



**MEDICAL UNIVERSITY - PLEVEN
FACULTY OF MEDICINE**

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

PREPARED BY DEPARTMENT OF NEPHROLOGY

PLEVEN, 2020

MYELOMIC NEPHROPATHY

1. Definition

Impairment of renal tubulointerstitial structures, resulting by myelomic cells production of monoclonal immunologically sterile immunoglobulins and mainly their precursor light chains (kappa and lambda) limiting the kidneys concentration and diluting ability and impetus development of chronic renal insufficiency.

2. Brief analysis of the disease stages

The students report the clinical manifestation of the multiplex myeloma. The assistant highlights the location of renal impairment in this disease, namely:

1. In proliferation impetus of myelomic cells and surplus production of light chains and their accumulation and precipitation in the tubular lumens.
2. The satiated capacity role of tubular epithelial cells to metabolise and reabsorb the light chains decomposed products.
3. The disrupted blood reology in peritubular capillaries by the high plasma protein.
4. The disrupted calcium-phosphorus exchange.

3. Handling of a patient having myelomic nephropathy.

Anamnesis: The students have to know in details the commencement of the disease as a term and symptoms. Hypertension, anaemia, bone (joint and spinal-vertebral) pains are considered and it is accented on the time of occurring of the proteinuria, polyuria and nocturia. Special attention is paid to the first symptoms of nitric retention, namely: nausea, morning sickness, lack of appetite, changed (bitter) mouth taste. The manifestations of renal or other organs' infection or system (fever, dysuria, sore throat, cough...) are checked.

Physical examination (status):

The students have to consider the characteristic skin and mucous membrane paleness of the patient, tongue dryness with brownish coating with increased rates of urea and creatine.

For the cardio-vascular system it is accented upon the arterial hypertension with accented second aortic tone and systolic noise of Erb-spot.

In the respiratory system there are often signs of parenchyma inflammatory infiltration (small, damp rale).

The limb joints are often painful without swelling.

Tests:

- ❖ The blood test - when the disease is clinically manifested shows typical anaemia (Hb < 110 g/l) normochrome, normocellular, determined by the bone marrow plasmocellular pathologic clone proliferation as erythropoietin deficiency is combined to it in case of nephropathy.
- ❖ From the biochemical tests – protein profile and protein exchange deviations are characteristics
 - The proteinogram is characteristic with high rates of total protein, above 86 g/l, sometimes above 100 g/l plasma for the account of the highly increased M-gradient (zone between β and γ globulin)
 - Excess of immunoglobulin light chains (kappa and lambda) is found. The assistant has to emphasise the light chains' nephrotoxicity.
 - After several impetuses of the disease it is found that urea has increased to > 8,3 ml/l and creatine > 125 μ mol/l which marks the commencement of chronic renal insufficiency.
- ❖ Urine tests - presence of Bence-Jones proteinuria is considered to be a pathologic sign and the severe tubular affection is proven by the high level of β_2 - microglobulin in the urine.
- ❖ If there is no urinary tract infection, the urine sediment is clear of leukocytes and erythrocytes. Otherwise leukocyturia, bacteriuria and bacteria significant growth are present.
- ❖ The kidney concentration ability is impaired functionally early as well as their role for the alkaline-acid equilibrium leading to acidosis.
- ❖ The glomerular filtration rate is affected at a later stage as when it decreases below 40 ml/min creatine clearance results in chronic renal insufficiency.
- ❖ Ultrasound tested the kidneys are not with decreased dimensions, even in the stage of renal insufficiency (100-110 mm).
- ❖ Venous urography is not made.
- ❖ X-ray of flat bones (skull, hip bone, ...) shows typical osteolytic changes as “moth-eaten”.

4. Diagnosis.

1. Diagnosis of myeloma:
 - ❖ Bone-marrow biopsy of sternum and proving of myelomic infiltration.

- ❖ Bone pains and bone X-ray changes.
 - ❖ Hyperproteinemia > 100 g/l and presence of “M-gradient” from the proteinogram.
2. Diagnoses of myelomic nephropathy:
- ❖ Proteinuria – Bence-Jones type
 - ❖ Light chains isolation from proteinuria, Kappa and Lambda type
 - ❖ Polyuria with low specific weight
 - ❖ Tendency for metabolite acidosis
 - ❖ Renal insufficiency.

Writing in Latin of the working diagnosis.

5. Differential diagnosis.

1. Macroglobulinemia of Waldenström:
 - More rarely occurring disease
 - Rarely resulting in renal destruction.
2. Amyloidosis:
 - Hypoalbuminaemia
 - Hypergammaglobulinaemia
 - Amyloid residues are easily found in biopsy material taken from rectum and gingiva.

6. Therapy.

1. Radical:
 - Bone-marrow transplantation
 2. For achieving a remission:
 - *Vincristin 2 mg – the first day*
 - *Cyclophosphamid from the first to the fifth day x 600 mg*
 - *Prednisolon t. 5 mg 60-8- mg/daily.*
 3. Prevention of myelomic nephropathy development, it is necessary:
 - Hydration – taking liquids $> 2l/24$ h.
 - Polyuria > 3000 ml/24 h.
 - Alkalinization of urine – aim – urine pH $> 8,0$ with Soluran.
 4. In case of infections energetic antibiotic therapy.
 5. In case of terminal chronic renal insufficiency – haemodialysis.
- ❖ Writing a prescription