

Alveolar Osteitis

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Teeth are routinely extracted in a general dental practice because they are deemed hopeless due to tooth decay or periodontal disease. Recently in the dental emergency clinic, I have noticed that I am frequently dealing with alveolar osteitis on a regular basis. Alveolar osteitis or dry socket is a complication that may follow a tooth extraction. It tends to occur when the blood clot in an extraction socket is disrupted prematurely leaving bone unprotected and exposed to the oral environment. Once opened to the oral environment it can become packed with food and bacteria products which can lead to further dissolution of the blood clot. Dry socket normally presents as postoperative pain of increasing severity in and around the extraction site one to three days after extraction. The patient may also complain of halitosis

Aetiology and prevalence

Dry socket is a normal complication that occurs anywhere from 0.5% to 5% during routine extractions. The probability of the dry socket increases when extractions are done in the mandible and especially during impacted wisdom teeth extraction (up to 35%). The aetiology of alveolar osteitis is not fully understood and several mechanisms and factors have been postulated that can increase the risk of its presentation.

Oral Micro-Organisms

Oral micro-organisms have long been postulated to contribute to dry socket. This is due to the increase frequency of dry socket seen with poor oral hygiene. Pre-existing local infections and advanced periodontal disease have shown an increased prevalence of dry socket among these populations. This relationship has been further strengthened by the incidence of dry socket decreasing in conjunction with antibacterial measures like a chlorhexidine mouth rinse. Many attempts have occurred to culture potential bacteria in relationship with dry socket. Although it has been difficult to discover these organisms, it has been found that significant anaerobes are common in dry sockets. In one study by Nitzen et al. they found *Treponema denticola*, which is known to be a putative micro-organism in the development of periodontal disease. This is further reinforced where children never experience dry socket especially because this bacterium hasn't colonized the mouth yet.

Another theory for bacteria induced dry socket is that bacteria constantly secrete pyrogens at a basal level and are indirect activators of fibrinolysis.

Difficulty and Trauma during Surgery

Many studies have determined a link between trauma and the difficulty of the

extraction. Surgical extractions that involve the reflection of flap and sectioning of the tooth with some bone removal have been reported to increase the causes of dry socket. A study found that the most common complication, for inexperienced dentists, during the removal of third molars is dry socket. This is directly related to trauma where it is known to delay wound healing by compression of the bone lining the socket impairing its vascular penetration. This causes inflammation of the alveolar bone marrow and subsequent release of direct tissue activators into the alveolus; where they increase precipitate fibrinolytic activity and play a role in pathogenesis of dry socket.

Roots and Bone Fragments Remaining in the Socket

Although one would think that having remaining roots and bone fragments in the socket would increase the risk of dry socket. However, a lack of scientific evidence exists between this relationship and it doesn't affect the development of a dry socket at all.

Excessive Irrigation or Curettage of the Alveolus after Extraction

It has been also postulated that excessive irrigation and curettage interferes with clot formation and gives rise to infection. Again, what we find is poor scientific evidence to prove this idea as well.

Physical Dislodgement of the Clot

Every dentist tells their patient that forceful spitting, sucking through a straw, coughing and sneezing all contribute to an increase risk of dry socket. Surprisingly there is little supportive evidence that these activities lead to an increase in the dry socket prevalence!

Local Blood Perfusion and Anaesthesia

The use of vasoconstrictor has also been thought to play a part in the development of dry socket. The thought process for this is that the blood can't profuse the socket thoroughly during the initial hemostasis, however this has been debunked by the fact that dry socket also happens in patients that have undergone general anaesthesia with no local being used during an extraction.

Also, injection technique has also been suggested to play a part of the aetiology of dry socket. Some investigators claimed that an increase in dry socket was seen in extractions that were done with periodontal ligament injections rather than a block or infiltration injections. This theory is attributed to the idea that it helps bacteria spread into the surrounding alveolus. Although other studies have shown that this is not the case and injection location has no factor, this is still a hotly debated topic which further research is necessary.

Oral Contraceptives

Women also appear to have a higher prevalence when compared to men. An

explanation of the gender difference is that changes in the clotting mechanism occur during the menstrual cycle along with the use of contraceptives - increases the risk. It has been proposed that the oestrogens will activate the fibrinolytic system indirectly and increase the risk of dry socket by lysis of the clot. Fibrinolytic activity is the lowest between days 23 to 28 days of the menstrual cycle. Normally oral contraceptives are shown to increase many factors including: II, VII, VIII, X and plasminogen. Unfortunately, we have very little published evidence of women not using oral contraceptives and the effect their menstrual cycle has on the probability of dry socket.

Smoking

This is single handily the largest factor in increasing the risk of dry socket. Studies have shown that patients who smoked at least half a pack of cigarettes a day had a five-fold increase in dry socket (roughly 12%). They also found that patients who smoked a full pack a day had a 20% chance and if they smoked that day or the first postoperative day it would increase to 40%.

The theory behind this very large increase is that containments are introduced into the clot and act to disrupt the surgical site. Unfortunately, there is no literature correlating the effects of the heat from the burning tobacco, containments in the smoke or the systematic effects of the ingredients on dry socket prevalence.

Pathogenesis

The research is clear in that an increase in the fibrinolytic activity is seen. Destruction of the clot is caused by tissue kinases liberated during inflammation through direct or indirect activation of plasminogen in the blood. When direct tissue activators are released after trauma, plasminogen is converted to plasmin resulting in the breakdown of the clot by disintegrating the fibrin. Intrinsic activators originate within plasma components. These include Factor XII and urokinase.

Direct extrinsic factors originate from outside of the blood and plasma components. These include: tissue plasminogen activators and endothelial plasminogen activators. These factors are found in many different tissue types including alveolar bone.

Indirect activators include streptokinase and staphylokinase. These activators are produced by bacteria and bind to plasminogen to form an activate complex that cleaves plasminogen molecules to plasmin. This is what strengthens the theory of bacteria inducing dry socket.

The pain associated with dry socket is attributed to the formation of kinin which produces intense pain. Plasmin is also involved in the conversion of kallikerins to kinins. Therefore, plasmin is thought to be involved in the two main characteristics in dry socket: clot disintegration and pain.

Prophylactic Management

There is still a large dilemma for clinicians if they should be prophylactically treat dry socket. The existing scientific literature is split between non-pharmacological and pharmacological preventive measures.

Non-pharmacological measures include comprehensive treatment planning with identification and possible elimination of risk factors.

These factors include:

- Previous experience with dry socket
- Deeply impacted wisdom teeth
- Poor oral hygiene
- Active or recent history of acute ulcerative gingivitis or pericoronitis
- Smoking more than a pack a day
- Use of oral contraceptives
- Immunocompromised patients

The measures that we would perform as a non-pharmacological approach include:

- Careful planning of the surgery
- Use of good surgical principles
- Extraction done with minimal trauma and maximum amount of care
- Confirm the presence of a blood clot
- Preoperative oral hygiene to reduce plaque levels
- Encourage patient to stop smoking and avoid vigorous mouth rinsing for 24 hours post op
- For patients taking oral contraceptives extractions should be performed through days 23-28
- Patient given pre- and post-operative instructions both written and verbal

Pharmacological Prevention

Pharmacological prevention has long been sought in the prevention of dry socket. There is a wide array of research that compares many different materials to each other and which one are effective. These can be broken down into 5 categories: Antibacterial agents, antiseptic agents and lavage, antifibrinolytic agents, steroid anti-inflammatory agents and obtundent dressings.

Antibacterial Agents

It has been shown that prophylactic antibiotics systemically or locally has decrease the prevalence of dry socket. The risk is further reduced if the antibiotics are given before the procedure vs postoperatively. Although this has been shown to decrease the rate of dry socket we must consider that we are possibly contributing to the development of drug resistant bacteria along with possible side effects of hypersensitivity and destruction of the host commensals.

Antiseptic Agents

Chlorohexidine has been shown to significantly decrease the risk of developing dry socket. This is true of both pre-operative and post-operative use. Therefore, this should be implemented when we perform complex mandibular third molar extractions or any complex extraction.

Antifibrinolytic Agents

Use of pure para-hydroxybenzoic acid (PHBA) in extraction sites post exodontia has been shown to decrease the risk of dry socket. The major downside of this product is that it is only available as a cone formulation with 32mg of ASA. ASA has a local irritating effect on bone, which causes inflammation within the socket and possibility resulting in dry socket.

Tranexamic acid has a reputation of preventing dry socket after extractions but no significant scientific study has shown its effectiveness against a placebo in reducing dry socket. Therefore, given the lack of evidence these agents shouldn't be implemented into post extraction care.

Steroid Anti-Inflammatory Agents

Steroids are known to decrease inflammation. However, no studies using topical steroids for the prevention of dry socket have shown any significant results. Again, this treatment modality would not be appropriate in prophylactic treatment.

Obtundent Dressings

A recent study has shown that placement of eugenol containing dressings have significantly reduce the risks of dry socket. However, this material does cause a local irritation and will delay wound healing.

Symptomatic Management of Alveolar Osteitis

Dry socket is complication that clinicians can only manage as the underlying aetiology is still not fully understood. Scientific literature is filled with different ways to manage dry socket.

Interestingly the placement of a dressing within exposed sockets is more based on empirical and reported effectiveness in reducing patient discomfort than scientific reasoning. No significant research has proven the complete effectiveness of these dressings. It has been postulated that the use of intra-alveolar substances allows greater local concentration of substances that can normally be expected from systemic circulation during clot formation. Also closing the socket to food debris also greatly improves comfort as well. Therefore, intra-socket dressings should be used to supplement non- dressing interventions. These non- dressing interventions include:

- Removal of any sutures to allow for adequate exposure of the extraction site
- Irrigate the socket with saline or local anaesthetic
- DO NOT attempt to curette the socket as this will increase the pain for the patient
- Prescription of oral analgesics
- The patient given a syringe with a curved tip for at home irrigation with a chlorhexidine solution or saline and instructed to keep the socket clean. Once clean and closed then irrigation can be discontinued

Take Home Message

Dry socket is a complicated process in which we still don't fully comprehend. The full aetiology has yet to be established despite are numerous theories. One thing that we can take away from all this is that we should be minimizing risk as much as possible. No single method is available to eliminated dry sockets from happening. As we look to improve our post-operative care for the prevention of dry sockets, we look back to the first rule of medicine which is to do no harm. Following these principles, the best treatment in reducing dry socket would be the use of a chlorhexidine mouth rinse post operatively for all extractions. This is due to the reported prophylactic benefit and lack of adverse effects that make it justified in the use of post-operative care and for the prevention of dry socket.

References

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