Mouth Inflammation

Oral inflammations associated with excessive use of irritants, such as tobacco, can cause an increase in salivary proteins, such as cystine and methionine.

Related terms:
Granulomatosis with Polyangiitis, Serositis, Lesion, Periodontal Disease, Saliva, Granulomatous Inflammation, Periodontitis, Third Molar

Inflammatory vascular diseases

Dirk M. Elston, in Dermatopathology (Second Edition), 2014

American College of Rheumatology criteria
Nasal or oral inflammation; chest X-ray with nodules; infiltrate or cavities; microscopic hematuria or red cell casts; granulomatous inflammation on biopsy (two criteria give >88% sensitivity, >92% specificity).

Differential Diagnosis
Palisaded granulomatous dermatitis with stellate abscess formation may be seen in Wegener’s granulomatosis (giant cells peripherally, neutrophils centrally), Churg–Strauss syndrome (epithelioid cells peripherally, eosinophils centrally), atypical mycobacterial infection, sporotrichosis, nocardiosis, cat scratch disease, lymphogranuloma venereum, and tularemia.

Fig 11-9. Wegener's granulomatosis, palisading granuloma with stellate abscess

Fig 11-10. Wegener's granulomatosis, granulomatous vasculitis

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URL: https://www.sciencedirect.com/science/article/pii/B978070205527000011X

Orbital Disease

TED H. WOJNO, in Primary Care Ophthalmology (Second Edition), 2005

Treatment

- In cases of idiopathic inflammation, oral corticosteroids are administered.
- In cases of a specific inflammation the underlying disorder is treated.
- In cases of infectious agents the following apply:
  - Oral, broad-spectrum antibiotics (e.g., amoxicillin/clavulanate [Augmentin] 500 to 875 mg two times a day) is used for bacterial infection. If the patient is allergic to penicillin, oral erythromycin (250 to 500 mg 4 times a day) is administered.
In cases of viral infection, the underlying disorder is treated.

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Lymphomatoid Granulomatosis

In Diagnostic Pathology: Lymph Nodes and Extranodal Lymphomas (Second Edition), 2018

Wegener Granulomatosis

- Systemic necrotizing vasculitis
  - Primarily involves upper and lower respiratory tracts and kidneys
- Clinical criteria
  - Nasal or oral inflammation
  - Pulmonary nodules, infiltrates, or cavities
  - Abnormal urinary sediment (usually microscopic hematuria)
  - Necrotizing granulomatous inflammation involving small arteries (by biopsy)
- Limited Wegener granulomatosis
  - Female predominance; disease confined to lungs
- Hallmark histologic features
  - Liquefactive &/or coagulative necrosis, geographic-shaped
  - Eosinophils (+); multinucleated giant cells without forming well-defined granulomas
  - Destructive, leukocytolytic angiitis involving arteries and veins

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Neuroendocrine tumors

Otto P. Rorstad MD, in Endocrine Biomarkers, 2017

9.3.5 Glucagonomas

The classical glucagonoma syndrome has components of glucose intolerance, a characteristic rash termed necrotic migratory erythema, oral and peri-oral inflammation (stomatitis, glossitis, angular cheilitis), thromboembolism, diarrhea, and anemia [1–3,48]. The blood glucagon level is increased. Potential false positive elevations can be caused by renal or hepatic failure, diabetes mellitus, fasting, and sepsis. Glucagonomas almost always arise in the pancreas and are rarely associated with the MEN1 syndrome. Metastases are present in 60%–80% of patients.

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URL: https://www.sciencedirect.com/science/article/pii/B9780128034125000094

Pediatrics in Systemic Autoimmune Diseases

S. Ozen, Y. Bilginer, in Handbook of Systemic Autoimmune Diseases, 2016

Clinical Manifestations

Patients frequently present with malaise, fever, sinusitis, epistaxis, and hematuria.

Diagnosis according to the ACR criteria requires two out of the four following
criteria: (1) nasal–oral inflammation, (2) abnormal chest X-ray, (3) microscopic hematuria or red blood cell casts, and (4) granulomatous inflammation on biopsy [60]. The triad of paranasal sinus involvement, pulmonary infiltration, and renal disease is characteristic of GPA (WG) [55]. The EULAR/PRES Endorsed Consensus Criteria for Classification of Wegener granulomatosis revised by pediatricians has introduced minor changes (Table 17.4); one of these was the inclusion of subglottic stenosis since this was a common pediatric feature.

Table 17.4. EULAR/PRINTO/PRES (Ankara 2008) Childhood Wegener Granulomatosis Criteria

At least three of the following six criteria:
- Histopathology
- Upper airway involvement
- Laryngo-tracheo-bronchial stenoses
- Pulmonary involvement
- ANCA positivity
- Renal involvement


Upper respiratory tract symptoms include rhinorrhea, nasal mucosal inflammation, epistaxis, persistent cough, hoarseness, and paranasal sinus pain.

Pulmonary involvement (cough, dyspnea, and hemoptysis) occurs in 74% of children [55]. It may be in the form of nodules, fixed infiltrates, or cavities [60] (Fig. 17.3). Multifocal infiltrates with or without small peripheral nodules were the commonest thoracic CT manifestations [61]. The skin disease may be in the form of palpable purpura, papules, vesicles, ulcers, and nodules [55]. Most features in children are similar to those of adults; however, some features such as subglottic stenosis and nasal deformity are more common in children [55]. Blurred vision, eye pain, conjunctivitis, episcleritis, persistent otitis media, myalgia, and arthralgia are also common [62]. CNS and cardiac involvement are less common.

In the pediatric series reported by Rottem et al. [55] (23 patients), Stegmayr et al.
Lesions of the Oral Cavity

Lindsay Montague, ... Jerry Elmer Bouquot, in Gnepp's Diagnostic Surgical Pathology of the Head and Neck (Third Edition), 2021

Pathologic Features and Differential Diagnosis

Early cases of oral submucous fibrosis present as chronic inflammatory cell infiltration of subepithelial connective tissues (Fig. 4.8B). This otherwise nonspecific infiltrate usually contains a number of eosinophils, cells seldom found in routine oral inflammation. Older lesions demonstrate reduced vascularity, reduced numbers of inflammatory cells, and dense bundles and sheets of collagen immediately beneath the epithelium. The eventual thick band of hyalinized subepithelial collagen shows varying extension into submucosal tissues, typically replacing the fatty or fibrovascular tissues normal to the site.

Minor salivary glands in the area of habitual quid placement often demonstrate a chronic inflammatory infiltrate and replacement of acinar structures by a hyalinized fibrosis. The hyalinized stroma can mimic amyloid deposits, but Congo red and/or thioflavin-T staining can rule this out.66

The epithelium is atrophic, with or without excess surface keratin, and demonstrates intracellular edema. One-fourth of the biopsied cases will demonstrate epithelial dysplasia at the time of biopsy. When squamous cell carcinoma is seen, it has the same features of carcinoma as those seen in persons without the betel quid chewing habit.

Dental Considerations

General:

- Avoid aspirin and NSAIDs to prevent GI irritation and excessive bleeding.
- Examine patient carefully for signs of opportunistic infections, mucositis, blood dyscrasias, stomatitis, and bleeding.
- Chlorhexidine mouth rinse prior to and during chemotherapy may reduce severity of oral inflammation.
- Patient may be taking prophylactic antiinfective drug.
- Place patient on frequent recall because of adverse oral effects of drug.

Consultations:

- Consult physician to determine disease status and ability of patient to tolerate dental procedures.
Consult physician to determine need for prophylactic or therapeutic antiinfective drug if oral surgery or periodontal therapy is planned.

Consult physician to determine patient's immunologic and coagulation status.

Teach Patient/Family to:

- Beware of oral adverse effects of drug.
- Use effective, atraumatic oral hygiene measures to prevent soft tissue inflammation.
- Report oral lesions, soreness, or bleeding to dentist.
- Update health and medication history regularly.

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S

In Mosby's Dental Drug Reference (Eleventh Edition), 2014

Dental Considerations

General:

- Patients on chronic drug therapy may rarely have symptoms of blood dyscrasias, which can include infection, bleeding, and poor healing.
- Potential for increased adverse events in patients at risk of thromboembolism.
- Assess salivary flow as a factor in caries, periodontal disease, and candidiasis.
- Avoid prescribing in last trimester of pregnancy.
- Should oral inflammation or lesions occur, refer to physician and consider palliative treatment for the lesions.
- Consider semisupine chair position for patient comfort because of GI side effects.

Consultations:

- Medical consultation may be required to assess disease control.
- In a patient with symptoms of blood dyscrasias, request a medical consultation for blood studies and postpone dental treatment until normal values are reestablished.

Teach Patient/Family to:

- Report oral lesions, soreness, or bleeding to dentist.
- Use caution to prevent injury in use of oral hygiene aids.
- Encourage effective oral hygiene to prevent soft tissue inflammation.
- When chronic dry mouth occurs, advise patient to:
  - Avoid mouth rinses with high alcohol content because of drying effects.
  - Use daily home fluoride products to prevent caries.
  - Use sugarless gum, frequent sips of water, or saliva substitutes.

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URL: https://www.sciencedirect.com/science/article/pii/B9780323169165000195

Immunology of Diseases of the Oral Cavity

Stephen J. Challacombe, ... Martin H. Thornhill, in Mucosal Immunology (Fourth Edition), 2015
Relationship with Systemic Diseases

In recent years there has been considerable interest in the relationship between periodontal diseases and certain systemic diseases including rheumatoid arthritis. The nature of this association is not completely clear, nor whether any mechanisms involved are immunologically based. Patients with rheumatoid arthritis suffer from a higher risk of periodontal attachment loss and increased oral inflammation and increased loss of periodontal attachment and alveolar bone can be detected in patients with early rheumatoid arthritis (Wolff et al., 2013). Interestingly, one study has shown that periodontal therapy in chronic periodontitis lowers gingival crevicular fluid IL-1β and the disease severity score (DAS28 score) in the associated rheumatoid arthritis (Bıyıkoğlu et al., 2013). Although the presence of DNA from oral bacteria has been found in synovial fluid, others caution about drawing too strong an inference about the association until studies all use the same definitions and severity scores of periodontal disease (Linden and Hertzberg, 2013).

In recent years, there has been much emphasis on the relationship between periodontal disease and a number of systemic diseases including rheumatoid arthritis, diabetes, low birth weight, and others, with the underlying hypothesis that CIPD reflects a genetically similar type of inflammatory response (Shangase et al., 2013). The pathogenesis of PD bears similarities to rheumatoid arthritis (RA), where chronic joint inflammation can lead to severe damage to the cartilage and underlying bone. Recently, Th17 cell and IL-17 pathways have been shown to have a potentially important role in the pathogenesis of RA, particularly in the regulation of bone destruction (Lubberts, 2010; Miossec and Kolls, 2012). In addition, mounting evidence indicates that increased proportions of Th17 cells and increased IL-17 levels play a critical role in the pathogenesis of inflammatory and autoimmune diseases including RA and inflammatory bowel disease (Song and Qian, 2013). However, the role of IL-17/Th17 cells in the pathogenesis of PD is still unclear (see above).

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The Journal of the American Dental Association