Dry socket -Prevalence and risk factors in a Pakistani Population

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Summary

Purpose: The aim of this study is to determine the prevalence and risk factors of dry socket in a Pakistani population at AIDM (Altamash Institute of Dental Medicine).

Material and methods: A two week clinical follow-up on extraction patients (all extraction sites included) at AIDM in Pakistan were conducted. Two questionnaires were distributed to all operators. The first questionnaire at the time of the extraction and the second questionnaire were completed on the 4th day after extraction.

Results: 117 extractions were carried out in 94 patients. The overall prevalence of dry socket was 13.8%. There were no statistically significant association between the stated risk factors and dry socket.

Conclusion: This study, due to the short period of time and the relatively small sample of patients did not identify significant risk factors in the development of dry socket. Even though the results were not statistically significant, certain trends such as female preponderance and distribution of dry socket posterior in the mandible can be seen.

Introduction

The term dry socket was first used by Crawford in 1896 (1). It is one of the most common postoperative complications and many different terms have since then been used to describe the condition. For example: Alveolar osteitis, localized osteitis, postoperative alveolitis, alveolalgia, alveolitis sicca dolorosa, septic dry socket, necrotic socket, localized osteomyelitis and fibrinolytic alveolitis. In his thesis Birn used the name “fibrinolytic alveolitis” (2) to describe the complication. This name reflects his theory of etiology and is probably the most accurate one, but not used widely in the literature. The more generic name, dry socket, tends to be used in most cases. In this article the condition will be referred to as dry socket from now on.

The exact etiology and pathogenesis of dry socket is not well understood. However, fibrinolysis causing disintegration of the blood clot seems to be a widely accepted theory (2). Several general and local factors have been associated with an increased risk for dry socket. They include; preoperative infection (especially pericoronitis) (2,4), poor oral hygien (4) difficult/traumatic extraction (2,7), gender (9), smoking (8), site of extraction (4,7) age (2), herpes simplex virus type 1 (22) and use of oral contraceptives (6,4).
Dry socket has a peak prevalence in the 40-45 year-old age group (13). The prevalence of dry socket is found to be 1-4% for all routine dental extractions, and 5-30% for impacted mandibular third molars (13,14). Dry socket affects molar extraction sites in the mandible up to 10 times more often than in the maxilla (15). Birn states that the frequency of dry socket after removal of wisdom teeth is about 20% (2).

Different methods have been suggested to reduce the prevalence of dry socket. Prophylactic treatment includes antibiotics, chlorhexidin, antifibrinolytic agents, steroids, eugenol dressings and cloth supporting agents. To date no single method has gained universal acceptance despite numerous published articles. (3).

The condition of dry socket cannot be fully treated as long as the underlying etiology is not completely understood. The management of dry socket is primarily symptomatic, seeking to relieve the pain and reassure the patient during the healing process. It usually involves cleaning and irrigation of the affected socket as well as insertion of a medicated pack with antibacterial and/or obtunding dressing. (3).

The aim of this study was to determine the prevalence and risk factors of dry socket in a Pakistani population at AIDM (Altamash Institute of Dental Medicine). This article will also describe the possible etiology and pathology of dry socket.

Clinical features

The clinical appearance of the disease was first described in 1896 by Crawford (1,2): “Two or three days after removal of the tooth, disintegration of the normal blood clot occurs. The alveolus is empty with complete or partially denuded, very sensitive bone surfaces, covered by a grayish-yellow layer of detritus and necrotic tissue. The surrounding gingiva often shows inflammatory reactions. The patient complains of heavy pain of a neuralgic character. From an alveolus of the mandible the pain irradiates towards the ear and the temporal region. From an alveolus in the maxilla the pain irradiates towards the eye and the frontal region. Halitosis is pronounced, and the patient complains of bad taste in the mouth. Swelling of the regional lymph nodes is rather common. General symptoms such as increased temperature are hardly ever seen, but the patient may be physically affected because of heavy pain and feel unwell due to lack of sleep and appetite.”

Blum later came up with a descriptive definition that could be used universally as a standardized definition of diagnostic criteria: “Postoperative pain in and around the extraction site, which increases in severity any time between 1 and 3 days after the extraction accompanied by a partially or totally disintegrated blood clot within the alveolar socket, with or without halitosis” (3).

Etiopathogenes

To understand the pathogenesis of dry socket we found it necessary to first go through the process of normal wound healing.
Normal healing

There are two ways in which traumatic injuries can be caused and that is physical or chemical. E.g. extremes of temperature or irradiation, incision or crushing, desiccation and obstruction of arterial inflow or venous outflow are ways of producing physical tissue damage. Chemicals which can cause injury are those that disrupt protein integrity, those with unphysiologic pH or tonicity and those which can cause ischemia by producing thrombosis or vascular constriction.

Epithelium, which has been injured, has a genetically programmed regenerative ability. Proliferation, migration and a process known as contact inhibition, allow the epithelium to reestablish its integrity.

Whatever the cause of the injury is, there is a stereotypic process that initiates and works to restore tissue integrity. This process is called wound healing and consists of three basic stages; 1) inflammatory, 2) fibroblastic, and 3) remodeling. (23).

Inflammatory stage

The moment tissue injury occurs the inflammatory stage begins and usually lasts 3 to 5 days. There are two phases in the inflammatory stage: vascular and cellular. The vascular phase starts with an initial vasoconstriction of disrupted vessels. This slows blood flow into the area of injury and promotes blood coagulation. Prostaglandins E1, E2 and histamine elaborated by white blood cells cause vasodilatation within minutes and open small spaces between endothelial cells. This allows leukocytes to migrate and plasma to leak into interstitial tissues. Lymphatic obstruction is caused by fibrin from the transudated plasma, and the transudated plasma, which is aided by obstructed lymphatics, accumulates in the area of the injury to dilute contaminants. This is called edema. (23)

The cellular phase starts when the activation of serum complement takes place after tissue trauma. Particularly C3a and C5a of the complement-split products act as chemotactic factors and cause neutrophils to stick to the side of blood vessels and then migrate through the vessel walls. The neutrophils release their lysosomes which then work to destroy bacteria and other foreign materials and to digest necrotic tissue. Monocytes, such as macrophages, also help out with the clearance of debris. With time lymphocytes accumulate at the site of tissue injury.

Because the inflammatory stage is the period during where no significant gain in wound strength occurs, this stage is sometimes referred to as the lag phase. The most important material holding a wound together during this stage is fibrin, which possesses little tensile strength. (23)

Fibroplastic stage

When blood coagulation takes place, strands of fibrin crisscross the wound to form a latticework. This is where fibroblasts can begin laying down ground substance and tropocollagen. The ground substance acts to cement collagen fibers together. The fibroblasts transform local and circulating pluripotential mesenchymal cells which begin tropocollagen
production on the third or fourth day after tissue injury. Fibroblasts also secrete fibronectin that helps stabilize fibrin, assist in recognizing foreign agents, acts a chemotactic factor for fibroblasts and helps to guide macrophages along fibrin strands for eventual phagocytosis of fibrin by macrophages.

New capillaries which bud from existing vessels along the margins of the wound will also use the fibrin network and run along fibrin strands to cross the wound. As this process continues, with increasing in growth of new cells, fibrinolysis takes place. This is caused by plasmin brought in by the new capillaries to remove the fibrin strands which now have become unnecessary.

Tropocollagen undergoes cross-linking to produce collagen. Initially this is produced in excessive amounts and is laid down in an irregular manner. Since the poor orientation of fibers decreases the wound strength, an overabundance of collagen is necessary to strengthen the healing wound initially. (23)

Remodeling stage

This stage is also known as wound maturation. It is the last stage of wound repair and continues indefinitely. This is when many of the previous randomly laid collagen fibers are replaced by new collagen fibers in a different manner, to better resist tensile forces on the wound. A final process is wound contraction. The exact mechanism that contracts a wound is still unclear. During wound contraction the edges of the wound migrate toward each other. If the edges of the wound are not or will not be placed in apposition, wound contraction will only diminishes the size of the wound. (23)

Healing of extraction sockets

Extraction of a tooth initiates the same sequence seen in prototypic skin or mucosal wounds. The extraction socket heals by secondary intention (a gap is left between the edges of a wound), and many months must pass before a socket heals completely. When a tooth is extracted, the remaining empty socket consists of the following: cortical bone covered by torn periodontal ligaments and a rim of oral epithelium left at the coronal portion. (23)

Blood fills the socket a few minutes after the extraction has been completed. Simultaneously an intravascular coagulation, which clogs up the torn vessels, takes place. A clotting of the blood in the alveolus then seals the socket from the oral environment. This clot will consolidate within the first 24 hours by network polymerization of the fibrin fibers formed, and cause shrinkage of the clot. Because of this the clot may be detached from the alveolar wall. The marginal gingival will fold over the extraction wound though, and thus prevent the occurrence of a cleft with a communication to the oral cavity. In the same period the clot will be infiltrated by leukocytes. Within 3 days the reaction will subside and be replaced by proliferative processes. (2)

The inflammatory stage takes place during the first week of healing and so does fibroplasia with the regrowth of fibroblasts and capillaries. The epithelium migrates down the socket wall
until it either encounters the bed of granulation tissue under the blood clot, over which it can migrate, or reaches a level at which it contacts epithelium from the other side of the socket.

Eventually, during the first week of healing, osteoclasts gather along the crestal bone. During the second week a large amount of granulation tissue fills the socket, and osteoid deposition begins along the alveolar bone lining the socket. This process continues during the third and fourth weeks of healing, with epithelialization of most sockets complete at this time. Resorption of cortical bone and apposition of trabecular bone continues for 4 to 6 months before the bone is completely healed. (23)

**Healing in dry socket**

Investigations into the healing process in dry socket have been carried out on experimental animals only, where it has been produced experimentally. It seems as if all investigations show the same course of healing. After 1 to 3 days complete or partial disintegration of the blood clot is seen. Where remains of the blood clot are found, they are infiltrated to a large extent with inflammatory cells and show signs of disintegration. Lamina dura is necrotic at large areas and empty osteocyte lacunae can be found in the bone tissue. The inflammatory stage has spread into the surrounding marrow spaces and in many cases into the periosteum. The gingiva has a heavy subepithelial inflammation. In the marrow spaces necrotic tissue is often found closest to the alveolus.

The histologic picture is typical for an acute or subacute osteomyelitis which has thrombosed vessels and heavy infiltration by mononuclear and polymorphonuclear leukocytes in the marrow spaces. Since the inflammatory reaction is so violent, the reparative processes start late and are preceded by extensive osteoclastic activity. This can sometimes cause sequester formation. Granulation tissue starts to grow into the alveolus through the perforations of the lamina dura at varying times after the extraction. From here the healing takes its normal course. (2)

**Blood coagulation and fibrinolysis**

Blood coagulation involves a biological amplification system. Relatively few initiation substances activate a cascade of circulating precursor proteins (the coagulation factor enzymes) by proteolysis. This results in the generation of thrombin, which in turn converts soluble plasma fibrinogen into fibrin.

A β-globulin proenzyme called plaminogen is converted to the serine protease plasmin, by activators either from the vessel wall or from the tissues. The endothelial cells release tissue plasminogen activator (tPA), which is a serine protease that binds to fibrin. This enhances its capacity to convert thrombus-bound plaminogen into plasmin. Stimuli such as trauma, exercise or emotional stress stimulate the release of tPA.

Fibrinolysis is stimulated by activated protein C. This is achieved by destroying plasma inhibitors of tPA. On the other hand thrombin is inhibited by fibrinolysis by activating thrombin-activated fibrinolysis inhibitor (TAFI). The role of fibrin is to enmesh the platelet
aggregates at the site of vascular injury and to convert the unstable primary platelet plug to a definitive, firm and stable haemostatic plug. (24)

**Etiopathogenesis of fibrinolysis in dry socket**

The significance of locally increased fibrinolytic activity in the pathogenesis of dry socket has been shown in clinical and laboratory studies (3). The two most important characteristics, dissolution of blood clot and violent pain, are explained by this common pathogenesis. Increased fibrinolytic activity gives rise to dissolution of the blood clot and the formation of kinins. The cause of the violent pain is due to kinins. (2)

During or immediately after the extraction and trauma, an infection will develop that cause inflammation of the marrow spaces of the alveolar bone. This leads to the liberation of tissue activators, which will convert plaminogen to plasmin. The blood clot will dissolve and at the same time release kinin from kininogen, which is also present in the clot. The result will be violent pain and dissolution of the blood clot (2). According to Goldstein et al. bacterial endotoxins are able to release fibrinolytic activity from leukocytes. Thus, there is a possibility of further fibrinolytic activity that originates from cells linked up with the inflammation (25).

The connective tissue type of bone marrow, which is rich in cells and vessels, seems to be particularly rich in stable tissue activator. This can be a good explanation of the age distribution in dry socket since this type of bone marrow is characteristic of the age group 20-40 years.

Dry socket seems to be more frequent in women. This might in part be because of the increased fibrinolytic activity in blood and saliva in women in the menstrual phase. Therefore, teeth extracted in that period will show a greater frequency of the development of dry socket.

Different areas of the jaw have the same fibrinolytic activity. The preferred location in the mandibular molar area may be explained by the trauma and/or the risk of infection which is greater in this area.

The explanation for the time of onset of dry socket is that the blood clot contains antiplasmin. This must be used up before dissolution of the clot can take place. At the same time the degree of inflammation will be decisive for the degree of fibrinolytic activity and therefore the time for inactivation of the antiplasmin.

In all extractions increased fibrinolytic activity is seen as a complication. What differs dry socket from this, is the fibrinolytic activity which reaches such levels that dissolution of the blood clot takes place. (2)

**Riskfactors**

**Gender**

Studies show that dry socket occurs up to five times more often in female patients (9). This is thought to be attributed to the use of oral contraceptives (6). It has been shown that the estrogen in oral contraceptives can elevate fibrinolytic activity in plasma. This could affect
the post-extraction clot stability (10). Other studies have shown a female preponderance of dry socket regardless of oral contraceptive use. In one study females were found to be at almost five times higher risk of getting dry socket than males (9). Other studies have shown that the male:female ratio is 2:3 (7).

Age

Dry socket is extremely rare after extraction of deciduous teeth and almost never occurs in childhood (2). The prevalence increases with the patients age and McGregor’s study placed the highest prevalence in the third and forth decade of life (7), though the higher prevalence of smoking at this age should be considered a confounding factor (16).

Smoking

Sweet and Butler (8) showed that the prevalence of dry socket is substantially higher in smokers than in non smokers. Those who smoked half a pack (10 cig/ day) had a four to five fold increase in dry socket (12% vs. 2.6%). The prevalence increased to over 20% among patients smoking more than 1 pack a day, and to 40 % among patients who smoked on the day of the surgery or on the first postoperative day. This phenomenon could be due to the introduction of a foreign substance that acts as contamination at the extraction site, combined with cloth removal due to suction and negative pressure during smoke inhalation (8). In addition, smoking has been shown to reduce neutrophil chemotaxis and phagocytosis and impede production of immunoglobin. Meechan also showed that smoking reduced the immediate post-extraction filling of sockets with blood and a higher prevalence of dry socket in patients smoking more than 20 cigarettes/ day compared to non-smokers (17).

“Sisha” (water pipe) smoking has shown similar effects to post-extraction dry socket as cigarette smoking (18).

Traumatic extraction

Trauma and difficult surgery is considered to be related to the development of dry socket (2, 12). Excessive trauma has been known to result in delayed wound healing. A reduction in blood perfusion is caused by compression of the alveolar bone. The trauma may also cause thrombosis in the underlying vessels (3). Birn suggests that damage to the alveolar bone cells cause inflammation, releasing tissue activators of fibrinolytic activity into the alveolus thus causing dry socket (2).

Birn also suggest that roots or bone fractures remaining in the wound are possible causes (2). Other studies have shown that these remnants do not necessarily cause complications during healing as they are often externalized by the epithelium (19). Despite lack of studies to support Birn’s suggestion it seems reasonable to assume that tooth and bone fragments combined with other debris can cause delayed wound healing (3).
Pre-operative infection – Oral micro organisms

Patients with poor oral hygiene (4) and pre-existing local infection such as pericoronitis and advanced periodontal disease (14) have been found to have a higher frequency of dry socket. One study performed by Kay (20) on 942 patients showed that 14.1% of patients with pericoronitis developed a dry socket compared to 6.6% of patients without the condition. A significant reduction was seen when antibiotics were given as prophylaxis (13, 20).

There have been attempts to isolate a specific causative organism. Nitzan et al. (21) showed a possible significance of anaerobic organisms to dry socket, which are also predominant in pericoronitis. Nitzan observed high plasmin-like fibrinolytic activities from Treponema Denticola. This organism is also known to be associated with development of periodontal disease. Further, dry socket is rarely seen during childhood when T. Denticola has not yet colonized the mouth.

Herpes

Hedner et al. found in their study that extraction of a mandibular third molar could be the cause of reactivation and recurrence of herpes simplex virus type 1 (HSV-1). Dry socket patients also reported a high frequency of oral cold sores in their history (64%) compared with the controls (33%) (22).

Methods and Materials

This study was performed at a dental institute (Altamash Institute of Dental Medicine) in Karachi, Pakistan.

Data were collected over a period of two weeks using two questionnaires. The first questionnaire (appendix 1) was completed by the operator at the time of the extraction and included items such as age, gender, smoking, medications, site of extraction, indication for extraction and level of experience of the operator. History of previous possible oral herpes reactivation was evaluated based on self-reported occurrence of oral cold sores. The second questionnaire (appendix 1) was completed by one of the students or teachers working in the department, on the 4th day after extraction. This included data such as socket affected, signs and symptoms, which lead to the diagnosis dry socket.

Participants in the study who did not feel the need to return for a follow-up visit were contacted by phone on the 4th day after the extraction, and asked for any post-operative pain. Patients with post-operative pain on the 4th day were asked to come back for a second visit. Patients who claimed to suffer no postoperative pain on the 4th day were excluded from the diagnosis dry socket.

Blum’s definition was used to diagnose patients with dry socket. “Post operative pain in and around the extraction site, which increases in severity at any time between 1 and 3 days after the extraction accompanied by a partially or totally disintegrated blood clot within the
alveolar socket with or without halitosis.” Thus, pain alone was not sufficient to result in the diagnosis.

The study was strictly observational without any interference with any of the clinical procedures normally followed at AIDM.

During the study period, 117 tooth extractions were carried out in 94 patients. There were 47 (50%) male patients and 47 (50%) female patients. The age of the patients ranged from 6 to 80 years with an average of 36.2 years.

The data were analyzed using a chi-square test. The critical level of significance was set at $P<0.05$.

**Results**

A total of 120 patients had their first questionnaire filled in at the time of extraction. From this group, 26 patients never returned for a check-up visit or could not be reached by phone, why they got excluded from the study. Remaining were 94 patients with a total of 117 extractions.

The prevalence of dry socket for all extractions in this study resulted in 13.8%, (13 out of 94 patients) although there were 12.8% (15 out of 117) sockets that got the diagnosis dry socket. The prevalence was higher, 23%, when the tooth was removed surgically (3/13). The prevalence of dry socket in patients undergoing non surgical extractions was 12.3% (10/81).

There were 15 extraction sockets in 13 patients (4 males and 9 females) aged 20 to 70 years with a mean age of 38 years who met the inclusion criteria for dry socket. (Table 1)

**Table 1. Patient age groups, gender and distribution of dry socket**

<table>
<thead>
<tr>
<th>Age group (yr)</th>
<th>Male,n (%)</th>
<th>Female,n (%)</th>
<th>Total,n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>20-30</td>
<td>1(7.7)</td>
<td>3(23.1)</td>
<td>4(30.8)</td>
</tr>
<tr>
<td>30-40</td>
<td>2(15.4)</td>
<td>3(23.1)</td>
<td>5(38)</td>
</tr>
<tr>
<td>40-50</td>
<td>1(7.7)</td>
<td>2(15.4)</td>
<td>3(23.1)</td>
</tr>
<tr>
<td>&gt;50</td>
<td>0</td>
<td>1(7.6)</td>
<td>1(7.7)</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>4(30.8)</strong></td>
<td><strong>9(69.2)</strong></td>
<td><strong>13(100)</strong></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Jaw</th>
<th>Right</th>
<th>Left</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maxilla</td>
<td>2(13.3)</td>
<td>2(13.3)</td>
<td>4(26.7)</td>
</tr>
<tr>
<td>Mandible</td>
<td>6(40)</td>
<td>5(33.3)</td>
<td>11(73.3)</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>8(53.3)</td>
<td>7(46.6)</td>
<td>15(100)</td>
</tr>
</tbody>
</table>

1) Female/male $P = 0.525$ [Chi2 = 2.233]
2) Maxilla/mandible $P= 0.451$ [Chi2 = 2.636]
There was no significant difference found for the distribution of dry socket between the genders or the jaws in this study.

In the maxilla 4/50 (8%) of the extraction sites developed dry socket. In the mandible the development was 11/61 (18%). Of the extracted teeth 13/15 teeth were molars, 1/15 were a premolar and 1/15 a canine.

*Figure 1. Reason for extraction.* Pre-operative diagnoses were mainly advanced caries (7/15) followed by advanced periodontal disease (3/15), Broken down root (3/15), fracture (1/15) and peri-apical disease (1/15).
Figure 2 shows the status of the operators. Most extractions were carried out by 4th year dental students, which in some more difficult cases, received help from House officers or a Demonstrator. All surgical extractions were carried out by House officers or Demonstrators.

In total 16/94 patients admitted to smoking. Of patients later diagnosed with dry socket 2 male patient disclosed tobacco use. One of them smoked 20 cig/day, the other one used snuff 2-3 times/day.

The presence of underlying systemic disease was elicited in 31% (hypertensive heart disease, previous episode of breast cancer, arthritis and arthritis). One patient suffered from migraine.

Time (>30 minutes) and surgical extraction were used as parameters of traumatic extraction. 3/15 extractions were surgical whereas 12/15 extractions were non surgical.
Figure 3. Distribution of dry sockets at different extraction times. The red curve shows how many extractions that were performed at each 5-minute interval and the blue curve is how much percentage of those that had complications in form of dry socket. For example; at the 30 minute level we had 11 extractions and 18% of these 11 extractions developed a dry socket.

**Figure 3**

Table 2. Rating of patients and selected risk factors.

<table>
<thead>
<tr>
<th>Risk factors</th>
<th>Kolumn1</th>
<th>n= 13, n(%), Rank</th>
</tr>
</thead>
<tbody>
<tr>
<td>Female gender</td>
<td>11(85) 1st</td>
<td></td>
</tr>
<tr>
<td>Posterior tooth (molar)</td>
<td>11(85) 1st</td>
<td></td>
</tr>
<tr>
<td>Mandibular tooth</td>
<td>8(62) 2nd</td>
<td></td>
</tr>
<tr>
<td>Age between 30-50 yr</td>
<td>8(62) 2nd</td>
<td></td>
</tr>
<tr>
<td>Traumatic extraction*/surgical extraction</td>
<td>6(46) 3rd</td>
<td></td>
</tr>
<tr>
<td>Underlying systemic disease</td>
<td>4(31) 4th</td>
<td></td>
</tr>
<tr>
<td>Previous infection**</td>
<td>3(23) 5th</td>
<td></td>
</tr>
<tr>
<td>Smoking/other tobacco</td>
<td>2(15) 6th</td>
<td></td>
</tr>
</tbody>
</table>

*traumatic extraction > 30 min. **Advanced caries was not considered as previous infection.
All patients received verbal postoperative instructions (appendix 2).

The typical symptom was pain from the socket (12/13 patients). On examination 11/15 sockets showed bare bone and 13/15 sockets were empty. One of the patients had halitosis.

Only 5/94 patients admitted to previous episodes of oral herpes/cold sores. None of these were later diagnosed with dry socket. At the follow up visit none of the patients were diagnoses with oral herpes/ blisters/cold sores. Thus no antiviral drugs were prescribed.

Table 3. Use of antibiotics and dry socket – all extractions.

<table>
<thead>
<tr>
<th></th>
<th>Antibiotics</th>
<th>No antibiotics</th>
<th>total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dry socket</td>
<td>4</td>
<td>9</td>
<td>13</td>
</tr>
<tr>
<td>No dry socket</td>
<td>36</td>
<td>45</td>
<td>81</td>
</tr>
<tr>
<td>Total</td>
<td>40</td>
<td>34</td>
<td>94</td>
</tr>
</tbody>
</table>

Antibiotics prescribed were most often Augmentin (amoxicillin and clavulanate potassium) or Cedrox (Cephalosporin).

Four out of the 13 patients later diagnosed with dry socket were prescribed antibiotics at the time for extraction. 2/4 patients were prescribed Cedrox, whereas 2/4 were put on Augmentin.

Treatment of all patients diagnosed with dry socket included; NSAIDS and/or paracetamol (acetaminophen), irrigation of the alveolus and insertion of Alvogyl (eugenol packing).

**Discussion**

The prevalence of dry socket is found to be 1-4% for all routine dental extractions and 5-30% for impacted mandibular third molars (13, 14). Nusair et al found that the prevalence following surgical extractions was 20.1 % (27). In this study the prevalence was 12.3% for all routine dental extractions and 23% for all surgical extractions. Thus, this study agrees with previous results of dry socket following surgical extractions. However, the result for routine dental extractions was higher than most of the previous studies. This can be due to many different reasons.

The study was performed in a country where the everyday oral hygiene reasonably may not always be the patient’s top priority. We experienced that many of the patients in the study had poor oral hygiene, even though this was not documented on the forms. Previous studies have shown that patients with poor oral hygiene and pre-existing local infection have a higher frequency of dry socket. (4, 14)

All of the patients were given postoperative instructions, but the compliance is a factor that is difficult to control. Smoking is believed to be one of the risk factors for dry socket. Therefore it is advised not to smoke after the extraction. Some patients may not understand or ignore the benefits of this instruction. Another postoperative instruction is to gently rinse the mouth with
hot salt water for 5 days. This can be considered as either waste of money or too much of an effort by some patients.

The way extractions were performed differs from the extraction technique we are taught at the Dental Program of Karolinska Institute. We experienced that the patients might have the mentality of a harder school and expected the extractions to be done rapidly. This together with the fact that the operators in most cases were students practicing could have made the extraction more traumatic. Even though the level of experience has not been established as significant for the level of trauma caused, it is likely that an inexperienced surgeon needs more time and causes more injuries to the bone. Trauma and difficult surgery are considered to be related to the development of dry socket according to previous studies. (2, 12)

Previous studies of dry socket have shown a peak incidence in the 40-45 year old age group (13). In this study the peak was found to be in the 30-40 year old age group (38%). However the prevalence in the 20-30 year old age group was 30.8% and the prevalence in the 40-50 year old age group was 23.1%. Thus, the prevalence of dry socket does not differ a lot between the ages 20-50. The amount of dry sockets in this study was quite small which can explain the small difference between the different age groups. The result is however not very remarkable since dry socket has been said to occur above all between the age 20-40.

Dry socket is extremely rare after extraction of deciduous teeth and almost never occurs in childhood (2). There were very few cases of children (11/94) participating in the study which makes it difficult to exclude this group from not developing dry socket.

As in most previous studies the most prevalent site for dry socket is in the posterior of the mandible. Dry socket affects molar extraction sites in the mandible up to 10 times more often than in the maxilla (15). This study agrees with previous findings although the difference between maxilla and mandible was not as high as previously shown.

According to former studies women seem to be predisposed to dry socket (9). This study agrees to this. Of all dry sockets, 69% was developed in women. This might be in part because of the increased fibrinolytic activity in blood and saliva in the menstrual phase (2). However this was not something that was looked into in this study.

Smoking is said to be one important risk factor in developing dry socket (8). Only two patients that developed dry socket disclosed tobacco use. One of them was smoking 20 cig/day, the other one used snuff 2-3 times/day. We believe this result is due to the fact that a lot of patients denied tobacco use since they might think this has a negative sound to it, especially with the doctor. Also the amount of dry sockets might be too small to give a significant value.

Only 5 patients admitted to previous episodes of HSV-1. Since oral herpes is a common virus, approximately 60-95% of adults worldwide are infected (26), it is likely that more than five patients in this study are infected with the virus. The low number can be due to unawareness to the symptoms of HSV-1. Many patients suffer from intra oral lesions due to other infections, burns and bites and may there for not classify the lesion as herpes. There was also a cultural reluctance to disclose HSV-1.
In agreement with Blum’s diagnosis criteria (3) all patients showed tender sockets and total or partial clot loss. Halitosis was not a common finding in this study. This could be due to an already compromised oral hygiene prior to extraction.

Pre existing infection is considered a predisposing factor (14) and the role of oral microorganisms were validated by some authors (21). Most teeth in this study were extracted due to advanced caries and not due to what has been classified as pre existing infections (advanced periodontal disease or pericoronitis) in previous studies. It is however likely that a tooth severely damaged or broken down by caries is a reservoir for a significant number of bacteria.

In conclusion; this study, due to the short period of time and the relatively small sample of patients, did not identify significant risk factors in the development of dry socket. Even though the result was not statistically significant certain trends such as female preponderance and distribution of dry socket posterior in the mandible can be seen. Control of preoperative infections, a good oral hygiene, non traumatic extractions/ avoidance of surgery may decrease the prevalence of dry socket.
Appendix 1

Dry socket in Altamash Institute of Dental Medicine

Extraction sheet

Please check all that apply

File nr: __________  Date: ______

Patient’s name: _______________  Patient’s contact no.: _______________

Patient speaks English:  ρ Yes  ρ No

Gender:  ρ Male  ρ Female  Age: ______ years

Medical history: _______________________________________________________

Previously known episodes of oral cold sores (herpes)  ρ Yes  ρ No

Medications: __________________________________________________________

Smoking:  ρ No  ρ Yes __________ cigarettes / day

Other tobacco use: _______________  amount / day: _______________

Teeth extracted: ______________________________________________________

Reason for extraction:  ρ Advanced caries  ρ Periapical disease

ρ Pericoronitis  ρ Advanced periodontal disease

ρ Orthodontic treatment  ρ Others, specify _______________

Extraction technique:  ρ Non surgical  ρ Surgical

Duration of extraction: _________ minutes

Operator:  ρ 4th year dental student  ρ Senior supervisor

ρ House officer  ρ Oral surgeon

ρ Demonstrator

Postoperative instructions given?  ρ Yes  ρ No

Postoperative medications prescribed: ______________________________________
Dry socket in Altamash Institute of Dental Medicine

Dry socket sheet

Please tick all that apply

File nr: __________  Date: ______

Patient’s name: _______________  Patient’s contact no.: _______________

Gender:  ρ Male  ρ Female  Age: ______ years

Socket affected:

Signs and symptoms:

ρ Pain
ρ Empty socket
ρ Bare bone
ρ Halitosis
ρ Oral cold sores (herpes)
ρ Others: ________________________________

Diagnosed with dry socket:  ρ Yes  ρ No

Treatment provided:

ρ Irrigation with saline
ρ Packing with Alvogyl
ρ Medications: ________________________________

ρ Antiviral drug
ρ No treatment
Appendix 2

Post-op info

ON THE DAY OF THE EXTRACTION:
1. Don’t rinse mouth with water for at least 24 hrs
2. Avoid hot fluids, hard or chewy foods. Soft foods and cool drinks only.
3. Do not chew on/with the treated side.
4. Smokers should avoid smoking.
5. If the wound starts to bleed, apply a small compress of cotton wool or gauze or a handkerchief, place it on the bleeding point & bite firmly for 10-15 mins.
6. Seek professional help if bleeding continues.
7. Pain & soreness can be relieved by prescribing medication

STARTING 24 HRS LATER:
Gently rinse mouth with hot salt water for 5 days (3-5 times a day)
References