹⁰ advocated the significance of anaerobic organisms in relation to the development of dry socket by observing the fibrinolytic activity from the anaerobic culture of *Treponema denticola* which is considered to be the causative organism in development of periodon-

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tal conditions. Certain bacterial species secrete pyrogens that are indirect activators of fibrinolysis *in vivo*. Catellani¹¹ studied the efficacy of bacteria in thromboembolic conditions by injecting the pyrogens intravenously which resulted in a sustained increase in fibrinolytic activity.

Incidence of alveolar osteitis increases with excessive trauma during extraction, especially in procedures that involve reflection of flap and excessive removal of bone.¹² Mandibular third molar surgery is a relatively difficult and long procedure involving flap reflection, grinding into dense bone and tooth splitting. Hence, the third molar area is the most common site of dry socket occurrence.¹² Excessive trauma causes compression of the bone lining the socket impairing vascular penetration. Subsequently excessive trauma can lead to the thrombosis of the underlying vessel. Some authors have narrated that trauma leads to decrease tissue resistance and wound infection.¹³ Birn¹⁴ and Nusair¹⁵ propose that excess trauma to the bone causes inflammation of the bone marrow and results in direct tissue activator release into alveolus and predispose to dry socket.

Birn¹⁴ and Simpson¹⁶ have contradictory views concerning the contribution of tooth and bone fragments in the incidence of dry socket. Birn¹⁴ suggests that bone and tooth fragments have a significant role in the onset of dry socket.

Certain studies indicate that irrigation can dislodge the newly formed blood clot.¹⁷ Newer literature however provides proof that provision of sufficient irrigation is one of the key factors in preventing dry socket.¹⁸ It allows for the removal of enamel and dentin powder formed during tooth splitting and bone fragments from the extraction socket that may act as an impediment in the healing process. Curettage was suggested to have a deleterious effect on the healing process in previous literature. Newer studies present surprisingly favorable results in prevention and healing of dry socket wound.

Thick cortical bone is thought to be responsible for poor perforation of blood supply to the posterior mandibular region hence resulting in a higher incidence of dry socket.¹⁹ However, certain studies contest the fact by pointing out the larger blood network in the posterior region as compared to the anterior.¹⁴ Some investigations have suggested the vasoconstrictors in intraligamentary injection are responsible for dry socket.²⁰ Sufficient literature is present challenging the earlier notion.²¹

There is significant documented evidence linking a higher incidence of alveolar osteitis with smoking.¹² Sweets and Butler¹² associated the negative pressure built within the mouth during smoking as one of the

possible reasons for clot dislodgment. However, this assertion remains largely conflicted. Cigarette smoking is also believed to retard the healing process of dry sockets.²²

The use of contraceptives increased after the 1960s, as did the incidence of dry socket. Several studies postulate that there appears to be a direct correlation of contraceptive use with dry socket. Alveolar osteitis occurs more frequently within the age group of 20-40 years⁽⁵⁾. Contraceptives which contain estrogen effect the coagulation system.²³ Ygge²⁴ found an increase in fibrinolytic activity in women taking contraceptives. Ygge's results were confirmed by Hedlin and Monkhouse²⁵ who demonstrated an increase in fibrinolysis after 24 hours of the first dose of contraceptives. A decrease of fibrinolytic activity was noted when the use of contraceptives was discontinued; pyrogens cause indirect activation of fibrinolysis and contribute to the occurrence of dry socket by increasing fibrinolysis. Oral contraceptive use favors the appearance of dry socket and postoperative pain after extraction.¹¹

PATHOGENESIS

Fibrinolysis is a normal physiologic process that removes fibrin deposits by enzymatic digestion of the fibrin meshwork into smaller soluble fragments. Fibrin is continually being laid down and removed in the body as injury and repair occur. Local increase in fibrinolysis occur in response to bleeding. Minor variations in plasma fibrinolytic activity in individuals and between individuals are normal but gross changes are associated with disease.¹¹

Lysis and destruction of the blood clot is caused by tissue kinases liberated during inflammation by a direct or indirect activation of plasminogen in the blood. When direct tissue activators are released after trauma to the alveolar bone cells, plasminogen (which is laid down in the fibrin network as it is formed) is converted to plasmin, resulting in the clot dissolution by disintegration of fibrin. This conversion is accomplished in the presence of tissue or plasma pro-activators and activators.

These activators have been recently classified as direct (physiologic) and indirect (nonphysiologic) and further sub classified according to their origin as intrinsic (in the plasma) and extrinsic (outside the plasma) activators.²⁶ Direct extrinsic activators include tissue plasminogen activators and endothelial plasminogen activators. Indirect activators include substances such as streptokinase and staphylokinase, which are produced by bacteria and bind to plasminogen to form an activator complex that then cleaves other plasminogen molecules to plasmin. This strengthens the theory of the involvement of micro-organisms in the development of dry socket.

Birn²⁷ attributes the cause of pain to the presence and formation of kinin locally in the socket. It has been shown that kinin in concentrations as low as 1 mg/ml is able to produce intense pain.²⁷

PREVENTION OF DRY SOCKET

Prevention of alveolar osteitis can be either pharmacological or non pharmacological. Non pharmacological measures include taking a good history, identification and if possible, elimination of risk factors.

Pharmacological intervention can be done by one of the following agents;

Systemic antibacterials are reported to have some benefit in the prevention of alveolar osteitis. Studies showing favorable results with Penicillin, Clindamycin, Erythromycin and Metronidazole use are available.⁸ Some researchers however, have found no significant difference in the incidence of dry socket with the use of systemic antibiotics.²⁸

The use of chlorhexidine as both a mouth rinse and as a preoperative irrigant has shown to significantly reduce the quantity of oral microbial population. It is reported to have a significant contribution in lowering the incidence of dry socket.²⁹

Certain studies have associated investigated the implication of the use of topical parahydroxy benzoic acid and tranexamic acid in the prevention and treatment of dry socket. The studies have mostly found no beneficial effect associated with the use of these agents.^{30,31}

Studies have revealed that placement of obtundent eugenol containing dressing following extraction results in reduction of dry socket.² Eugenol is however also associated with delaying the healing process.³²

Polymer polylactic acid was used in 1980 as the ultimate solution of dry socket and is available today by the name of Drilac. Investigations conducted by Hooley and Gordon have reported a higher incidence of dry socket associated with the use of polylactic acid.³³

Antimicrobial photodynamic therapy seems to be a new and promising possibility for the prevention of dry socket. Antimicrobial photodynamic therapy (aPDT) with HELBO Blue and TheraLite laser enables local decontamination of the extraction socket.³⁴ However, due to the limited research available on this option, the authors believe photodynamic therapy should be investigated further.

MANAGEMENT

Management of dry socket (alveolar osteitis) can be divided into irrigation and surgical intervention, and dressing placement.

Currently available dressing options are antibacterial dressing, obtundent dressing, topical anaesthetic dressings or combinations of the three. Placement of dressing is controversial in literature and lacks concrete evidence. Placement is repeatedly suggested as an adjunct to surgical intervention. Case reports regarding the occurrence of other local complications have been described in the literature.³⁵

Thorough irrigation and adequate suctioning of the socket with normal saline cleanses the socket of all bone, tooth and food debris. Good pain control can be established by potent analgesics. Home instructions for maintenance of oral hygiene and gentle warm saline rinses assist in gradual healing of the socket.³⁶

Curettage is often discouraged due to induction of more pain. The Oral and Maxillofacial Department at Khyber College of Dentistry has investigated surgical intervention as a treatment option for dry socket. The technique involves administration of anesthesia, surgical debridement of socket and primary closure by advancement flap. Primary closure promotes clotting and enhances healing by primary intention. This provides immediate relief from pain and has shown consistent results in many cases.

DISCUSSION

It is well established that alveolar osteitis is by far the most common complication encountered following tooth extraction, especially after removal of third molars. Documented literature indicates that a sterile surgery and minimal tissue damage during the surgical process can minimize chances of dry socket occurrence. Provision of sufficient irrigation following tooth surgery was previously thought to be one of contributors to dry socket.¹⁷ It was common belief that the continuous flow of water dislodges the newly formed blood clot. Newer researches contradict this notion.¹⁸ It is now known that prolonged irrigation can prevent accumulation of bone and tooth tissue debris in the socket and assists in quick and uneventful healing.

Smoking is known to be a predisposition to dry socket.³⁷ Pathogenesis of the process is not quite yet understood. The Oral and Maxillofacial Surgery Department at Islamic International Dental Hospital is currently investigating the correlation between smoking and dry socket.

Pharmacological preventive measures of antiseptic agents and clot promoting agents have been tested continuously for efficacy. The absence of predisposing factors can render these agents completely useless. The adaptation of clean surgical techniques is critical in minimizing the incidence of dry socket.

Curettage is slowly becoming a popular option as a therapeutic measure for dry sockets. Cleansing the

walls of the socket from necrosed tissue under sufficient analgesia ensures better chances of recovery. However, very little literature documents the success of this treatment option.

Eugenol based dressing (tradename Alvogyl) contain benzocaine as a local anesthetic, balsam of Peru vehicle and eugenol as an obtundant. Placement of the dressing provides immediate relief from pain. A course of 2-3 placements is usually required at alternate days.³⁸ This treatment option is particularly recommended by us because of its low cost, ease of use and good outcome.

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