Lecture
for the 5th year students of foreign students training faculty:


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Lecture plan

- Etiology of Candidiasis
- Pathogenesis of oral candidosis
- Classification of candidiasis
- Clinic, diagnosis, differential diagnosis of different forms of candidiasis.
- Treatment of oral candidosis
- Kinds of allergic reaction.
- Clinic, diagnosis, differential diagnosis of anaphylactic shock
- Clinic, diagnosis, differential diagnosis of nettle rash
- Clinic, diagnosis, differential diagnosis of erythema multiform
- Clinic, diagnosis, differential diagnosis of Stevens-Johnson syndrome
- Clinic, diagnosis, differential diagnosis of Lyell's disease.
Etiology of Candidiasis

Infective agent:

* Candida albicans,*

* C. glabrata,*

* C. krusei,*

* C. tropicalis,*

* C. parapsilosis.*
Etiology of Candidiasis

*C. albicans* accounts for about 50% of oral candidiasis cases, and together, *C. albicans*, *C. tropicalis* and *C. glabrata* account for over 80% of cases.

Candidiasis caused by non-*C. albicans* Candida (NCAC) species is associated more with immunodeficiency. For example, in HIV/AIDS, *C. dubliniensis* and *C. geotrichium* can become pathogenic.
Candida albicans

Courtesy of M. McGinnis

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Local factors of Candidiasis

- Poor oral hygiene,
- Xerostomia,
- Mucosal damage,
- Dentures,
- Antibiotic mouthwashes
- High sugar diet
- Corticosteroid inhalers
Systemic factors

- Broad-spectrum antibiotics,
- Steroids, immunosuppressive drugs,
- Radiation,
- HIV infection,
- Hematological malignancies,
- Neutropenia,
- Iron-deficiency anemia, diabetes mellitus,
- Cellular immunodeficiency, endocrine disorders.
Pathogenesis
5 levels of cooperation between fungus and human cells. Which were made a conditions for development of immunodeficiency:

- **Fungus localized on the epithelium surface and OMM without disturbance of cell and without penetration of tissue;**
- **Fungal disturbance of epithelium as a result cell reaction developed on the skin and mucous surface;**
Fig. 1. Molecular interaction between the cell wall of C. albicans and surfaces of the oral cavity (for Cannon, 1999):

a - a schematic representation of the structure and chemical composition of the cell wall of C. albicans;

b - the interaction of C. albicans with the molecules and the surfaces of the oral cavity, which can be subjected to colonization.
The host defenses against opportunistic infection of candida species are comprised of:

- The host defenses against opportunistic infection of candida species are comprised of:
- The oral epithelium, which acts both as a physical barrier preventing micro-organisms from entering the tissues, and is the site of cell-mediated immune reactions.
The host defenses against opportunistic infection of candida species are comprised of:

- Competition and inhibition interactions between candida species and other micro-organisms in the mouth, such as the many hundreds of different kinds of bacteria.

- **Saliva**, which possesses both mechanical cleansing action and immunologic action, including salivary **immunoglobulin A** antibodies, which aggregate candida organisms and prevent them adhering to the epithelial surface; and **enzymatic** components such as **lysozyme**, **lactoperoxidase** and antileukoprotease.
Classification of candidiasis:

**Acute**
- Pseudomembranous (thrush);
- Atrophic (erythematous)

**Chronic candidiasis:**
- Atrophic (denture stomatitis);
- Hyperplastic (candidal leyplakia);
- Granulematous
Classification of candidiasis:

- Denture related stomatitis
- Angular cheilitis
- Candida-associated lesions
- Hyperplastic
- Erythematous
- Chronic multifocal oral candidiasis
- Chronic mucocutaneous candidiasis
Diagnosis of Candidiasis

- **Cytology examination:** of tissue culture;
- **Mycological or bacteriological examination**
- **Molecular diagnosis of candidiasis:** chromatography polymerized chain reaction
- **Biopsy:** only in chronic cases.
Angular cheilitis.
Chronic hyperplastic candidiasis. Milky-white patches on the mucous membrane little change language
Pseudomembranous candidiasis

Clinically characteristic

• by curdy, creamy-white, slightly elevated, removable spots or plaques. The lesions may be localized or generalized, and appear more frequently:
• on the buccal mucosa, soft palate, tongue, and lips.
• Xerostomia,
• a burning sensation, and an unpleasant taste are the most common symptoms
Erythematous (atrophic) candidiasis

Caused by:
- HIV infection
- Broad-spectrum antibiotics or steroids relatively

Clinic picture:
- Erythematous patches or large areas, usually located on the dorsal of the tongue and palate.
- A burning sensation is a common symptom.
- In severe cases there may be erosions and vesicles associated with these changes.
Pseudomembranous candidiasis
Pseudomembranous candidiasis

263 Oral candidosis (thrush) in HIV disease.
Erythematous Candidiasis and Pseudomembranous candidiasis

264 Oral erythematous and pseudomembranous candidosis, with extensive caries in HIV disease.
Pseudomembranous candidiasis

262 Oral candidosis in HIV disease, showing typical lesions of thrush (pseudomembranous candidiosis).
Atrophyc candidiasis
Hyperplastic candidiasis
Differential diagnosis

- Leukoplakia,
- Hairy leukoplakia,
- Lichen planus,
- Syphilitic mucous patches,
- White sponge nevus,
- Traumatic lesions,
- Cinnamon contact stomatitis,
- Lupus erythematosus.
Therapy

- **Mild disease**: topical antifungal agent (Nystatin)
- **Moderate disease**: a systemic antifungal agent may be indicated (Ketoconazole).
- **Severe disease**: systemic antifungal agent (Amphotericin B). This medication requires intravenous administration and is highly nephrotoxic.
Treatment

- **Topical antifungal agents:** (Nystatin, Azoles derivatives, Amphotericin B).
- **Systemic azoles** (Ketoconazole, Fluconazole, Itraconazole).
The allergic reaction divided on:

- The disease close connected with immediate type of reactions:
  - anaphylactic shock;
  - angioneurotic Quinck’s oedema;
  - nettle rash.
- The diseases connected with delayed type of reaction:
  - fixed medicamental stomatitis;
  - toxico-allergic stomatitis;
  - Systemic toxico-allergic diseases:
    - Lyell’s disease;
    - Erythema multiforme;
    - Stevens-Johnson syndrome;
    - Chronic recurrent aphthous stomatitis;
    - Behchet’s syndrome;
    - Sjogren syndrome.
Pathogenetic mechanism of allergic reaction accordant to I.D. Ado (1978) following stages:

- Immunological stages;
- Pathochemical stage;
- Pathophysiological stage.
Four types of allergic reactions accordant to the mechanism of its development:

1 type – immediate reaction;
2 type – cytotoxical reaction;
3 type – precipitant reaction;
4 type – cell’s type of reaction, delayed type.
Anaphylactic shock

A syndrome involving the skin and subcutaneous submucosal tissues, occurring in a hereditary and a sporadic form

Clinic types of anaphylactic shock:
- Haemodynamic type;
- Cerebral type;
- Asphyxial type;
- Abdominal type.
Treatment of anaphylactic shock:

- restriction of allergen, infusion of 0,1% Adrenaline.
- the patient lay down in horizontal position, make a artificial breathing.
- increase the arterial pressure with injection of 0,5ml 0,1% Adrenaline, 0,3-1,0ml 0,1% Mesathone, control the pressure.
- Antihistamine preparation: 0,1% solution of Dimedroli, 2,5% sol. Diprazine, Suprastine, Hydrocortezone 50-150ml intra vein, Dexamethazone 4-20ml
- bronchospasm to cup off with Eupheline intra vein 5-10 ml with 40% sol. of Glucosa
- if it necessary neuroleptic, tranquilizative preparations.
Nettle Rash

Etiology of disease

Symptoms may include a particular kind of medicines: non-steroidal anti-inflammatory drugs (NSAIDs), antibiotics, and ACE-inhibitors,

- viral or bacterial infections,
- food contact,
- with pollen or fungal spores,
- contact with animals or creatures, such as sunlight, water, sweating or pressure and substances perfumes.
Clinic symptoms

During an allergic reaction, chemical called histamine is released by cells in the skin. Histamine makes the blood vessels dilate and become more permeable (leaky) so that fluid seeps out into the skin. The rash itself is due to a local build up of an excessive amount of fluid in the skin (oedema). In severe cases this may be followed by swelling in other parts of the body as well. If swelling occurs around the eyes, lips, tongue, larynx or hands it is called angioneurotic oedema.
ERYTHEMA MULTIFORME

Erythema multiforme is an uncommon, often recurrent, immune-mediated vesiculo-bullous eruption, seen especially in younger men. It is characterized by serosanguinuous exudates on the lips, and often target-like lesions on the skin.
ERYTHEMA MULTIFORME
Aetiology

Although the aetiology is unclear in most patients, in some it is precipitated by infections (such as herpes simplex or mycoplasma), drugs (sulphonamides, barbiturates, hydantoins and others) or a range of other triggers such as hormonal changes.
ERYTHEMA MULTIFORME
Clinical features of Erythema Multiforme

The oral lesions often recur but the condition usually resolves after six or seven episodes. Usually attacks occur for 10 to 14 days once or twice a year but the periodicity can vary from weeks to years. Erythema multiforme may affect the mouth alone, or skin and/or other mucosa.
ERYTHEMA MULTIFORME
The **minor form** of erythema multiforme is much more common and affects only one site.

**Oral lesions include:**

- **Lips** – cracked, bleeding, crusted, swollen.
- **Ulcers** – diffuse and widespread. Oral lesions progress through **macules to blisters and ulceration**, typically most pronounced in the anterior parts of the mouth. Extensive oral ulceration may be seen.
ERYTHEMA MULTIFORME
Other lesions:

- Rashes – various but typically ‘iris’ or ‘target’ lesions or bullae on extremities
- Ocular changes: resemble those of mucous membrane pemphigoid, and dry eyes and symblepharon may result
- Genital lesions: balanitis, urethretitis and vulval ulcers.
ERYTHEMA MULTIFORME
Stevens-Johnson syndrome

Toxic epidermal necrolysis

The Stevens-Johnson syndrome is the most severe acute form of EM:

Prodromal stages of diseases:
- upper respiratory tract infection, fever, malaise, nausea, and arthralgia may occur during the early.

The **major form** (SJS) causes widespread lesions affecting mouth, eyes, skin and genitals, with fever and toxicity, bullous and other rashes, pneumonia, arthritis, nephritis or myocarditis. Toxic epidermal necrolysis (TEN) presents similarly but is usually drug-related.
Stevens-Johnson syndrome
Lyell's disease

Toxic epidermal necrolysis (Lyell's disease), first described in 1956.

Toxic Epidermal Necrolysis is an acute onset, potentially life-threatening, idiosyncratic mucocutaneous reaction, usually occurring after commencement of a new medication.

Widespread full-thickness epidermal necrosis develops producing erythema, large blisters and/or exfoliation of large sheets of skin leaving a raw base. The skin has an appearance similar to a scald. It usually affects the trunk, face and one or more mucous membranes.
Lyell's disease
Topical issues

- Laboratory diagnosis of candidiasis.
- Clinic symptoms of thrush in oral cavity.
- Prescribe the examination and laboratory diagnosis methods for patient with candidiasis.
- Determine the clinic forms of candidiasis.
- Keeping preventive and treatment measures for patient with candidiasis.
- Etiological factors of allergic reactions.
- Pathogenesis of immediate type of allergic reaction.
- Clinic symptoms of allergic reactions.
- Determine the etiological reason of allergic reaction.
- Distinguish the clinic symptom of allergic reaction (AR).
- Complete the treatment and preventive plan personal for the patient with AR.
THANKS FOR YOUR ATTENTION!