Oral Disease, Part 1

by Helena Pasieka, MD

	CLINICAL	PATHOLOGY	TREATMENT	ASSOCIATIONS
DEVELOPMENTAL CO				
Fordyce Granules	1-2mm yellowish papules on buccal mucosa and vermillion. Asymptomatic.	"Free" sebaceous glands.	None indicated.	
Geographic Tongue	Well-defined red patches on lateral & dorsal tongue with a serpiginous white border.	Psoriasiform mucositis.	If symptoms, potent topical steroids.	
Fissured Tongue	Multiple grooves on dorsum of tongue. ("Scrotal tongue") Asymptomatic.	Numerous grooves on dorsum of tongue.	None indicated.	Melkerson-Rosentha & Down syndrome. Occasionally in Cowden, pachyo- nychia congenita & acromegaly (in set- ting of macroglossia
Hairy Tongue	Hair-like elongation of lengthening of papillae on dorsum of tongue, especially in central area.	Pronounced accumulation of para- keratosis at tips of otherwise normal filiform papillae.	Identification & cessation of cause and if symptoms, scraping or brushing tongue.	Smoking, poor hygiene, oxidizing mouthwashes, and hot beverages.
Median Rhomboid Glossitis	Well-demarcated, diamond-shaped eroded area in midline of posterior dor- sal tongue.	Loss of filiform papillae. Consistently associated with candidiasis.	Anticandidal treatment, such as clotrimazole troches or PO fluco- nazole.	
PERIODONTAL AND	GINGIVAL DISEASE			
Necrotizing Ulcerative Gingivitis	Necrosis and/or ulceration of the inter- dental papillae "punched-out papillae". Painful and hemorrhagic.	Non-specific, as disease etiology is bacteria from normal oral flora.	Debridement of necrotic areas, oral hygiene instruction, and con- trol of pain.	
Desquamative Gingivitis	Diffuse painful gingival erythema. Epithelium readily mechanically sloughs leaving behind a smooth appearance.	Depends on the vesiculoerosive dis- ease it represents. Biopsy for H&E + DIF should be performed.	Treatment directed at underlying dx. Meticulous dental prophylaxis & hygiene can decrease severity of lesions.	Erosive lichen pla- nus, cicatricial pem- phigoid, pemphigus vulgaris, lichenoid mucositis, linear IgA bullous dermatitis, SLE, EBA, or chronic ulcerative stomatitis
Intraoral Dental Sinus Tract	Soft, non-tender, erythematous papule on alveolar process +/- diffuse, tender swelling of facial soft tissues.	Necrosis surrounding a non-vital tooth.	Elimination of the focus of infec- tion (extraction).	
PHYSICAL & CHEMI	CAL INJURIES			
Fibroma	Firm, smooth, nodule in the mouth; usually the same color as surround- ing tissue. Most commonly on buccal mucosa.	Unencapsulated mass of hyperplastic fibrous connective tissue with minimal inflammation. Overlying epithelium may show atrophy or hyperkeratosis from friction/biting.	Excision is curative.	
Chemical Burn	Initially, erythema. In time, a wrinkled white membrane representing superficial necrosis appears.	Coagulative necrosis, extending from surface either partially or fully into the epithelium.	Should self resolve after preven- tion of exposure to the offending agent.	
Morsicatio Buccarum	Bilateral shaggy white or shredded lesions of the anterior buccal mucosa that approximate the occlusal plane.	Marked hyperparakeratosis of the sur- face epithelium with ragged morphol- ogy +/- spongiosis in the superficial portion of the epithelium. Colonization by bacteria can also be seen.	Benign condition, requires no treatment.	
Traumatic Ulcer	Mild erythema surrounding a central ulcer covered by a yellow fibrinopurulent membrane. Can have a white border of hyperkeratosis adjacent to the ulcer. Most commonly on the tongue, lips and buccal mucosa.	Normal histology with mixed inflam- mation.	As traumatic ulcers clinically mimic oral SCCs, biopsy is indi- cated if the ulcer does not resolve within 2 weeks.	
Drug-Related Gingival Hyperplasia	Gingival enlargement of the interdental papillae of the anterior teeth. Edentulous areas usually spared, but can involve areas under poorly maintained den- tures.	Redundant tissue of normal com- position or an increased amount of collagen with a normal density of fibroblasts. Often with a plasmocytic infiltrate.	Discontinuation of the offending drug or substitution with another drug of the same class may result in cessation/regression. Extirpation of the excess gingival tissue is the treatment of choice.	
ALLERGIC AND CONT	TACT DISEASE			
Contact Stomatitis	Variable; cinnamon and dental amalgam most common. Cinnamon causes shag- gy, white hyperkeratotic areas on the lat- eral tongue & buccal mucosa. Amalgam causes superficial erosions w/radiating white striae localized to the buccal mucosa adjacent to a restoration.	show hyperkeratosis, basilar crowding and atypia, atrophy of the spinous	Discontinuation of the offending product. Polishing or replacing the dental work with inoffensive material.	
Recurrent Aphthous Stomatitis	Painful, <5 mm diameter, round or oval creamy-colored "cookie-cutter" ulcers with an intense erythematous halo.	Non-specific. Early lesions show spon- giosis, usually show prominent fibrin- ous neutrophilic membrane.	Mostly supportive. Potent topical steroids may shorten duration of ulcer. Complex aphthosis may require therapy with oral colchi- cine, dapsone or thalidomide.	



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Oral Disease, Part 1 (continued)

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	CLINICAL	PATHOLOGY	TREATMENT	ASSOCIATIONS	Do you have an
ALLERGIC AND CON Behçet's Disease	TACT DISEASE (continued) Aphthae usually multiple, <6 mm diam- eter, lasting approximately 1-3 weeks. Ocular involvement is the clue.	Perivascular lymphocytic and mono- cytic cellular infiltration +/- fibrin deposition in the vessel wall and sur- rounding tissue necrosis.	Topical anesthetics, NSAIDs for symptoms of arthritis and inflam- matory skin lesions. Colchicine, dapsone or thalidomide for mucocutaneous lesions. Ocular involvement is organ-threatening and requires systemic steroids +/- immunosuppresives.		interesting Boards' Fodder? We're expanding our Boards' Fodder archives and you can be a part of it. Contact Dean Monti, managing editor, special publications, at dmonti@aad.org.
Eosinophilic Ulcer of the Oral Mucosa	Rapidly enlarging, firm nodule(s) that develop a central ulceration with elevated borders and indurated base. Becomes 1-2cm in size. +/- pain. Typically covered with a fibrinous exu- date. Usually tongue, but the buccal, labial, alveolar and palatal mucosa can also be affected.	Ulcer w/dense infiltrate of eosinophils and pleomorphic mononuclear cells extending deep into the submucosal tissue and underlying striated muscle. Large atypical cells are CD30+ T lym- phocytes. Base of ulcer is composed of poorly formed granulation tissue +/- increased capillaries with prominent endothelial cells.	Most resolve spontaneously within a few months, no treatment necessary.		
Orofacial Granulomatosis	Initially intermittent, then persistent non- tender swelling of the lip and/or face.	Non-necrotizing granulomatous inflam- mation.	Intralesional corticosteroids, sometimes repeatedly. Dapsone, clofazimine, hydroxychloroquine, thalidomide,TNF inhibitors, or systemic corticosteroids have also been used.	Often with undiag- nosed GI disease. Referral to GI for scopes to rule out enteric involvement.	Watch for Oral Disease, Parl in the next Boards' Fodder.
Wegener's Granulomatosis	Pathognomonic friable 'strawberry gums'. Necrotizing granulomatous vas- culitis of the upper and lower respiratory tract. Destructive ulcerated lesions of the oral and nasal cavity.	Skin biopsy can demonstrate leuko- cytoclastic vasculitis and/or granulo- matous inflammation. Such classic features are infrequently observed in oral biopsy specimens.	Systemic corticosteroids and cyclophosphamide.		Need more Boards Fodder? Visit
EPITHELIAL PATHOL					the Directions in
Oral Leukoplakia	Sharply demarcated, homogeneous or speckled white plaque, often on floor of mouth, lateral & ventral surfaces of the tongue, and the soft palate.	Simple hyperkeratosis seen most fre- quently. +/- epithelial dysplasia (mild to severe). Carcinoma in situ or even invasive SCC possible.	Cessation of smoking. Biopsy mandatory, as considered pre- malignant. If no or mild dysplasia evident, site of the lesion dictates treatment. Low-risk sites (buccal mucosa, labial mucosa, hard palate) warrant periodic clinical follow-up. Moderate or severely dysplastic lesions require com- plete removal.		Residency archive listed under "Publications" at www.aad.org.
Oral Erythroplakia	Flat or slightly elevated, velvety, sharply circumscribed plaque. The lesions are often asymptomatic.	Advanced epithelial dysplasia w/90% of lesions demonstrating carcinoma in situ or invasive carcinoma at the time of biopsy.	Prompt surgical removal and careful follow-up. Cessation of smoking.		
Nicotine Stomatitis	Posterior hard palate & anterior soft palate w/grayish-white mucosa. Sometimes, umbilicated papules w/red central puncta are found, representing inflamed palatal mucous salivary gland orifices.	Hyperkeratosis and parakeratosis, acanthosis, and mild chronic sialo- dochitis.	Cessation of smoking typically results in complete resolution within 1-2 weeks.		
Actinic Keratosis/ Cheilitis	Loss of normal dermatoglyphics, atro- phy, and blurring of vermilion border. The development of hyperkeratotic scal- ing suggests evolution to precancerous actinic cheilitis.	Thickening of epithelium and hyper- keratosis. Different degrees of epi- thelial dysplasia possible in the same lesion so multiple sections of biopsy should be reviewed to find areas of severe dysplasia, carcinoma in situ, or invasive SCC.	Cryosurgery, topical agents such as 5-FU or imiquimod, and Blu-U. CO2 laser ablation has also been used, but only after the pres- ence of invasive SCC has been excluded.		
Oral Squamous Cell Carcinoma	May present as ulcer, exophytic mass, or endophytic process w/varying degrees of induration. The surface is typically irregular, rough or granular. Most commonly on lateral and ventral surfaces of the tongue and floor of the mouth.	Histologically similar to those occur- ring elsewhere.	Surgery, radiation therapy, or both; chemotherapy + radiation therapy may decrease the risk of mets. 90% of recurrences occur w/in 2 years of initial treatment.		
Verrucous Carcinoma	White exophytic papillomatous or warty proliferations w/well-defined borders, most commonly on palate, buccal mucosa, & alveolar processes. Slow growing. Uncommonly ulcerated.	Epithelium with hyperkeratosis & acan- thosis w/a papillary or verrucous sur- face. Well differentiated epithelium and few mitotic figures. A dense infiltrate of chronic inflammatory cells can be present. Evaluate multiple sections, as 25% show foci of typical SCC.	therapy with imiquimod and/or		

Bolognia JL, Jorizzo JL, Rapini RP eds. Dermatology. 2nd ed. Mosby; 2007.
Rapini R. Practical Dermatopathology. Mosby; 2005.

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