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## Non-Microbial Periodontal Conditions

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*Gingival and periodontal inflammation is commonly induced by plaque. However, we all occasionally struggle with inflammation that does not respond to conventional antimicrobial therapy.*

*We will use a practical diagnostic approach and clinical cases to look at major conditions to consider in these patients.*

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### Conflict of Interest





I was an investigator in clinical trials of these products 1998-2009. I received no direct compensation for this:  
 Brush Test: **OralCDx™** (Oral Scan Laboratories)  
 Reflectance Test, Toluidine Blue: **ViziLite™** and **ViziLite Plus™** (Zila Pharmaceuticals)

Neither I, nor members of my family, have any financial interests to disclose related to this presentation.

2

 This course is purely for informational purposes

 Patient care, safety and confidence are paramount. In addition to any direct legal or ethical guidance, adhere to best practices and reasonable professional clinical judgment.

 Use caution if using limited knowledge to integrate any new techniques into your practice. Be cautious about dangers of incorporating techniques and procedures into your practice if the course has not given you adequate, supervised clinical experience in the technique/procedure to allow you to perform it competently.

3

### Learning Objectives

At the end of this educational session, the attendee will be able to:

- 1. Define non-microbial gingivitis.**
- Describe a diagnostic approach based on the suspected tissue of origin and the suspected disease process.
- Differentiate typical features of benign vs malignant lesions.
- Formulate a logical differential diagnosis for a patient with gingival inflammation that is not responsive to conventional antimicrobial therapy.

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### Non-microbial gingivitis

*“Non-dental plaque biofilm-induced gingival diseases include a variety of conditions that are not caused by plaque and usually do not resolve following plaque removal. Such lesions may be manifestations of a systemic condition or may be localized to the oral cavity.*

*“Dental plaque-induced gingivitis has a variety of clinical signs and symptoms, and both local predisposing factors and systemic modifying factors can affect its extent, severity, and progression...”*

• Chapple ILC, Mealey BL, et al. *Periodontal health and gingival diseases and conditions on an intact and a reduced periodontium: Consensus report of workgroup 1 of the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions.* J Periodontol. 2018;89(Suppl 1):S74–S84. <https://doi.org/10.1002/JPER.17-0719>

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### Learning Objectives

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- Differentiate typical features of benign vs malignant lesions.
- Formulate a logical differential diagnosis for a patient with gingival inflammation that is not responsive to conventional antimicrobial therapy.

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**My DAMIEN method asks two key questions about a lesion to solve difficult diagnoses:**

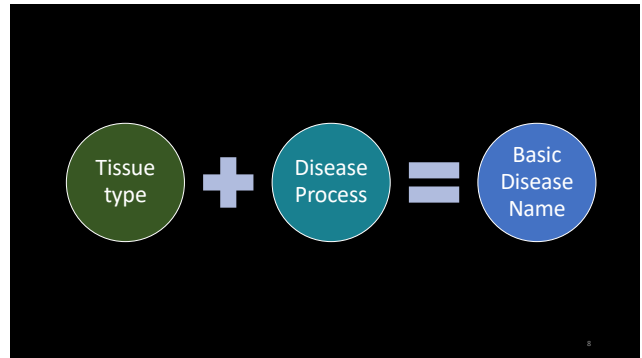
*What **tissue** do you think it started in?*

- Common simple disease name prefix

*What do you think is the **disease process**?*

- Common simple disease name suffix

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**Tissue type + Disease Process = Many Basic Disease Names**

<b>Epithelium</b> • By cell type: i.e. Squamous Cell; By gland type: i.e. Adeno-, Mucoepithelial-	<b>Developmental</b> • Larger: hyperplasia; Smaller: hypoplasia; Failure: to form: atresia
<b>Blood vessels &amp; cells</b> • By type: i.e. Vasculo-, Arterio-, Angio-, Phlebo-, Lympho-	<b>Allergic/immune</b> • -itis
<b>Soft tissue</b> • Fibrous: Fibro-; Fat: Lipo-; Muscle by type: i.e. Myo-, Leiomyo-; Rhabdomyo-; Nerve: Neuro-	<b>Metabolic:</b> • (none specific)
<b>Hard tissue</b> • Bone: Osteo-, Cartilage: Chondro-, Enamel: Amelo-; Dentin: Dentino-	<b>Infectious</b> • -itis
	<b>Environmental</b> • Larger overall: hypertrophy; More cells: hyperplasia; Smaller: atrophy
	<b>Neoplasia</b> • Benign: -ma (usually); Malignant: -carcinoma, sarcoma

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A cloud-shaped graphic containing a list of disease names: Neuroma, Fibromatosis, Foreign body gingivitis, Squamous cell carcinoma, Desquamative gingivitis, Localized juvenile spongiotic gingival hyperplasia, Melanoma, Osteosarcoma, Fibroma, Angioma, Ameloblastoma, Lymphoma, Necrotizing ulcerative periodontitis, and Vasculitis.

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**What tissue do you think it started in?**

**Epithelium**  
 • Squamous Cell

**Blood vessels & cells**  
 • Vasculo-, Angio-, Lympho-

**Soft tissue**  
 • Fibrous: Fibro-; Nerve: Neuro-

**Hard tissue**  
 • Bone: Osteo-, Enamel: Amelo-; Dentin: Dentino-

Especially consider these tissues in periodontal areas...

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**What do you think is the disease process?**

**DAMIEN method**

D	A	M	I	E	N
Developmental	Allergic / Auto-immune	Metabolic	Infectious	Environmental	Neoplastic (Benign or malignant)

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**D**

**Developmental diseases typically**

- Congenital (present at birth)
- Or acquired (develop after birth)
- Stable once established
- Don't respond to antimicrobials - unless secondarily infected
- Some risks may be reduced with appropriate environmental or behavioural modification
- Consider social determinants of health

**A**

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**Developmental diseases rarely**

- Linked to predisposition to other disease processes i.e. malignancy, infection, etc.

**Today's example**

- Hereditary gingival fibromatosis

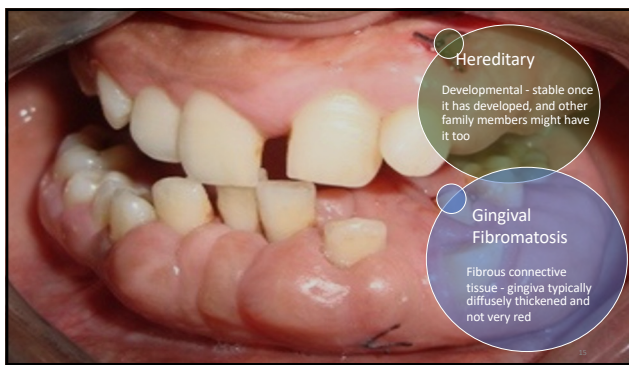
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## Hereditary gingival fibromatosis

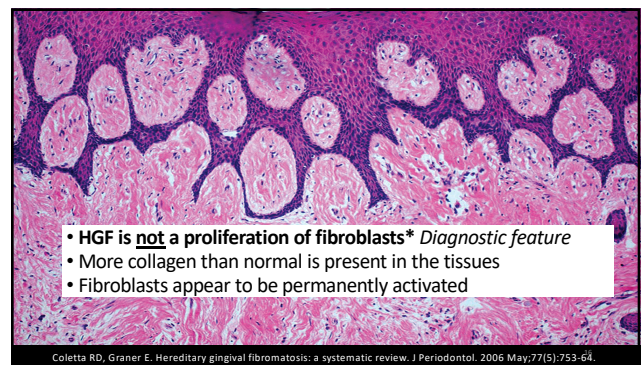
<b>Demographics</b>	Autosomal dominant or recessive	Begins young – some 1 <sup>o</sup> dentition, some 2 <sup>o</sup>
<b>Location</b>	Generalized. Not always symmetrical	Sporadic or in syndromes
<b>Appearance</b>	Slowly progressive enlargement	Range of severity
<b>Other</b>	Surgical excision	Mutations of "Son of Sevenless" genes + others

Hart TC, Zhang Y, Gorry MC, Hart PS, Cooper M, Marazita ML, Marks JM, Cortelli JR, Pallos D. A mutation in the SOS1 gene causes hereditary gingival fibromatosis type 1. Am J Hum Genet. 2002 Apr;70(4):943-54.  
Wai et al. A novel gene SH3BP2 causes hereditary gingival fibromatosis. eLife 11:e66446.  
Strzelec, K., Dziedzic, A., Łazarz-Bartyzel, K. et al. Clinics and genetic background of hereditary gingival fibromatosis. Orphanet J Rare Dis 16, 492 (2021). <https://doi.org/10.1186/s13023-021-02104-9>

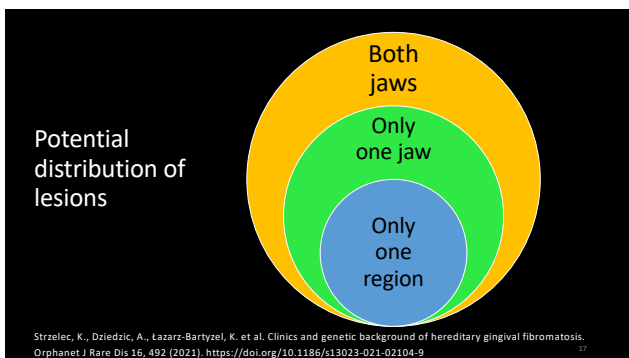
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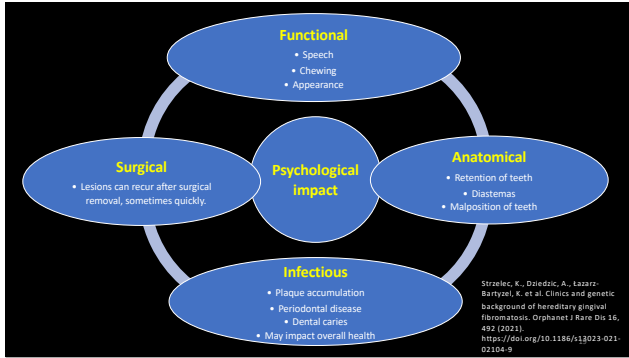
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Typical clinical progress: *slowly progressive, non-hemorrhagic, benign, fibrous gingival overgrowth*

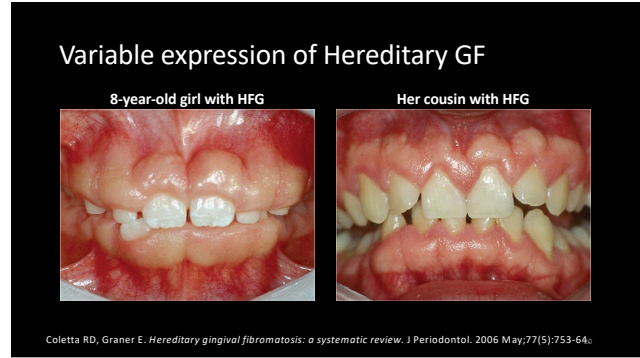
<b>Childhood:</b>	<b>Adolescence:</b>	<b>Adulthood:</b>
Extremely wide zone of keratinized gingiva. Coincides with tooth eruption	Connective tissue within gingiva thickens, mostly due to overproduction of collagenous proteins.	Except for recurrent cases, gingival overgrowth is only slowly progressive

Strzelec, K., Dziedzic, A., Łazarz-Bartyzel, K. et al. Clinics and genetic background of hereditary gingival fibromatosis. Orphanet J Rare Dis 16, 492 (2021). <https://doi.org/10.1186/s13023-021-02104-9>

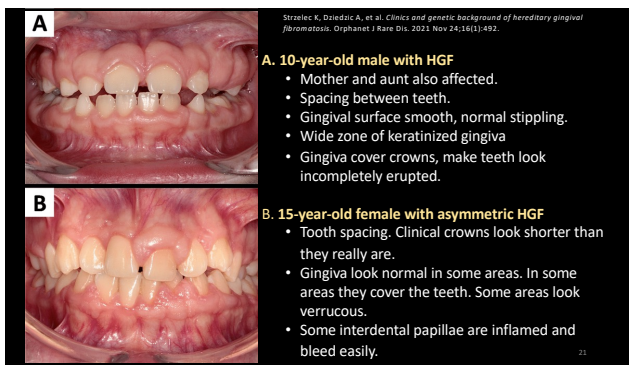
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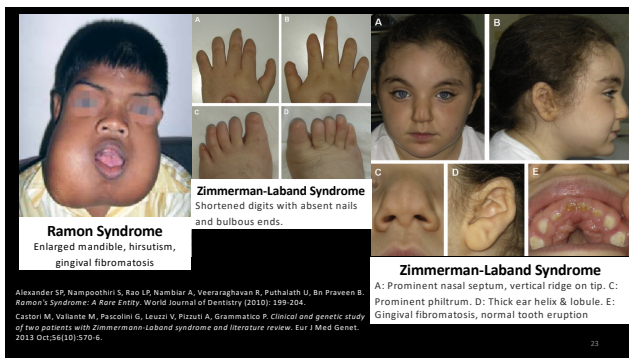


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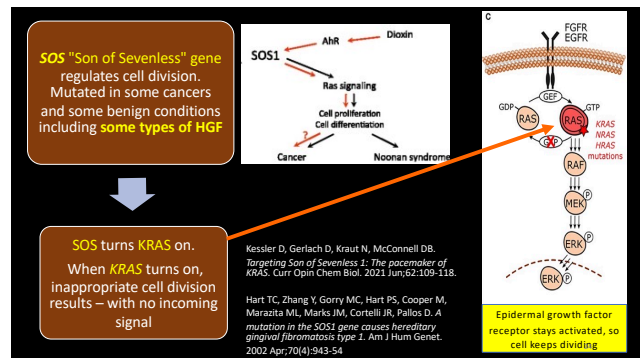
Syndromes with hereditary gingival fibromatosis	Genetic Pattern	Features
Gingival fibromatosis w/ hypertrichosis	AD	Hypertrichosis, intellectual delay
Ramon	AR	Hypertrichosis, intellectual delay, cherubism, seizures, stunted growth, +/- juvenile RA, +/- eye defects
Murray-Puretic-Drescher (Juvenile hyaline fibromatosis)	AR	Multiple hyaline fibromas, osteolysis of terminal phalanges, recurrent infections, stunted growth, premature death
Rutherford	AD	Corneal opacities, delayed tooth eruption
Cross	AR	Microphthalmia, intellectual delay, involuntary muscular writhing (athetosis), hypo-pigmentation
Zimmermann-Laband	AD	Ear and nose defects, dysplastic fingernails, hypoplastic terminal phalanges, hyperextensible joints, enlarged liver and spleen
G. fibromatosis w/ distinctive facies	AR	Macrocephaly, hypertelorism, bushy eyebrows, down-slanted palpebral fissures, flat nasal bridge, hypoplastic nares, Cupid's bow, high arched palate
Jones	AD	Progressive deafness
Prune-belly (Eagle-Barrett)	?	No abdominal muscles, abnormal urinary tract, undescended testes

Coletta RD, Graner E. Hereditary gingival fibromatosis: a systematic review. J Periodontol. 2006 May;77(5):753-64.

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**Allergic/immune diseases typically**

- Appear inflamed
- Develop relatively fast usually, may wax and wane
- Don't respond to antimicrobials unless secondarily infected
- May respond to immune modulators such as steroids
- Some risks may be reduced with appropriate environmental or behavioural modification
- Consider social determinants of health

**Allergic/immune diseases rarely**

- Linked to predisposition to other disease processes i.e. malignancy, infection, etc.


**Today's example**

- Crohn disease

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**Crohn Disease**

- "Gum to bum" granulomatous inflammation
- Most common presentation: Persistent diarrhea, rectal bleeding, and abdominal pain
- Oral lesions tend to coincide with GI flares



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**Systemic**

- Lesions in GI tract most prominent
- Fever, fatigue, loss of appetite, weight loss
- Growth failure, delayed sexual development
- Also may be perianal, skin, muscle, joints, liver, eye, lung

**Oral**

- 80% of patients - may be first sign of disease
- Tend to coincide with GI flares
- Non-specific: Aphthous-like ulcers
- Specific: Cobblestone appearance, typically in vestibule

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Demographics	Etiology
<ul style="list-style-type: none"> <li>• Any age but peaks in 20's</li> <li>• M = F</li> <li>• May be family history</li> <li>• Any race or ethnicity</li> <li>• Peak rates in Caucasian, Ashkenazi Jewish</li> </ul>	<ul style="list-style-type: none"> <li>• Cause still unknown</li> <li>• More common in urban, industrialized areas</li> <li>• More common in northern climates</li> <li>• High-fat and refined-food diet?</li> <li>• NSAIDs may worsen symptoms (but do not cause Crohn's)</li> </ul>

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**Inflammation – the double-edged sword**

- Attacks invading microbes, or
- Attacks native tissues





Illustration by Jason Holley in Nature | A. A Double-Edged Sword: Inflammation and Your Health, Cedars-Sinai Discoveries, 2021 Feb 12. <https://www.cedars-sinai.org/discoveries/inflammation.html>

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Black Death killed 30%-50% of the population of Africa and Eurasia (1346-1350)

Does this mutation carried by their descendants increase their risk of immune diseases like Crohn?



People survived often because of protective mutations – and their descendants inherited these mutations

Klunk, J., Vilgaly, T.P., Demeure, C.E. et al. Evolution of immune genes is associated with the Black Death. *Nature* (2022). <https://doi.org/10.1038/s41586-022-05349-x>

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**ERAP2 carriers were 40% more likely to survive plague**

- MHC class I Ag presentation & CD8<sup>+</sup> T cell activation
- Better at killing invading bacteria
- Strong inflammation - combats infection

**Descendants (ERAP2 carriers) have greater risk of Crohn's**

- Excess inflammation - autoimmune disease
- Same/related mutations in Ankylosing Spondylitis, Behçet's Disease, Psoriasis, Inflammatory Bowel Disease, Juvenile Idiopathic Arthritis, Diabetes Mellitus Type 1, Multiple Sclerosis, others

Klunk, J., Vilgaly, T.P., Demeure, C.E. et al. Evolution of immune genes is associated with the Black Death. Nature (2021). <https://doi.org/10.1038/s41586-022-06312-x>.  
Khor, B., Gardet, A. & Xavier, R. Genetics and pathogenesis of inflammatory bowel disease. Nature 474, 307–317 (2011). <https://doi.org/10.1038/nature10209>

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Intestinal homeostasis requires coordination of epithelium, innate immunity and adaptive immunity.

The diagram illustrates the interaction between the gut epithelium and the immune system. Microbes enter from the lumen, passing through the epithelium (P) and interacting with innate immune cells (IC) and L cells. This triggers innate immune cells and recruits adaptive immune cells (B cell, T<sub>H</sub>17, T<sub>H</sub>22). The balance of signals is affected by genetics, microbiome, diet, and immune factors, leading to the production of IgA by plasma cells.

**Balance of signals is affected by genetics, microbiome, diet, and immune factors**

Khor, B., Gardet, A. & Xavier, R. Genetics and pathogenesis of inflammatory bowel disease. Nature 474, 307–317 (2011). <https://doi.org/10.1038/nature10209>

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Onset of disease affected by **genetics, diet, immune factors, microbiome**

Social determinants of health

Potential treatment could address **diet, immune factors, microbiome**

Surgery for Crohn - now less common	Diet	Immune	Microbiome - Still being developed
<ul style="list-style-type: none"> <li>Remove a diseased segment to relieve blocked GI tract or fistula</li> </ul>	<ul style="list-style-type: none"> <li>Prevent malnutrition &amp; weight loss</li> <li>Promote healthy microbiome, healthy diet?</li> <li>Avoid foods that cause epithelial damage</li> </ul>	<ul style="list-style-type: none"> <li>Steroids</li> <li>Immune suppressants and modulators, stressors</li> <li>Biologic Rx - Block proteins that cause inflammation i.e. infliximab.</li> </ul>	<ul style="list-style-type: none"> <li>Antibiotics</li> <li>Probiotics</li> <li>Fecal microbiota transplantation</li> </ul>

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**Metabolic diseases typically**

- Often associated with a systemic condition
- May affect multiple body systems
- Don't respond to antimicrobials unless secondarily infected
- Respond best to correction of underlying metabolic problem, if possible
- Some risks may be reduced with appropriate environmental or behavioural modification
- Consider social determinants of health

**Metabolic diseases rarely**

- Linked to predisposition to other disease processes i.e. malignancy, infection, etc.

**Today's example**

- Localized juvenile spongiotic gingival hyperplasia (juvenile spongiotic gingivitis)

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**Localized juvenile spongiotic gingival hyperplasia (Juvenile spongiotic gingivitis)**

Demographics	Location	Presentation
<ul style="list-style-type: none"> <li>Onset average age 12-14 years - About puberty</li> <li>Female = male</li> </ul>	<ul style="list-style-type: none"> <li>Anterior gingiva, 2-10 mm</li> <li>Maxilla &gt;&gt; Mandible</li> </ul>	<ul style="list-style-type: none"> <li>Red, papillary areas bleed easily, painless</li> <li><b>No response to hygiene</b> like puberty gingivitis</li> </ul>

Darling MB, Daley TD, Wilson A, Wysocki GP. Juvenile spongiotic gingivitis. J Periodontol. 2007 Jul;78(7):1335-40.  
Mawardi HH, Almazrooa SA, Turkstani HA, Bakhair RS, Almasoudi AG, Bakhamis BA, Azzouz LZ, Alohareef TA, Alsulami SE, Alshahri SA, Albaraj AA. Management of localized juvenile spongiotic gingival hyperplasia: A systematic review. J Dermatol Dermatol Surg 2021;25:3-5

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**"Juvenile"?**

U Pittsburgh study	UCSF study
<ul style="list-style-type: none"> <li>28 cases - Average age 14</li> <li>4 patients under age 9; youngest 3</li> <li>8 patients over age 20; oldest 64</li> </ul>	<ul style="list-style-type: none"> <li>27 cases - Median age 13, Average age 18</li> <li>8 patients under age 10</li> <li>No patients ages 20-65</li> <li>3 patients over age 65: ages 65, 66, 72</li> </ul>

Vaziro RI, Bilodeau EA. Reappraising localized juvenile spongiotic gingival hyperplasia. J Am Dent Assoc. 2019 Feb;150(2):147-153.e2.  
Wang MZ, Jordan RC. Localized juvenile spongiotic gingival hyperplasia - A report of 27 cases. J Cutan Pathol. 2019 Nov;46(11):839-843

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### "Gingival"?

- 2 cases on alveolar ridge/palate (age 55, 78)
- 12 other extragingival lesions with this same microscopic appearance (mean age 49)
- Proposed new name "**Spongiotic hyperplasia**" of oral mucosa

Silveira HA, Toral-Rizo VH, Lara-Carrillo E, Dominguez MHL, Silva EV, Ribeiro-Silva A, Bufalino A, Almeida LV, León JE. Spongiotic hyperplasia of the oral mucosa: case series and immunohistochemical analysis. Oral Maxillofac Surg. 2022 Jun;26(2):333-337.

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### What is the most predictable treatment?

 <p><b>Scaling + CHX not effective</b> 1/1 case - No response to tx</p>	 <p><b>Surgical excision mostly effective</b> 12/ 97 cases recurred</p>	 <p><b>Other excisions effective</b> Cryotherapy 2/2 no recurrence Photodynamic therapy 1/1 no recurrence Surface cauterization + topical clobetasol 1/1 no recurrence</p>
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• Further studies are needed

Mawardi HH, Almazrooq SA, Turktani HA, Balkhair RS, Almasoudi AG, Bakhamis BA, Azzouz LZ, Alsharief TA, Atsulami SE, Alkhatfi SA, Albarqi AA. Management of localized juvenile spongiotic gingival hyperplasia: A systematic review. J Dermatol Dermatol Surg 2021;25:1-5

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### Recent research

- "...the exact cause of SGH and the triggers for activating the aforementioned pathways still need to be investigated in future studies, which may provide more specific answers to why debridement of plaque and calculus is ineffective.
- "Our results may lead to novel treatment approaches for SGH, which may involve medications that block IL-17, preventing surgery, and reducing esthetic gingival defects associated with surgical excision."
- Lin J, Jeong W, Dempster L, Chugh D, Posluns J, Magalhaes M. Spongiotic gingival hyperplasia: identifying new mechanisms and a survey of clinical approach. Oral Surg Oral Med Oral Pathol Oral Radiol. 2026 Jan;141(1):75-85. doi: 10.1016/j.oooo.2025.08.001. Epub 2025 Aug 28. PMID: 40967966.

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### Infectious diseases typically

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- Respond to appropriate antiviral, antibacterial or antifungal therapy
- Should not be treated primarily with immunomodulatory agents such as steroids unless inflammation is driving risk of infection – there are exceptions
- Some risks may be reduced with appropriate environmental or behavioural modification, vaccination, – or control of systemic disease
- Consider social determinants of health

**Infectious disease processes rarely**

- Linked to predisposition to other disease processes i.e. malignancy

**Today's example**

- Linear gingival erythema

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### Linear gingival erythema

- Distinct red linear band along gingival margin
- Now believed to be caused by Candida
- Associated with HIV+ status



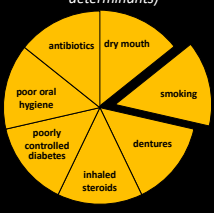
Taylor M, Brzuzecki M, Raja A. Oral Candidiasis. 2022 Sep 12. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2022. Jan. PMID: 31420826.

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### Oral and oropharyngeal candidiasis in HIV

Address these contributing factors if possible (consider social determinants)

- Declined since advent of HAART
- Still associated with CD4<200 and increasing viral load.
- Candida albicans #1 species
- Others also
- Drug-resistance



Clark-Ordóñez I, Callejas-Nagrete OA, Aréchiga-Carvajal ET, Mourillo-Pérez RN. Candida species diversity and antifungal susceptibility patterns in oral samples of HIV/AIDS patients in Baja California, Mexico. Medical Mycology. 2017;55(3):285-294.

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**Preferred Rx for oral candidiasis in patients with HIV**

- Fluconazole 100 mg PO 2x/day x 7-10 days
  - Best compliance
  - Ensure liver function OK

**Alternative Tx**

- Clotrimazole one 10-mg troche PO 5x/day x 7-10 days
- Troches generally have longer oral contact time than suspensions

**Not favored**

- Nystatin suspension 4-6 ml 4x/day x 7-10 days
  - High sucrose levels
  - Short contact time limits effectiveness

Panel on Opportunistic Infections in Adults and Adolescents with HIV. Guidelines for the prevention and treatment of opportunistic infections in adults and adolescents with HIV: recommendations from the Centers for Disease Control and Prevention, the National Institutes of Health, and the HIV Medicine Association of the Infectious Diseases Society of America. *Candidiasis (Mucocutaneous)*. May 26, 2020. [AIDSinfo].

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**Environmental diseases typically**

**D**

- Have identifiable Chemical, Physical, Thermal, or Electromagnetic cause

**A**

- Consider a direct cause-and-effect timeline although not always demonstrable

**M**

- Some risks may be reduced with appropriate environmental or behavioural modification

**I**

- Consider social determinants of health

**E**

**Environmental diseases rarely**

- Linked to predisposition to other disease processes i.e. malignancy, infection, etc.

**N**

**Today's example**

- Foreign Body Gingivitis

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**Foreign body gingivitis**

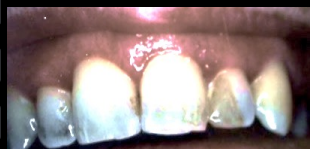
- First described in 8 patients by Daley & Wysocki in 1990
- They demonstrated that:
  - Foreign particles are not normally in connective tissue
  - Some gingivitis in patients with excellent oral hygiene could be caused by foreign material
- Suggested that particles might be from prophy paste

Daley TD, Wysocki GP. Foreign body gingivitis: an iatrogenic disease? *Oral Surg Oral Med Oral Pathol*. 1990 Jun;69(6):708-12.

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One of their cases:

<b>Patient</b>	• Woman in 40's
<b>Chief complaint</b>	• Inflamed anterior gingiva x 6 months
<b>History</b>	• Non-contributory
<b>Findings</b>	• Excellent oral hygiene



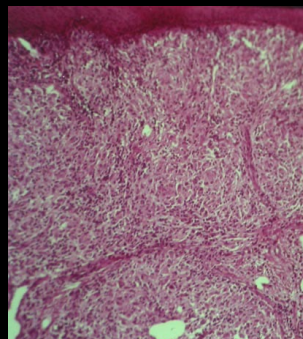
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**Biopsy**

- Granulomas with foreign particles deep in tissues
- Analysis: contain magnesium, silicon, tin

**Diagnosis**

- Foreign body gingivitis




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**1997: Two papers from one study**

- Analyzed 61 cases of FBG
- Correlated microscopic and clinical features with foreign elements
- Gave initial explanation of disease mechanism

Gordon SC, Daley TD. Foreign body gingivitis: clinical and microscopic features of 61 cases. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*. 1997 May;83(5):562-70.

Gordon SC, Daley TD. Foreign body gingivitis: identification of the foreign material by energy-dispersive x-ray microanalysis. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*. 1997 May;83(5):572-6.



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# Under the microscope

- Particles are small, easily overlooked
- Particles can be refractile or opaque
- Two main inflammatory patterns:
  - Lichenoid - dominated by T-cells
  - Granulomatous - dominated by macrophages
- Varying intensities of inflammation

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# Elements identified using Energy Dispersive X-Ray Microanalysis (EDX)

- Incident electron hits atom, ejects inner shell electron
- Higher-energy outer shell electron drops into vacancy
- Releases surplus energy as characteristic x-ray

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- Refractile (polarizable) material in lesion
- Elemental Analysis: Aluminum and silicon

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- Demographics**
  - F>M, peaks in 30s-40s, Persists 6 mo – 10 years
  - Not cumulative like most periodontal diseases
- Presentation**
  - Sore red, red/white, or colored gingiva
  - Does not respond to hygiene or to steroids
- Diagnosis**
  - Biopsy - minute particles of unexplained material
  - Elemental analysis confirms it is foreign
- Material origin**
  - Silicon-containing materials most prominent
  - C/W polishing, restorative metals, cements

Gordon SC, Daley TD. Foreign body gingivitis: clinical and microscopic features of 61 cases. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 1997 May;83(5):562-70.  
Gordon SC, Daley TD. Foreign body gingivitis: identification of the foreign material by energy-dispersive x-ray microanalysis. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 1997 May;83(5):571-6.

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- In some painful gingiva, inflammation is consistently associated with minute particles deep in the tissues
- These particles are not normal inside the body
- Elements in dental materials for polishing, restoring
- Conclusion:** These painful gingiva are caused by imbedded particles, usually of polishing or restorative material

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**Subsequent European validation study - 85 cases**

**Foreign body gingival lesions: distribution, morphology, identification by X-ray energy dispersive analysis and possible origin of foreign material**

Hanna Stromme Koppang<sup>1,3</sup>, André Roshan<sup>1</sup>, Ali Sraifzadeh<sup>1</sup>, Steinar Orbeck Stølen<sup>1</sup>, Rolf Koppang<sup>1</sup>

<sup>1</sup>Department of Pathology and Forensic Odontology, Institute of Clinical Dentistry, Faculty of Dentistry, University of Oslo, Oslo, Norway; <sup>2</sup>Laboratory for Pathology A.S. Oslo; <sup>3</sup>Department of Oral Biology, Faculty of Dentistry, University of Oslo, Oslo; <sup>4</sup>Section for Biomaterials Science, Institute of Clinical Dentistry, Faculty of Dentistry, University of Oslo, Oslo, Norway

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**Controversy: Ferreira et al (2019) studied 86 cases with foreign bodies in tissues ID'd by EDX**

- Si was most common element found, as in other studies
- Unlike other studies, *"We found a strong association between the presence of foreign material in gingival biopsy specimens and epithelial dysplasia."*
  - Verrucous hyperplasia in 59%
  - Epithelial dysplasia in 28%

Ferreira L, Peng HH, Cox DP, Chambers DW, Bhula A, Young JD, Ojcius DM, Ramos-Junior ES, Morandini AC. Investigation of foreign materials in gingival lesions: a clinicopathologic, energy dispersive microanalysis of the lesions and in vitro confirmation of pro-inflammatory effects of the foreign materials. Oral Surg Oral Med Oral Pathol Oral Radiol. 2019 Sep;128(3):250-267.

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**So is FBG a premalignant disease?**

- **No**
- Dysplasia and verrucous hyperplasia were **excluded** in all 8 cases of Wysocki & Daley, 61 cases of Gordon & Daley, and 85 cases of Koppang et al. (n=154)

**Does this study show Silicon can be associated with cancer?**

- **Yes**, this has been known for decades in the lungs.
- Also silicosis / fibrosis known.
- Association with oral cancer is unproved and interesting to investigate

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Second most abundant element on Earth	Constituent of marine organisms' shells
<b>Silicon</b>	
5-10% of dry weight of many grasses	Necessary to make bone and collagen

Schaechter: More Respect For the Silicon Cycle, Please. Small Things Considered. <https://schaechterasmblab.org/schaechter/2014/08/more-respect-for-the-silicon-cycle-please.html>

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**Relation of cancer and silicon is variable, complex**

Borm PJ, Tran L, Donaldson K. The carcinogenic action of crystalline silica: a review of the evidence supporting secondary inflammation-driven genotoxicity as a principal mechanism. Crit Rev Toxicol. 2011 Oct;41(9):756-70.

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**Prevention of Foreign Body Gingivitis**

- Avoid introducing particles of restorative or polishing material into the soft tissues
  - Especially if gingiva are ulcerated
    - If it occurs during a cleaning, or from LP/LM, etc., let them heal before polishing

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**Diagnosis of Foreign Body Gingivitis**

- When gingivitis doesn't respond to conventional therapy and there is no obvious non-bacterial etiology, perform a biopsy.
- The biopsy may yield a diagnosis of Foreign Body Gingivitis.
- Foreign bodies may also be associated with diseases causing atrophy of oral mucosa.
- One study associated them with some potentially premalignant conditions.

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### Treatment of Foreign Body Gingivitis

- Lesions may regress spontaneously with time
- *Conservative* excision of *localized* lesions may help
- Steroids and oral hygiene don't help

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### Learning Objectives

At the end of this educational session, the attendee will be able to:

1. Define non-microbial gingivitis.
2. Describe a diagnostic approach based on the suspected tissue of origin and the suspected disease process.
- 3. Differentiate typical features of benign vs malignant lesions.**
4. Formulate a logical differential diagnosis for a patient with gingival inflammation that is not responsive to conventional antimicrobial therapy.

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### Neoplastic diseases typically

- May be benign or malignant
- Benign lesions *might* have different characteristics but it may require a biopsy to be certain
- Some risks may be reduced with appropriate environmental or behavioural modification
- Consider social determinants of health

Neoplastic diseases rarely

- "Come and go and come back" – once established, usually either progress or don't

### Today's example

- Squamous Cell Carcinoma

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### What is a neoplasm?

Originating cell mutates and proliferates

Many Descendant Cells

More mutations

Benign tumors are formed of cells with **NO** mutations that allow spread

Malignant tumors are formed of cells with mutations that allow it to spread

Some tumors never develop mutations that allow invasion

Some tumors develop mutations allowing spread

A neoplasm arises from one originating cell that has mutated.

The nature of its mutations determine whether the neoplasm is benign or malignant

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**Benign** Localized growth only

Develops capsule

Freely-movable in soft tissue  
Well-defined on x-ray

**Malignant** Invasive growth

No capsule

Indurated in soft tissue  
Ill-defined on x-ray

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Hyperplasia → Mild Dysplasia → Moderate Dysplasia → Severe Dysplasia → Cancer

Most benign lesions are not dysplastic. Dysplasia is premalignant change.

If dysplastic mutations continue and progress, lesion may eventually become malignant

Then it could invade and metastasize.

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# Cases!

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