Color Atlas of Oral Diseases

Diagnosis and Treatment

George Laskaris

Fourth Edition

Thieme


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Diagnosis and Treatment

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*This book is dedicated to my sweetest son **Christos G. Laskaris**, whose memory continues to inspire and give me the strength to create, love, and offer.*

*To my wife **Vivi**, constant and unique partner to any step along the family, social, and scientific way.*

*To my daughters, **Christina** and **Marina,** as a small indication of reward for their love.*

Nikon Medical Camera

This camera has been an irreplaceable technological instrument, which, for 43 years, has helped me impress in color photographs, accurately, a great variety of oral and skin diseases in thousands of patients.

Contents

Foreword

It is a pleasure and privilege for me to write the foreword to the new English edition of Professor Laskaris' *Color Atlas of Oral Diseases*. This work reflects the vast experience and expertise that Professor Laskaris has gained in clinical practice and research during his professional life spanning over 40 years.

Since the publication of the first edition of the atlas, there have continued to be immense social and medical changes across the globe that have led to a substantial increase in the number of individuals with diseases that require intervention by specialists in oral medicine. For example, HIV disease led to many affected persons developing a wide range of oral disorders, including oral hairy leukoplakia and Kaposi's sarcoma that oral specialists had never encountered previously, and while now becoming less frequent (in view of the widening availability of antiretroviral therapy), they are also now recognized as complications of iatrogenic immunosuppression. Life expectancy has continued to improve, and with this there are many more patients than in the past developing the oral consequences of immunologically mediated disease, oral and other malignancy, and paraneoplastic disease. Similarly, as early life health care improves, so the number of children and adults with complex genetically based orofacial disease increases, while at the same time the widening availability of novel agents of systemic disease (e.g., disease-modifying biologicals, other immunotherapies, and antiretrovirals among others) has led to an increase in the frequency and spectrum of disorders of the oral mucosa and salivary glands.

All of these, and many other, changes in oral disease and the need for appropriate treatment are reflected in the contents of Professor Laskaris' book. This is not merely an atlas providing excellent clinical examples of common (as well as uncommon, rare, and very rare!) diseases, but a book that provides synopses that are of significant benefit to both the nonspecialist and specialist to safely and effectively manage patients with diseases that can greatly lessen their quality of life. Professor Laskaris is to be commended on his discipline of securing such high-quality images of diseases and industry in writing such an informative and practically useful text.

This is an important and essential book for the practice of oral medicine across the globe.

Professor Stephen Porter, MD, PhD, FDSRCS (Eng)
Institute Director and Professor of Oral Medicine
UCL Eastman Dental Institute
London, UK

Preface

Oral medicine is a demanding clinical specialty. Beyond possessing adequate theoretical knowledge, the oral physician (stomatologist) must be a keen and diligent observer and an astute clinician with extensive outpatient and inpatient experience. The visual approach is the most powerful tool at the disposal of the oral physician. Acquisition of the skill to "see" lesions in a particular way requires extensive and intensive clinical training and is rewarded with a high degree of diagnostic accuracy. To this effect, I felt that the format of juxtaposition of text and pictures is ideal and was chosen for the presentation of the subject matter of this text.

Twenty-nine years have elapsed since the first edition and 3 years since the third edition of this book were published in Greek, and the impact was beyond the wildest expectation.

The three English editions that followed were embraced by the international community and had an impressive track record. The numerous favorable comments by international leaders in the field of oral medicine and reviews in the most prestigious specialty journals, where the reviewers ranked this book among the standard texts forming the canon of the specialty, are cherished rewards to the author.

The tally so far is three editions in Greek, four editions in English, translations into seven more languages, and Greek and international prizes. This fourth English edition contains the distillation of my experience and service at the Medical School, University of Athens, Department of Dermatology, "A. Sygros" Hospital and other major hospitals of the city as well as in my private practice.

This new edition has been entirely rewritten and adapted to contemporary publishing and scientific demands. Numerous changes and additions have been made. Ten new chapters, enriched with many new clinical entities, have been added. The pictorial material has been renewed and enriched at a level of over 90% with high-standard color pictures from the 130,000 pictures and slides comprising my personal collection.

The structure of the text has changed. In the beginning of the book, rules for the diagnostic and therapeutic approach to the patient with oral disease have been codified. For every disease entity, the basic characteristics (key points) and the differential diagnosis are presented in boxes. The clinical description is precise, followed by clear, representative pictures. The histopathologic findings and the rest of the laboratory tests are then presented, followed by the therapeutic strategies in detail. At the end of the book, there is an appendix with tables of differential diagnosis of oral lesions classified according to morphology and color, and with tables presenting the performance of biopsy and interpretation of the histopathologic findings. These changes will provide the reader with a concise, comprehensive, and reliable book. Furthermore, students of medicine and dentistry can acquire basic and up-to-date knowledge, which, in combination with the rich pictorial material, provides an excellent introduction to clinical oral medicine. Finally, I hope that the book will be a useful diagnostic and therapeutic tool in the medical practice of oral medicine specialists, dentists, otorhinolaryngologists, dermatologists, pediatricians, and internists.

George Laskaris, MD, DDS, PhD

Acknowledgments

Creating a scientific book requires discipline and is a lonely journey during which you come closer to people who will contribute in their own different ways. Therefore, I would like to thank the following individuals.

First of all, I wish to acknowledge the important contribution of **Dr. Annie Argyriades Porter**, specialist in special care dentistry, for her initial excellent translation of the Greek text to English. There is no doubt that without her help it would have been difficult for me to complete this book.

My special thanks also go to **Dr. Stephen Porter**, Professor of Oral Medicine, University of London, for the final editing and the foreword of the book.

To **Dr. Christos Kittas,** Professor of Pathology, Medical School, University of Athens, I owe special recognition for over 40 years of scientific collaboration and the useful suggestions in the pathology part of this book.

Many thanks to **Dr. Eleana Stoufi, Dr. Marina Doukas,** and **Dr. Eleni Georgakopoulos,** my collaborators in oral medicine, for their constant help during this project.

Finally, my thanks go to the staff of Thieme Publishers for the excellent professional commitment they have shown.

The following figures were sourced from the collection of the colleagues listed below and who I thank greatly.

Dr. G. Goumenos for figs. 11.4, 11.26; Prof. A. Katsabas for fig. 30.17; Prof. I. Iatrou for fig. 42.7; Prof. A. Katsarou for figs. 25.18, 25.19, 25.20; Dr. A. Panagiotopoulos for fig. 28.9; Prof. N. Papadogeorgakis for fig. 39.57; Dr. Ahsan A. (India) for figs. 2.5, 30.9, 43.4; Prof. Almeida O. (Brazil) for figs. 19.19, 19.20; Profs. Blozis G. and Neiders M. (USA) for figs. 25.91, 25.92; Dr. Ribeiro (Brazil) for fig. 20.4; Prof. Carozzo M. (England) for fig. 25.45; Dr. Delgando-Azanero W. (Peru) for figs. 18.25, 20.6; Dr. Glick M. (USA) for fig. 17.33; Drs. Romero de Leon and Aguirre A. (USA) for fig. 20.5; Prof. Scully C. (England) for figs. 25.93, 27.27, 31.8; Prof. Shklar G. (USA) for figs. 5.10, 5.11, 31.9.

Introduction to Oral Medicine

Diagnostic and Therapeutic Approach

This brief introduction offers codified information that a practicing oral physician (stomatologist) must implement in order to successfully overcome diagnostic and therapeutic dilemmas. Oral medicine is an important subspecialty of general internal medicine. A specialist in oral diseases is required to possess a certain level of clinical and laboratory expertise in medicine and dentistry along with clinical experience. These will allow the practitioner to negotiate with diagnostic and therapeutic challenges successfully.

The training framework of the specialist in oral medicine includes internal medicine, dermatology, otorhinolaryngology, pediatrics, clinical pharmacology, therapeutics, histopathology, and others.

The mouth and the structures within the oral cavity offer significant clinical advantages: (1) It is a cavity readily accessible for inspection and palpation, (2) it is easy to be biopsied, (3) it is repeatedly examined because dental and gingival problems are common, thereby necessitating repeated visits to the dentist, and (4) it is readily accessible for self-examination. On the other hand, the oral cavity also offers many diagnostic difficulties due to the following factors: (1) A plethora of local and systemic diseases with morphologically similar lesions manifest in the mouth, and (2) local factors, such as saliva, dentures, foodstuffs, and the mechanical action of the teeth themselves may alter the appearance of the elementary lesion. The clinical diagnostic methodology follows fundamental principles that we must adhere to in order to arrive at a correct diagnosis. These principles are summarized in **Table I.1**. Laboratory tests follow the clinical evaluation. The laboratory is an aid and not a substitute for sound clinical methodology. Clinicians should be familiar with laboratory errors and the disorientation that they may cause in the diagnostic process. Thus, it is a basic principle of practice that laboratory results are always evaluated by the clinician in relation to the clinical presentation of the disease.

A correct decision for therapy is based on a correct diagnosis. The rules and parameters that must be followed in implementing appropriate therapy are summarized in **Table I.1**. In cases of therapeutic failure, provided all the rules and guidelines have been followed, the clinician must revise the diagnostic hypothesis and investigate other alternatives.

The reader of this book is encouraged to study the cited bibliography at the end of this Introduction for additional information on oral medicine, pathology, dermatology, internal medicine, immunology, and therapeutics that were used by the author in this book as basic, classic reference material.

Table I.1 Diagnostic and therapeutic approach

I. Diagnosis

• Complete history and physical examination
• Evaluation of oral lesions
• Examination and evaluation of skin and mucosal lesions elsewhere in the body
• Evaluation of symptoms and signs in other systems
• Intake of medications prior to the beginning of the disease
• Grouping of disease entities according to clinical criteria
• Reevaluation of symptoms and signs in cases where the diagnosis is doubtful
• Consultation with the family physician if necessary
• Performance of biopsy on the basis of clinical indications
• Histopathologic examination by an experienced pathologist
• Evaluation of pathology report by the clinician, in relation to the symptoms and signs of the patient
• Repeat biopsy if in doubt
• Further laboratory evaluation if clinically indicated
• Is the diagnosis correct
• Reevaluation of the patient after initiation of therapy

II. Differential diagnosis

• Initiation of symptoms and signs
• Duration (acute–subacute–chronic course)
• General symptoms (fever, pain, malaise, anorexia, weight loss, arthralgias)
• Lesion morphology (macules, papules, blisters, bullae, pustules, plaques, nodules)
• Color of the lesions (white, red, black, brown, yellow)
• Location (tongue, buccal mucosa, palate, floor of the mouth, gingiva, lips)
• Coexisting skin lesions, mucosal lesions
• Biopsy and histopathologic examination
• Laboratory work-up (microbiology, immunology, hematology, imaging studies, molecular studies)

III. Therapy

• Choice of the right medication
• Administration of the medication in the right form (pills, injections, ointments)
• Exact dosage and dosage schedule
• Consideration of clinical pharmacology issues, such as interaction between the administered drug and other medications that the patient is taking
• Extreme care concerning contraindications for the drug that we intend to administer
• Side effects due to our therapy
• Adherence to the therapeutic program
• Evaluation of the therapeutic result 2–4 days after initiation of therapy
• In case of therapeutic failure, reevaluation of the diagnostic hypothesis
• It is a general principle that correct therapy presupposes correct diagnosis

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1 Normal Mucosal Variants

Leukoedema

Key points

- Common normal variation of the oral mucosa.
- It is more frequent in black-skinned people than in whites.
- Leukoedema usually occurs on the buccal mucosa, frequently bilaterally and rarely on the lateral border of the tongue and the mucosal surface of the lips.
- The diagnosis is based on the clinical features.

Introduction

Leukoedema is a common normal oral mucosal variant due to increased thickness of the epithelium and intracellular edema of the spinous layer cells. A similar clinical pattern may be seen at the mucosa of the larynx and the vagina. It occurs more frequently in black-skinned people, with a prevalence ranging between 70 and 90%. In contrast, the prevalence rate in whites is much less.

Clinical features

As a rule, leukoedema occurs bilaterally and involves most often the buccal mucosa and rarely the lateral border of the tongue (**Figs. 1.1**, **1.2**). Clinically, the oral mucosa presents as opalescent or grayish-white with slight wrinkling, which characteristically disappears if the mucosa is distended, by pulling or stretching the cheek. Leukoedema has a normal consistency on palpation.

The disorder is usually discovered as an incidental finding by the patient or during a routine oral examination by the dentist. The diagnosis is based exclusively on the clinical features.

Differential diagnosis

- Chronic biting
- Pseudomembranous candidiasis
- Lichen planus
- Leukoplakia
- Cinnamon contact stomatitis
- White sponge nevus
- Hereditary benign intraepithelial dyskeratosis

Pathology

It is not necessary. On histologic examination, leukoedema demonstrates thickness of the epithelium, increased parakeratosis, and acanthosis with broad and elongated rete ridges. Characteristically, the cells of the spinous layer are large with small pyknotic nuclei due to marked intracellular edema.

Treatment

No treatment is necessary.

Racial Pigmentation

Key points

- Physiologic findings due to increased melanin production.
- The pigmentation is more common in black-skinned people and in dark-skinned whites.
- The gingiva and the buccal mucosa are the most frequently affected areas.
- The disorder is asymptomatic and benign.

Introduction

Melanin is a normal skin and oral mucosal pigment produced by melanocytes. Increased melanin deposition in the oral mucosa may occur in various diseases. Dark discoloration may often be a normal finding in black- and dark-skinned people.

Clinical features

In healthy people there may be clinically asymptomatic black or brown areas of varying size and distribution in the oral mucosa, usually on the gingiva, buccal mucosa, palate and less often on the tongue, floor of the mouth, and the lips (**Fig. 1.3**). The pigmentation is more prominent in areas of pressure or friction and becomes more intense with aging. A biopsy may be justified if the clinical features are atypical, causing a diagnostic dilemma.

Differential diagnosis

- Smoking-associated melanosis
- Pigmentation due to drugs
- Freckles
- Lentigo simplex
- Lentigo maligna
- Pigmented nevi
- Melanoma
- Addison's disease
- Peutz-Jeghers syndrome
- Albright's syndrome
- Amalgam tattoo

Pathology

Histologically, increased melanin production is observed while the number of melanocytes is normal. The overlying epithelium is normal.

Treatment

No treatment is required.

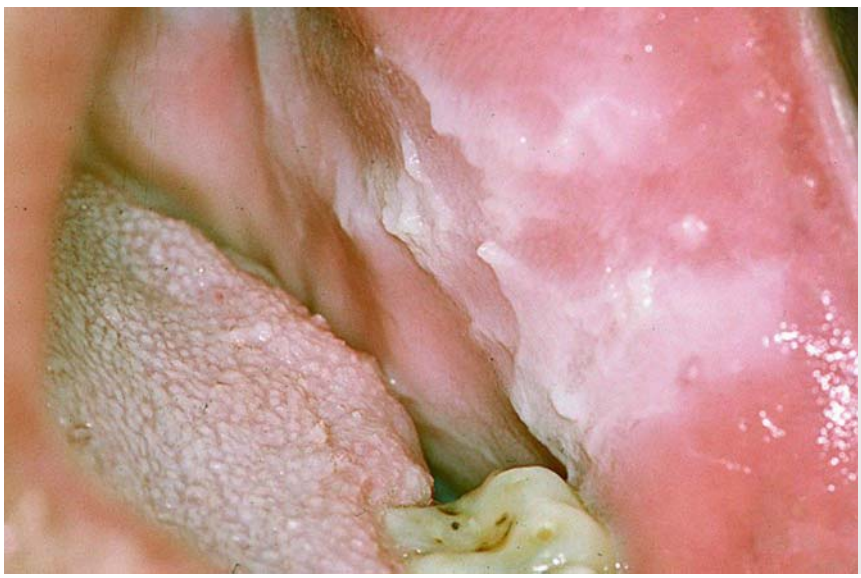

Fig. 1.1 Leukoedema of the buccal mucosa.

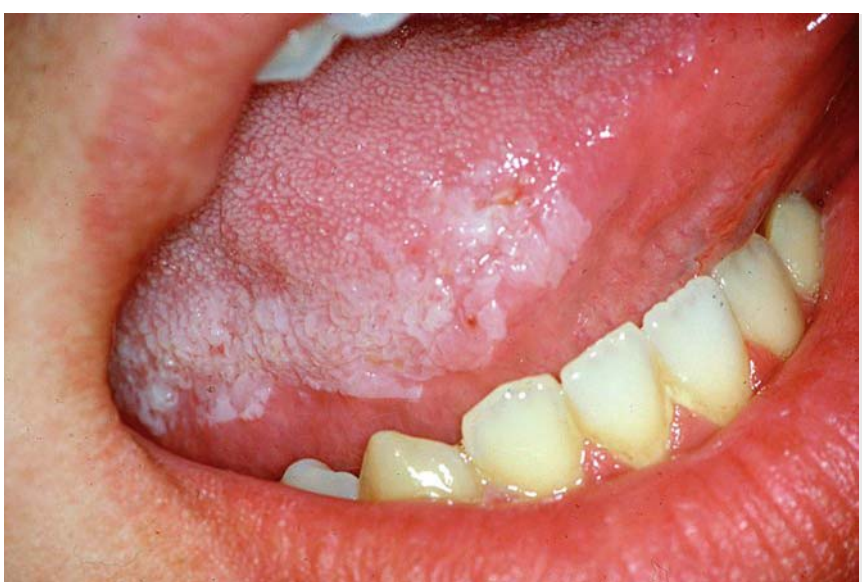

Fig. 1.2 Leukoedema of the tongue.

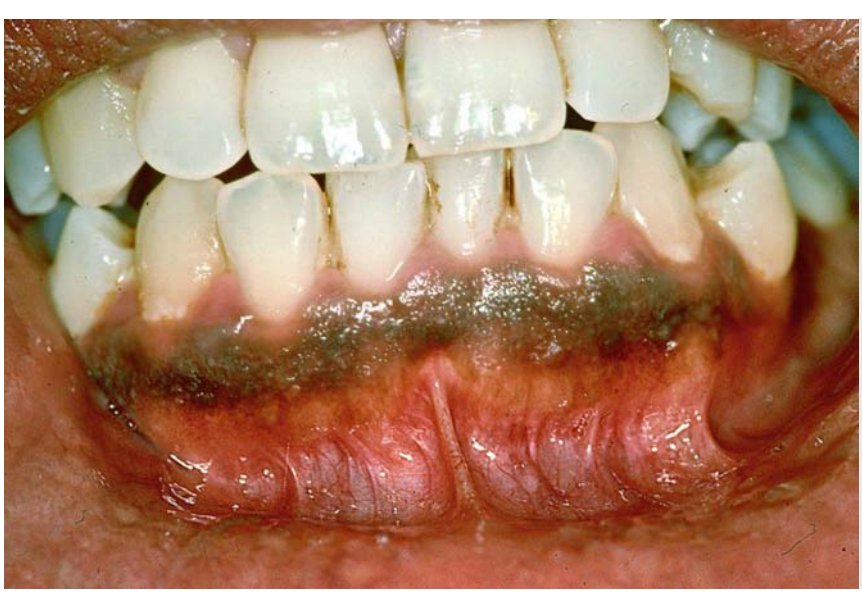

Fig. 1.3 Normal pigmentation of the gingiva.

Linea Alba

Key points

- Linea alba is a common alteration of the buccal mucosa, and is usually bilateral.
- It is located on the buccal mucosa at the level of the occlusal line, usually behind the premolars.
- Mechanical pressure or irritation from the buccal surface of the teeth is the etiologic factor.

Introduction

Linea alba is a relatively common linear elevation of the buccal mucosa, extending from the corner of the mouth to the third molar, at the occlusal line. It is more prominent along the premolar and molar teeth. Usually, it is associated with mechanical irritation or sucking from the buccal surfaces of the teeth. The change is more prominent around occlusal defects.

Clinical features

Clinically, linea alba presents as unilateral or usually bilateral linear elevation of normal or slightly whitish color and normal consistency on palpation (**Fig. 1.4**). Occasionally, it may be scalloped and characteristically it appears on the buccal mucosa along the occlusal level. Linea alba varies in prominence and can be more frequent in obese people. The diagnosis is based exclusively on the clinical features.

Differential diagnosis

- Chronic biting
- Leukoplakia
- Candidiasis
- Cinnamon contact stomatitis
- Leukoedema

Pathology

It is not needed. On histologic examination, the epithelium is normal with slight hyperorthokeratosis and intracellular edema of the spinous cells.

Treatment

No treatment is required.

Fordyce's Granules

Key points

- Fordyce's granules represent a normal anatomical variation characterized by collection of sebaceous glands in the oral mucosa.
- They occur in approximately 70 to 80% of the population.
- The upper lip, commissures, buccal mucosa, and retromolar area are most frequently affected.
- The diagnosis is based on the clinical features.

Introduction

Fordyce's granules are ectopic sebaceous glands in the oral mucosa which are functionally inactive. A similar pattern may be seen on the genital mucosa. They are a frequent oral finding that occurs in approximately 70 to 80% of population, in both sexes. With aging, the granules may become more prominent as a result of moving toward the mucosal surface.

Clinical features

Clinically, Fordyce's granules appear as multiple small, slightly raised whitish-yellow, well-circumscribed spots that rarely coalesce to form plaques (**Figs. 1.5**, **1.6**). They most often occur in the mucosal surface of the upper lip, commissures, buccal mucosa adjacent to the molars, and the retromolar region, in a symmetrical bilateral pattern. The majority of the patients have multiple lesions while some may have only a few. Characteristically, the granules become more evident when the mucosa is stretched. They are asymptomatic and the diagnosis is usually made during a routine oral examination or incidentally by the patients.

Differential diagnosis

- Lichen planus
- Candidiasis
- Leukoplakia

Pathology

It is not necessary. On histologic examination, multiple, well-formed, ectopic sebaceous glands, without hair follicles, can be seen in the superficial part of the connective tissue.

Treatment

No treatment is required as Fordyce's granules represent a normal anatomical variation of the oral mucosa.

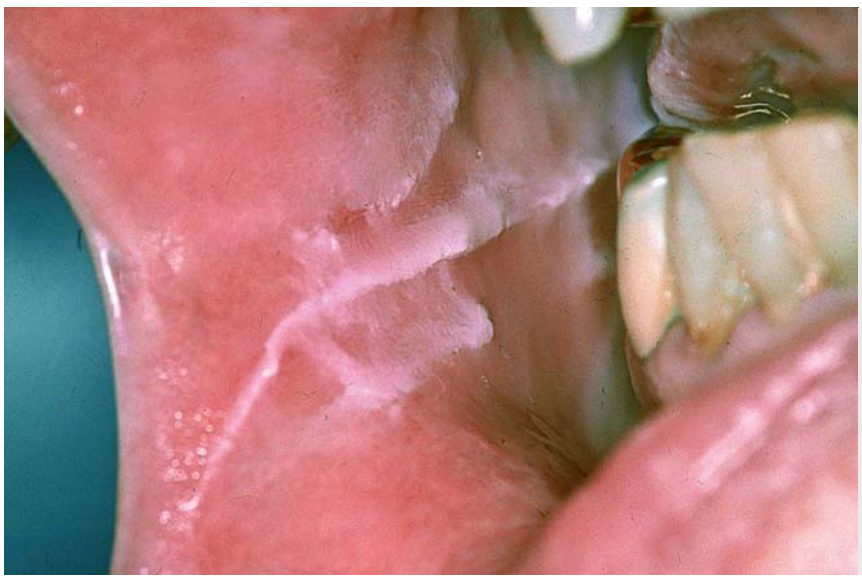

Fig. 1.4 Linea alba.

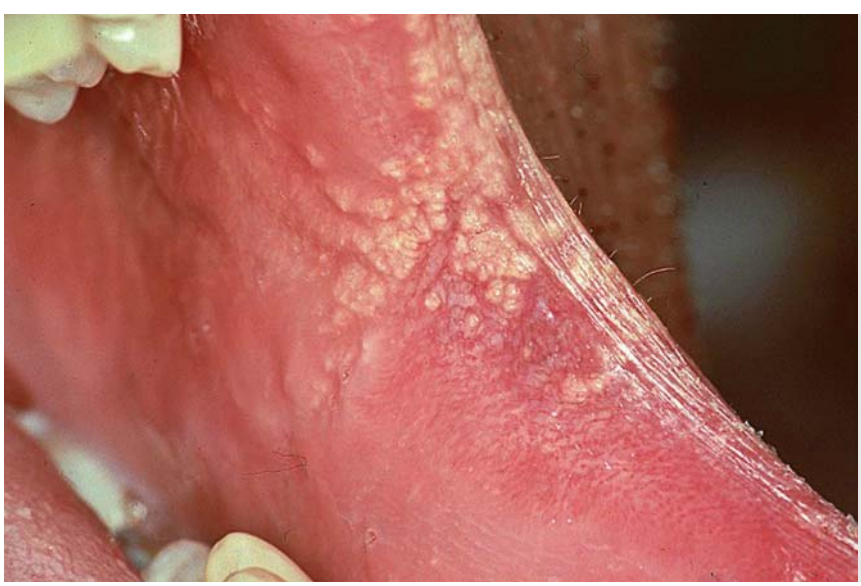

Fig. 1.5 Fordyce's granules in the buccal mucosa.

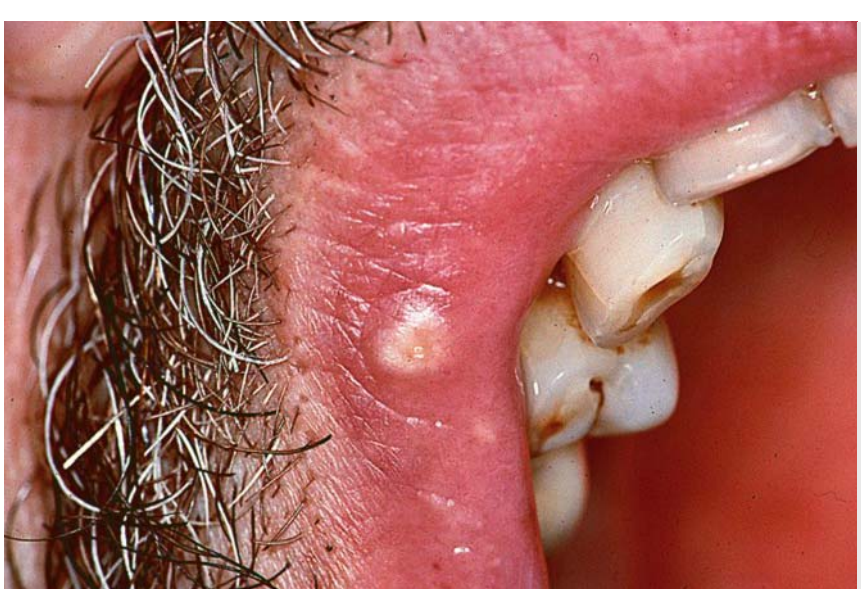

Fig. 1.6 Sizeable Fordyce's granule of the upper lip.

2 Developmental Defects

Orofacial Clefts

The fetal development of the face and the oral cavity is a marvelous multilateral and complicated procedure which results in the formation of the most exceptional part of the human body, the face. Every derangement of the formation and the completion of the tissues can cause disorders in the facial region, the facial clefts.

Types of facial clefts

- Cleft lip
- Cleft palate
- Cleft maxilla/mandible
- Bifid uvula
- Medium upper lip cleft
- Oblique facial cleft

Causes of cleft formation

- Genetic
- Environmental
- Smoking during pregnancy
- Alcohol abuse during pregnancy
- Medication during pregnancy
- Malnutrition and vitamin deficiency

Cleft Lip

Key points

- The most common cleft.
- It can be solitary or part of a syndrome.
- Often coexists with palatal cleft.
- It causes esthetic defect.

Introduction

Cleft lip is a derangement of the development which mainly involves the upper lip and very rarely the lower lip. It can present as a solitary disorder or as part of a genetic syndrome.

Clinical features

A unilateral or more rarely bilateral deficit on the lip with higher frequency on the left side is observed (**Fig. 2.1**). It occurs more often in males than females. It is usually combined with palatal or maxillary cleft. It has been estimated that the incidence of cleft lip is 1 per 9,000 births and in combination with palatal malformation it is 1 per 1,000 births. Usually, the defect is unilateral and it causes esthetic and functional deficit.

Treatment

Plastic reconstructive surgery.

Cleft Palate

Key points

- A common cleft.
- In several cases it coexists with maxillary and lip cleft.
- Many times it is part of genetic syndromes.
- It causes functional defects.

Introduction

The cleft palate is a developmental derangement caused by the failure to fuse the two embryonic palatal processes. The cleft palate often coexists with maxillary and lip cleft. The incidence of cleft palate alone varies from 0.29 to 0.56 per 1,000 births.

Clinical features

The deficit is located on the hard palate alone or on both hard and soft palates (**Figs. 2.2, 2.3**). In major cases functional and psychological problems may occur. Bifid uvula represents a minor expression of the cleft palate and may be seen alone or in combination with more severe malformations (**Fig. 2.4**).

Treatment

Early surgical treatment is recommended. The role of the orthodontist and the prosthodontist is pivotal for the functional restoration of the deficit.

Oblique Facial Cleft

Key points

- A rare form of cleft.
- It extends from the upper lip to the ocular orbit.
- The disorder causes major esthetic and functional defects.
- Often combined with palatal and labial cleft.

Introduction

Oblique facial cleft is a sporadic, exceedingly rare form of cleft. The incidence is 1 case per 1,300 facial clefts. The disorder is nearly always associated with cleft lip and palate. Occasionally it is incompatible with life.

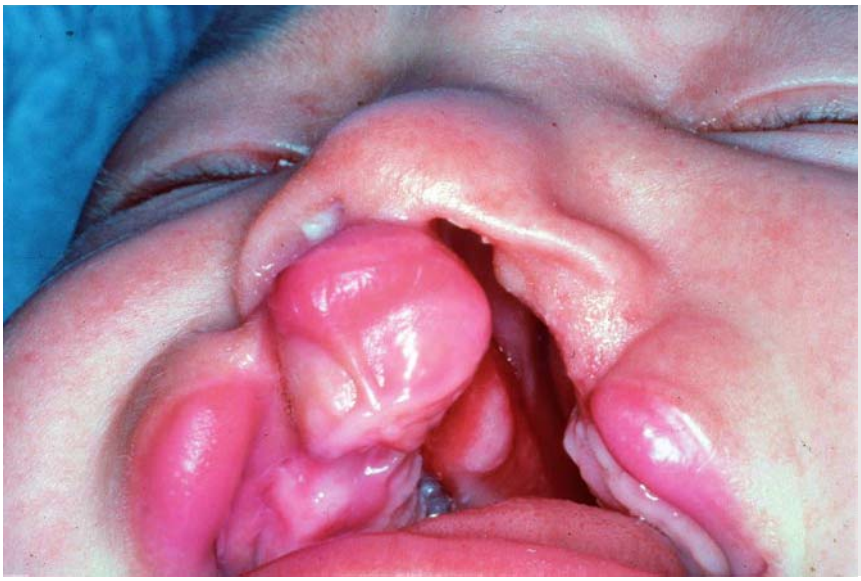

Fig. 2.1 Cleft lip and palate.

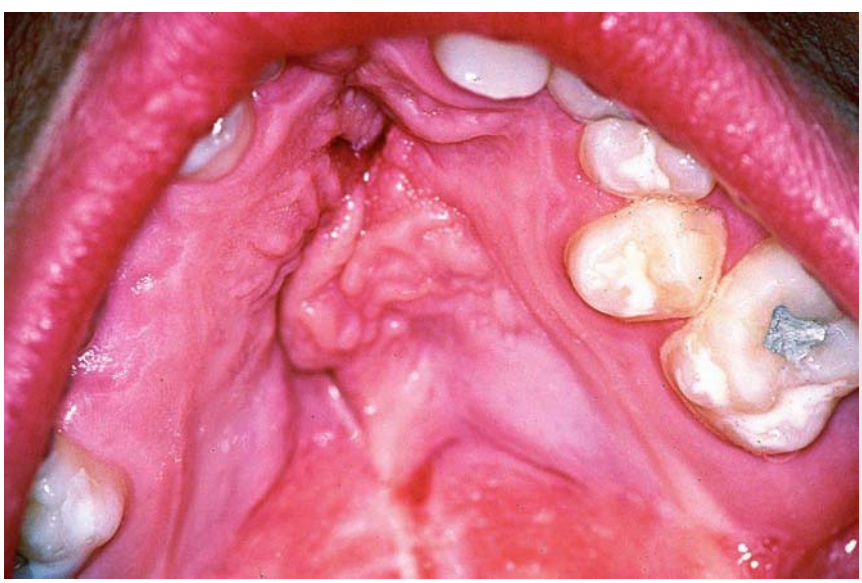

Fig. 2.2 Cleft palate.

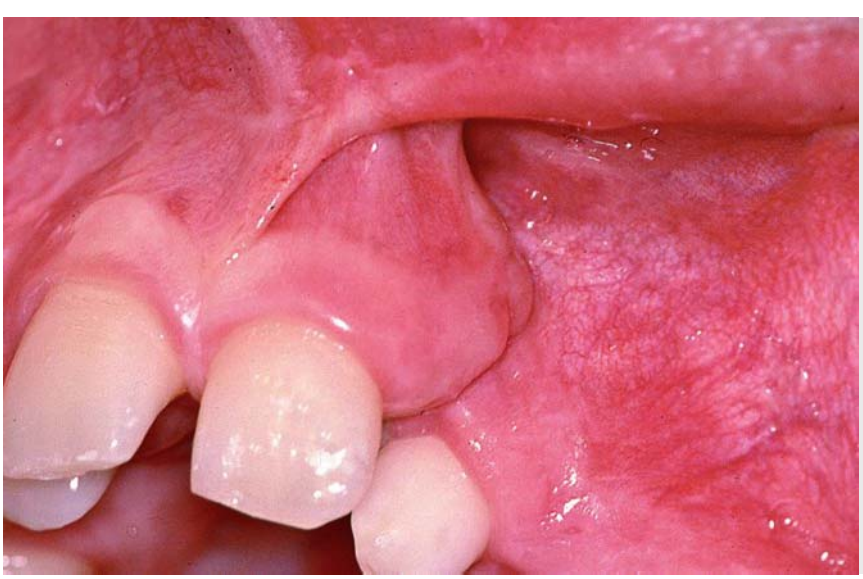

Fig. 2.3 Cleft maxilla.

Clinical features

Oblique facial cleft may extend through the upper lip to the nose to involve the eye (oro-ocular type) (**Fig. 2.5**). In 20 to 30% of the cases it is bilateral. It may coexist with other developmental malformations such as facial asymmetry, ocular and nasal abnormalities.

Treatment

Plastic reconstructive surgery.

Oral Hair

Key points

- Hair and hair follicles are extremely rare on the oral mucosa.
- Few cases have been reported, all in whites.
- Oral hair may offer an explanation of oral keratoacanthoma.

Introduction

Original hair and hair follicles are a very unusual phenomenon within the oral tissues. Only seven cases have been reported in the literature by 2015. All the reported cases have been registered in whites. There is no satisfactory explanation for the occurrence of oral hair although a developmental anomaly is the most possible cause.

Clinical features

Clinically, oral hair presents as a single, asymptomatic black hair of 0.3 to 3.5 cm in length, usually surrounded by a whitish mucosa (**Fig. 2.6**). Occasionally, the lesion may cause minor problems during kissing. The presence of oral hair may result in psychological problems and the patients can be quite anxious to solve the problem. The tongue, gingival and buccal mucosa are the most frequently affected sites. The presence of oral hair and follicles may offer an explanation for the rare occurrence of keratoacanthoma intraorally. The diagnosis of oral hair is based on the clinical features, but it can be confirmed by a biopsy.

Differential diagnosis

- Skin graft with hair
- Implantation of hair to the oral mucosa following an accident

Pathology

On histologic examination, hair follicles in the lamina propria are observed, associated with several sebaceous glands.

Treatment

Conservative surgical excision is the treatment of choice.

Congenital Lip Pits

Key points

- A rare developmental malformation.
- Occasionally, it may be associated with cleft lip and/or cleft palate.
- It usually occurs on the lower lip.
- The diagnosis is based on the clinical features.

Introduction

Congenital lip pits are relatively rare developmental invaginations that occur exclusively on the lower lip. They originate from persistent lateral sulci, on the embryonic mandibular arch. The malformation may occur alone or in combination with commissural pits, cleft lip, and/or cleft palate. Congenital lip pits are frequently inherited as an autosomal dominant trait.

Clinical features

Clinically, congenital lip pits present as bilateral, asymptomatic, and symmetric depressions in the middle of the vermilion border of the lower lip (**Fig. 2.7**). A small amount of mucous may accumulate at the deep aspect of the pit which is blind and may vary in size from 0.2 to 1 cm. The lip may be enlarged and swollen, but the defect is not associated with pain. The diagnosis is based on the clinical features and the history.

Differential diagnosis

- Mechanical trauma
- Surgical deformity
- van der Woude syndrome
- Other rare syndromes

Pathology

It is not usually required. On histologic examination, a sulcus lined by normal epithelium may be seen. A mild chronic inflammation by plasma cells and leukocytes is a common finding. In addition, minor salivary glands may communicate with the pits.

Treatment

Treatment of choice is reconstructive surgical repair of the irregularity for aesthetic purposes.

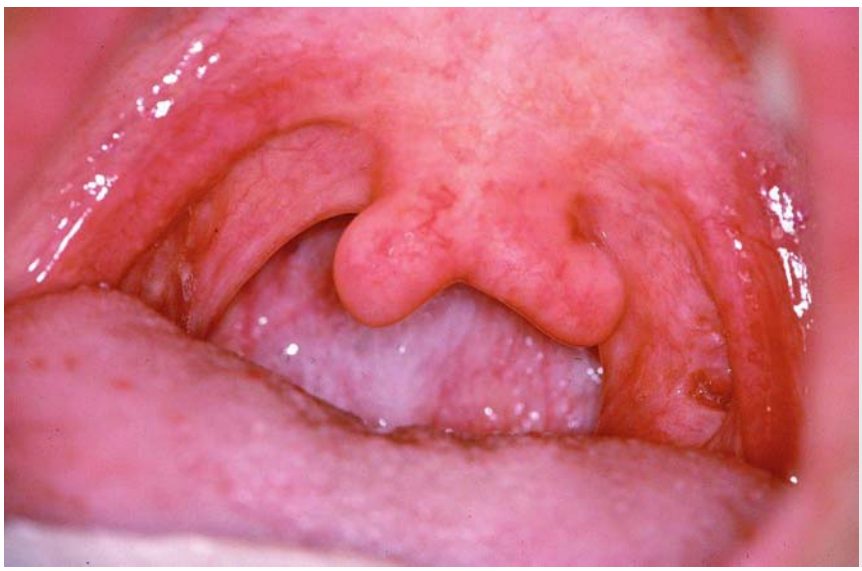

Fig. 2.4 Bifid uvula.

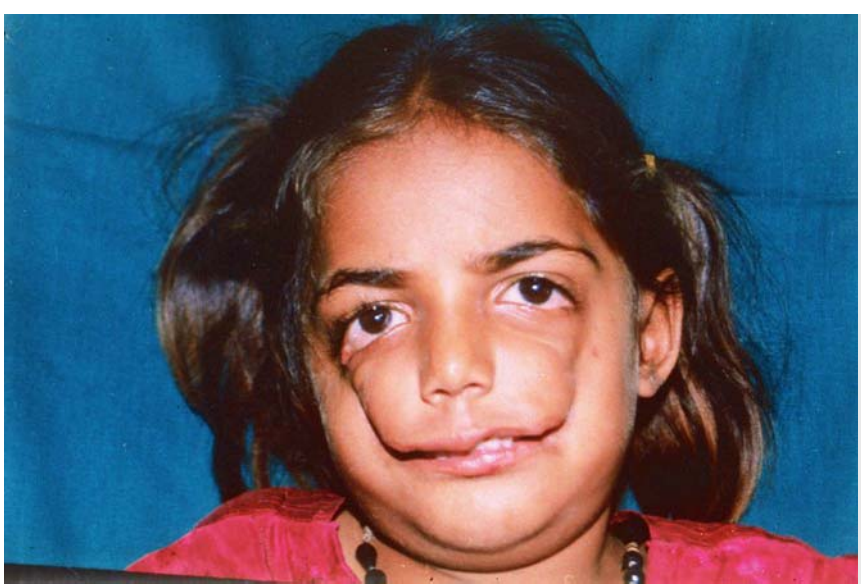

Fig. 2.5 Oblique facial cleft.

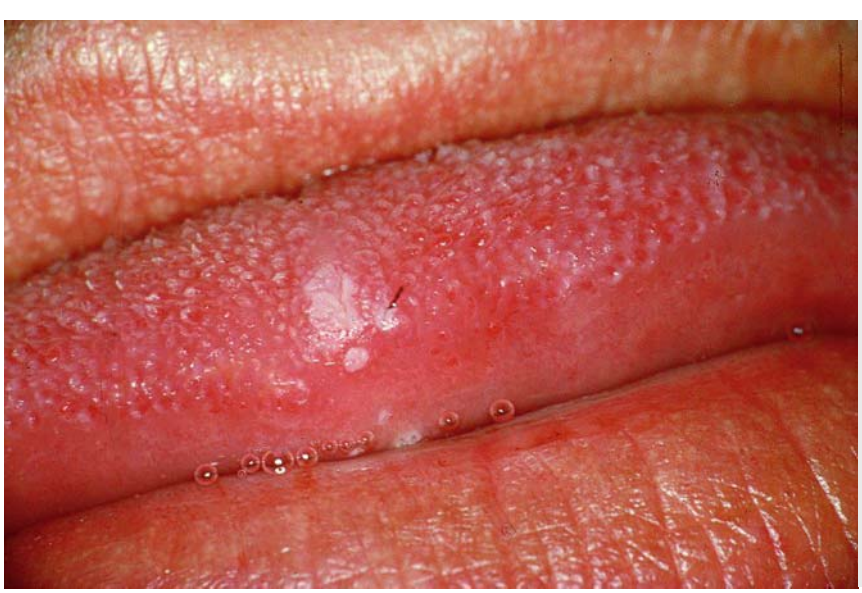

Fig. 2.6 Black hair with accessories on the tip of the tongue.

Commissural Lip Pits

Key points

- A relatively common developmental invagination that characteristically occurs at the corners of the mouth.
- Very rarely, the disorder may be associated with other orofacial defects.
- The diagnosis is based on the clinical features.

Introduction

Commissural lip pits are a developmental invagination due to failure of normal fusion of the embryonal mandibular and maxillary processes. Occasionally, an autosomal dominant trait has been recorded. They appear to be more common in adult men and are not obvious during childhood. Usually, the disorder is not associated with other orofacial defects.

Clinical features

Clinically, commissural lip pits usually present as bilateral small blind asymptomatic fistulas 1 to 3 mm in depth. Characteristically, the pits develop at the corner of the vermilion border of the lips (**Fig. 2.8**). Occasionally, small amount of mucous may accumulate at the deep aspect of the pits which becomes evident after squeezing its base. The diagnosis is based exclusively on the clinical features.

Differential diagnosis

- Mechanical trauma
- Surgical deformity

Pathology

It is not required. On histologic examination a small invagination lined by normal epithelium is the common feature. Usually, ducts from minor salivary glands may drain into the invaginated epithelium.

Treatment

No treatment is required. In rare cases where the pits may cause mild topical problems, reconstructive surgery is recommended.

Ankyloglossia

Key points

- A developmental malformation of the tongue.
- Ankyloglossia may occasionally cause speech defects or gingival recession, which may lead to periodontal disease.
- The diagnosis is based on clinical criteria and is frequently incidental.

Introduction

Ankyloglossia or tongue-tie is a rare developmental malformation characterized by a short and thick lingual frenum leading to limitation of tongue movement. Most often the anomaly is the result of fusion and attachment of the lingual frenum to the floor of the mouth or the alveolar mucosa. In other cases the short lingual frenum stems from the tip of the tongue.

Clinical features

Clinically, the lingual frenum is short and usually thick and inelastic (**Fig. 2.9**). Occasionally, it can be thin and short, extending from the floor of the mouth or the alveolar mucosa to the tip of the tongue. In severe cases the tongue is tied down with limited movement. In such cases it may lead to speech difficulties. However, in the majority of the cases the functional problems are minor or absent. Short and thick frenum of the lips may also occur (**Fig. 2.10**). The diagnosis is based on the clinical features.

Pathology

It is not required.

Treatment

In most cases treatment is not required. In severe cases, with speech and swallowing problems, frenectomy (surgical clipping of the lingual frenum) will correct the problems.

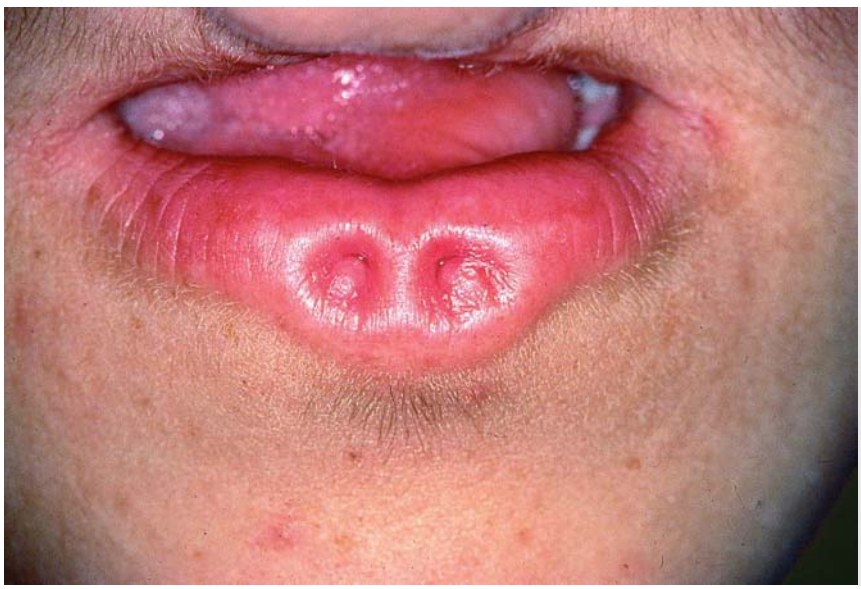

Fig. 2.7 Congenital lip pits.

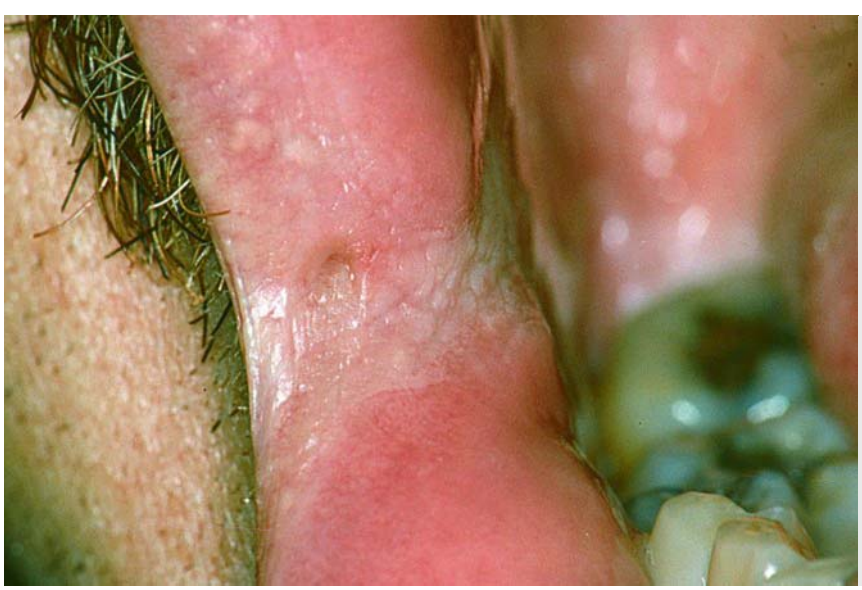

Fig. 2.8 Congenital commissural pits.

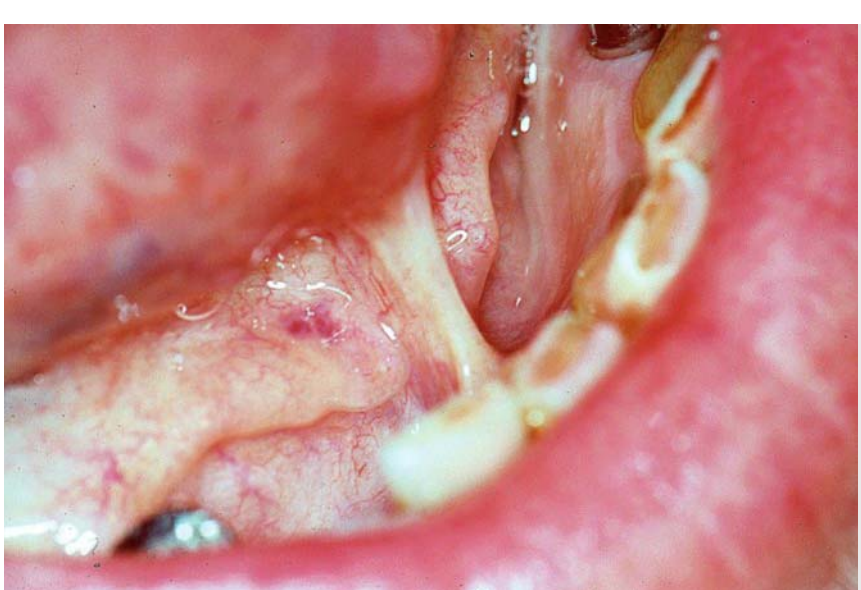

Fig. 2.9 Ankyloglossia.

Bifid Tongue

Key points

- A rare developmental malformation.
- It may be in complete or incomplete form.
- The lesion is usually asymptomatic.

Introduction

Bifid tongue is a rare developmental anomaly that may appear in *complete* or *incomplete* form. It may develop as an isolated defect, coexist with the oro-facial-digital syndrome or occur as a complication of tongue piercing.

Clinical features

Clinically, the incomplete form of bifid tongue manifests as a deep furrow along the midline of the dorsum of the tongue or as a double ending of the tip of the tongue (**Figs. 2.11, 2.12**). The complete form is very rare and appears as two separate parts of the anterior portion of the tongue, each of them controlled independently. The malformation is usually asymptomatic. The diagnosis is based on the clinical features.

Differential diagnosis

- Trauma of the tongue
- Surgical complication
- Lingual piercing complication
- Oro-facial-digital syndrome

Pathology

It is not required.

Treatment

The incomplete form usually requires no therapy. The complete form requires reconstructive surgical reconstruction.

Double Lip

Key points

- A rare oral malformation that usually affects the upper lip.
- Double lip may be congenital or acquired.
- It is more evident during smiling.

Introduction

Double lip is a rare oral malformation that affects more often the upper lip and less often the lower lip or both. The disorder is usually *congenital*, but it can also be *acquired*. The acquired form can occur as a result of trauma, surgical complication, radiation, and repeated sucking of the lip by the patient. Frequently, double lip is a part of Ascher's syndrome.

Clinical features

Clinically, double lip is characterized by an asymptomatic protruding horizontal fold on the mucosal surface of the lip (**Fig. 2.13**). The abnormality becomes prominent during speech or smiling, while when the lip is at rest the disorder is usually invisible. In a case of double lip, the clinician should bear in mind Ascher's syndrome with the classical clinical triad: blepharochalasis, nontoxic thyroid goiter, and congenital double lip. The diagnosis of double lip is based exclusively on the clinical features.

Differential diagnosis

- Ascher's syndrome
- Repeated mechanical trauma
- Surgical complication
- Radiation of the orofacial area

Pathology

It is not required.

Treatment

It is usually not required. In severe cases, surgical correction may be attempted for aesthetic reasons only.

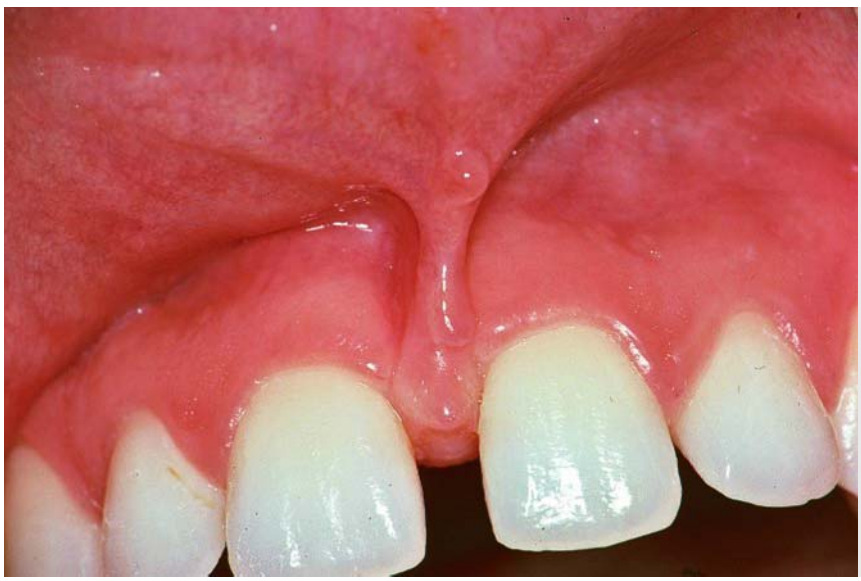

Fig. 2.10 Short bridle of the upper lip.

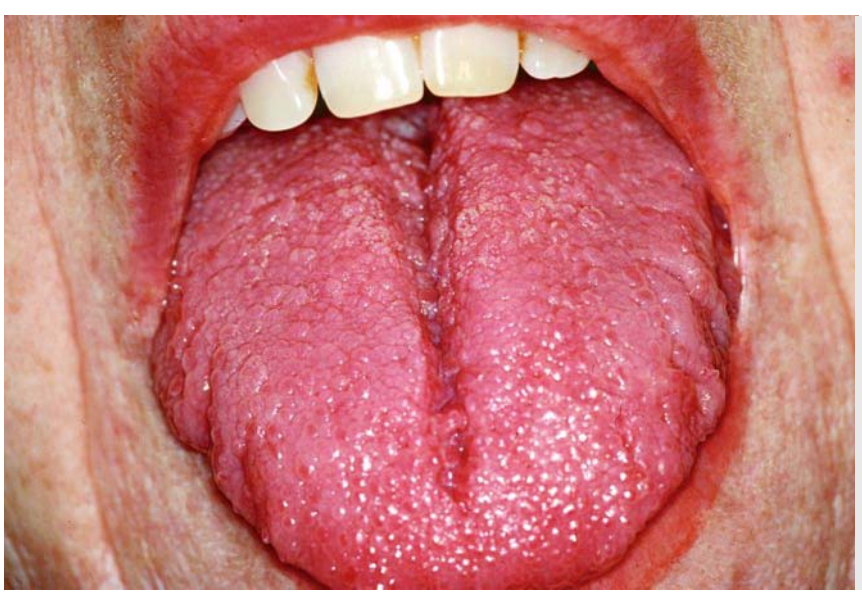

Fig. 2.11 Deep central fissure on the middle of the tongue.

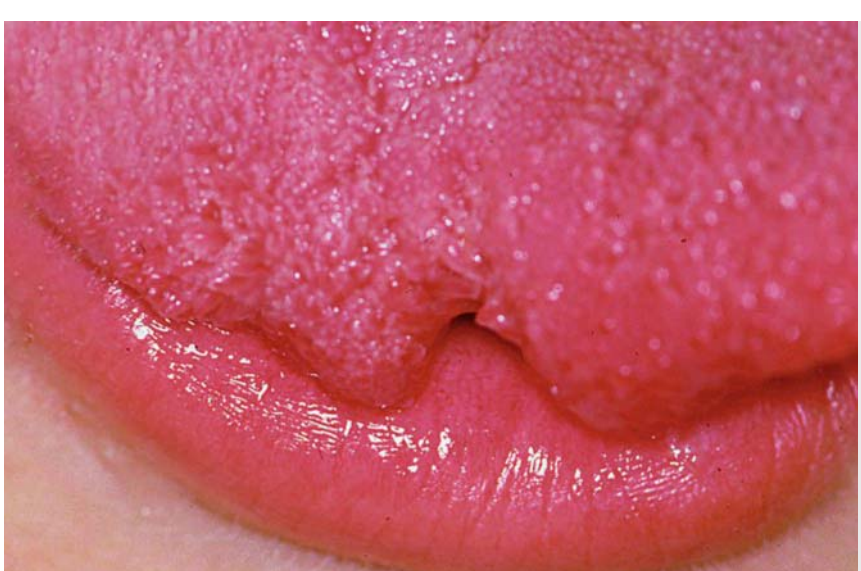

Fig. 2.12 Bifid tongue.

Torus Palatinus

Key points

- A relatively common bony exostosis.
- Characteristically, it occurs along the midline of the vault of the hard palate.
- The disorder is usually asymptomatic and the diagnosis is usually made incidentally during routine oral examination.
- The diagnosis is based on the clinical features.

Introduction

Torus palatinus is a bony exostosis that occurs along the midline of the hard palate. The etiology and pathogenesis remain uncertain. However, it is thought that genetic and environmental influences (multifactorial etiology) participate in the development of the lesion. The prevalence of torus palatinus varies from 20 to 30% or less with a female-to-male ratio of 2:1.

Clinical features

Clinically, torus palatinus appears as a sessile, asymptomatic, bony hard mass that develops during normal growth along the middle of the hard palate and is covered by thin normal mucosa. However, occasionally the mucosa may become ulcerated and painful if traumatized (**Fig. 2.14**). Recently, cases have been recorded with mucosal ulcerations and osteonecrosis of torus palatinus in patients who received bisphosphonates and RANKL inhibitor (denosumab). The size of the mass usually varies from 1 to 3 cm in diameter. There are several clinical forms of torus palatinus: the *nodular*, *lobular*, *spindled*, *flat*, and *completely irregular*. The lesion usually appears during the second or third decade of life. Because of its slow asymptomatic growth, it is usually an incidental finding during routine dental examination. The diagnosis is based on the clinical features.

Differential diagnosis

- Osteoma
- Gardner's syndrome
- Osteosarcoma

Pathology

It is usually not required. On histologic examination, a normal mass of lamellar cortical bone is recorded. The dental and occlusal radiographs are usually normal. However, large lesions may present as diffuse radiopaque lesions.

Treatment

Usually, no treatment is needed. However, surgical excision is necessary in edentulous patients if a full or partial denture is required and possibly in cases of osteonecrosis after bisphosphonates or denosumab therapy.

Torus Mandibularis

Key points

- A relatively common bony exostosis.
- It occurs along the lingual aspect of the mandible in the premolar area.
- Torus mandibularis usually develops bilaterally.
- The exostosis is usually asymptomatic and the diagnosis is based on the clinical features.

Introduction

Torus mandibularis is a relatively common bony exostosis that characteristically appears along the lingual aspect of the mandible superior to the mylohyoid ridge at the premolar area. In over 80 to 90% of the cases the lesions are bilateral. They may rarely be associated with torus palatinus. The prevalence ranges between 6 and 40%, affecting both sexes almost equally.

Clinical features

Clinically, torus mandibularis presents as slow-growing, asymptomatic, hard bony modules that may be single or multiple, three to five in number (**Fig. 2.15**). The size varies from 0.5 to several centimeters. Usually, the patients are not aware of the lesion unless secondary erosion or ulcer develops after mechanical trauma. Tori mandibulares usually reach their final size by the end of the third decade of life. The diagnosis is based on the clinical features.

Differential diagnosis

- Osteoma
- Gardner's syndrome
- Chronic dental abscess
- Osteosarcoma

Pathology

It is not usually required. On histologic examination, a mass of dense lamellar cortical bone is seen. Occlusal radiograph usually shows a dense radiopacity at the area of exostosis.

Treatment

It is usually not required. In severe cases surgical correction is needed particularly if a full or partial denture has to be constructed.

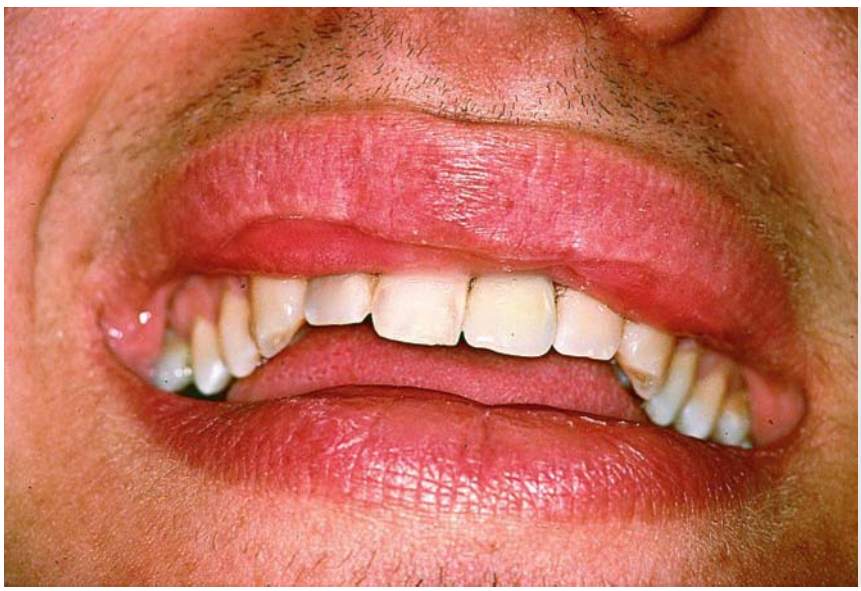

Fig. 2.13 Double lip.

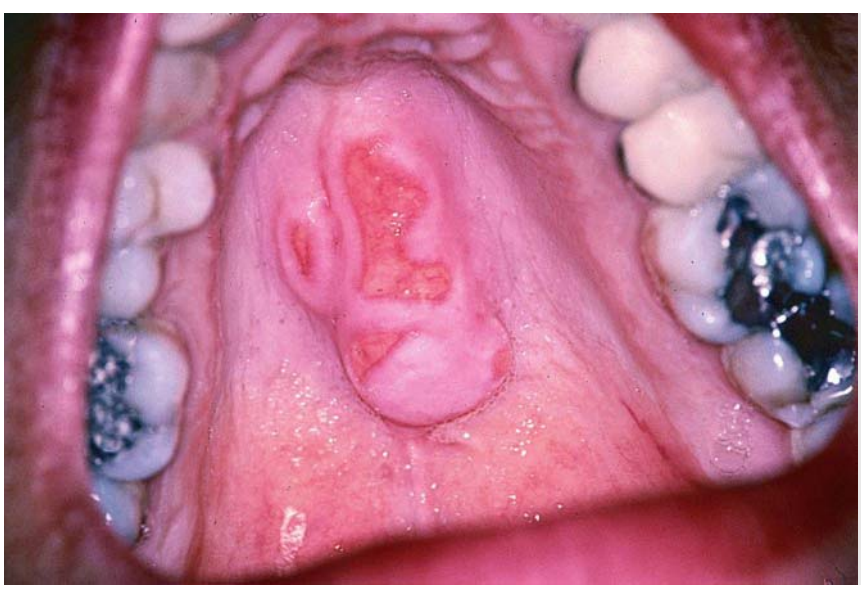

Fig. 2.14 Torus palatinus on the center of the hard palate superficially ulcerated.

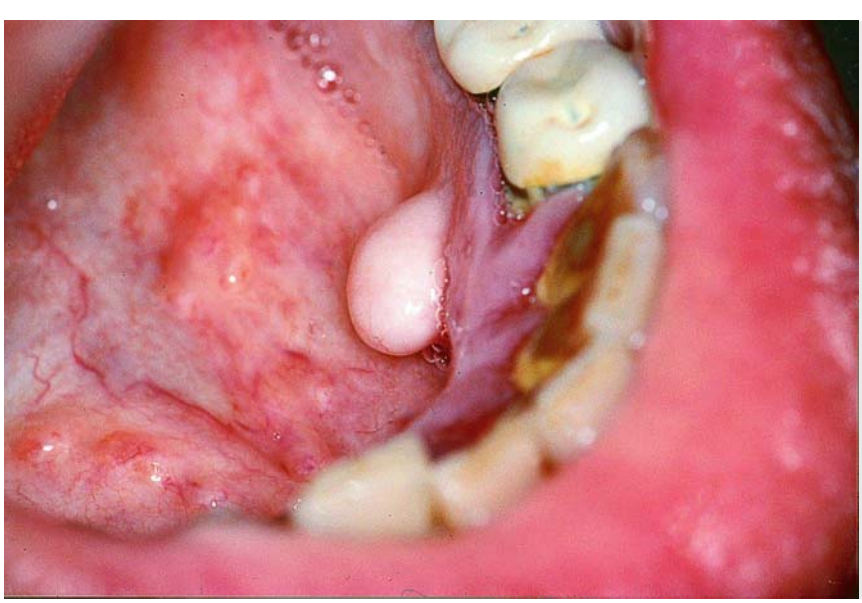

Fig. 2.15 Torus mandibularis.

Multiple Exostoses

Key points

- A relatively rare type of bony exostoses.
- They occur on the buccal aspect of the maxilla and the mandible, usually bilateral.
- Their occurrence is related to increasing age.
- Rarely multiple exostoses may be concurrent with torus palatinus and/or torus mandibularis.

Introduction

Multiple exostoses occur as multiple bony hard masses along the buccal aspect of the alveolar ridges of the jaws. The lesions usually develop bilaterally and are more common in the maxilla than the mandible. They occur less frequently than torus palatinus and torus mandibularis, and may rarely coexist with them. Occasionally, a single exostosis may be seen in the tuberosity area of the maxilla. The etiology of multiple exostoses involves interplay of multifactorial genetic and environmental factors.

Clinical features

Clinically, multiple exostoses present as multiple, asymptomatic, nodular bony elevations along the buccal aspect of the alveolar bone of the jaws, covered by normal mucosa (**Fig. 2.16**). However, occasionally ulceration may develop on the overlying mucosa, following mechanical trauma, leading to pain. The diagnosis is based on the clinical features.

Differential diagnosis

- Multiple osteomas
- Gardner's syndrome
- Paget's disease of bone
- Fibrous dysplasia

Pathology

It is not usually required. On histologic examination, a mass of normal dense lamellar cortical bone is seen.

Treatment

No treatment is required. Problems may be encountered during full or partial denture fitting. In such cases surgical recontouring can be performed.

Fibrous Developmental Malformation

Key points

- A rare developmental malformation of fibrous proliferation.
- It usually occurs in the maxillary tuberosity region and less frequently in the retromolar region of the mandible.
- The lesions usually occur bilateral in a symmetrical pattern.

Introduction

Fibrous developmental malformation is a rare fibrous overgrowth of unknown etiology. The lesions most frequently begin during childhood or puberty and may exhibit rapid or insidious growth. Usually, after the third decade of life the lesions remain stable.

Clinical features

Clinically, fibrous developmental malformations appear as a painless fibrous overgrowth that classically develops bilaterally, in a symmetrical pattern in the maxillary tuberosity region and rarely in the retromolar region of the mandible (**Figs. 2.17, 2.18**). The surface of lesion is smooth and is covered by normal mucosa with pale color. It is firm on palpation. The fibrous mass is firmly attached to the underlying bone, but on occasion may be movable. The lesion is asymptomatic and benign. Its size may extend to several centimeters and occasionally may even touch each other causing problems in speech, mastication, and swallowing. The diagnosis is based on the clinical criteria and a biopsy is rarely necessary.

Differential diagnosis

- Fibroma
- Hereditary gingival fibromatosis
- Neurofibroma
- Oral focal mucinosis

Pathology

Microscopic examination of fibrous developmental malformation shows large amount of dense collagen with few spindle-shaped cells and mild, chronic, scattered inflammation, mainly of plasma cells and lymphocytes. The covering epithelium is thin with flat rete ridges.

Treatment

Conservative surgical excision is the treatment of choice.