Pharmaceutical Society of Singapore

PRACTICE GUIDE FOR MINOR AILMENTS

Oral Health (Ulcers, Cheilitis, Xerostomia, Gingivitis)

PSS SELF-CARE GUIDELINE FOR COMMUNITY PHARMACISTS: Oral Health (Ulcers, Cheilitis, Xerostomia, Gingivitis)

Ulcers

Introduction

Oral ulcers, also known as aphthous stomatitis, are commonly seen in the community setting. In most cases, they are painful and can affect eating, drinking and speaking. It occurs with loss or erosion of part of the mucous membrane lining the inside of the mouth.

Ulcers can be a result of many conditions. The most common cause is injury, e.g. accidentally biting the mucosa of the cheek. Other causes include aphthous ulceration, certain medications, skin rashes in the mouth, viral, bacterial and fungal infections, chemicals and some medical conditions like Crohn's disease or celiac disease.[1] This guideline aims to provide information on oral ulcers to better equip community pharmacists in its management.

Objectives

1) Describe the epidemiology, risk factors and clinical presentation of the condition
2) Explain the diagnosis and differential diagnosis of the condition
3) Identify the goals of treatment for the condition
4) List the pharmacological and non-pharmacological options for treatment and prevention of the condition.
5) Discuss monitoring parameters for the condition.

Outline

1) Introduction to Ulcers
2) Epidemiology and Pathophysiology
3) Etiology and Risk factors
4) Symptoms
5) Diagnosis
6) Potential complications
7) Goals of treatment
8) Management
9) When to refer
10) References

1) INTRODUCTION TO ULCERS

1.1. A mouth ulcer is the loss or erosion of part of the delicate tissue which lines the mucous membrane of the mouth.[1]

1.2. Mouth ulcers can be classified based on the duration of their onset, number of ulcers and etiological factors[14]. For example, a person with a traumatic oral ulcer may present with a single, painful and irregular ulcer[5]. On the other hand, ulcers may be a long-standing problem for some patients[5].

1.3. Recurring mouth ulcers are also known as aphthous ulcers, the most common ulcerative condition of the oral mucosa. They present as painful punched-out sores on the oral mucous membrane, and are also called aphthae, aphthosis, recurrent aphthous stomatitis (RAS) or canker sores.[1,3]

1.4. Benign aphthous ulcers tend to be small (less than 1 cm in diameter) and shallow.[4] Aphthous ulcers that occur in conjunction with symptoms of uveitis, genital ulcerations, conjunctivitis, arthritis, fever or adenopathy should prompt a search for a serious etiology[4].

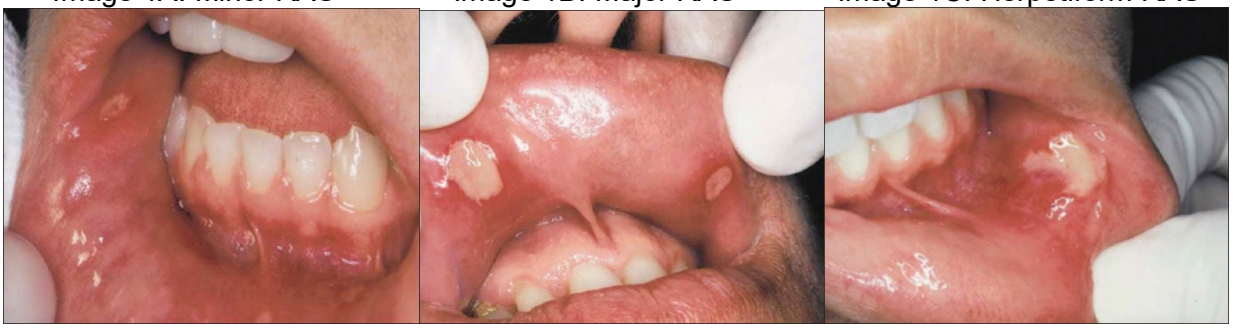
1.5. RAS is the most common cause of mouth ulcers, occurring mainly in otherwise healthy individuals[15, 16]. Although in most people there is no clear cause for these painful round or oval ulcers, risk factors or triggers have been identified[4, 7]. RAS can be classified into three subtypes with the following features[5]:

1.5.1. Minor RAS (most common of the 3 subtypes) (Image 1A)
- Occurs in groups of around 5 small ulcers
- Each ulcer is less than 1cm in diameter
- Affects non-keratinised sites in the mouth, such as buccal mucosa, labial mucosa or the floor of the mouth
- Usually heals within 10 to 14 days

1.5.2. Major RAS (Image 1B)
- Occurs as 1 to 3 ulcers at any one time
- Ulcers are greater than 1cm in diameter
- Involve any oral sites
- May take several weeks to heal

1.5.3. Herpetiform RAS (Image 1C)
- Involves between 10 and 50 small ulcers at non-keratinised sites that heal within 10 to 14 days
- These small ulcers may be present at the same time and may coalesce to form larger confluent areas of ulcer, usually with marked erythema[6]

Image 1A: Minor RAS[6] Image 1B: Major RAS[6] Image 1C: Herpetiform RAS[6]



2) EPIDEMIOLOGY AND PATHOPHYSIOLOGY

2.1. Anyone can get an aphthous ulcer; 20% of the population have one or more, at least occasionally[3]. The 3-month recurrence rates are as high as 50%[6].

2.2. Aphthous ulcers more commonly affect young adults[4]. They usually first appear in childhood or adolescence, and more commonly affect females than males[3].

2.3. In most patients, RAS resolves or spontaneously subsides with age.[15]

2.4. Approximately 40% of people who get aphthous ulcers have a family history of aphthous ulcers[3].

2.5. Pathophysiology of aphthous ulcers is poorly understood[4].

3) ETIOLOGY AND RISK FACTORS

3.1. Young adults are more commonly affected by aphthous ulcers, and a familial tendency may exist[4].

3.2. Proposed etiologic factors include stress, physical or chemical trauma, food sensitivity and infection[4].

- 3.2.1. Physical trauma or injury is usually due to accidental biting of the cheek or tongue, brushing, poorly-fitted dentures or sports mishaps[4,7]
- 3.2.2. Toothpastes and mouth rinses containing sodium lauryl sulfate may also trigger ulcers[7]
- 3.2.3. In particular, food sensitivities arising from chocolate, coffee, strawberries, eggs, nuts, cheese, and spicy or acidic foods[7]
- 3.2.4. Infectious agents such as *Helicobacter pylori* (the bacteria that causes peptic ulcers[7]) and herpes simplex virus have been investigated but have not been consistently found in aphthous ulcers[4]

3.3. Other possible triggers[7] include:

- 3.3.1. A diet lacking in vitamin B12, zinc, folate or iron
- 3.3.2. An allergic response to certain bacteria in the mouth
- 3.3.3. Hormonal shifts during menstruation

3.4. Other possible conditions and diseases[7] that may also present with ulceration

- 3.4.1. Celiac disease, a serious intestinal disorder caused by a sensitivity to gluten, a protein found in most grains
- 3.4.2. Inflammatory bowel diseases, such as Crohn's disease and ulcerative colitis
- 3.4.3. Behcet's disease, a rare disorder that causes inflammation throughout the body, including the mouth
- 3.4.4. A faulty immune system that attacks healthy cells in the mouth instead of pathogens, such as viruses and bacteria
- 3.4.5. Human Immunodeficiency Virus (HIV)/ Acquired Immunodeficiency Syndrome (AIDS), which suppresses the immune system

3.5. Paradoxically, smoking offers a somewhat protective effect against recurrent aphthous ulcers[4].

4) SYMPTOMS

4.1. Most aphthous ulcers are round or oval with a white or yellow center and a red border[7].

4.2. They form inside the mouth – on or under the tongue, inside the cheeks or lips, at the base of the gums, or on the soft palate[7].

4.3. A tingling or burning sensation may be experienced a day or two before the sores actually appear[7].

5) DIAGNOSIS

5.1. Clinical assessment of an ulcer includes inspection and palpation for the consistency of the base and fixation to underlying structures[6]

5.2. Important features to note when examining a patient with oral ulceration[6]:

- 5.2.1. Family history
- 5.2.2. Frequency of ulceration
- 5.2.3. Duration of ulceration
- 5.2.4. Number of ulcers
- 5.2.5. Site of ulcers (non-keratinized or keratinized)
- 5.2.6. Size and shape of ulcers
- 5.2.7. Associated medical conditions
- 5.2.8. Genital ulceration
- 5.2.9. Skin problems
- 5.2.10. Gastrointestinal disturbances
- 5.2.11. Drug history
- 5.2.12. Edge of ulcer, base of ulcer, and surrounding tissue

5.3. In diagnosing RAS, clinical features in Table 1 should be identified through a detailed and accurate clinical history and examination of the ulcers. An external examination including palpation of the cervical lymph nodes should be conducted[6].

Character	Type of RAS		
	Minor	Major	Herpetiform
Peak age of onest (decade)	Second	First and second	Third
Number of ulcers	1-5	1-3	5-20 (up to 100)
Size of ulcers (mm)	<10	>10	1-2
Duration	7-14 days	2 weeks-3 months	7-14 days
Heal with scarring	No	Yes	No
Site	Non-keratinized mucosa especially labial/buccal mucosa. Dorsum and lateral borders of the tongue	Keratinized and non-keratinized mucosa, particularly soft palate	Non-keratinized mucosa but particularly floor of the mouth and ventral surface of the tongue
RAS: Recurrent aphthous stomatitis			

Table 1: Clinical features of RAS[6]

- 5.3.1. For persistent RAS, the following investigation tests are recommended:
  - Hemoglobin and full blood count

- Erythrocyte sedimentation rate/C-reactive protein
- Serum B12
- Serum/red cell folate
- Anti-gliadin, and anti-endomysial autoantibodies

5.4. Aphthous ulcers should be differentiated from other stomatologie mucocutaneous diseases that have ulcerative manifestations[6]. Benign aphthae tend to be smaller and are more often self-limited when compared to more serious conditions[4].

5.5. Major aphthous ulcers can be associated with human immunodeficiency virus (HIV) infection; clinicians should consider HIV testing when ulcers are large and slow to heal[4].

5.6. Usually, more serious conditions can be differentiated based on the location of the lesion or the presence of an additional symptom. Table 2 highlights clinical features in making a differential diagnosis. If RAS is associated with a systemic condition, a referral should be made to the appropriate specialist for further investigations[6].

Differential Diagnosis of Aphthous Ulcers

DIAGNOSIS	*POTENTIAL DIFFERENTIATING FEATURES*
Infection	
Viral	
Herpesvirus	Vesicular lesions, Tzank stain positive for inclusion-bearing giant cells
Cytomegalovirus	Immunocompromised patient, biopsy positive for multinucleated giant cells
Varicella	Characteristic skin lesions
Coxsackievirus	Hand/foot/buttock lesions, typically in children
Treponemal	
Syphilis	Risk factors, other skin lesions, RPR/FTA test is positive
Fungal	
Cryptosporidium, mucormycosis, histoplasma	Immunocompromised patient, chronicity, biopsy and culture positive
Autoimmune	
Behçet's syndrome	Genital ulceration, uveitis, retinitis
Reiter's syndrome	Uveitis, conjunctivitis, HLA B27 arthritis
Inflammatory bowel disease	Recurrent bloody or mucous diarrhea, other GI ulcerations
Lupus erythematosus	Malar rash, ANA-positive
Bullous pemphigoid	Diffuse skin involvement
Pemphigus vulgaris	Diffuse skin involvement
Hematologic	
Cyclic neutropenia	Periodic fever, neutropenia
Neoplasm	
Squamous cell carcinoma	Chronicity, head/neck adenopathy, biopsy positive

RPR/FTA = rapid plasma reagin/fluorescein treponema antibody test; GI = gastrointestinal; ANA = antinuclear antibody.

Table 2: Differential Diagnosis of Aphthous Ulcers[4]

6) POTENTIAL COMPLICATIONS

6.1. Most aphthous ulcers heal without a problem[8]. However, potential complications may include the following[9]:

- 6.1.1. Cellulitis of the mouth, due to secondary bacterial infection that can result from acute ulcerations[8]
- 6.1.2. Dental infections (tooth abscesses)
- 6.1.3. Oral cancer
- 6.1.4. Spread of contagious disorders to others

6.2. Major aphthous ulcers and Behcet ulceration may heal with scarring[8].

6.3. Chronic ulceration due to oral lichen planus predisposes to oral squamous cell carcinoma (in ~5%)[8].

7) GOALS OF TREATMENT

7.1. Treatment of RAS is aimed at achieving the following[6]:
- 7.1.1. Decrease in symptoms
- 7.1.2. Reduction in ulcer number and size
- 7.1.3. Increase in disease-free periods

7.2. The approach to treatment should be determined by[6]:
- 7.2.1. Disease severity (pain)
- 7.2.2. Patient's medical history
- 7.2.3. Frequency of flare-ups
- 7.2.4. Patient's ability to tolerate the medication

8) MANAGEMENT

8.1. As there is a lack of clarity regarding the etiology of aphthous ulcers, the treatment is largely empiric.[4] Drug therapy is considered for patients who experience multiple episodes of RAS each month and/or present with symptoms of severe pain and difficulty in eating.[6]

8.2. Treatment for aphthous ulcers include antibiotics, anti-inflammatory agents, anesthetics and alternative (herbal) remedies.[4]

Forms of Treatment	**Medication/ Product (Forensic classification in Singapore)**	**Recommended Usage**	**Remarks**
Antibiotics	**Tetracycline 250mg capsule** (Prescription-only medication (POM))	Contents of a 250mg capsule can be dissolved in 180mL of water and used as a "swish and swallow" or "swish and spit" treatment four times a day for 4 to 5 days in adult patients.	Tetracycline and minocycline are most commonly used to help reduce pain and duration of ulcerations. In children and in women who may be pregnant, tetracyclines should be avoided because of its tendency to discolor teeth and the possibility of causing fetal harm.

	Minocycline 100mg capsule (POM)	Contents of a 100mg capsule can be dissolved in 180mL of water and the mixture can be used as a rinse twice daily for 4 to 5 days.	
Anti-inflammatory agents	**Triamcinolone 0.1% paste/ lotion** Pharmacy only (P-only): - Keno Oral Paste (Triamcinolone acetonide 1mg/g) - Oracort E Dental Paste (Triamcinolone acetonide 0.1% w/w, Lignocaine hydrochloride 3% w/w) POM: - Oramedy Paste (Triamcinolone acetonide 1mg/g) - Orrepaste (Triamcinolone acetonide 0.1% w/w) - Oral Aid Lotion (Triamcinolone acetonide 0.1% w/v, Lignocaine 2.5% w/v, Chlorhexidine hydrochloride 0.5% w/v)	Oral Paste: - Dab or press a small amount of paste on the affected area using a clean, dry finger or cotton swab. A smooth, slippery film forms after application. Do not rub the paste in, as the paste will become crumbly, grainy or gritty. - Apply to the affected area 2 to 3 times daily after meals or at bedtime as directed by doctor or dentist. - Do not eat or drink immediately after applying the oral paste. - Do not apply more than four times in 24 hours. Oral Lotion: - Apply 3 or 4 drops on the affected area, every four hours if required. - Do not eat or drink immediately after applying the oral lotion	The paste provides a protective local coating and using it early may result in a more rapid response. Localised application can promote quick healing and relieve symptoms in the management of minor recurrent aphthous stomatitis (RAS). Common side effects: Burning, itching, irritation, dryness, or redness of the treated area may occur. If any of these effects persist or worsen, consult a doctor or dentist. Do not use for longer than 5 days without medical advice.
	Dexamethasone elixir (0.5mg/5mL) POM: - SP-Cordexa Elixir (0.5mg/5mL) - SW-Dexasone Elixir (0.5mg/5mL)	May be used as a rinse and expectorated three to four times daily.	For more extensive oral ulceration. Patients should be warned of the potential for secondary fungal infection when using a steroid rinse. Systemic steroids are

			generally not recommended in the management of aphthous ulcers.
	Benzydamine hydrochloride[5] General Sales List (GSL): - Difflam anti-inflammatory lozenge with antibacterial (Benzydamine 3mg, Cetylpyridinium chloride 1.33mg)	- 1 lozenge as required, works for up to 3 hours - Severe cases: 1 lozenge should be sucked slowly every 1 to 2 hours as required, up to a maximum of 12 lozenges per day.	- Flavours available: Honey Lemon, Raspberry, Menthol and Eucalyptus - Contains 31g isomalt per 12 lozenges - Excess consumption of products containing isomalt may have a laxative effect. - Not recommended for use in children below 6 years of age
	- Difflam-C Anti-Inflammatory Antiseptic Solution (Benzydamine hydrochloride 22.5mg/15mL, Chlorhexidine gluconate 18mg/15mL)	- When used as a gargle, 15mL should be gargled for at least 30 seconds at 1.5 to 3 hourly intervals, as needed. - When used as a rinse for oral lesions, 15mL should be held in the mouth and swished around for at least 30 seconds. Repeat usage every 1.5 to 3 hours throughout the day. - As it is indicated for use as a rinse or gargle, it should not be swallowed but rather, expectorated after each use.	- Should generally be used undiluted but if burning or stinging occurs, it may be diluted with water - If used as an alternative to usual oral hygiene procedures, it should be swished around in the mouth for at least a minute. - Not intended for prolonged use except under dental or medical supervision. - Most commonly reported reactions are oral numbness, occasional burning or stinging sensations, dryness or thirst, tingling, warm feeling in the mouth and altered taste.

			- A common side effect associated with chlorhexidine gluconate oral rinses is staining of the teeth and other oral surfaces, which is harmless and can be minimised by thorough teeth brushing before administration. - Chlorhexidine can also cause an increase in calculus formation and an alteration in taste perception. No serious systemic adverse reactions associated with its use have been reported from clinical studies.
	P-only: - Difflam lozenge (Benzydamine hydrochloride 3mg)	- 1 lozenge should be sucked slowly every 2 to 3 hours as required, up to a maximum of 12 lozenges per day.	Most commonly reported adverse reaction is oral numbness
	- Difflam Plus Anaesthetic Sore Throat Lozenges (Benzydamine hydrochloride 3mg, Lignocaine hydrochloride 4mg, 2,4 Dichlorobenzyl Alcohol 1.2mg)	- 1 lozenge should be sucked slowly every 1 to 2 hours as required, up to a maximum of 12 lozenges per day.	- Flavours available: Honey Lemon, Menthol and Eucalyptus - Most commonly reported adverse reactions are oral numbness, occasional burning and stinging sensation.
	- Difflam solution (Benzydamine hydrochloride 22.5mg/15mL)	- As a rinse for oral lesions, 15mL should be held in the mouth and swirled around for at	- Should generally be used undiluted, but may be diluted with water if stinging occurs.

		least 30 seconds, with repeat use every 1.5 to 3 hours as needed throughout the day.	- The solution should be expelled from the mouth after use. - Most commonly reported adverse reaction is oral numbness.
	- Difflam mouth gel (Benzydamine hydrochloride 10mg/g, Cetylpyridinium chloride 1mg/g)	Apply every 2-3 hours, up to a maximum of 12 times per day - Apply approximately 1cm of gel with a clean finger - Gently massage into sore area - Do not eat or drink for 15 minutes	Most commonly reported adverse reaction is oral numbness
	- Difflam Forte Anti-Inflammatory Throat Spray (Benzydamine hydrochloride 3mg/mL)	- 2 to 4 sprays directly onto the sore/inflamed area and swallow gently. Repeat every 1.5 to 3 hours as necessary.	Most commonly reported adverse reaction is oral numbness For all Difflam products: - Not recommended for children below 6 years of age - Uninterrupted treatment for symptomatic relief should not exceed 7 days unless under medical supervision. - Medical advice should be sought if symptoms persist or other symptoms develop after this time.

Anesthetics for symptomatic relief	**Lignocaine-containing oral preparations** are usually used. GSL: - Medijel gel (Lignocaine hydrochloride 0.66% w/w, aminacrine hydrochloride 0.05% w/w) P-only: - Oracort E Dental Paste (Lignocaine hydrochloride 3% w/w, Triamcinolone acetonide 0.1% w/w) POM with exemption - Oral Aid Lotion contains lignocaine HCl 2.5% w/v, triamcinolone acetonide 0.1% w/v, chlorhexidine HCl 0.5% w/v	 Apply a small quantity of Medijel on a clean fingertip directly to the abrasion, ulcer or painful area. Repeat the application every 20 minutes if necessary. Please see instructions for use under triamcinolone 0.1% paste/ lotion Please see instructions for use under triamcinolone 0.1% paste/ lotion	Aimed at local and systemic symptom relief in patients with aphthous ulcers.
	Over-the-counter products to coat aphthous ulcers and provide local protection: GSL: - Orabase (Gelatin 16.7%, Pectin 16.7%, Carboxymethylcellulose Sodium 16.7%)	Dab a small amount on affected area as often as needed, particularly after eating. Do not rub. Adheres to wet areas and mucous membranes.	
	Non-steroidal anti-inflammatory agents or paracetamol for very painful ulcers. GSL:		

	Soragel antiseptic pain relieving oral gel (Choline salicylate 8.7% w/w, Cetylpyridinium chloride 0.01% w/w) Bonjela Gel (Choline salicylate 8.714% w/w, Cetalkonium chloride 0.01% w/w) Panadol (paracetamol 500mg tablets) Nurofen tablet 200mg (OTC) (ibuprofen 200mg) Aleve tablet 220mg (OTC pack) (naproxen sodium 220mg)	For gels: - Apply with a washed finger, rubbing into affected area, every 3 hours. - Apply approximately 1 cm or enough gel to cover the fingertip every 3 hrs. For analgesic relief in adults, 1000mg can be taken 4 times a day when necessary. For analgesic relief in adults, swallow 1 or 2 tablets with water, up to three times a day as required. For analgesic relief in adults, take one tablet, every 8 to 12 hours when necessary.	Not suitable for babies under 4 months. For Nurofen and Aleve: - Side effects are rare but may include stomach pain, nausea, indigestion and occasional stomach ulcer and black tarry stools. Also, worsening of asthma, itchy skin, 'nettle rash' and very rarely skin peeling and bruising. - Other side effects which have been very rarely reported are impaired kidney and liver function, headache, ringing in the ears and dizziness
Alternative remedies/ treatment	**Zinc gluconate lozenges** - Anecdotally reported to provide local relief and speeding of healing time for aphthous ulcers	Suck on lozenges	None of these agents has been studied in randomized controlled trials.
	Vitamin C, vitamin B complex and lysine - May speed up healing when taken orally at the onset of		

	lesions		
	Sage and chamomile mouthwash - Created by infusing equal amounts of the two herbs in water	May be helpful when used four to six times a day	
	Echinacea - Reported to speed up healing, perhaps through its immune modulatory effect		
	Carrot, celery and cantaloupe juices - Reported as helpful complementary agents		

8.3. Treatment of aphthous ulcers also include the use of mouthwashes in relieving symptoms and maintaining oral hygiene[5]

8.3.1. Saline mouthwash

- Salt water mouthwash, prepared by dissolving half a teaspoon of salt in glass of warm water
- Should be used as a rinse at frequent intervals until the discomfort and swelling subsides

8.3.2. Antiseptic mouthwash

- Chlorhexidine gluconate 2mg/mL (0.2%) mouthwash available OTC
- Used as a gargle twice daily as instructed
- Should not be used within 30 minutes of using toothpaste, in view of possible interaction (and may cause an unpleasant taste in the mouth)

8.4. Prevention of oral ulcers

8.4.1. Maintain oral hygiene as far as possible and keep the mouth healthy

- Use high quality, soft toothbrushes to reduce the risk of damage to the mouth[11] and prevent irritation to delicate mouth tissues[12]
- Regular brushing after meals and flossing once a day[12]
- Avoid toothpastes and mouth rinses containing sodium lauryl sulfate[12] which may cause irritation

8.4.2. Protect the mouth by consulting a dentist about orthodontic waxes to cover sharp edges if braces or other dental appliances are worn.[12]

8.4.3. Dietary considerations

- Eat a balanced diet which is rich in vitamins A, C and E, including foods such as fresh fruit and vegetables (to lessen the risk of mouth cancer)[11]
- Prevent nutritional deficiencies by eat healthily with plenty of fruits, vegetables and whole grains.[12]

- Consuming sufficient amounts of vitamin B12 and folate may be a useful strategy to reduce the number and/or duration of RAS episodes.[6]
- Try to avoid food that can irritate the mouth, such as nuts, chips, pretzels, certain spices, salty foods and acidic fruits like pineapple, grapefruit and oranges
- Avoid any food that can trigger allergies or sensitivity[12]

8.4.4. Visit the dentist regularly[11]

8.4.5. Reduce stress, especially if the ulcers seem to be related to stress. Learn and practise stress-reduction techniques, such as meditation and guided imagery.[1]

9) WHEN TO REFER

9.1. In most cases, mouth ulcers are harmless and resolve by themselves within 10 to 14 days without the need for treatment.[1]

9.2. Seek medical attention and consult a doctor if any of the following occurs:

9.2.1. Persistent ulcers, lasting two weeks or more[7] as an ulcer that does not heal may be a sign of mouth cancer[1]

9.2.2. Recurring ulcers, with new ones developing before old ones heal, or frequent outbreaks[7], or if new symptoms develop[13]

9.2.3. Unusually large ulcers[7]

9.2.4. Ulcers that extend into the lips (vermilion border)[7]

9.2.5. Pain that is beyond control with self-care[7]

9.2.6. Extreme difficulty eating or drinking[7]

9.2.7. High fever along with ulcers[7]

9.3. See a dentist if there are any sharp tooth surfaces or dental appliances that seem to trigger the sores[7]

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Cheilitis

Introduction

Cheilitis is an inflammation of the lips, which could be acute or chronic[1], and may appear as an isolated condition or as part of certain systemic diseases/conditions[2]. The inflammation primarily arises in the vermilion zone (lip border)[2] but may extend to surrounding skin and less commonly, to the oral mucosa.[1]

It may be caused by various factors, including contact irritants or allergens, chronic sun exposure, and nutritional deficiencies such as anemia due to vitamin B12 or iron deficiency[2], as well as by various cutaneous and systemic illnesses[1]. It can also be due to local infections (e.g., herpes and oral candidiasis) or drug intake, especially retinoids[2]. Generally, the forms most commonly reported in the literature are angular, eczematous, actinic, glandular, granulomatous, exfoliative and plasma cell cheilitis. However, this chapter will focus mainly on reversible cheilitis, as most forms of irreversible cheilitis have either a rare or very rare occurrence, and usually calls for biopsy and histology for diagnosis.[2]

Objectives

1) Describe the epidemiology, risk factors and clinical presentation of the condition
2) Explain the diagnosis and differential diagnosis of the condition
3) Identify the goals of treatment for the condition
4) List the pharmacological and non-pharmacological options for treatment and prevention of the condition.
5) Discuss monitoring parameters for the condition.

Outline

1) Introduction to Cheilitis
2) Epidemiology
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1) INTRODUCTION TO CHEILITIS

1.1. Various types of cheilitis can be classified based on duration and etiology[2,3]:

1.1.1. Mostly reversible

- Angular cheilitis
- Eczematous cheilitis[12]
  i) Atopic cheilitis
  ii) Allergic contact cheilitis
  iii) Irritant contact cheilitis
- Exfoliative cheilitis
- Drug-related cheilitis

1.1.2. Mostly irreversible

- Actinic cheilitis
- Granulomatous cheilitis
- Glandular cheilitis
- Plasma cell cheilitis

1.2. In practice, it can be challenging to identify the precise type of cheilitis readily, thus proper diagnostic procedures are necessary to determine the exact disease based on the characteristics[2]

1.2.1. Angular cheilitis can occur spontaneously or may be related to several precipitating factors (e.g. systemic immune suppression, local irritation and moisture, fungal/bacterial infection)[5]

1.2.2. Eczematous cheilitis can be related to endogenous causes such as atopic dermatitis or exogenous causes like the effects of irritants (climatic, mechanical, caustic agents) or allergens (allergic contact cheilitis)[13]

1.2.3. Exfoliative cheilitis is usually accompanied by desquamation ('skin peeling') found on the lower lip and is common among young people who frequently moisturize their lips

1.2.4. Oral intake of retinoids such as isotretinoin or acitretin is a frequent cause of drug-related cheilitis

1.2.5. Actinic cheilitis is mostly a persistent form of cheilitis caused by chronic ultraviolet radiation and is considered a potentially malignant disorder

1.2.6. Granulomatous cheilitis is idiopathic but some predisposing factors include food allergy, genetics, infection and atopy[1]

1.2.7. The cause of glandular cheilitis is unknown, but several factors have been identified, including atopy, infection, chronic exposure to the sun, repeated licking, and use of tobacco.[1]

1.2.8. Some types of cheilitis last longer and are persistent, such as chronic actinic cheilitis, granulomatous cheilitis and plasma cell cheilitis

1.2.9. Cheilitis can also be seen in various skin or systemic diseases such as lupus erythematosus, lichen planus and atopic dermatitis

2) EPIDEMIOLOGY

2.1. Angular cheilitis may present at any age with an equal male to female ratio but is especially likely in older individuals wearing dentures. Children suffering from this have a history of recurrent infections or immune defects.[1]

2.2. Eczematous cheilitis is the most common form of cheilitis and is more prevalent in people with a history of allergies.[1] A local study found endogenous cheilitis (53%) to be the most common diagnosis, followed by allergic contact cheilitis (34%) and irritant contact cheilitis (5.4%). A personal history of atopy was recorded in 33% of the 202 patients studied, with no difference in prevalence among the sexes.[16]

2.3. Exfoliative cheilitis occurs slightly less frequently compared to other forms of cheilitis, and is more common among young people who frequently moisturize their lips, followed by individuals with nutritional deficiencies such as vitamin B12 or iron, oral candidiasis infection (in patients with or without HIV) or in patients with allergies.[2]

2.4. Drug-induced cheilitis is one of the most commonly reported adverse effect of oral systemic retinoids (e.g. isotretinoin) and the severity is often dose related.[12,15] Medications which

cause dry mouth have also been implicated in drug-induced cheilitis.[12]

2.5. Actinic cheilitis is more prevalent in geographic areas with high ultraviolet (UV) irradiation, and in fair-skinned individuals who work outdoors with chronic sun exposure (e.g. fishermen, farmers) of ages 40s to 80s.[1]

2.6. Granulomatous cheilitis is rare. It primarily affects young adults but can occur at any age, with an equal ratio in males to females.[1]

2.7. Glandular cheilitis, a form of cheilitis rarer in occurrence than granulomatous cheilitis, is more prevalent in older males, but younger individuals and women may be affected.[1]

2.8. Plasma cell cheilitis is a very rare type of cheilitis and its etiology is unknown.[2]

3) PATHOPHYSIOLOGY

3.1. Angular cheilitis is a result of softening of tissue from excessive moisture from saliva and secondary infection with *Candida albicans* or, less frequently, *Staphylococcus aureus*.[1] Diabetes and nutritional deficiencies (riboflavin, folate or iron) may also be contributory factors.[2]

3.2. Eczematous cheilitis could be due to loss of plasticity, delayed-type hypersensitivity or atopy, depending upon the type.[1] Often a combination of factors is present.[13]

- 3.2.1. Atopic cheilitis is a form of eczematous cheilitis due to endogenous factors, and is common in patients with atopic dermatitis or a history of atopic disease.[12]
- 3.2.2. Allergic contact cheilitis is a delayed type of hypersensitivity reaction to allergens (exogenous factors) which come into contact with the lips. Common allergens include cosmetics, oral hygiene products and certain foods.[13]
- 3.2.3. Irritant contact cheilitis is a form of eczematous cheiltis due to irritants (exogenous factors), such as constant lip licking, cosmetics, oral hygiene products, food or environmental factors.[13]

3.3. The etiology of exfoliative cheilitis is unknown, though associated factors include lip licking/picking, psychological distress and nutritional deficiencies such as vitamin B12 or iron.[2]

3.4. Drug-induced cheilitis refers to lesions due to drug intake, mainly retinoids (e.g. isotretinoin) or other medications (e.g. topical antibiotics, virostatic agents, lip care products, disinfectants, local anesthetics, creams with protection factors).[2]

3.5. Actinic cheilitis originates from the proliferation of atypical epidermal keratinocytes due to chronic sun exposure.[1]

3.6. Granulomatous cheilitis results from long-standing edema and perivascular inflammation of lip and facial tissue. As the infiltrates become more dense, pleomorphic and small focal granulomas are formed, which may be indistinguishable from systemic granulomatosis.[1]

3.7. Glandular cheilitis results from fibrosis surrounding the salivary glands. Dense chronic inflammatory infiltrates may be found in severe cases.[1]

3.8. The etiology of plasma cell cheilitis is unknown.[2]

4) RISK FACTORS

4.1. Angular cheilitis[4]

- 4.1.1. Oral thrush, which may be related to infancy, old age, diabetes, systemic corticosteroid or antibiotic use
- 4.1.2. Poor-fitting dentures, which may be associated with gum recession, malocclusion of teeth, and substantial weight loss may all contribute to sagging perioral skin[5]
- 4.1.3. Excessive saliva buildup which may be contributed by repeated lip-smacking and thumb-sucking behaviors[5]
- 4.1.4. Poor nutrition which can be linked to coeliac disease, iron deficiency, riboflavin deficiency
- 4.1.5. Systemic illness, particularly inflammatory bowel disease (ulcerative colitis and Crohn's disease)
- 4.1.6. Sensitive skin, especially atopic dermatitis
- 4.1.7. Genetic predisposition, e.g. in Down's syndrome
- 4.1.8. Oral retinoid medication e.g. isotretinoin for acne, acitretin for psoriasis

4.2. Eczematous cheilitis[8]

- 4.2.1. Atopic cheilitis
  - Family history of eczema or allergies
  - Stress
  - Having a cold or flu
  - Changes in hormone levels, especially in women
- 4.2.2. Allergic contact cheilitis
  - Lip cosmetics (e.g. lipsticks and lip balms) followed by toothpastes were the most common causes found in a local study[16]
  - Frequent cosmetic sensitizers include fragrances, nickel, castor oil (ricinoleic acid), dyes, preservatives and propolis (a bee product)[12]
- 4.2.3. Irritant contact cheilitis
  - The most common causes found in a local study was lip licking, lipsticks and medication[16]
  - Other risk factors include job or activities involving exposure to irritating substances, as well as environmental factors (e.g. cold, low humidity or wind)

4.3. Exfoliative cheilitis[2]

- 4.3.1. Young people who frequently moisturize their lips
- 4.3.2. Nutritional deficiencies (e.g. vitamin B12, iron)
- 4.3.3. Oral candidiasis infection (with or without associated HIV infection)
- 4.3.4. Bacterial infection (e.g. due to *Staphylococcus aureus*)
- 4.3.5. Psoriasis[17]
- 4.3.6. Self-damaging behaviour due to stress[17]

4.4. Drug-induced cheilitis[2,12]

- 4.4.1. Oral systemic retinoids (e.g isotretinoin, acitretin)
- 4.4.2. Oral medications that cause dry mouth
- 4.4.3. Topical antibiotics

4.5. Actinic Cheilitis[6]

- 4.5.1. Increased age, especially over 60 years
- 4.5.2. Fitzpatrick I and II skin types[7]
  - Fitzpatrick skin type I: Pale white skin, blue/green eyes, blond/red hair; Always burns, does not tan
  - Fitzpatrick skin type II: Fair skin, blue eyes; Burns easily, tans poorly
- 4.5.3. Genetic abnormalities affecting pigmentation such as albinism
- 4.5.4. Working outdoors for more than 25 years
- 4.5.5. History of non-melanoma skin cancer
- 4.5.6. Certain genetic conditions associated with increased susceptibility to solar damage may predispose individuals to acquire actinic cheilitis at an early age (e.g. xeroderma pigmentosum, oculocutaneous albinism).[1]

5) SYMPTOMS

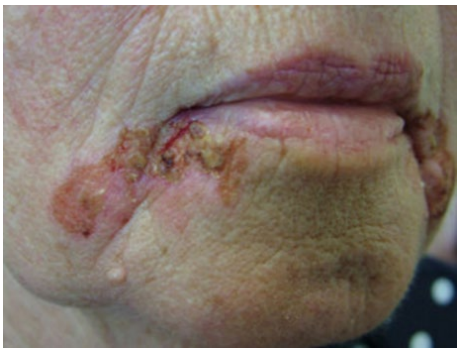
5.1. Common symptoms associated with cheilitis include the following[9]:

- 5.1.1. Dryness
- 5.1.2. Redness
- 5.1.3. Scaling or fissuring
- 5.1.4. Tenderness
- 5.1.5. Cracking or peeling
- 5.1.6. Swelling (edema)
- 5.1.7. Itching and burning sensation
- 5.1.8. Brown-black discoloration of the lips (seen with certain types of irritant contact cheilitis)

5.2. In particular, angular cheilitis (Image 2A) can present with:

- 5.2.1. Irritation and soreness in the corner(s) of the mouth[10], where one or both corners may be:
  - Bleeding
  - Blistered
  - Cracked
  - Crusty
  - Itchy
  - Painful
  - Red
  - Scaly
  - Swollen
  - Macerated[12]
- 5.2.2. Dry and uncomfortable lips, a bad taste in the mouth or sometimes the lips and mouth can feel like they are burning[10]
- 5.2.3. Difficulty eating if the irritation is strong, and this can lead to malnutrition or weight loss[10]

Image 2A: Angular Cheilitis[4]



5.3. Eczematous cheilitis[12]

5.3.1. Clinical manifestations of atopic cheilitis include erythema, dryness, scaling and fissuring. Its clinical manifestations may be indistinguishable from that of irritant or allergic contact cheilitis (Image 2B)

5.3.2. In allergic contact cheilitis, patients typically present with an erythematous, scaly eruption involving both the upper and lower lips, which often extends beyond the lip border to the perioral skin. Vesicles or superficial ulcers may also be seen (Image 2C)

5.3.3. Lip licking cheilitis is a form of irritant contact cheilitis, that presents as erythema with hyperpigmentation, scaling and fissures, surrounded by a well-demarcated ring around the lips (Image 2D)

Image 2B: Eczematous Cheilitis - Atopic cheilitis[1]

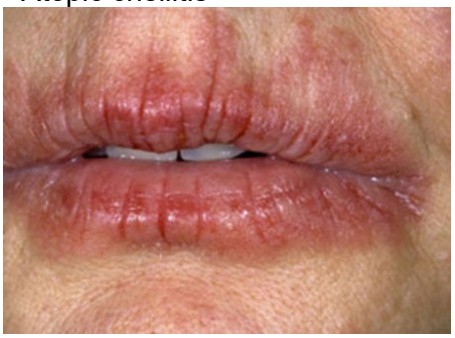


Image 2C: Eczematous Cheilitis - Allergic contact cheilitis[12]

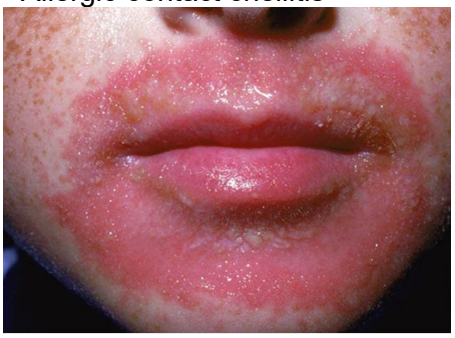
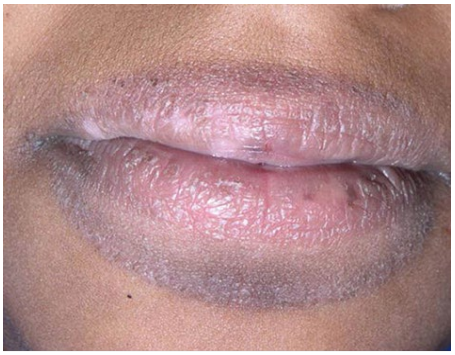


Image 2D: Eczematous Cheilitis - Irritant contact cheilitis[12]



5.4. In exfoliative cheilitis (Image 2E), symptoms may include the presence of a thick keratin scale on the lips.[9]

Image 2E: Exfoliative Cheilitis[17]

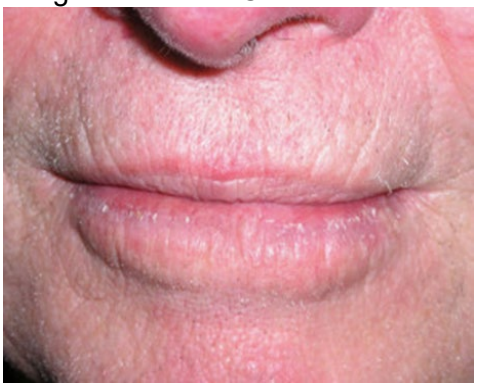
5.5. Drug-induced cheilitis due to oral retinoids causes dryness, scaling, erythema and lip fissures in nearly all patients.[12]

5.6. Actinic cheilitis (Image 2F) most commonly affects the lower lip (90%), presenting as[11]:

5.6.1. Dryness
5.6.2. Thinned, fragile, skin
5.6.3. Thickened, scaly papules and plaques.
5.6.4. Other clinical features may include:

- Swelling
- Redness
- Soreness
- Fissuring, focal ulceration and crusting
- Loss of demarcation between the lip border and its adjacent skin
- White thickened patches (leukokeratosis)
- Discoloured skin with pale or yellow areas
- Prominent folds and lip lines

Image 2F: Actinic Cheilitis[11]



6) DIAGNOSIS

6.1. Angular cheilitis

6.1.1. A potassium hydroxide preparation from lesions and oral mucosa may be needed to confirm or rule out *Candida* infection[12]
6.1.2. If cheilitis is recalcitrant, a lesion swab for bacterial and fungal culture should be obtained[12]
6.1.3. Diagnosis is based on physical examination with no special tests required.[5]

6.2. Eczematous cheilitis[12]
6.2.1. A general skin examination that includes the oral mucosa for signs of atopic dermatitis or other skin diseases that may involve the lips.
6.2.2. Assessment of personal or family history of atopic disease (atopic dermatitis, allergic rhinitis, asthma).
6.2.3. A detailed history that reviews the exposure to irritants or allergens (e.g. cosmetics, sunscreens, foods, hobbies, instruments, dental hygiene products)
6.2.4. Patch testing
6.2.5. Biopsy should be considered when standard treatments are not effective or in patients with persistent, painful, bleeding, hyperkeratotic, or eroded findings

6.3. Exfoliative cheilitis shares similar clinical appearance with conditions such as eczematous or acitinic cheiltis[12], and can be made worse by a secondary infection caused by *Candida* or *Staphylococcus* aureus.[17]
6.3.1. While it is a diagnosis of exclusion, with no specific diagnostic test available, tests may be required to rule out other similar looking conditions or secondary infection[17]
6.3.2. A psychiatric evaluation may be helpful if the condition is associated with a mood or anxiety disorder

6.4. Diagnosis of drug-induced cheilitis involves taking a careful medication history. Complete resolution of symptoms usually occurs soon after drug discontinuation.[12] Drug-induced cheilitis due to oral retinoids such as isotretinoin for acne, or acitretin for psoriasis may present as angular cheilitis.[4]

6.5. Chronic actinic cheilitis
6.5.1. A referral to the doctor for biopsy is required for confirmation of diagnosis[2] and important especially if there is suspicion of malignant transformation[1]

7) POTENTIAL COMPLICATIONS

7.1. Angular, eczematous, exfoliative and drug-induced cheilitis are mostly reversible forms of cheilitis[2]

7.2. Actinic cheilitis and less commonly glandular cheilitis can lead to squamous cell carcinoma[1]

7.3. Permanent disfigurement of the lip may be seen in severe forms of cheilitis, especially in the case of glandular cheilitis and granulomatous cheilitis[1]

8) GOALS OF TREATMENT

8.1. To remove or treat underlying cause(s)[9,12]

8.2. To provide relief of symptoms[12]

9) MANAGEMENT

9.1. Therapeutic management of cheilitis is symptomatic and dependent on etiology[1], as well as the type of cheilitis.

9.2. Angular cheilitis

9.2.1. Management includes the following general measures to reduce moisture pooling at the corners of the mouth[12]

- Maintain oral hygiene[1]
- Improve denture fit and cleaning[12]. Dentures must be removed before going to bed at night, brushed intensely, and then soaked in denture cleaner, or a solution of chlorhexidine gluconate or a dilute solution of bleach (10 drops of solution in a denture cup filled with water).[1]
- Use of barrier creams (e.g. zinc oxide paste) or petrolatum [12]
- Treat dry mouth symptoms (see chapter on Xerostomia)

9.2.2. Treatment of secondary infection where present[12]

- *Candida* infection:
  i. Antifungal creams (e.g. miconazole 2% or clotrimazole 1%) may be applied twice daily for 1 to 3 weeks and repeated as necessary.[12]
  ii. A topical steroid (e.g. hydrocortisone 1% cream or ointment) may be applied after the antifungal cream to reduce inflammation.[5]
  iii. Combination creams of either miconazole 2% or clotrimazole 1% with hydrocortisone 1% are also available in Singapore as P-only medicines.
  iv. Achieving good diabetic control would also help reduce the risk of *Candida* infection.[4]
- *Staphylococcal* infection:
  i. Topical mupirocin 2% ointment may be applied twice daily for 7 to 14 days[12]

9.2.3. Vitamin supplements could be given if nutritional or vitamin deficiencies are detected.[1,5]

9.3. Eczematous cheilitis

9.3.1. Removal of causative irritants or allergens from a patient's environment is the mainstay of treatment for all forms of eczematous cheilitis[1,12]

9.3.2. This includes the avoidance of lip licking, ricinoleic acid (castor oil) found in most lipsticks, other lip cosmetics or toothpastes containing flavours, preservatives, propolis, lanolin and other potential irritant or allergens[12]

9.3.3. Low to medium potency topical corticosteroids, applied twice daily for 1 to 2 weeks are helpful in reducing inflammation and itch[1,12]

- Low potency: Hydrocortisone 0.5% cream or 1% cream/ointment[18]
- Medium potency: Betamethasone 0.025% cream/ointment[18]
- Topical corticosteroids should be applied sparingly, and prolonged use avoided, as it can cause atrophy, which may exacerbate irritant cheilitis[12]

9.3.4. Simple emollients like petrolatum can be used liberally, in combination with topical corticosteroids[12]

9.4. Exfoliative cheilitis

9.4.1. This form of cheilitis is typically resistant to treatment, unless a predisposing or associated condition can be identified[17]. Some isolated reports of treatment success include topical corticosteroids or topical tacrolimus in combination with lip moisturizers[12]
9.4.2. Use of topical tacrolimus would be an off-label indication as Protopic® ointment (0.03% or 0.1%), the only 2 topical tacrolimus preparations registered in Singapore, should not be applied to the mucous membranes[19]
9.4.3. In some cases associated with a mood or anxiety disorder, psychotherapy or antidepressants have found to be helpful[2,12,17]

9.5. Drug-induced cheilitis
9.5.1. Use lip moisturizers and emollients[1]
9.5.2. Cease offending drugs for complete resolution of symptoms[1]

9.6. Other forms of cheilitis which are mostly irreversible, such as actinic, glandular or granulomatous cheilits[2], are best referred to the doctor for management

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Xerostomia

Introduction

Xerostomia is a subjective sensation of oral dryness.[1] Everyone has a dry mouth once in a while, especially when feeling nervous, stressed or upset.[2] It is a common, complex and under-recognized condition.[1] However, if it happens all or most of the time, it can be uncomfortable and can lead to more serious health problems or indicate that a more serious medical condition may exist.[2]

Saliva is a complex mixture of fluids that provides several protective functions, including cleansing the oral cavity, facilitating speech and swallowing, protecting oral tissues against physical and microbial insults, including teeth from decay.[1,2] Apart from keeping the mouth wet, it also helps with digestion of food[2] and maintaining a neutral pH.[1]

Chronic xerostomia remains a significant burden for many individuals.[3] With reduced salivary flow, it can cause difficulties in tasting, chewing, swallowing, and speaking;[1,2] it can also increase the chance of developing dental caries, demineralization of teeth, tooth sensitivity, mucosal infections[1] like oral fungal infections (e.g. candidiasis), taste changes, halitosis, or burning mouth.[3]

Objectives

1) Describe the epidemiology, risk factors and clinical presentation of the condition
2) Explain the diagnosis and differential diagnosis of the condition
3) Identify the goals of treatment for the condition
4) List the pharmacological and non-pharmacological options for treatment and prevention of the condition.
5) Discuss monitoring parameters for the condition.

Outline

1) Introduction to Xerostomia
2) Epidemiology
3) Pathophysiology
4) Etiology and Risk factors
5) Symptoms
6) Diagnosis
7) Potential complications
8) Goals of treatment
9) Management
10) References

1) INTRODUCTION TO XEROSTOMIA

1.1. Xerostomia is typically associated with salivary gland hypofunction[1] causing a reduced salivary flow, or caused by changes in the biochemical composition of saliva.[2] The normal stimulated salivary flow rate averages 1.5–2.0mL/min while the unstimulated salivary flow rate is approximately 0.3–0.4mL/min. A diagnosis of hyposalivation is made when the stimulated salivary flow rate is ≤0.5–0.7mL/min and the unstimulated salivary flow rate is ≤0.1mL/min. Xerostomia in patients with objective hyposalivation is diagnosed when the

rate of saliva flow is less than the rate of fluid absorption across the oral mucosa plus the rate of fluid evaporation from the mouth.[3]

1.2. There are a variety of potential causes of xerostomia, including dehydration, medication use, chemotherapy and/or radiation therapy of the head and neck, autoimmune diseases, other chronic diseases (e.g. Sjögren's syndrome), and nerve damage.[1] Other factors include depression, anxiety and stress, or malnutrition.[3]

1.3. It was reported that the most frequent cause of hyposalivation is the use of certain medications, such as anticoagulants, antidepressants, antihypertensives, antiretrovirals, hypoglycemics, levothyroxine, multivitamins and supplements, non-steroidal anti-inflammatory drugs, and steroid inhalers.[3]

2) EPIDEMIOLOGY

2.1. The estimated prevalence of xerostomia in the population varies widely depending on case definitions used and differences in study samples (e.g., age range, health status).[1] It can range from 5.5% to 46%[3], while a 2018 systematic review reported an overall estimated prevalence of xerostomia in approximately 22% of the global population.[1]

2.2. Studies have shown differences in the prevalence between the sexes and xerostomia appears to increase with increasing age.[3] Previous studies have reported xerostomia prevalence estimates ranging from 10 to 26% in men to 10 to 33% in women.[1] On the other hand, xerostomia affects 30% of patients older than 65 years and up to 40% of patients older than 80 years.[1] Its prevalence is generally higher among older individuals, typically due to polypharmacy where several xerogenic drugs may be taken concurrently for chronic conditions[3]. Also, with age, various medical conditions develop over time.[1]

3) PATHOPHYSIOLOGY

3.1. All major and minor salivary glands have nerve supplies, and upon stimulation, salivatory nuclei in the medulla generate an efferent response. The efferent nerve impulses release acetylcholine, which acts on muscarinic (M3) receptors, thereby stimulating salivary glands to produce saliva.[4]

3.2. Histologically, major salivary glands are made up of salivary acini and ducts, which produce 2 types of fluids, serous and mucinous. Pathology arises when there is a dysfunction of gland innervation, or the gland itself.[4]

4) ETIOLOGY AND RISK FACTORS

4.1. Side effect of certain medications

4.1.1. Drugs commonly associated with xerostomia:[1,2]

- Antidepressants (particularly tricyclic antidepressants) and psycholeptics
- Urologic medications e.g. those for urinary incontinence
- Selective Serotonin Reuptake Inhibitors (SSRIs), particularly when combined with benzodiazepines

- Diuretics
- Antihypertensive drugs
- Angiotensin-converting enzyme inhibitors (ACE inhibitors)
- Oral hypoglycemics, mainly sulfonylureas[3]
- Acetylsalicylic acid
- Iron supplements
- Antihistamines
- Antiseizure/antispasmodics

4.1.2. Other drugs include those used to treat obesity, diarrhea, asthma (certain bronchodilators), and Parkinson's disease.[2]

4.2. Aging

4.2.1. Xerostomia affects the elderly population significantly, typically due to polypharmacy with concurrent use of xerogenic drugs[3] and the development of chronic conditions with age[1]

4.3. Cancer therapy[1]

4.3.1. Oral complications of cancer chemotherapy or head and neck cancer radiotherapy can be acute (i.e., develop during therapy) or chronic (i.e., develop months to years after therapy). These therapies can cause xerostomia/salivary gland hypofunction via direct toxicity to salivary glands and oral tissues or indirect damage due to regional or systemic toxicity.

4.3.2. Xerostomia can also occur following hematopoietic stem-cell transplantation and as part of salivary gland graft-versus-host disease

4.3.3. Sialadenitis, or infection of the salivary gland, is another potential acute oral toxicity associated with chemo/radiotherapy

4.3.4. Radioactive iodine, which is used to treat some thyroid cancers, can damage salivary glands (primarily the parotid glands) in a dose-dependent fashion

4.4. Side effect of certain diseases and infections[2]

4.4.1. Sjögren's syndrome
4.4.2. HIV infection
4.4.3. Alzheimer's disease
4.4.4. Diabetes
4.4.5. Anemia
4.4.6. Rheumatoid Arthritis
4.4.7. Hypertension

4.5. Nerve damage[2]

4.5.1. May result from an injury to the head or neck

4.6. Dehydration[2]

4.6.1. Can be a result of fever, excessive sweating, vomiting, diarrhea, blood loss, and burns

4.7. Surgical removal of the salivary glands[2]

4.8. Lifestyle factors such as:

4.8.1. Smoking or chewing tobacco[2]
4.8.2. Continuous breathing through an open mouth[2]
4.8.3. Alcohol use[1]

4.8.4. Consumption of excessive caffeine or spicy food[1]

5) SYMPTOMS

5.1. The most commonly reported symptoms of xerostomia[2] include the following:
- 5.1.1. Thirst
- 5.1.2. Saliva that seems thick
- 5.1.3. Stringy, dry, sticky feeling in the mouth
- 5.1.4. Halitosis (bad breath)
- 5.1.5. Dry, hoarse throat
- 5.1.6. Dry, irritable and scratchy tongue
- 5.1.7. Burning or tingling sensation of the tongue
- 5.1.8. Difficulty speaking
- 5.1.9. Inability to chew, swallow or taste food
- 5.1.10. Dry nasal passages
- 5.1.11. Painful sores of the mouth and tongue
- 5.1.12. Chapped lips
- 5.1.13. Increased plaque
- 5.1.14. Tooth decay
- 5.1.15. Gum disease

5.2. Physical examination of the oral cavity may reveal the following:[2]
- 5.2.1. Oral mucosa may be dry and sticky, or it may appear erythematous due to an overgrowth of *Candida albicans*. The red patches often affect the hard or soft palate and dorsal surface of the tongue. Occasionally, pseudomembranous candidiasis will be present, appearing as removable white plaques on any mucosal surface.
- 5.2.2. Little or no pooled saliva in the floor of the mouth
- 5.2.3. Tongue may appear dry with decreased numbers of papillae
- 5.2.4. Saliva may appear stringy, ropy or foamy
- 5.2.5. Dental caries may be found at the cervical margin or neck of the teeth, the incisal margins or the tips of the teeth

6) DIAGNOSIS

6.1. Diagnosis of xerostomia may be based on evidence obtained from the patient's history, an examination of the oral cavity and/or sialometry, a procedure that measures the flow rate of saliva.[2]

6.2. A thorough medical history should be obtained in order to identify any known causes of xerostomia.[3] Xerostomia should be considered if the patient complains of dry mouth, particularly at night, or of difficulty eating dry foods such as crackers. When the mouth is examined, a tongue depressor may stick to the buccal mucosa. In women, the “lipstick sign,” where lipstick adheres to the front teeth, may be a useful indicator of xerostomia.[2]

6.3. A careful oral examination is fundamental to identify clinical signs pathognomonic for hyposalivation. Several helpful signs have been proposed by Osailan et al:[3]
- 6.3.1. Sticking of an intraoral mirror to the buccal mucosa or tongue

6.3.2. Frothy saliva
6.3.3. No saliva pooling in floor of mouth
6.3.4. Loss of papillae of the tongue dorsum
6.3.5. Altered/smooth gingival architecture
6.3.6. Glassy appearance to the oral mucosa (especially the palate)
6.3.7. Lobulated/deeply fissured tongue
6.3.8. Cervical caries (more than two teeth)
6.3.9. Mucosal debris on palate (except under dentures)

6.4. Several office tests and techniques can be utilized to ascertain the function of salivary glands.[2]
6.4.1. In sialometry, or salivary flow measurement, collection devices are placed over the parotid gland or the submandibular.
6.4.2. Sialography is an imaging technique that may be useful in identifying salivary gland stones and masses. It involves the injection of radio-opaque media into the salivary glands.
6.4.3. Salivary scintigraphy can be useful in assessing salivary gland function. Technetium-99m sodium pertechnate is intravenously injected to ascertain the rate and density of uptake and the time of excretion in the mouth.

6.5. Patients complaining of xerostomia should be interviewed and their medication history should be reviewed in order to identify medications that can reduce the saliva flow.[2,3] It may be possible to change medications or dosages to provide increased salivary flow. Symptoms of xerostomia are often worse between meals, at night and in the morning. Therefore, consider modifying drug schedules to achieve maximum plasma levels when the patient is awake.[2]

7) POTENTIAL COMPLICATIONS

7.1. Xerostomia can lead to these localised conditions:
7.1.1. Markedly increased dental caries[5]
7.1.2. Increased plaque, tooth decay and gum disease[6]
7.1.3. Parotid gland enlargement[5]
7.1.4. Sores or split skin at the corners of the mouth[6]
7.1.5. Inflammation and fissuring of the lips (cheilitis)[5]
7.1.6. Inflammation or ulcers of the tongue and buccal mucosa[5,6]
7.1.7. Oral thrush/candidiasis[5,6]
7.1.8. Salivary gland infection (sialadenitis)[5]
7.1.9. Halitosis[5]
7.1.10. Cracking and fissuring of the oral mucosa[5]

7.2. Xerostomia can often contribute to both minor and serious health problems
7.2.1. It can result in poor nutrition, mainly due to chewing and swallowing issues[,6]
7.2.2. It can affect dental and psychological health[5]
7.2.3. It is commonly associated with constant sore throat, burning sensation, difficulty speaking and swallowing, hoarseness and/or dry nasal passages[5]
7.2.4. Xerostomia is an original hidden cause of gum disease and tooth loss in 3 out of every 10 adults. If left untreated, xerostomia decreases the oral pH and significantly increases the development of plaque and dental caries.[5]

7.3. Referral to the dentist or doctor should be made to prevent and treat complications

8) GOALS OF TREATMENT

8.1. To identify the possible cause(s) of xerostomia[1] to facilitate appropriate management

8.2. To relieve discomfort[1] from symptoms and effects of xerostomia

8.3. To increase salivary flow[3]

8.4. To prevent complications such as dental caries and periodontal infections[1]

9) MANAGEMENT

9.1. Management of xerostomia and hyposalivation should emphasize patient education and lifestyle modifications.[1]

9.2. Various palliative and preventive measures, including pharmacologic treatment with salivary stimulants, topical fluoride, saliva substitutes, and use of sugar-free gum/mints, may alleviate some symptoms of dry mouth and may improve a patient's quality of life.[1]

9.3. Coping strategies for relieving dry mouth include:[1]
- 9.3.1. Sipping water or sugarless, caffeine-free drink
- 9.3.2. Sucking on ice chips
- 9.3.3. Using lip lubricants frequently (e.g. every 2 hours) to soothe dry or cracked area[7]
- 9.3.4. Chewing sugar-free gum or sucking on sugar-free candy - Products that contain xylitol may also help prevent cavities. However, in some people, xylitol, which is often found in sugar-free gum or sugar-free candies, may cause gas or diarrhea if consumed in large amounts.[7]
- 9.3.5. Avoiding salty or spicy food, or dry, hard or crunchy food
- 9.3.6. Avoiding sticky, sugary foods
- 9.3.7. Avoiding irritants such as alcohol (including alcohol-containing mouth rinses), tobacco, and caffeine
- 9.3.8. Avoid OTC antihistamines and decongestants[7]
- 9.3.9. Drinking fluids to ensure proper hydration[3]
- 9.3.10. Using a humidifier at night
- 9.3.11. Breathing through the nose instead of the mouth, and consider seeking treatment for snoring if needed[7]

9.4. Saliva is important to maintain the health of teeth and mouth, and protecting the teeth may help with dry mouth. Dental and oral health-specific recommendations include the following for patients with dry mouth:[1]
- 9.4.1. Brush teeth gently at least twice a day with fluoridated toothpaste
- 9.4.2. Floss teeth every day
- 9.4.3. Schedule dental visits at least twice a year (with yearly bitewing radiographs)
- 9.4.4. Consult a dentist if it can be beneficial to use prescription fluoride toothpaste, toothpaste containing betaine, or a tooth gel to neutralize bacteria acids.[7]

9.4.5. Prompt treatment of oral fungal or bacterial infections
9.4.6. Application of 0.5% fluoride varnish to teeth
9.4.7. Dental soft- and hard-tissue relines of poorly fitting prostheses and use of denture adhesives

9.5. Salivary Stimulants

9.5.1. Should be considered in patients with residual salivary gland function[1] as their effect depends on the presence of functional glandular tissue[3]

9.5.2. Sugar-free chewing gum, candies, and mints can be used to stimulate salivary output.[1] In particular, chewing gums have been shown to increase saliva secretion and decrease oral mucosal friction.[3]

9.5.3. The US FDA has approved two oral sialagogues with similar benefit[3] to treat dry mouth:

- Pilocarpine tablet (Salagen® 5mg (POM)) is typically administered at a dose of 5mg three times a day for at least 3 months[1]
- Pilocarpine is a parasympathomimetic medication with muscarinic action[3]
- Pilocarpine is contraindicated in individuals with narrow-angle glaucoma and iritis, and should be used with caution in individuals with chronic pulmonary disease, asthma, or cardiovascular diseases.[3]
- Cevimeline hydrochloride capsules (not available in Singapore) are prescribed at a dose of 30 mg three times a day for at least 3 months[1]
- Cevimeline has a stronger affinity for M3 muscarinic receptors[3]
- Adverse effects include sweating, cutaneous vasodilation, nausea and vomiting, diarrhea, hiccup, hypotension and bradycardia, increased urinary frequency, bronchoconstriction, and vision problems[1]
- Both pilocarpine and cevimeline are relatively contraindicated in patients with uncontrolled asthma or chronic pulmonary disease and in β-adrenergic blocker users, and should be used with caution in patients with active gastric ulcers or uncontrolled hypertension.[3]

9.6. Saliva Substitutes and other oral care products[1,3]

9.6.1. Saliva substitutes aim to increase viscosity and mimic natural saliva without altering the salivary flow

- They typically contain a combination of carboxymethylcellulose or hydroxyethylcellulose and glycerin to increase viscosity, as well as buffering and flavoring agents (e.g., sorbitol, xylitol), and minerals such as fluoride, calcium and phosphate ions.
- Provide temporary relief of symptoms (but does not cure dry mouth), and may be used as often as needed[1]

9.6.2. Other OTC oral care products for dry mouth include alcohol-free mouth rinses, lozenges, moisturizing oral sprays and gels, or specially formulated toothpastes

- Some of these topical agents contain olive oil, betaine and xylitol, and may be effective in improving xerostomia secondary to medication use.[3]
- When oral lubricants are considered, the gel formulation appears to be the most efficient and appreciated by patients.[3]
- Patients taking oral lozenges of anhydrous crystalline maltose showed an increase in saliva production and a decrease in perceived symptoms of xerostomia.[3]
- Other remedies include mucoadhesive lipid-based bio-erodible tablets or mucin spray, although their efficacy for management of xerostomia remains

controversial. Mucin-containing lozenges provided benefit for the treatment of xerostomia when compared to a placebo.[3]

- Other oral sprays, specifically oxygenated glycerol tri-ester, serve as an alternative treatment for dry mouth and have been proven to be more effective than other commercially available saliva substitutes.[3]

Some OTC Saliva Substitutes and Other Xerostomia Products Available in Singapore		
Trade Name	**Primary Ingredients**	**Points to note**
Bioxtra Dry Mouth Gel Mouth Spray	Natural enzymes, moisturizers, fluoride, xylitol	• Spread liberally to the gums and under dentures • Remove or spit out excess • Do not rinse mouth after application. Repeat as necessary • Contains traces of milk and egg white proteins. Do not use if allergic to any of the ingredients
Bioxtra Dry Mouth Oral Gel	Natural enzymes, moisturizers, xylitol	• Apply liberally to gums and tongue or under dentures, whenever mouth feels dry, especially at night • Remove or spit out excess • Do not rinse mouth after application. Repeat as necessary • Contains traces of milk and egg white proteins. Do not use if allergic to any of the ingredients • Contains dextrose. Use with caution in diabetics
Bioxtra Dry Mouth Ultra Mild Mouthrinse	Natural enzymes, moisturizers, fluoride, xylitol	• Use about 10ml of mouth rinse for 30 seconds after brushing • Do not rinse with water immediately after use • Use regularly throughout the day (maximum of 5 times a day) • Not recommended for children below 6 years old • Contains traces of milk and egg white proteins. Do not use if allergic to any of the ingredients
Oral Seven Moisturising Mouth Gel	Natural enzymes, fluoride, calcium	• Use as needed daily, especially at night • Up to 7 hours of relief for mouth and throat dryness • Squeeze 1-2cm onto index finger, place gel on tongue, then use tongue to spread around mouth • Safe to swallow • Does not contain sugar or saccharin

Oral Seven Moisturising Mouthwash	Natural enzymes, xylitol, calcium	• Rinse mouth with a capful of mouthwash for 30 seconds after brushing • Use 2-3 times a day or as needed • Does not contain sugar or saccharin
Oral Seven Moisturising Toothpaste	Natural enzymes, fluoride, calcium	• Use 2-3 times a day, for brushing thoroughly especially after each meal • For children 2-6 years old: use a pea-size amount for supervised brushing to minimize swallowing • Does not contain sugar or saccharin
Oral Seven Mouth Spray	Natural enzymes, xylitol	• Use as often as required with a gentle spray • May be swallowed as it does not contain fluoride • Use with caution in children 12 years and below

9.7. Changes in medications[3]

9.7.1. For medications known to induce salivary gland hypofunction, the physician may consider decreasing the dosage, or potentially replacing the medications with less xerogenic drugs. Studies have shown that xerostomia then becomes more manageable.

9.8. Other therapies[3]

9.8.1. Intraoral electro-stimulation has been tested to increase salivary flow.

9.8.2. Intraoral appliances, such as the saliva stimulation device Saliwell Crown or the electrostimulating device GenNarino, have been effective in reducing dry mouth and increasing the production of saliva.

9.8.3. Acupuncture may be a useful adjunct for the stimulation of salivary flow in some patients with xerostomia and in patients with irradiation-induced xerostomia. However, additional larger studies are necessary to confirm these findings.

9.8.4. Patients who undergo radiation of the head and neck region may benefit from the use of intensity-modified radiation therapy and/or of amifostine (cytoprotective agent).

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Gingivitis

Introduction

Gingivitis, also known as gum inflammation, is an inflammatory condition of the gingival tissue, most commonly caused by bacterial plaque (dental biofilm) that accumulates daily on the teeth.[1] Unlike periodontitis, there is no attachment loss in gingivitis and thus, no migration of the junctional epithelium. The inflammation is restricted to the soft-tissue area of the gingival epithelium and connective tissue.[2]

The gingival tissues may present with redness, slight swelling, a shiny surface, tenderness, "puffiness" and bleeding on tooth brushing or gentle probing.[1,2] Gingivitis seldom causes spontaneous bleeding and is commonly painless, therefore many patients do not recognise the disease and fail to seek attention.[2] Treatment involves thorough professional tooth cleaning and effective daily removal of dental plaque by tooth brushing and cleaning between the teeth.[1]

Objectives

1) Describe the epidemiology, risk factors and clinical presentation of the condition
2) Explain the diagnosis and differential diagnosis of the condition
3) Identify the goals of treatment for the condition
4) List the pharmacological and non-pharmacological options for treatment and prevention of the condition.
5) Discuss monitoring parameters for the condition.

Outline

1) INTRODUCTION TO GINGIVITIS

1.1. Various forms of gingivitis differ based on clinical appearance, duration of infection, severity, and etiology. However, the chronic form of gingivitis that is caused by plaque is considered to be the most frequent variant.[2]

1.2. Necrotising gingivitis is an atypical, acute form of bacteria-related gingivitis that is rarely found in developed countries. It is a more serious condition that is mainly found in developing countries and is associated with people with severe malnutrition or people living with HIV with low CD4 counts.[1]

1.3. Many other potentially serious conditions (congenital or acquired, and a number of genetic syndromes) may feature inflammation or lesions of the gingiva and these must always be considered within the differential diagnosis.[1]

2) EPIDEMIOLOGY

2.1. Gingivitis is the most common among periodontal diseases and is commonly seen in children and adults. It is more prevalent in males as compared to females as it has been found that females tend to follow better oral care regimes.[2]

2.2. Studies have found gingivitis to be more prevalent in people with low socioeconomic status, as people with high socioeconomic status tend to show a more positive attitude towards maintaining oral hygiene and they generally have better access to healthcare.[2]

2.3. Studies reveal that the prevalence of gingivitis is higher among pregnant women than non-pregnant women, and more severe forms of gingivitis have more often been seen in pregnant women.[2]

2.4. The most frequently seen types of gingivitis are plaque-induced, hormonal, acute ulcerative necrotizing, drug-induced, or spontaneously presenting hyperplastic gingivitis. Among them, the more predominant form of gingivitis is plaque-induced, which accounts for far more cases than all other variants combined.[2]

3) PATHOPHYSIOLOGY

3.1. Gingivitis can be divided into initial, early, and established stages, and periodontitis has been indicated as the advanced stage as illustrated by Image 3.[2,5]

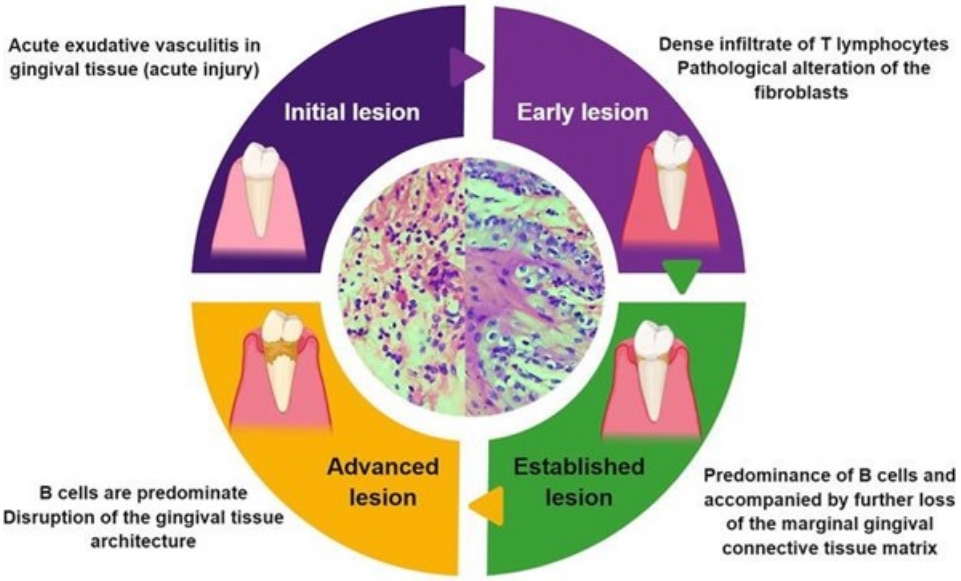

Image 3: Progression of Stages for Gingivitis[5]

3.1.1. Initial Lesion

- Characterized by an acute exudative inflammatory response, a raised gingival fluid flow, and the migration of neutrophils from the blood vessel of the

subgingival plexus located in the gingival connective tissue to the gingival sulcus

- An alteration of the matrix of the connective tissue located next to vessels results in the accumulation of fibrin in the area
- Initial lesion is seen within 4 days of the initiation of plaque accumulation
- There is a destruction of collagen caused by collagenase and other enzymes secreted by the neutrophils. About 5% to 10% of the connective tissue is occupied by the inflammatory infiltrate in this stage.

3.1.2. Early Lesion

- The early lesion is consistent with delayed hypersensitivity
- Usually appears after one week from the beginning of plaque deposition
- Clinical signs of gingivitis, such as redness and bleeding from the gingiva start appearing
- Inflammatory cells that predominate in this lesion are lymphocytes accounting for 75% of the total, and macrophages. A small number of plasma cells are also seen. Along with the inflammatory infiltration that occupies 5% to 15% of the connective tissue of the gingival margin, there is loss of collagen in the affected area that reaches 60% to 70%. Furthermore, the local fibroblasts undergo a series of pathological changes, and the gingival fluid flow and the number of leukocytes migrating to the region continue to increase. Neutrophils and mononuclear cells are also increased in the junctional epithelium.
- The duration of the early lesion has not yet been determined, and it can remain longer than previously expected.

3.1.3. Established Lesion

- Increased collagenolytic activity is seen along with a rise in the number of macrophages, plasma cells, T and B lymphocytes. However, the predominant cells are plasma cells and B lymphocytes.
- In this stage, a small gingival pocket lined with a pocket epithelium is created. The lesion exhibits a high degree of organization. It has been suggested that the severity of gingivitis correlates with a growth in the B cells and plasma cells population, and a decrease in the number of T cells.
- An established lesion may follow two paths:
  - It can either remain stable for months or years, or
  - Progress to a more destructive lesion, which appears to be related to a change in the microbial flora or infection of the gingiva
- This stage has shown to be reversible after an effective periodontal therapy that results in an increase in the number of microorganisms associated with periodontal health that directly correlates with a reduction in the plasma cells and lymphocytes.

3.1.4. Advanced Lesion

- This stage is a transition to periodontitis
- Characterized by attachment loss that is irreversible
- Inflammatory changes and bacterial infection start to affect the supporting tissues of the teeth and the surrounding structures such as gingival, periodontal ligament, and alveolar bone resulting in their destruction and may eventually result in tooth loss.

4) ETIOLOGY AND RISK FACTORS

4.1. Gingivitis is caused by the microbial plaque deposits located in or close to the gingival sulcus. The microorganisms more strongly associated include species of *Streptococcus*, *Fusobacterium*, *Actinomyces*, *Veillonella*, and *Treponema*. *Bacteroides*, *Capnocytophaga*, and *Eikenella* are also potentially linked to the etiology of the disease. There may be other local or systemic etiologic factors that intensify plaque deposition or the vulnerability of the tissue to microbial attacks.[2]

4.2. Based on the etiology, gingivitis can be classified into different types.

4.3. Plaque-induced Gingivitis[2] (Image 3)

4.3.1. The most common cause of gingivitis

4.3.2. Poor oral hygiene can result in the formation of a thin film of plaque on the tooth surface, and if not regularly removed, can harden up and form calculus. As plaque harbors a large number of bacteria, inflammation can occur in the gingival tissue.

4.3.3. Local factors that can contribute to plaque formation include:

- Crowding of teeth which makes plaque removal difficult
- Misaligned teeth which makes cleaning more difficult and often requires orthodontic correction
- Dental prosthesis that does not have an adequate fit or is not properly finished

4.3.4. Often associated with eruption gingivitis among children undergoing tooth eruption, where plaque accumulation tends to increase in the area where primary teeth are exfoliating, and permanent teeth are erupting. Oral hygiene may be difficult to maintain in these areas.

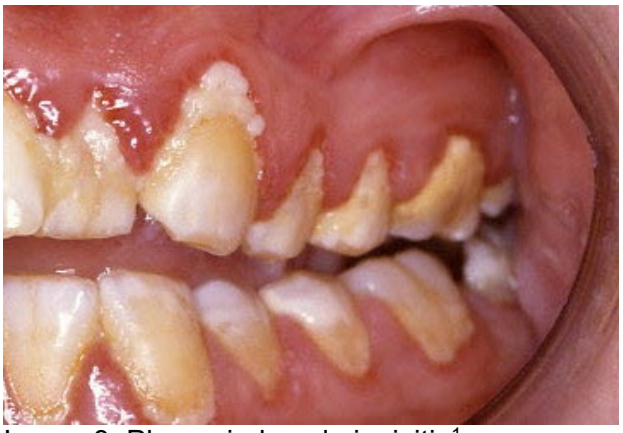

Image 3: Plaque-induced gingivitis[1]

4.4. Nutritional Gingivitis[2]

4.4.1. May result from vitamin C deficiency

4.4.2. It has been found that a modern lifestyle with the intake of an increased amount of refined carbohydrates and an increased ratio of omega-6 to omega-3 fatty acids can promote the inflammatory process.

4.4.3. Carbohydrates with a high glycemic index can promote the inflammatory process through activation of NFkB and oxidative stress.

4.5. Hormonal Gingivitis[2]

4.5.1. With hormonal changes in pregnancy, there is a greater predisposition to dilating blood vessels. These factors contribute to an exaggerated inflammatory response by the gingival tissues even to a small amounts of plaque accumulation. It has been suggested that the levels of estrogen determine the severity of gingival inflammation created against the biofilm at the gingival margin.

4.5.2. In puberty, puberty gingivitis can occur, where hormonal changes can also affect how the gingival tissue reacts to plaque accumulation. During adolescence, gingivitis appears earlier in girls (11 to 13 years) than in boys (13 to 14 years). It has been found that receptors for both estrogens and testosterone are present in the cytoplasm of the cells of the gingiva, making it an easy target organ for these steroid hormones, resulting in gingivitis.

4.6. Drug-induced Gingivitis[2]

4.6.1. Various drugs used for systemic conditions can cause gingivitis as a side effect

- Phenytoin
- Calcium channel blockers
- Anticoagulants and fibrinolytic agents
- Oral contraceptive agents
- Protease inhibitors
- Vitamin A and analogs
- Anti-rejection drugs like ciclosporin[3]

4.6.2. It was postulated that gingival inflammation was brought about by the ability of the metabolites of these drugs to induce the proliferation of fibroblasts. An imbalance between the synthesis and the degradation of the extracellular matrix leads to the accumulation of immature proteins in the extracellular matrix, particularly collagen, and in turn, results in gingivitis.

4.7. Other risk and influencing factors[2] that can contribute to the development of gingivitis include:

4.7.1. Smoking and tobacco chewing

- Thought to reduce gingival blood flow (thereby masking/suppressing the signs and symptoms of gingivitis), impair wound healing and increase production of inflammation-mediating cytokines[3]

4.7.2. Systemic conditions

4.7.3. Genetic factors (hereditary gingival fibromatosis)

4.7.4. Local conditions (dry mouth, crowded teeth, calculus, overhanging restorations, partial dentures[3])

4.7.5. Diabetes[3] (increased risk of developing periodontal diseases)

- Poorly controlled diabetes enhances the signs and symptoms of gingivitis and periodontitis and has an adverse effect on wound healing, making treatment more difficult

5) SYMPTOMS AND DIAGNOSIS

5.1. Healthy gingival tissue looks pink or pigmented in dark-skinned patients, firm, with no signs of redness or swelling, and with no bleeding after gently passing a periodontal probe along the gingival crevice. On periodontal probing, healthy gingiva shows less than 3 mm crevice and there is no bone loss on x-rays.[2]

5.2. In many instances, gingivitis may go unnoticed by the patient as the disease may exist and progress without any symptoms.[2]

5.3. When symptomatic, the following are usually seen[2]:
- 5.3.1. Bleeding from the gingiva while brushing, flossing, and sometimes eating hard food
- 5.3.2. Halitosis that does not resolve even after performing oral hygiene
- 5.3.3. Physical examination of the oral cavity revealing the presence of an inflamed and tender gingiva that usually bleeds on gentle probing
- 5.3.4. A more rounded and shiny aspect, instead of gingival tissue with stippled aspect and gingival margins with a knife-edge appearance found in healthy gingiva
- 5.3.5. Significant plaque and calculus deposits

5.4. Gingivitis can be differentiated from periodontitis by the attachment loss undergone in the latter and it can be seen clinically during periodontal probing. They can also be differentiated histologically and radiographically.[2]

6) POTENTIAL COMPLICATIONS

6.1. If identified and treated, gingivitis can easily be resolved as it is reversible and the altered tissues can return to normal once the dental biofilm has been removed.[2]

6.2. The most common complication of chronic gingivitis is the progression of the inflammation towards the underlying tissue and bone, resulting in periodontitis. If gingivitis progresses to periodontitis, connective tissue attachment loss and bone destruction will occur, which may ultimately lead to tooth loss.[2]

6.3. Of note, gingivitis is a precursor of periodontitis but gingivitis does not always progress to periodontitis.[2]

6.4. Referral to the dentist should be made to prevent and treat complications where needed.

7) GOALS OF TREATMENT

7.1. Treatment is aimed primarily at reduction of etiologic factors to reduce or eliminate inflammation, thereby allowing gingival tissues to heal.[4]

7.2. In individuals with chronic gingivitis, initial treatment should be directed at reducing oral bacteria and associated calcified and noncalcified deposits.[4]

8) MANAGEMENT

8.1. Plaque-induced gingivitis
- 8.1.1. Dental plaque deposits should be removed to reduce inflammation[2]
- 8.1.2. Remove supra-gingival plaque, calculus and stain and sub-gingival deposits and, if possible, correct any local plaque retentive factors[3]

8.1.3. Early stages of gingivitis can be easily managed if the patient starts following oral hygiene protocol, which includes regular tooth brushing with an appropriate technique and interproximal hygiene, such as dental flossing, or the use of interproximal brushes[2]

8.1.4. Patients with chronic gingivitis, but without significant calculus, alterations in gingival morphology, or systemic diseases that affect oral health, may respond to a therapeutic regimen consisting of improved personal plaque control alone[4]

8.1.5. However, many patients lack the motivation or skill to attain and maintain a plaque-free state for significant periods of time and may not achieve long-term inhibition of gingivitis without periodic professional reinforcement[4]

8.1.6. Removal of plaque and calculus can also be professionally done by scaling and root planning using hand, sonic, or ultrasonic instruments[4], according to the severity of the condition[2]. These procedures aim to remove plaque and calculus to reduce subgingival bacteria below a threshold level capable of initiating clinical inflammation[4].

8.1.7. Medications in the form of antiseptic mouthwash that contains chlorhexidine can also be prescribed in conjunction with the mechanical removal of plaque. It has been suggested that the use of chlorhexidine mouthwashes in addition to the usual toothbrushing and interproximal cleaning leads to a significant decrease in the build-up of dental biofilm. The concentration of the chlorhexidine rinse does not affect its effectiveness.[2]

8.1.8. Among individuals who do not perform excellent oral hygiene, supragingival irrigation with or without medicaments is capable of reducing gingival inflammation beyond that normally achieved by tooth brushing alone. This effect is likely due to the flushing out of subgingival bacteria.[4]

8.2. Other types of gingivitis

8.2.1. In drug-induced gingivitis, the doctor can consider changing the medication to improve the condition[2]

8.2.2. If gingivitis is due to nutritional deficiency, supplements can be prescribed[2]

8.2.3. For conditions related to systemic factors (e.g. diabetes, pregnancy, etc), gingival health may be attained once the systemic condition is resolved and plaque control is maintained[4]

8.3. Herbal remedies[2]

8.3.1. Medicinal or herbal plants (e.g. pomegranate, tea, and chamomile) have anti-inflammatory properties and can resolve both gingival bleeding and inflammation

8.3.2. Flavonoids and tannins found in these plants are potent anti-inflammatory and astringent phytochemicals.

8.3.3. Some studies proved that there is a synergistic effect when the herbal plants are prescribed along with conventional mechanical procedures of plaque removal, such as scaling.

8.4. Prevention/Patient education

8.4.1. Maintenance of good oral hygiene can prevent plaque formation and thus, gingivitis[2]

8.4.2. Oral Hygiene TIPPS behaviour change strategy can be used to improve patient's oral hygiene[3]

- Talk to the patient about causes of periodontal disease, the importance of oral hygiene, and how to achieve good plaque removal

- Instruct the patient on the use of oral hygiene tools and demonstrate brushing techniques, as well as how to use dental floss or interdental brushes
- Practise cleaning teeth - can have the patient do so in front of the healthcare provider
- Plan to make effective plaque removal/good oral hygiene a habit
- Support the patient with follow-ups on the recommendations at subsequent appointments

8.4.3. Correct brushing technique according to individual needs, frequency of brushing, and use of interproximal hygiene must be taught[2]
8.4.4. Regular dental visits should be emphasized[2]
8.4.5. Use of mouthwash may also be advised[2]
8.4.6. Appropriate supportive periodontal maintenance that includes personal and professional care is important in preventing re-initiation of inflammation[4]

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