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ORAL CANDIDIASIS

Risk factors. Clinical forms. Diagnostic and Treatment
OUTLINE

- Etiology
- Pathogenesis
- Classification
- Clinical characteristics
- Course and evolution
- Diagnosis
- Therapy
- Prognosis
INTRODUCTION

CANDIDA SPP. - MORPHOLOGICAL CHARACTERISTIC

Candida albicans is a diploid fungus that grows both as yeast and filamentous cells and a causal agent of opportunistic oral and genital infections in humans.

To infect host tissue, the usual unicellular yeast-like form of C. albicans reacts to environmental cues and switches into an invasive, multicellular filamentous form, a phenomenon called dimorphism.
Candida spp. as **commensal** are carried in the mouths of about 90% of the population as a normal component of the oral microbiota. **Overgrowth is prevented by other microorganisms.**

This candidal carriage state is not considered a disease, but when Candida spp become pathogenic and invade host tissues, oral candidiasis can occur.

This change usually constitutes an **opportunistic infection** of normally harmless micro-organisms because of local, or systemic factors altering host immunity.

**SOURCE**
http://www.helpyourautisticchildblog.com/category/candida/
Candidiasis occurs in the very young, the very old, or the very sick.

Candidiasis is a disease of the diseased!

Prof. T. Djemileva
SPECIES

The causative organism is usually *Candida albicans* or less commonly other Candida species such as (in decreasing order of frequency):

- C. tropicalis (1)
- C. glabrata (2)
- C. parapsilosis (3)
- C. krusei (4)
- C. dubliniensis (5)
- other
**ORAL CANDIDIASIS**

### Risk factors. Clinical forms. Diagnostic and Treatment

<table>
<thead>
<tr>
<th><strong>C. albicans</strong></th>
<th>70-80% (50%)</th>
<th>Highly virulent</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>C. glabrata</strong></td>
<td>5-10% (10-15%)</td>
<td><strong>HIV</strong>, leukaemia, elder patients, diabetic patients.</td>
</tr>
</tbody>
</table>

C. Albicans and C. glabrata account for over 80% of cases.
PATHOGENESIS

The host defenses against opportunistic infection of candida species are:

1. **The oral epithelium**, which acts both as a physical barrier preventing microorganisms from entering the tissues, and is the site of cell mediated immune reactions.

2. **Competition and inhibition interactions between Candida spp and other microorganisms** in the mouth.

3. **Saliva**, which possesses both mechanical cleansing action and immunologic action, including salivary IgAs antibodies, which aggregate candida organisms and prevent them adhering to the epithelial surface; and enzymatic components such as lysozyme, lactoperoxidase and antileukoprotease.

**Disruption to any of these local and systemic host defense mechanisms constitutes a potential susceptibility to oral candidiasis**, which rarely occurs without predisposing factors.
PATHOGENESIS

Candidal carriage state is not considered a disease, but when Candida spp become pathogenic and invade host tissues, oral candidiasis can occur:

The predisposing factors are:

• LOCAL FACTORS
  – Dentures;
  – Low pH of the saliva;
  – Neglected hygiene etc.

• SYSTEMIC FACTORS
  – Systemic diseases;
  – HIV/AIDS;
  – Immunosuppression
  – Oncological treatment;
  – Drugs misuse;
  – Antibiotic treatment etc.
**PATHOGENESIS.** Macro-organism – systemic factors

**Immunodeficiency/immunocompromise**

- Acute pseudomembranous candidiasis occurs in about *5% of newborn infants*. Candida species are acquired from the mother's vaginal canal during birth. *At very young ages, the immune system is yet to develop fully* and there is no individual immune response to candida species, an infant's antibodies to the bacteria are normally supplied by the mother's breast milk.

- In non-infants, **immunodeficiency** is also a cause, e.g., as a result of AIDS/HIV or chemotherapy.

- **Topical or systemic corticosteroids**, e.g., for treatment of asthma may also result in oral candidiasis:

- **Active cancer and oncological treatment**, chemotherapy or radiotherapy
**PATHOGENESIS.** Macro-organism – systemic factors

**Diet**

- **Malnutrition**, whether by malabsorption or poor diet, especially hematinic deficiencies (iron, vitamin B12, folic acid) can predispose to oral candidiasis by causing diminished host defense and epithelial integrity.

- For example, iron deficiency **anemia is thought to cause depressed cell-mediated immunity**. Some sources state that deficiencies of vitamin A or pyridoxine are also linked.

- There is evidence that a **diet high in carbohydrates predisposes to oral candidiasis**.

- In vitro and studies show that Candidal growth, adhesion and biofilm formation is enhanced by the presence of carbohydrates such as glucose, galactose and sucrose.
**Drug induced conditions**

- **Broad-spectrum antibiotics** eliminate the competing bacteria and disrupt the normally balanced ecology of oral micro-organisms. Acute oral candidiasis occurring due to medication with corticosteroids or broad-spectrum antibiotics (e.g., tetracycline).

**Smoking**

- Smoking, especially heavy smoking, is an important predisposing factor but the reasons for this relationship are unknown. One hypothesis is that cigarette smoke contains nutritional factors for C. albicans, or that local epithelial alterations occur that facilitate colonization of candida species.
ALTERED BARRIER FUNCTION OF THE ORAL MUCOSA

• Presence of certain mucosal lesions, especially those that cause hyperkeratosis and/or dysplasia, e.g., lichen planus. Such changes in the mucosa predispose it to secondary infection with candidiasis.

• Other physical mucosal alterations are sometimes associated with candida overgrowth, such as Fissured tongue (rarely) or Tongue piercing.
QUANTITY AND QUALITY OF SALIVA

• Both the quantity and quality of saliva are important oral defenses against Candida spp.
• Decreased salivary flow rate or a change in the composition of saliva, collectively termed salivary hypofunction or hyposalivation is an important predisposing factor.
• Xerostomia is frequently listed as a cause of candidiasis, but xerostomia can be subjective or objective, i.e., a symptom present with or without actual changes in the saliva consistency or flow rate.
IMBALANCE OF THE ORAL MICROBIOTA

- **Broad-spectrum antibiotics** which eliminate the competing bacteria and disrupt the normally balanced ecology of oral micro-biota. Acute oral candidiasis occurring due to medication with corticosteroids or broad-spectrum antibiotics.
- **Denture wearing**, and poor denture hygiene, particularly wearing the denture continually rather than removing them during sleep, is another risk factor, both for candidal carriage and for oral candidiasis. **Dentures provide a relative acidic, moist and anaerobic environment because the mucosa covered by the denture is sheltered from oxygen and saliva.** Loose, poorly fitting dentures may also cause minor trauma to the mucosa, which is thought to increase the permeability of the mucosa and increase the ability of C. albicans to invade the tissues.
**Pathogenesis.** Candida’s pathogenic factors

Pathogenesis 1

- Adhesion is an important determinant of *Candida’s* virulence
  - *Candida* produces a large number of adhesins that mediate adherence to host epithelial and endothelial cells
  - Strains with faulty adhesins are avirulent
PATHOGENESIS. Candida’s pathogenic factors

Pathogenesis 2

- *Candida* produces many enzymes that contribute to its pathogenicity
  - Produces 9 proteinases involved in invasion of tissues by degradation of extracellular matrix proteins
  - Produces adenosine which blocks neutrophil degranulation, thus impairing phagocytosis
**PATHOGENESIS.** Candida’s pathogenic factors

**Pathogenesis 3**

- *Candida* adapts rapidly to changes in host environment
  - Shifts between phenotypes in a reversible and random fashion
  - Produces genetically altered variants at a high rate
  - This adaptation makes it difficult for host defenses to attack and eliminate infection
Head&Neck radiotherapy – acute mucositis.
Head&Neck radiotherapy – radiogenic caries
ORAL CANDIDIASIS

Risk factors. Clinical forms. Diagnostic and Treatment

Head&Neck radiotherapy – severe xerostomia
Head & Neck radiotherapy – severe xerostomia (0.1 ml saliva for 5 min.), decreased pH (6.4) and low buffer capacity. Saliva is viscous and sticky.
Neutropenia after chemotherapy

Acute pseudomembranous candidiasis in the same patient
External stigmas of advanced diabetes.
Such a patients are prone to develop different fungal infections
• Erythematous candidiasis usually occurs on the dorsum of the tongue in persons who use **corticosteroid inhalators** due to asthma treatment
• In individuals who have developed candidiasis secondary to the use of inhaled steroids, rinsing out the mouth with water after taking the steroid, and using a spacer device to reduce the contact with the oral mucosa (particularly the dorsal tongue) may be beneficial
Imbalance of the oral microbiota. Broad-spectrum antibiotics, which eliminate the competing bacteria and disrupt the normally balanced ecology of oral micro-organisms.

Lingua nigra villosa and antibiotic sore tongue after oral intake of suspension of Augmentin
ORAL CANDIDIASIS

TONGUE piercing

Microbiological sampling and Candida albicans grown in the laboratory
ORAL CANDIDIASIS

Risk factors. Clinical forms.
Diagnostic and Treatment

SEM – resin surface

SEM – metal surface
C. albicans biofilms may form on the oral piercing surfaces of different materials.
Presence of certain mucosal alterations are sometimes associated with Candida spp overgrowth, such as *Epulis fissuratum*
Presence of certain mucosal alterations are sometimes associated with Candida spp overgrowth, such as fissured tongue (Lingua plicata)
Good denture hygiene involves regular cleaning of the dentures, and leaving them out of the mouth during sleep. This gives the mucosa a chance to recover. In oral candidiasis, the dentures may act as a reservoir of Candida species.
CLASSIFICATION of the clinical forms

**Acute candidiasis**
- Pseudomembranous candidiasis (oral thrush)
- Erythematous (atrophic) candidiasis

**Chronic candidiasis**
- Erythematous (atrophic) candidiasis
- Hyperplastic candidiasis (Candida leukoplakia)
- **Candida-associated lesions in oral cavity**
  - Angular cheilitis
  - Denture related stomatitis
  - Median rhomboid glossitis
  - Linear gingival erythema (?)

Acute Pseudomembranous Candidiasis (Thrush)

- Acute pseudomembranous candidiasis is a classic form of oral candidiasis. Overall, this is one of the most common type of oral candidiasis, accounting for about 35% of oral candidiasis cases.
- It is characterized by a coating or individual patches of pseudomembranous white slough that can be easily wiped away to reveal erythematous, and sometimes minimally bleeding mucosa beneath. These areas of pseudomembrane are sometimes described as "curdled milk". The white material is made up of debris, fibrin, and desquamated epithelium that has been invaded by yeast cells and hyphae that invade to the depth of the stratum spinosum.
Due to the fact that an erythematous surface is revealed beneath the pseudomembranes, some consider pseudomembranous candidiasis and erythematous candidiasis stages of the same entity.

Pseudomembranous candidiasis can involve any part of the mouth, but usually it appears on the tongue, buccal mucosae or palate.
Acute Pseudomembranous Candidiasis (Thrush)

- It is classically an acute condition, appearing in infants, people taking antibiotics or immunosuppressant medications, or immunocompromising diseases.

- However, sometimes it can be chronic and intermittent, even lasting for many years. Chronicity of this subtype generally occurs in immunocompromised states, (e.g., leukemia, HIV) or in persons who use corticosteroids topically or by aerosol.
• White, cheesy, creamy, loose patches that can be easily rubbed off

• Underlying mucosa is erythematous and easily bleeds

• Disturbed taste sensations (disgeusia)
Acute Pseudomembranous Candidiasis
Acute Pseudomembranous Candidiasis
Acute Pseudomembranous Candidiasis
Acute Pseudomembranous Candidiasis I usually observed in:

- Newly born (first weeks after birth)
- Old patients (marasmus)
- Leukemia
- Chemotherapy/radiotherapy
- Severe xerostomia
- Local application of corticosteroids
- Immunosuppression e.g. HIV/AIDS
Oral candidiasis in an infant. At very young ages, the immune system is yet to develop fully.
Acute erythematous (atrophic) candidiasis

- Erythematous (atrophic) candidiasis is where the condition appears as a red, raw-looking lesion.
- It may precede the formation of a pseudomembrane, be left when the membrane is removed, or arise de novo.
Acute erythematous (atrophic) candidiasis

- The erythematous candidiasis accounts for 60% of oral candidiasis cases. Where it is associated with inhalation steroids, erythematous candidiasis commonly appears on the palate or the dorsum of the tongue.

- Some authors consider denture-related stomatitis, angular stomatitis, median rhomboid glossitis, and antibiotic-induced stomatitis as subtypes of erythematous candidiasis, since these lesions are commonly erythematous/atrophic.
Atrophic (Erythematous) Candidiasis

- Acute erythematous candidiasis usually occurs on the dorsum of the tongue in persons taking long term corticosteroids or antibiotics, but occasionally it can occur after only a few days of using a topical antibiotic. This is usually termed "antibiotic induced stomatitis" because it is commonly painful as well as red.

- Chronic erythematous candidiasis is more usually associated with denture wearing.
Erythematous candidiasis can mimic geographic tongue. Erythematous candidiasis usually has a diffuse border that helps distinguish it from erythroplakia, which normally has a sharply defined border.

geographic tongue
Candida-associated denture induced stomatitis

- The **major risk factor** for the development of this condition is wearing an **upper complete denture**, particularly **when it is not removed during sleep and cleaned regularly**. Older dentures are more likely to be involved. Other factors include xerostomia (dry mouth), diabetes or a high carbohydrate diet.

- The local environment under a denture is **more acidic and less exposed to the cleansing action of saliva**, which favors high Candida enzymatic activity and may cause inflammation in the mucosa.

- Poorly fitting dentures may cause pressure on the mucosa and mechanical irritation.
Candida-associated denture induced stomatitis

- Denture-related stomatitis is usually painless and asymptomatic.
- The appearance of the involved mucosa is erythematous (red) and edematous (swollen), sometimes with petechial hemorrhage.
- This usually occurs beneath an upper denture. Sometimes angular cheilitis can coexist, which is inflammation of the corners of the mouth, also often associated with Candida albicans.
- The affected mucosa is often sharply defined, in the shape of the covering denture.
- Stomatitis rarely develops under a lower denture.
Candida-associated denture induced stomatitis

The Newton classification divides denture-related stomatitis into three types based on severity.

• **Type 1** - Localized inflammation or pinpoint hyperemia
• **Type 2** - More diffuse erythema (redness) involving part or all of the mucosa which is covered by the denture
• **Type 3** - Inflammatory nodular/papillary hyperplasia usually on the central hard palate and the alveolar ridge

Type one may represent an early stage of the condition, whilst type two is the most common and type three is uncommon.
Type 1 - Localized inflammation or pinpoint hyperemia
Type 2 - More diffuse erythema (redness) involving part or all of the mucosa which is covered by the denture
Type 3 - Inflammatory nodular/papillary hyperplasia usually on the central hard palate and the alveolar ridge
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Median rhomboid glossitis is a condition characterized by an area of redness and loss of lingual papillae, situated on the dorsum of the tongue in the midline immediately in front of the circumvallate papillae.

Median rhomboid glossitis is thought to be related to a chronic fungal infection.
CLINICAL CHARACTERISTICS

Glossitis rhombica mediana

- Apart from the appearance of the lesion, there are usually no other signs or symptoms.
- Rarely is any soreness associated with the condition.
- The typical appearance of the lesion is an oval or rhomboid shaped area located in the midline of the dorsal surface of the tongue, just anterior of the sulcus terminalis.
CLINICAL CHARACTERISTICS

Glossitis rhombica mediana

- The lesion is usually **symmetric, well demarcated, erythematous** and depapillated, which has a **smooth, shiny surface**.
- Less typically, the lesion may be hyperplastic or lobulated and exophytic.
- The lesion is typically 2 – 3 cm in its longest dimension.
Sometimes an approximating erythematous lesion is present on the palate as tongue touches the palate frequently - a "kissing lesions"
Sometimes an approximating erythematous lesion is present on the palate as tongue touches the palate frequently - a "kissing lesions"
Angular cheilitis is inflammation at the corners (angles) of the mouth, very commonly involving Candida species. Candida spp alone are responsible for about 20% of cases, and a mixed infection of C. albicans and Staphylococcus aureus for about 60% of cases. Signs and symptoms include soreness, erythema (redness), and fissuring of one, or more commonly both the angles of the mouth, with edema seen intraorally on the commisures.
Angular cheilitis is generally occurs in elderly people and is associated with denture related stomatitis.

Sometimes dentures become very worn, or they have been constructed to allow insufficient lower facial height (occlusal vertical dimension), leading to over-closure of the mouth. This causes pronouncement of the skin folds at the corners of the mouth, in effect creating an intertriginous areas where angular cheilitis can develop.
Chronic Hyperplastic Candidiasis

- The most common appearance of hyperplastic candidiasis is a persistent white plaque that does not rub off.
- The lesion may be rough or nodular in texture.
- Hyperplastic candidiasis is uncommon, accounting for about 5% of oral candidiasis cases, and is usually chronic and found in adults.
- The most common site of involvement is the commisural region of the buccal mucosa, usually on both sides of the mouth.
Chronic Hyperplastic Candidiasis

- Another term for hyperplastic candidiasis is "candidal leukoplakia". This term is a largely historical synonym for this subtype of candidiasis, rather than a true leukoplakia. Indeed it can be clinically indistinguishable from true leukoplakia, but tissue biopsy shows candidal hyphae invading the epithelium.

- Some sources use this term to describe leukoplakia lesions that become colonized secondarily by Candida species, thereby distinguishing it from hyperplastic candidiasis.
Chronic Hyperplastic Candidiasis

- White plaque that does not rub off
- Commonly found on buccal mucosa along occlusal line in V-shape and widening as it approaches commissure
- Biopsy is conditional
The oral candidiasis may occasionally be a herald of a more sinister undiagnosed pathology, such as HIV/AIDS.

Oral candidiasis is rare if CD4 counts are above 500. Outbreaks are more common as the count drops to 100, when it may be harder to treat.
A more serious condition of the throat and windpipe, called esophageal candidiasis, is on the list of AIDS defining illnesses, affecting up to 1 in 5 of people with AIDS.

- It often occurs together with oral candidiasis.

- Symptoms include chest pain, nausea and painful or difficult swallowing, causing patients to not want to eat.
ORAL CANDIDIASIS AND HIV/AIDS

Pseudomembranous Candidiasis

SOURCE:
Classification of oral diseases of HIV-associated immune suppression (ODHIS) Glick M, Abel SN, Flaitz CM, Migliorati CA, Patton LL, Phelan JA, Reznik DA (ODHIS Workshop Group-USA, Dental Alliance for AIDS/HIV CARE – DAAC)
ORAL CANDIDIASIS AND HIV/AIDS

Oesophageal Candidiasis

SOURCE:
Classification of oral diseases of HIV-associated immune suppression (ODHIS) Glick M, Abel SN, Flaitz CM, Migliorati CA, Patton LL, Phelan JA, Reznik DA (ODHIS Workshop Group-USA, Dental Alliance for AIDS/HIV CARE – DAAC)
ORAL CANDIDIASIS

Risk factors. Clinical forms. Diagnostic and Treatment

ORAL CANDIDIASIS AND HIV/AIDS

Hyperplastic Candidiasis

SOURCE:
Classification of oral diseases of HIV-associated immune suppression (ODHIS)
Glick M, Abel SN, Flaitz CM, Migliorati CA, Patton LL, Phelan JA, Reznik DA
(ODHIS Workshop Group-USA, Dental Alliance for AIDS/HIV CARE – DAAC)
ORAL CANDIDIASIS AND HIV/AIDS

Erythematous Candidiasis

SOURCE:
Classification of oral diseases of HIV-associated immune suppression (ODHIS) Glick M, Abel SN, Flaitz CM, Migliorati CA, Patton LL, Phelan JA, Reznik DA (ODHIS Workshop Group-USA, Dental Alliance for AIDS/HIV CARE – DAAC)
ORAL CANDIDIASIS AND HIV/AIDS

This is a localized or generalized, linear band of erythematous gingivitis. It was first observed in HIV infected individuals and termed "HIV-gingivitis", but the condition is not confined to this group. Candida species are involved, and in some cases the lesion responds to antifungal therapy.

SOURCE:
Classification of oral diseases of HIV-associated immune suppression (ODHIS) Glick M, Abel SN, Flaizt CM, Migliorati CA, Patton LL, Phelan JA, Reznik DA (ODHIS Workshop Group-USA, Dental Alliance for AIDS/HIV CARE – DAAC)
ORAL CANDIDIASIS AND HIV/AIDS

Angular cheilitis associated with Candidiasis

SOURCE:
Classification of oral diseases of HIV- associated immune suppression (ODHIS) Glick M, Abel SN, Flaitz CM, Migliorati CA, Patton LL, Phelan JA, Reznik DA (ODHIS Workshop Group-USA, Dental Alliance for AIDS/HIV CARE – DAAC)
ORAL CANDIDIASIS AND HIV/AIDS

Kaposi sarcoma associated with Candidiasis

SOURCE
Sol Silverman, Jr., D.D.S., University of California, San Francisco
http://hardinmd.lib.uiowa.edu/cdc/6058.html
DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS

• The diagnosis can typically be made from the clinical appearance alone, but not always.

• As candidiasis can be variable in appearance, and present with white, red or combined white and red lesions, the differential diagnosis can be extensive.

• In general Candida spp are grown in the laboratory on solid growth media or in liquid broths

• Special investigations to detect the presence of candida species include oral swabs, oral rinse or oral smears.

• Molecular diagnosis of Candida spp using real-time polymerase chain reaction (RT-PCR), Monoclonal Antibody and Rapid Latex Agglutination (RLA)

• If candidal leukoplakia is suspected, a biopsy may be indicated. Smears and biopsies are usually stained with Periodic acid-Schiff, which stains carbohydrate in fungal cell walls magenta
In pseudomembraneous candidiasis, the membranous slough can be wiped away to reveal an erythematous surface underneath. This is helpful in distinguishing pseudomembraneous candidiasis from other white lesions in the mouth that cannot be wiped away, such as lichen planus, oral leukoplakia etc.
Aside from infection and mechanical trauma, inflammatory reactions of the mucosa beneath a denture can also result from irritation or allergy (allergic contact stomatitis) caused by the materials in the denture itself (acrylic, cobalt, chromium), or in response to substances within denture adhesives. Incomplete curing of the acrylic resin may also be an involved factor.
In general Candida spp are grown in the laboratory on solid growth media or in liquid broths.

- Candida albicans growing on Sabouraud agar
- Candida appears as large, round, white or cream colonies with a yeasty odor on agar plates at room temperature.
ORAL CANDIDIASIS
Risk factors. Clinical forms. Diagnostic and Treatment
Smears are collected by gentle scraping of the lesion with a spatula or tongue blade and the resulting debris directly applied to a glass slide.

Oral swabs are taken if culture is required. Some recommend that swabs be taken from 3 different oral sites.

Oral rinse involves rinsing the mouth with phosphate-buffered saline for 1 minute and then spitting the solution into a vessel that examined in a pathology laboratory. Oral rinse technique can distinguish between commensal candidal carriage and candidiasis.

Gram staining is also used as Candida stains strongly Gram(+).
Routine diagnostic of Candida spp – DIRECT MICROSCOPY

C. tropicalis  C. parapsilosis  C. lusitaniae  C. krusei
C. kefyr  C. guilliermondii  C. lipolytica  C. glabrata
C. albicans ferments glucose and maltose to acid and gas, sucrose to acid, and does not ferment lactose, which help to distinguish it from other Candida species.

- API test at 20°C
SEROLOGICAL TESTS

- Whole cell agglutination
- Immunofluorescence
- immunoenzyme analysis for detection of IgG antibodies against Candida spp
- Radio-immunological analysis
CANDIDAL LEUKOPLAKIA - HISTOPATHOLOGIC APPEARANCE

- Spores and hyphae are identifiable (1)
- Hyperkeratosis and orthokeratosis (2)
- Defined chronic inflammation (3, 4, 5)
- N.B. Unlike other forms of candidiasis, hyperplastic candidiasis may show dysplasia
CANDIDAL LEUKOPLAKIA - HISTOPATHOLOGIC APPEARANCE

SOURCE
http://www.medadvocates.org/diseases/opportunistic/candidiasis/
Molecular diagnosis of Candida spp using real-time polymerase chain reaction (RT - PCR)

Real-time PCR detection of Candida DNA from samples may aid in diagnosis of oral candidiasis in high risk populations. This assay's ability to discriminate between C. albicans and non-C. albicans species, including C. glabrata, C. krusei, and C. parapsilosis, may allow for implementation of species-specific therapies, when necessary.

SOURCE
PCR-based gene targeting in Candida albicans
Diagnostic CAT test

- 5 sterile paper points for sampling
- 1 instruction leaflet
- 4 containers for transportation
  - 1 container for mixed sampling (T, transparent)
  - 3 single sample containers from different locations (G, B, R)
Diagnostic CAT test

Sampling and transportation:

- Mixed sample or single site samples (max. 5 samples)
- Periodontal pocket, cheek, tongue etc
- Buffer solution is no needed (DNA analysis)
Diagnostic CAT test

Shipping to laboratory and further sample proceeding

- Bar code and identification
- DNA isolation, MagNA Pure 96 (Roche)
- PCR proceeding, Microlab STARlet IVD (Hamilton)
- Real-Time-PCR, LightCycler 480 II (Roche)
The identification of Candida species is very important in the diagnostic laboratory, because such identification shows prognostic and therapeutically significance, allowing the early and correct antifungal therapy.
TREATMENT – common recommendations

- Treatment of co-existing systematic diseases
- Proper diet, probiotics
- The candida load in the mouth can be reduced by improving oral hygiene measures, such as regular toothbrushing and use of anti-microbial mouthwashes
Treatment with ANTI-FUNGAL drugs

- Oral candidiasis can be treated with topical anti-fungal drugs, such as nystatin, miconazole, gentian violet or amphotericin B. Topical treatment is recommended for patients with normal immune function.

- Patients who are immunocompromised, either with HIV/AIDS or as a result of chemotherapy, may require systemic treatment with oral anti-fungals.
SYSTEMIC TREATMENT of acute oral candidiasis

- **Fluconazole**
  50 – 150mg/daily for 7-14 days
- **Itraconazole**
  2x 100 mg/daily for 7-14 days
- **Posaconazole** (Noxafil)
  200 mg/first day. 100 mg/daily for the rest 7-14 days.

Systemic treatment is advocated in case of relapse
TREATMENT of acute pseudomembranous candidiasis

Before treatment

After treatment
TREATMENT of acute pseudomembranous candidiasis
LOCAL TREATMENT of erythematous candidiasis

- **Nystatin** (100000 IU/ml - 50 ml)
  4-6x/daily for 7-14 days

- **Natamycin** (10mg)
  4-6x/daily for 7-14 days. It is not absorbed in GIT

- **Miconazole** *(Dactarin oral gel 2%)*
  4-6x/daily for 7-14 days.
TREATMENT MODALITIES FOR DENTURE-RELATED STOMATITIS

• The most important aspect of treatment is improving denture hygiene, i.e. removing the denture at night, cleaning and disinfecting it, and storing it overnight in an antiseptic solution. This is important as the denture is usually infected with C. albicans which will cause re-infection if it is not removed.

• Substances which are used include solutions of alkaline peroxides, acids (e.g. benzoic acid), yeast lytic enzymes and proteolytic enzymes (e.g. alcalase protease).

• The other aspect of treatment involves resolution of the mucosal infection, for which topical antifungal medications are used (e.g. nystatin, amphotericin, miconazole, fluconazole or itraconazole).

• Often an antimicrobial mouthwash such as chlorhexidine is concurrently prescribed.
Candida-associated denture induced stomatitis - Type 2

Before treatment

After denture removal
Candida-associated denture induced stomatitis - Type 2

Alkalizing and Dactarin gel applications

Two weeks after treatment
Candida-associated denture induced stomatitis - Type 3

Laser ablation with CO2 laser
TREATMENT of angular cheilitis associated with C. albicans

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dose</th>
<th>Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clotrimazole 1% cream</td>
<td>Apply to affected areas bid</td>
<td>7–10 days</td>
</tr>
<tr>
<td>Ketoconazole 2% cream</td>
<td>Apply to affected areas bid</td>
<td>7–10 days</td>
</tr>
<tr>
<td>Miconazole 1% cream</td>
<td>Apply to affected areas bid</td>
<td>7–10 days</td>
</tr>
</tbody>
</table>

Treatment of the infection and inflammation of the lesions with topical antifungal medication, such as clotrimazole, amphotericin B, ketoconazole, or nystatin cream is recommended. Some antifungal creams are combined with corticosteroids such as hydrocortisone or triamcinolone to reduce inflammation, and some antifungals such as miconazole cream also have some antibacterial action.
PROGNOSIS of **angular cheilitis associated with C. albicans**

- Most cases of angular cheilitis respond quickly when antifungal treatment is used.

- In more long standing cases, the severity of the condition often follows a relapsing and remitting course over time.

- The condition can be difficult to treat and can be prolonged.
**Clinical case:** A 56-year-old woman presented with black discoloration and hairy appearance of her tongue, which she had had for 6 days, accompanied with pain sensation. She had been taking doxycycline for the past week for an upper respiratory tract infection.

She did not smoke or use oral tobacco products. Physical examination revealed brown discoloration of the tongue. Microbiological sampling confirmed *Candida albicans* infection.
The patient received a topical application of 0.5ml MB solution at 500 μg/ml. After this the patient was underwent light activation with 810nm of diode laser light (treatment parameters 2.5 W, CW) with a cylindrical diffuser for 30sec. to cover the affected area. The treatment was repeated 3 times at intervals of a day for one week.
Resolution of the lingua villosa nigra associated with Candida after 1 week's treatment with MB-mediated photodynamic therapy with diode laser (810nm).
The prognosis of oral candidiasis is usually excellent after the application of topical or systemic treatments.

However, oral candidiasis can be recurrent. Individuals continue to be at risk of the condition if underlying factors such as reduced salivary flow rate or immunosuppression are not rectifiable.

Candidiasis can be a marker for underlying disease, so the overall prognosis may also be dependent upon this.