



**MEDICAL UNIVERSITY - PLEVEN  
FACULTY OF MEDICINE**

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**DISTANCE LEARNING CENTRE**

**DEPARTMENT OF “NEPHROLOGY,  
HEMATOLOGY AND GASTROENTEROLOGY”**

# **PRACTICAL EXERCISES – THESES**

**FOR E- LEARNING IN NEPHROLOGY**

**ENGLISH MEDIUM COURSE OF TRAINING**

**SPECIALTY OF MEDICINE**

**ACADEMIC DEGREE: MASTER**

**PROFESSIONAL QUALIFICATION: DOCTOR OF MEDICINE**

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## **IB. LUPUS NEPHROPATHY**

### **1. Definition.**

Autoimmune process of renal connective tissue – inflammation and destruction affecting mainly the glomerular structures (autoimmune glomerulonephritis) and to a smaller extent the tubulointerstitial apparatus and the blood vessels in kidneys, clinically characterised by oedemas, hypertension and renal insufficiency.

### **2. Brief analysis of Lupus erythematoses disseminatus (LED).**

The students define the location of the kidneys impairment in LED as a manifestation of the beginning of the stage of visceralisation. During the discussion the assistant describes the so-called “pathomorphosis” of the disease:

1. Man – women ratio: 1:4
2. The beginning of the disease is changed more often. It commences directly with renal impairment rather than long years of “skin form”.
3. More and more often recently the renal impairment is the only clinically noticeable organ manifestation.
4. The diversity of renal impairment in LED makes it “ a great imitator” of a great number of renal diseases.

### **3. Handling of a patient having lupus nephropathy.**

**Anamnesis:** The students ask in details about:

1. Preceding virus infections before LED clinical manifestation.
2. The students inquire about oral contraceptives (estrogens) or pregnancy.
3. They place in chronological order the manifestations from different organs before the occurrence of the renal ones, e.g. “purple erythema” – butterfly of the cheekbones and the back of the nose, joint’ pains, breathlessness, e. t. c.
4. They determine the beginning of the arterial hypertension and the oedema syndrome.
5. They define the consecutive number of the attack as well as the remissions’ duration.
6. The students interrogate about the treatment until now.

**Physical examination** (status) of the patient:

The students have to consider the presence or lack of dermal changes: “butterfly”, “discondia” and lesions. It is of great importance for the students, to find a generalised swelling periorbitally, of the body, the abdomen wall and “ad sacrum”, the legs, the ankles and the feet. During the joint examination the assistant emphasises the “testability” of the present oedema.

Arterial hypertension with high diastolic value above 160/100 is especially characteristic, sometimes with a malevolent course and complications.

**Tests:** The assistant submits to the students the following test:

- Haematological
- Biochemical
- Urine
- Immunologic.

These tests have to support the formulation of the working diagnosis and the differential diagnosis.

- ❖ haematological tests – They are often changed as Hb < 110 g/l; Erythrocytes and Ht are reduced respectively because of the presence of antierythrocyte autoantibodies determining the haemolytic anaemia development of extra-erythrocyte origin with impetus course.
- ❖ biochemical tests are changed if there is a nephrosis syndrome defined by massive proteinuria:
  - hypoproteinemia < 66 g/l
  - hypoalbuminemia < 36,0 g/l
  - hyperalpha-2- and gammaglobulinemia
  - lack of hyperlipidemia
  - urea above 100 ml/l; creatine > 140 µmol/g, are found transitorily or permanently at the beginning of chronic renal insufficiency stage.
- ❖ immunologic tests characteristic for LED are discussed:
  - Anti-DNA antibodies against the double chain (native) DNA.
  - Anti-nuclear antibodies for diagnosing LED impetus – anti SM antibodies.
  - It is discussed with the students, why LED is called “display of antibodies”.
- ❖ urine tests:
  - proteinuria is discussed - moderate to massive 2 – 6 – 8 g/24h, glomerular in composition and highly unselective (selectivity index >0,5-0,6). The more severe structure impairment of the glomeruli with formation of fibrinoid dystrophy and necrosis; the more unselective is proteinuria.
  - erythrocytes with dysmorphism and cylinders (hyaline and granular).
- ❖ The renal function is limited, when there is an impetus of the disease. When it is treated, the glomerular filtration is recovered partially. Chronic renal insufficiency is developed after a series of impetuses with creatine clearance below 50-40 ml/min.

#### **4. Preparing of working diagnosis.**

The assistant summarises together with the students the syndromes ensuing from the anamnesis, the physical test and the applied tests:

- hypertension-vascular
- nephrotic
- nephritic
- dermal
- cardiac
- of renal insufficiency

❖ Writing in Latin the working diagnosis.

### **5. Discussion of the differential diagnosis.**

1. Chronic glomerulonephritis – LED nephritis might resemble all forms of chronic glomerulonephritis. The puncture renal biopsy shows changes – fibrinoid dystrophy and necrosis (“wire stitches”). There are changes and symptoms in the joints, skin and heart.
2. Other collagenosis nephropathies
  - Shonline-Henoch nephritis – capillary-toxicosis – haemorrhagic dermal rash, increased YgA.
  - Periarteritis nodosa nephritis – very severe hypertension syndrome and fast developing renal insufficiency.

### **6. Therapy.**

1. Pathogenetic therapy in all diffuse forms:
  - It is mainly relied on corticosteroids 1-2 mg/kg body weight/daily *rednison* or *prednisolon* for 6-8 weeks
  - *Cyclophosphamid*; it is preferred also *Imuran* – tabl. of 50 mgs for 6-8 weeks.
  - Pulse therapy is undertaken very often in the severe clinical and immunological cases with:
    - *Urbason* 10 -12 mg/kg daily – **3 days**
    - *Cyclophosphamid* 10 mg/kg/daily - **1 day**
  - Anticoagulant therapy:
    - *Heparin* 30-40 000 E/24h and following up t of coagulation and KKB
    - *Fraxiparin* 2 x 0,6 E and following coagulation samples
  - Plasmapheresis in very severe forms and superactive autoimmune process.  
10 procedure every 48 hours
2. Symptomatic therapy:
  - antihypertensive drugs
  - Protein products /prosthesis/ in case of severe nephrotic syndrome

- Cardiotoxic therapy in heart weakness *l*
- Haemodialysis in:
  - Acute renal insufficiency from very acute forms
  - Permanent advanced stages of chronic renal insufficiency (creatinine above 600-650  $\mu\text{mol/l}$ ).

❖ **Writing a prescription**