## **IMPORTANT!!!**

DO HOMEWORK ONLY IN WORD FILE AND ATTACH IT TO MY EMAIL svetlanataneva@abv.bg DO NOT SEND HOMEWORK BY WETRANSFER, GOOGLE DOCUMENTS, ETC. THE NAME OF THE FILE SHOULD CONTAIN YOUR NAME, FACULTY (MF/FDM), GROUP, WEEK NUMBER (8). HOMEWORK CANNOT BE CHECKED AS A PHOTO!FORWARD YOUR COLLEAGUES.

**TASK: MARK 10 KEY POINTS OF THE PRESENTATION BELOW** 

# Part 1 NON-PLAQUE INDUCED INFLAMMATORY GINGIVAL LESIONS



## ASSOC. PROF. A. MLACHKOVA, DM

- WHILE PLAQUE INDUCED GINGIVITIS IS ONE OF THE MOST COMMON INFLAMMATORY DISEASES, THERE ARE UNPROVOKED BY THE PLAQUE DISEASES OF GINGIVA WHICH ARE RARELY ENCOUNTERED, BUT FREQUENTLY OF GREAT SIGNIFICANCE FOR PATIENTS.
- NON-PLAQUE INDUCED GINGIVAL LESIONS MAY BE MANIFESTATION OF SYSTEMIC STATES BUT CAN ALSO APPEAR AS PATHOLOGICAL CHANGES CONFINED ONLY TO GINGIVA.
- A CLASSIFICATION HAS BEEN SUBMITTED, BEING PART OF THE NEW CLASSIFICATION SCHEME OF PERIODONTAL DISEASES, BASED ON ETHIOLOGY OF THESE LESIONS AND INCLUDING:

- GENETIC / DEVELOPMENTAL DISORDERS;
- > SPECIFIC INFECTIONS;
- > INFLAMMATORY AND IMMUNE STATES AND LESIONS;
- > REACTIVE PROCESSES;
- > NEOPLASMS;
- > ENDOCRINE, NUTRITIONAL AND METABOLIC DISEASES;
- > TRAUMATIC LESIONS;
- > GINGIVA PIGMENTATION.

ALTHOUGH NON- PLAGUE INDUCED LESIONS ARE NOT DIRECTLY CAUSED BY PLAQUE, THEIR CLINICAL MANIFESTATION CAN BE SIGNIFICANTLY INFLUENCED BY THE AVAILABILITY OF SUCH AND THE CONSEQUENT BY ITS PRESENCE INFLAMMATION OF THE GINGIVAL TISSUE.

DENTAL MEDICS ARE THE FIRST LINK WHEN DIAGNOSING AND PLANNING TREATMENT OF PATIENTS AFFECTED BY SUCH LESIONS.

THEY SHOULD BE FAMILIAR WITH THEIR CHARACTERISTICS IN ORDER TO BE ABLE TO DIAGNOSE, TREAT, OR SEND FOR SPECIALIST TREATMENT IN CASE OF DOUBT OF NON - ASSOCIATED PLAQUE LESIONS ASSOCIATED WITH SYSTEMIC DISEASE.

# 1.GENETIC DISORDERS OF DEVELOPMENT 1.1. HEREDITARY FIBROMATOSIS (HGF) 2. SPECIFIC INFECTIONS 2.1. BACTERIAL ORIGIN

PERIODONTAL DISEASES, OCCURRING WITH NECROSIS AND CAUSED BY: (BACILLUS FUSOBACTERIUM, PREVOTELLA INTERMEDIA, ETC.) NEISSERIA GONOREAE - GONORRHEA; TREPONEMA PALLIDUM -SYPHILIS; (MYCOBACTERIUM TUBERCULOSIS) - TUBERCULOSIS; STREPTOCOCCAL GINGIVITIS - STREPTOCOCCUS STRAINS.

#### **2.2. VIRAL ORIGIN**

COXSACKIE VIRUS (HAND-FOOT-MOUTH); HERPES SIMPLEX VIRUS 1/2 (PRIMARY OR RECURRENT); VARICELLA-ZOSTER VIRUS (VARICELLA AND HERPES ZOSTER AFFECTING TRIGEMINUS), HUMAN PAPILLOMA VIRUS (SQUAMOUS CELL PAPILLOMA, CONDYLOMA ACUMINATUM, VERRUCCA VULGARIS AND FOCAL EPITHELIAL HYPERPLASIA).

## 2.3. FUNGAL ORIGIN - CANDIDIASIS AND OTHER MYCOSIS-(HISTOPLASMOSIS, ASPERGILLOSIS).

3. INFLAMMATORY AND IMMUNE STATES OF LESIONS 3.1. HYPERSENSITIVITY REACTIONS CONTACT ALLERGY; PLASMOCELLULAR GINGIVITIS; ERYTHEMA MULTIFORME

**3.2. AUTO-IMMUNE DISEASES OF THE SKIN AND ORAL MUCOSA** 

- PEMPHIGUS VULGARIS;
- PEMPHIGOID;
- LICHEN PLANUS;
- LUPUS ERYTHEMATOSUS.

## **3.3.GRANULOMATOUS INFLAMMATORY CONDITIONS** (OROFACIAL GRANULOMATOSIS)

- CROHN'S DISEASE;
- SARCOIDOSIS;
- 4. REACTIVITY PROCESSES 4.1. EPULIDES FIBROUS EPULIS CALCIFYING FIBROBLASTIC GRANULOMA;
- PYOGENIC GRANULOMA VASCULAR EPULIS
- PERIPHERAL GIANT-CELL GRANULOMA

#### **5. NEOPLASMS**

- **5.1. PREMALIGNANT**
- LEUKOPLAKIA;
- ERYTHROPLAKIA

## **5.2 MALIGNANT**

- SQUAMOUS CELL CARCINOMA;
- LEUKEMIA ;
- LYMPHOMA

6. ENDOCRINE, NUTRITIONAL AND METABOLIC DISEASES 6.1. VITAMIN DEFICIENCY

VITAMIN C DEFICIENCY

## **7. TRAUMATIC LESIONS**

## **7.1. PHYSICAL/MECHANICAL CAUSES**

- KERTOSIS FROM FRICTION;
- GINGIVA ULCERATIONS CAUSED BY BRUSHING AND SELF-INJURY
- **7.2. CHEMICAL (TOXIC) CAUSES**
- DUE TO ETCHING;
- CHLOROXIDINE;
- ACETYLSALICYLIC ACID;
- **COCAINE;**
- HYDROGEN PEROXIDE;
- DETERGENTS;
- PARAPHORMALDEHYDE OR CALCIUM HYDROXIDE

## **7.3 THERMAL CAUSES**

• BURNS ON MUCOUS MEMBRANE

## **8. GINGIVA PIGMENTATION**

- MELANOPLAKIA;
- MELANOSIS IN SMOKERS;
- DRUG-INDUCED PIGMENTATION; (ANTIMALARIAL MINOCYCLINE)
- AMALGAM TATTOO

**1.1 HEREDITARY FIBROMATOSIS-GENERALIZED FIBROUS GINGIVA HYPERPLASIA, LOCALIZED ALONG FREE AND ATTACHED GINGIVA AND RETRO-MOLAR SPACE.** 

CAN BE A SIDE EFFECT OF USAGE OF SOME MEDICATIONS SYSTEMATICALLY SUCH AS: PHENYTOIN, CYCLOSPORINE AND NIFEDIPINE (COLETTA & GRANER). THIS TYPE OF HYPERPLASIA DEPENDS ON PLAQUE EXISTENCE.

PROPER GINGIVAL HYPERPLASIA HAS A GENETIC ORIGIN (HGF) AND IT IS ALSO CHARACTERIZED BY DIFFUSIVE GINGIVAL HYPERPLASIA, SOMETIMES ENCOMPASSING PART OF THE TEETH, AS WELL. GINGIVAL HYPERPLASIA DEVELOPS INDEPENDENTLY OF THE EFFICIENT PLAQUE REMOVAL. \*HGF CAN BE ISOLATED OR PART OF SYNDROMES (GORLIN ET AL.), ASSOCIATED WITH OTHER CLINICAL MANIFESTATIONS, SUCH AS:

- HYPERTRICHOSIS (HORNING ET AL.; CUESTAS-CARNEIRO & BORNANCINI);
- EPILEPSY (RAMON ET AL);
- HEARING LOSS (HARTSFIELD ET AL);
- GROWTH RETARDATION (BHOWMICK ET AL);
- AND LIMB ANOMALIES (NEVIN ET AL;
- **SKRINJARIC & BASIC).**



HEREDITARY GINGIVAL FIBROMATOSIS. FRONTAL VIEW WITH PARTIAL TEETH COVERAGE.

#### THE SAME PATIENT - GINGIVAL MAXILLA FIBROMATOSIS IS SEVERE AND LEADS TO JAW DEFORMATION.



MOST CASES INVOLVE AUTOSOMAL DOMINANT MODE OF INHERITANCE. HOWEVER, THERE ARE CASES DESCRIBED WITH AUTOSOMAL RECESSIVE INHERITANCE (EMERSON; JORGENSON & COCKER SINGER ET AL). THE MOST COMMON CLINICAL MANIFESTATIONS OF HEREDITARY GINGIVAL FIBROMATOSIS ARE: HYPERTRICHOSIS, EPILEPSY AND MEMORY PROBLEMS.

USUALLY HGF IS A SOLID, DENSE, ELASTIC, NON-TOUCH FIBROUS TISSUE THAT COVERS THE ALVEOLAR PROCESSES AND SPREADS TO THE TOOTH CROWNS, RESULTING IN THE PRESENCE OF EXTENSIVE PSEUDOPOCKETS. THE COLOR OF GINGIVA CAN BE NORMAL OR ERYTHEMATOUS, IF INFLAMED. DEPENDING ON THE DEGREE OF GINGIVA ENLARGEMENT, PATIENTS COMPLAIN OF FUNCTIONAL AND AESTHETIC PROBLEMS.

ENLARGED GINGIVA LEADS TO PROTRUSION OF THE LIPS, PATIENTS MASTICATE HYPERPLASTIC GINGIVAL TISSUE, WHICH COVERS DENTAL CROWNS.

HGF IS RARELY OBSERVED AT BIRTH, BUT CAN BE DIAGNOSED AT AN EARLY AGE. IF HYPERPLASIA IS AVAILABLE BEFORE THE TEETH ERUPTION, THE SOLID FIBROUS TISSUE MAKES THE TEETH ERUPTION DIFFICULT (SHAFER ET AL). HISTOLOGICAL PICTURE FACILITATES DIFFERENTIAL DIAGNOSIS OF OTHER GENETICALLY DETERMINED PROLIFERATIONS OF GINGIVA, SUCH AS FABRY DISEASE, WHICH IS CHARACTERISED BY TELANGIECTASIA.

HGF TREATMENT IS SURGICAL THROUGH SEQUENCE OF GINGIVECTOMIES, BUT RECURRENTS ARE COMMON. IF THE VOLUME OF ENLARGEMENT IS VERY LARGE, THE TECHNIQUE OF APICAL FLAP REPOSITION IS APPLIED TO AVOID DIRECT EXPOSURE OF CONNECTIVE TISSUE IN GINGIVECTOMY AND ACHIEVE MORE COMPLETE ELIMINATION OF PSEUDOPOCKETS. 2.1. WITH BACTERIAL ORIGIN-PERIODONTAL DISEASES OCCURRING WITH NECROSIS (ULCERO-NECROTIC GINGIVITIS, ULCERO-NECROTIC PERIODONTITIS AND ULCERO-NECROTIC STOMATITIS).

PAPILLARY NECROSIS ULCERATIONS ARE TYPICAL, WHICH MAY RESULT IN SIGNIFICANT TISSUE DESTRUCTION WITH CRATER FORMATION. SUCH CONDITION IS CAUSED BY NEGLECTED ORAL HYGIENE OF THE ORAL CAVITY, SMOKING AND STRESS. IT IS FOUND IN IMMUNOCOMPROMISED HIV PATIENTS. IT HAS A CHARACTERISTIC CLINICAL PICTURE.



CLINICAL PICTURE OF ULCERO-NECROTIC GINGIVITIS

## NECROTIZING PERIODONTAL DISEASES ARE COMMON IN IMMUNOCOMPROMISED PATIENTS, ESPECIALLY IN HIV (+) OR WITH AIDS





GONORRHEA-SEXUAL TRANSMISSIBLE INFECTION, THAT CAN ALSO BE OBSERVED AS A PRIMARY ORAL INFECTION.

NONSPECIFIC LESIONS (ROUNDED GRAY- WHITISH SPOTS), WITH ULCERATIONS COVERED WITH PSEUDOMEMBRANES AND FIERY RED MUCOSA WITH OR WITHOUT SYMPTOMS.

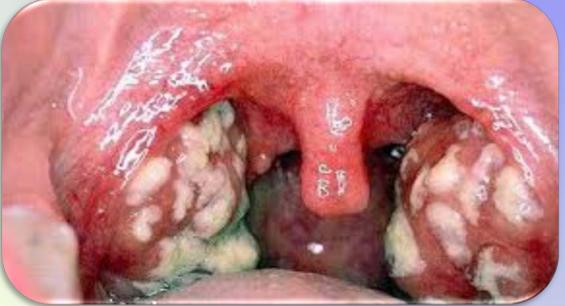
LESIONS ARE OBSERVED IN THE TONGUE, SOFT PALATE, CHEEKS, TONSILS AND OROPHARYNX.

IT MAY BE ASSOCIATED WITH PAINFUL PHARYNGITIS; PATIENTS ARE WITH LYMPHADENOPATHY AND FEVER.

#### THERE IS USUALLY A GENITAL INFECTION OF THE SEXUAL PARTNER

## THE LESIONS MAY RESEMBLE LICHEN OR STOMATITIS HERPETICA

#### MICROBIOLOGICAL AGENT IDENTIFICATION NEISSERIA GONORRHEA OR FLUORESCENCE TESTING IS REQUIRED FOR DIAGNOSIS



## GONORRHEA - GRAY-WHITISH SPOTS ON THE TONSILS

#### ORAL GONORRHEA. A GRAY-WHITE SPOT IS OBSERVED, COVERED WITH PSEUDOMEMBRANOUS COATING.





GONORRHEA - ULCERATIONS COVERED WITH COATING ON THE BACK OF THE TONGUE SYPHILIS - SYPHILIS IS A SEXUALLY TRANSMITTED DISEASE CAUSED BY TREPONEMA PALLIDUM.

PRIMARY SYPHILITIC ULCER IN THE MOUTH IS A HARD CHANCRE. IT IS A FIERY RED EDEMATOUS AND OFTEN PAINFUL ULCER OF SIZE 2 - 3 MILLIMETERS, SMOOTH COPPER COLORED BOTTOM, FLAT EDGES WITH CARTILAGE DENSITY AND WITHOUT RED HALO.

THE ULCER CAN BE COVERED WITH WHITISH INFECTED SEROUS MATTER, WHICH IS HIGHLY CONTAGIOUS!

THE SECOND STAGE OF SYPHILIS BEGINS ON AVERAGE FROM 1 TO 6 MONTHS. AFTER THE INITIAL INFECTION MANIFESTATIONS ARE VARIED, ITS DURATION IS FROM 6 MONTHS UP TO 4-5 YEARS. THE SECONDARY STAGE IS CHARACTERIZED WITH VARIOUS MANIFESTATIONS - SYMPTOMS MOST COMMONLY AFFECTING SKIN, MUCOUS MEMBRANES AND LYMPH NODES.

MOST SIGNIFICANT SYMPTOMS ARE SYPHILIC RASHES, (SYPHILIDES), THAT ARE FOUND OVER THE WHOLE BODY, THEY ARE PAINLESS AND DO NOT CAUSE ITCHING.

✤ ON THE ONSET THESE ARE SMALL AND DISCRETE PALE PINK LESIONS. AS THE DISEASE PROGRESSES, THEY DEVELOP INTO LARGE AND NOTICEABLE RASHES, MOSTLY LOCATED OVER THE PALMS, SOLES, ABDOMEN, CHEST AND BACK. THESE RASHES CAN BECOME MACULAR OR PUSTULAR. THEY MAY ALSO FORM FLAT, WIDE, WHITISH WART LESIONS KNOWN AS FLAT MUCOSAL CONDYLOMAS.

ALL THESE LESIONS ARE BACTERIAL FOCI AND CONTAGIOUS. OTHER SYMPTOMS INCLUDE: FEVER, SORE THROAT, MALAISE, WEIGHT LOSS, HAIR LOSS AND HEADACHE. IF SYPHILIS IS LEFT UNTREATED IN THE THIRD PHASE OF THE DISEASE, GUM ULCERS MAY DEVELOP IN THE ORAL CAVITY!!!

- THE LAST STAGE CALLED LATE SYPHILIS BEGINS THREE OR MORE YEARS AFTER THE ONSET OF INFECTION. THE PATIENT MAY NOT BE INFECTED WITH IT, BUT THE BACTERIA MULTIPLY AND SPREAD THROUGHOUT THE WHOLE BODY, DAMAGING THE HEART, EYES, BRAIN, NERVOUS SYSTEM, BONES AND JOINTS.
- CARDIOVASCULAR DISEASES AORTIC ANEURYSM AND AORTIC VALVE INSUFFICIENCY, DEGENERATIVE DISEASES OF THE CENTRAL NERVOUS SYSTEM LEADING TO DEMENTIA, TREMOR, LOSS OF MUSCLE COORDINATION (ATAXIA), PARALYSIS, ANISOCORIA AND BLINDNESS ARE ALSO OBSERVED.

TUMOR-LIKE PLAQUES (SYPHILITIC GUMMAS) DEVELOP ON THE SKIN, BONES, ETC. THESE ARE ENTITIES THAT ARE INITIALLY HARD AND RESEMBLE RUBBER BALLS. THEY ARE NOT SO FREQUENT COMPARED TO THE TYPICAL FOR THE SECONDARY STAGE RASH. SUCH SYPHILITIC GUMMAS ARE LOCATED UNDER THE SKIN AND ARE FLEXIBLE RELATING TO IT.

OVER TIME, THEY GROW, SOFTEN AND STICKY MATTER, CONSISTING OF NECROTIC TISSUE AND INFLAMMATORY EXUDATE DISCHARGES.

THEY LEAD TO LESIONS OVER THE SKIN AND SUBCUTANEOUS TISSUES, HEAL SLOWLY AND CAUSE DEFORMITIES AND SCARRING.

# DD: TRAUMATIC ULCER, TUBERCULOSIS, HERPES SIMPLEX, ERYTHEMA MULTIFORME, LICHEN PLANUS EROSIVA.



PRIMARY SYPHILIS -SYPHILITIC ULCER OF THE TONGUE (HARD CHANCRE) -ULCUS DURUM.





## TERTIARY SYPHILIS - TUMOR-LIKE PLAQUE - SYPHILITIC GUMMA

