Clinical Outline of Oral Pathology:

DIAGNOSIS AND TREATMENT

LEWIS R. EVERSOLE

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To

Chris and Laura

Preface

This book is written with the full knowledge that many comprehensive texts dealing with the information recorded herein are available to the dental clinician and student of oral diseases. Indeed, some of these textbooks on oral diseases explore them in such detail that the student is inundated with description and may find himself in a quandary regarding salient features, resulting in a lack of assimilation and retention of the knowledge originally sought. In addition to this dilemma, in the conventional textbook approach disorders are considered on a pathogenetic or etiologic basis (e.g., developmental, inflammatory, neoplastic diseases, etc.). Unfortunately, patients do not present themselves for diagnosis and treatment in this fashion but, rather, manifest certain signs and symptoms. The pathogenetic approach to the presentation of oral pathologic entities is under no criticism here; indeed, initial confrontation with the basic concepts of oral disease is perhaps dealt with most logically in this fashion. Once these basic concepts have been studied, however, the student must filter through this knowledge to construct a differential diagnosis dependent upon the presenting signs and symptoms. Herein lies the value of this clinical outline.

The diseases of the oral cavity have been categorized according to presenting signs and symptoms, with a primary objective being the development of a differential diagnosis. Each chapter outlines diseases according to these features, and the various clinical parameters used to differentiate among lesions manifesting common signs and symptoms are enumerated. Whereas microscopic characteristics are not featured in detail, for the emphasis is on clinical diagnosis, the vital role of biopsy for the confirmation of a clinical impression must be remembered. As stated, the diseases discussed in this outline are categorized according to their most common presenting features; since, however, exceptions can and do arise, a diagnosis must be considered tentative

until confirmed by biopsy or, in some instances, by use of certain laboratory tests.

Another goal of this book is to rank disorders according to frequency and life-threatening importance. Each chapter considers the most common pertinent entities and emphasizes those diseases with serious prognostic implications. With each entity, references are given for standard texts and publications which pursue more detailed accounts of the subject; in this respect, this outline may be used as an adjunctive text.

Finally, once a definitive diagnosis has been evolved, the matter of treatment becomes paramount. For each entity, the most widely accepted form of treatment, be it surgical, medical, psychological or radiotherapeutic, is given. Oral abnormalities may be manifestations of diseases of a systemic nature, and, under these circumstances, patient management becomes the responsibility of a physician. Recommendations of this nature are dealt with under the treatment sections.

It is my hope that this text will integrate the precepts of basic oral pathology, oral radiology and oral medicine into a usable chairside outline, enabling the clinician to construct a differential diagnosis, arrive at a definitive diagnosis and ultimately provide appropriate management and treatment for the patient.

San Francisco, California

Lewis R. Eversole

Acknowledgments

This book was designed as a practical, useful chairside aid to clinical diagnosis of oral lesions. Its utility depends upon the use of representative illustrative material. In this regard, I am deeply indebted to many people who provided me with clinical photographs from previous publications and from their own personal collections. In particular, I wish to acknowledge the cooperation of Dr. Sheldon Rovin, Dean, University of Washington, School of Dentistry; Dr. Henry Cherrick, Assistant Dean, University of California at Los Angeles, School of Dentistry; Dr. Alan Leider, Associate Professor of Pathology, University of the Pacific, School of Dentistry; Dr. Robert Gorlin, Chairman of Oral Pathology, University of Minnesota; Dr. Carl Witkop, Chairman of Human and Oral Genetics, University of Minnesota, School of Dentistry; Dr. Stefan Levin, Department of Otolaryngology, the Johns Hopkins University, School of Medicine; and Dr. Charles Tomich, Department of Oral Pathology, Indiana University, School of Dentistry.

My appreciation is also warmly extended to Dr. Willard Fee, Department of Otolaryngology, Stanford University, School of Medicine, and to Dr. Roger Bowles, Chairman of Otolaryngology, University of California at San Francisco, School of Medicine, for contributing illustrative materials for the chapter on extraoral soft tissue swellings.

One of the most tedious tasks in the preparation of a textbook is, of course, the typing of the manuscript. I am deeply indebted to my secretaries, Miss Myrna Pantangco and Miss Doris Low, for their patience and diligence in this regard. Mr. Arnold Eilers, Director of Audiovisual Aids, University of the Pacific, School of Dentistry, deserves my gratitude for his preparation of literally hundreds of photographs used to illustrate the various disorders compiled in this text.

Lastly, I wish to extend appreciation to my family, children and friends for their encouragement and patience with me during the months of work required to prepare this book.

White Lesions

GENOKERATOSES

- Leukoedema
- White Sponge Nevus
- Hereditary Benign Intraepithelial Dyskeratosis
- Keratosis Follicularis
- Pachyonychia Congenita
- Incontinentia Pigmenti

LEUKOKERATOSIS

- Frictional Hyperkeratosis, Parakeratosis and Acanthosis
- Idiopathic and Tobacco-associated Leukokeratosis
- Verruciform Hyperkeratosis
- Snuff Keratosis
- Stomatitis Nicotina
- Actinic Cheilitis
- Dysplasia, Carcinoma in Situ, Squamous Cell Carcinoma, and Verrucous Carcinoma

DERMATOSES

- Lichen Planus
- Lupus Erythematosus

INFLAMMATIONS

- Koplik Spots of Measles
- Mucous Patches of Syphilis
- Chemical Burns
- Candidiasis

MISCELLANEOUS WHITE SPOTS

- Fordyce's Granules
- Dental Lamina Cysts, Bohn's Nodules and Epstein's Pearls

Contents

White Lesions

1

White lesions of oral mucous membranes appear thus because (1) one or more of the epithelial layers is thickened or (2) an extrinsic or intrinsic pseudomembrane is adherent to the surface mucosa. The white lesions encountered in practice may be subdivided into groups on the basis of pathogenesis or etiologic factors. Developmental white lesions are generally bilateral in distribution. Patients will relate that a familial pattern exists, since most of the developmental white lesions are genetically inherited (genokeratosis).

As a gardener's hands become calloused from hours of friction from the hoe, so may the oral epithelium become calloused or thickened in response to chronic trauma. Tobacco in its many forms is also considered a local irritant, and indeed is associated with carcinomatous transformation in white lesions of the oral mucosa. The white lesions associated with physical or tobacco-product irritation are grouped under the heading *leukokeratoses,* as are other white lesions of undetermined origin. Since tobacco-associated and idiopathic white lesions may show microscopic evidence of malignancy, biopsy must be performed when (1) a cause cannot be detected or (2) the suspected traumatic agent is eliminated yet the lesion fails to involute or regress.

White lesions of nonhereditary diseases may coexist with skin lesions. These keratotic dermatoses may, however, occur as oral lesions without skin involvement. As no etiologic agent can be discerned in oral keratotic dermatoses, biopsy must be undertaken to confirm the diagnostic impression when classical clinical features of these disorders are lacking.

Inflammatory lesions which may produce thickened epithelium and/or a surface pseudomembrane will appear white, and in these instances the surface coating may be rubbed away with gauze. The diagnosis of inflammatory white lesions can be secured by obtaining a thorough history, including questions regarding use of drugs, sexual habits or promiscuity, recent contact with persons manifesting similar illness or contact with injurious chemicals.

The last group of white lesions is characterized by multifocal spots on the mucosa which are generally diagnostic on a clinical basis for the entities in this group.

This chapter illustrates and enumerates the features of white lesions in the aforementioned context. The differential diagnosis for a given white lesion will depend upon obtaining a history, with primary considerations of: (1) a familial pattern, (2) presence or absence of a causative agent with notations on oral habits, particularly tobacco use, (3) presence or absence of associated skin lesions, (4) questions relating to contact with individuals showing similar lesions, (5) presence or absence of fever and (6) exploration of the nature of the white patch as to tenacity to underlying structures and ease of removal by rubbing. Based upon responses and findings regarding the above considerations, a ranking of entities in the differential diagnosis may ensue and ultimately biopsy, or in certain instances serology and blood count, must be performed to obtain a definitive diagnosis.

The most frequently encountered white lesions are: (1) frictionally induced leukokeratosis (e.g., denture irritation), (2) lichen planus, (3) candidiasis, (4) leukoedema.

White lesions associated with a potentially lethal outcome include: (1) premalignant leukokeratoses and (2) lupus erythematosus.

GENOKERATOSES

Leukoedema

Age: No predilection
Sex: No predilection

Clinical Features: Leukoedema occurs in blacks and dark-skinned Caucasians and is so frequently encountered that it should be considered a normal variation in these individuals. The lesion is bilateral and diffuse, involving the buccal mucosa, and shows a filmy, mother-of-pearl appearance, often with delicate overlapping curtain-like mucosal folds. When the mucosa is stretched the white appearance is diminished.

Microscopic Features: The epithelium displays acanthosis, parakeratosis and spongiosis.

Differential Diagnosis: The diffuse delicate mother-of-pearl sheen and the propensity to involve members of the Negro race are characteristics which permit differentiation from other hereditary white lesions of the genokeratosis group. The primary entities in the differential diagnosis include white sponge nevus and hereditary benign intraepithelial dyskeratosis, both of which are thicker, plaque-like, and show specific microscopic features.

Treatment: No treatment is necessary.

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White Sponge Nevus

Age: Childhood onset
Sex: No predilection

Clinical Features: White sponge nevus, inherited as an autosomal dominant trait, is characterized by diffuse and sometimes patchy white

FIG. 1–1. Pale mucosa with a slight tendency to form curtain-like folds in leukoedema.

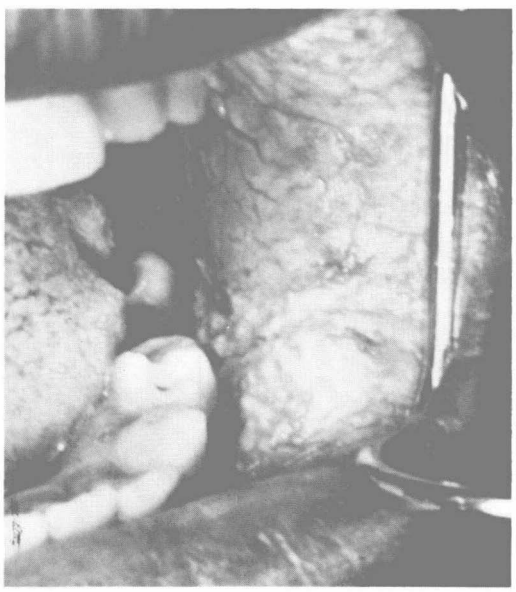

FIG. 1–2. Thickened, diffuse white lesion of buccal mucosa in white sponge nevus.

lesions of the buccal mucosa, bilaterally, with involvement of the tongue and occasionally other oral mucosal sites. The conjunctivae are spared; however, vaginal, esophageal and anal mucous membranes may harbor white lesions similar to those of the oral tissues.

Microscopic Features: The epithelium shows parakeratosis and acanthosis with spongiosis. Parallel striae of condensed parakeratin traverse the surface layers in oblique planes. Individual cell keratinization may be seen in the spinous cell layer.

Differential Diagnosis: Other white lesions do not tend to be as thick and diffuse as white sponge nevus; however, hereditary benign intraepithelial dyskeratosis, lichen planus, and candidiasis should be considered in the differential diagnosis. Biopsy will disclose characteristic features.

Treatment: No treatment is necessary.

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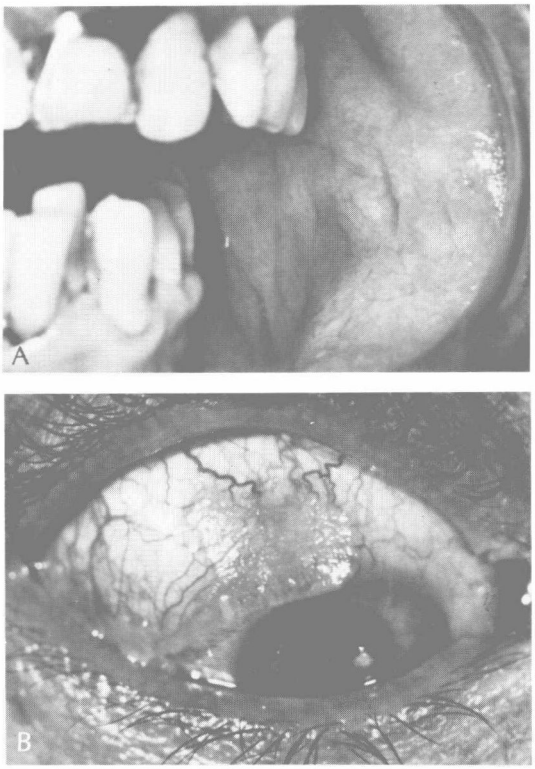

FIG. 1–3. Hereditary benign intraepithelial dyskeratosis. *A*: Buccal mucosal white lesions. *B*: Conjunctival telangiectasia and gelatinous plaque. (Courtesy of Dr. Carl J. Witkop.)

Hereditary Benign Intraepithelial Dyskeratosis

Age: Childhood onset
Sex: No predilection

Clinical Features: Hereditary benign intraepithelial dyskeratosis (HBID) is inherited as an autosomal dominant trait and, as with other genokeratoses, manifests diffuse white plaques of the buccal mucosa and tongue. In addition to the oral signs are coexistent conjunctival telangiectasias and occasionally thickened plaques of the bulbar conjunctiva which may eventuate in blindness. The disease was originally described in a racial isolate group in the Carolina states.

Microscopic Features: Acanthosis with both hyperorthokeratosis and hyperparakeratosis is present within the epithelium. The pathognomonic features seen are benign dyskeratotic changes consisting of the cell-within-a-cell phenomenon and individual cell keratinization, similar to those features encountered in Darier-White's disease; however, no intraepithelial cleavage occurs.

Differential Diagnosis: The oral white lesions must be differentiated from those in white sponge nevus, pachyonychia congenita and lichen planus. The combination of eye and oral lesions in this disease should not be confused with the muco-oculocutaneous syndromes in which the oral lesions are either ulcerative, bullous, or erythematous.

Treatment: No treatment is necessary. Because of the potential for blindness, genetic counselling may be in order.

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Keratosis Follicularis (Darier-White's Disease)

Age: Generally adult onset
Sex: No predilection

Clinical Features: Keratosis follicularis is a dermatologic disorder manifesting orange-yellow papular and white keratotic lesions of skin. It is inherited as an autosomal dominant trait. Oral lesions are not universally seen in all patients, yet when present they are papular and usually keratotic in appearance. Buccal mucosa, lips, palate, and tongue are the favored oral locations. Other mucous membranes including vulva, vagina and anus may be involved.

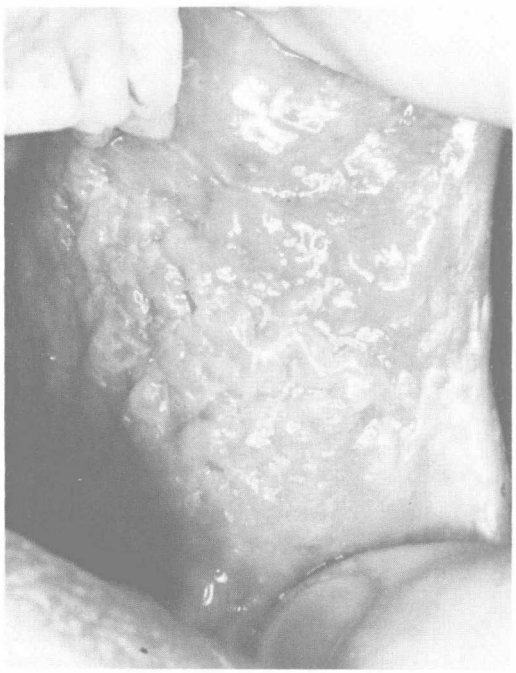

FIG. 1–4. Multiple keratotic papules in keratosis follicularis. (From Weathers, D. R. and Driscoll, R. M.: Darier's disease of the oral mucosa, Oral. Surg. *37*:711, 1974.)

Microscopic Features: Surface hyperkeratosis is present. Within the spinous cell layer are dyskeratotic changes represented by the cell-within-a-cell phenomenon known as *corps ronds* and pyknotic elongated nuclei termed *grains*. A suprabasilar cleavage is present with acantholysis; formation of wandering rete ridges manifesting cleft-like spaces produces a villous pattern. The microscopic appearance may be confused with pemphigus vulgaris and familial pemphigus (Hailey-Hailey disease).

Differential Diagnosis: The multipapular or cobblestone appearance and characteristic skin lesions differentiate Darier-White's disease from the other genokeratoses which generally manifest oral white lesions in a more diffuse or plaque-like pattern. This white papular eruption may be confused with other oral lesions showing multiple polyps or papules, including pyostomatitis vegetans, verrucous carcinoma, oral florid papillomatosis and denture papillomatosis. Biopsy will afford a definitive diagnosis.

Treatment: Vitamin A has been employed in high doses, the rationale being an attempt to eliminate the degree of skin keratinization. This treatment is of limited value.

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Pachyonychia Congenita

Age: Childhood onset
Sex: No predilection

Clinical Features: Pachyonychia congenita is an autosomal dominant inherited disorder man-

ifesting oral white lesions and laminated thickening of the finger- and toenails. The oral white lesions are located bilaterally on the buccal mucosa, tongue, and other sites and are usually limited in terms of the extent of mucosal tissues affected.

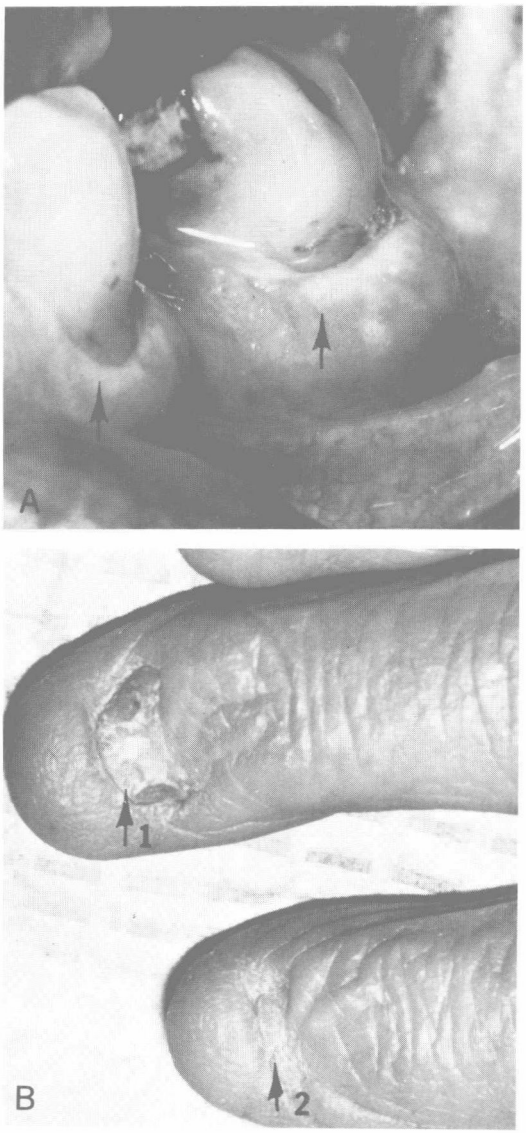

FIG. 1–5. *A:* Gingival white lesions in pachyonychia congenita. *B:* Dystrophic nail changes (arrow #1) and surgically removed fingernail (arrow #2). (From Young, L. L. and Lenox, J. A.: Pachyonychia congenita; a long-term evaluation of associated oral and dermal lesions. Oral Surg. *36*:663, 1973.)

Microscopic Features: A nonspecific thickening of the parakeratin and spinous cell layers is seen.

Differential Diagnosis: The bilateral mucosal white lesions of pachyonychia congenita may be confused with white sponge nevus, HBID, and lichen planus; however, the fingernail changes, when considered in conjunction with the oral lesions, provide the diagnostic clinical features of the disease. Candidiasis of the oral mucosa with fingernail involvement should be considered in the differential diagnosis and can be ruled out by obtaining an oral cytologic smear which can be stained for hyphae.

Treatment: No treatment is necessary.

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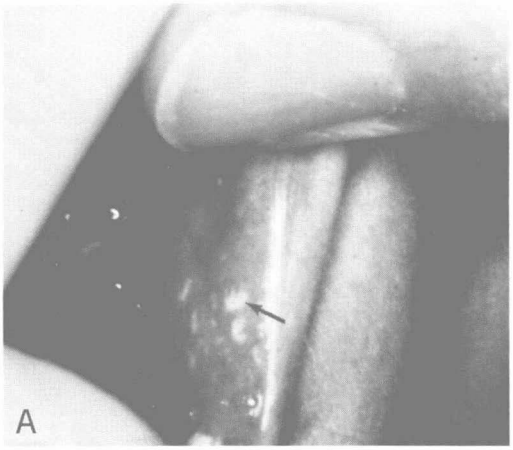
Incontinentia Pigmenti

Age: Begins during infancy
Sex: Females

Clinical Features: Incontinentia pigmenti is inherited as a dominant trait, is lethal in males, and manifests both skin and dental defects. The skin lesions vary from reticulated slate-grey pigmentations to vesicles and verrucous keratoses. Oral mucosal white lesions may occur on the buccal mucosa, yielding a patchy plaque or verrucous appearance. Partial anodontia, strabismus with nystagmus, and epilepsy may also be seen.

Microscopic Features: Skin lesions in the vesicular stage show intraepithelial vesicle formation with numerous eosinophils. Pigmented zones display incontinence of melanin granules from the basal cells with accumulation in dermal or submucosal macrophages. White lesions are characterized by hyperorthokeratosis, hyperparakeratosis and acanthosis. Individual cell keratinization may be present.



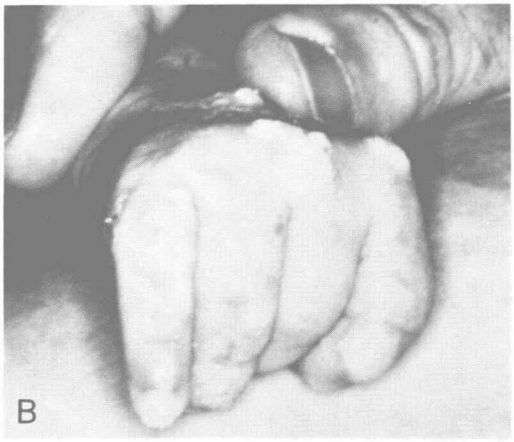


FIG. 1–6. *A*: Oral lesions on buccal mucosa in an infant with incontinentia pigmenti. *B*: Keratoses and focal pigmentations of skin. (Courtesy of Dr. Sheldon Rovin.)

Differential Diagnosis: The white lesions encountered intraorally may resemble those seen in other genokeratoses yet are generally focal and patchy in distribution. The other components of this disorder, including skin discoloration and keratosis as well as partial anodontia, separate this entity from other genetic diseases manifesting oral white lesions.

Treatment: There is no treatment.

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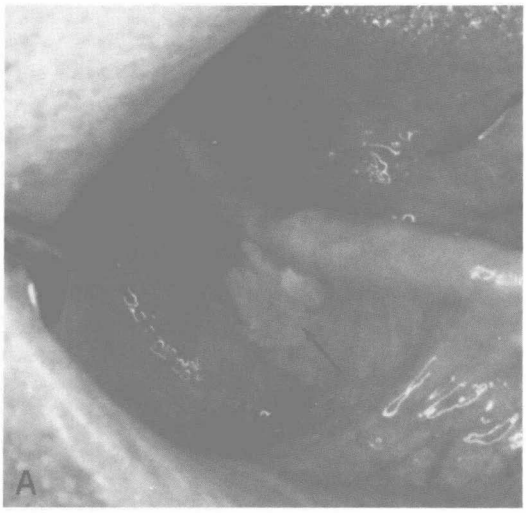
LEUKOKERATOSES

Frictional Hyperkeratosis, Parakeratosis and Acanthosis

Age: No predilection
Sex: No predilection

Clinical Features: Frictional keratoses represent callus formation from chronic trauma with resultant thickening of one of the epithelial cell



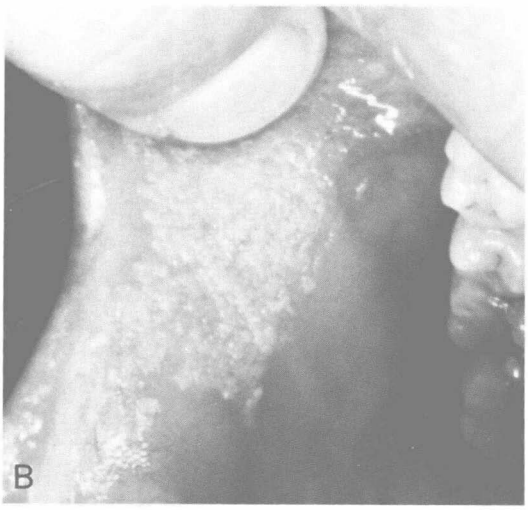


FIG. 1–7. *A*: Hyperkeratosis of mandibular alveolar ridge due to ill-fitting denture. *B*: Cheek-biting keratosis.