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

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PROFESSIONAL QUALIFICATION: DOCTOR OF MEDICINE

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II B. GOUT NEPHROPATHY

1. Definition

In its most common form the gout nephropathy is manifested as chronic interstitial gouty nephritis as a result of deposit in the renal interstice of crystal pure uric acid in the form of depots "tophi" leading to abacterial inflammatory process followed by fibrosis. Uric acid deposit is a consequence of its high plasma level – above 416 μmol/l.

2. Brief analysis of the disease stages

The students report the clinical manifestation of gout and mainly of gouty arthritis. The assistant emphasises, that it is a disease of many years and the gradual accumulation of symptoms, revealing that the pathologic process is in the kidney. Other forms of renal impairment in case of gout are discussed briefly, namely:

1. Acute hyperuriemic crises – acute renal insufficiency.
2. Uric nephrolithiasis.
3. Form with proteinuria.

3. Handling of a patient having chronic interstitial gouty nephritis

Anamnesis: The students have to know in details the commencement of the disease as a term and symptoms. They are asking about arthritic manifestations in metatarsal-phalangeal joints, wrists, ankles and vertebrae. Anamnesis is sought for nephrolithiasis colic and easily friable orange calculi excretion. The assistant draws the attention to more discrete signs, such as: easy fatigue, dull pains or heaviness bilaterally in the girdle area, increased amount of diuresis with excretion of lighter urine, night urinating, front and back headache, nausea and morning sickness.

Physical examination (status):

The students consider the characteristic skin and mucous membrane paleness.

For the cardio-vascular system it is found arterial hypertension with accented second aortic tone. The feet joints, mainly metatarsal-phalangeal of the 1st toe are deformed with "tophi". The small joints of the hands are often deformed, too.

Tests:

- *The blood test* when the disease is clinically manifested and the kidneys are affected by the pathologic process shows data for normochrome, normocytic anaemia with Hb < 110 g/l as

the erythrocytes are normal in shape, size and haemoglobin content. The assistant highlights the fact that the anaemic syndrome occurs before chronic renal insufficiency and it is a result of the decreased rate of renal erythropoietin. Leukocytes – normal in number – up to 10^9/l. Speed of erythrocyte sedimentation accelerated.

- *From the biochemical tests* – leading is the uric acid plasma level elevation > 416 μmol/l before increasing the rate of urea and creatine. After years of evolution urea surpasses 8,3 mmol and creatine - 130 μmol/l thus the stage of chronic renal insufficiency commences. Gout often is combined with decreased glucose tolerance and even Diabetes Mellitus type II with glucose > 6,0 mmol/l.
- *From the urine tests* proteinuria is present, slight to moderate in volume 2,0 – 3,0 g/24 h. In qualitative composition it refers to tubular proteinuria for the account of microglobulins (mainly β_2 – microglobulin). Proteinuria does not lead to hypo- and dysproteinemia. There is no nephrotic syndrome.
  - Urine pH is acid > 6,0
  - The sediment is characteristic with abundance of large uric acid crystals and erythrocytes. Erythrocyturia rarely becomes macroscopic hematuria. In the classical case of interstitial nephritis it refers to dysmorphous (with disrupted contours) erythrocytes originating from the glomerular capillaries and interstice.
- The functional tests give considerably early limitation of renal plasma flow as the glomerular filtration rate is preserved.

  Limitation of the alkalifying renal ability leading to metabolite acidosis is found early before the occurrence of chronic renal insufficiency. The assistant presents and discusses with the students pH-metry of a concrete patient. Chronic renal insufficiency is developing slowly, for years, but steadily leads to terminal stage.
- *Ultrasound test* reveals information for:
  - Early diminishing the kidneys dimensions < 100 mm (still before chronic renal insufficiency)
  - Great thinning out of the parenchyma zone (<10-12 mm)
  - Presence or lack of calculi in the renal hollow system.
- *Venous urography* gives information for bilateral, symmetrical diffuse inflammatory process. The X-ray signs of renal inflammatory process are discussed (slowed excretion, smooth papillary impressions, elongated cervix, atonic pyelon). Venous urography is not made in the cases of chronic renal insufficiency or impetus of hyperuricemia.
- *Isotope nephrogram* (ING) shows bilateral, symmetrical retarded tubular secretory and excretory phases. ING is seen.

4. Diagnosis

1. Diagnosis of gout:

❖ Hyperuricemia

❖ Pains and deformation of the small joints

2. Diagnosis of chronic gouty nephritis

❖ Anaemia before chronic renal insufficiency

❖ Arterial hypertension

❖ Metabolite acidosis before chronic renal insufficiency

❖ Small size of the kidneys with smooth parenchyma

❖ Tubular proteinuria

❖ Acid urine.

❖ Writing in Latin of the working diagnosis

5. Differential diagnosis

1. Other forms of chronic interstitial nephritis, in particular analgesic nephritis:
   - anamnesis – years of taking Sedalgin
   - reduced values of uric acid
   - without joint impairments>
2. Balkan endemic nephropathy:
   - Endemicity, heredity
   - Tumors of urothelium.
3. Chronic renal insufficiency
   - Anamnesis for treated for years another renal disease.
4. Chronic pyelonephritis.

6. Therapy

1. Dietary therapy:

It is of great importance before and after development of nephritis that

- The consummation of edible offal is forbidden: brain, liver, tripe, guts, kidneys and foods made from them.
- The consummation of meat from young animals: broiler; kid goat, lamb game is forbidden, as they are rich in purines.
- The consummation of meat not more than 1 portion/daily. Food rich in nourishing proteins (from egg and milk origin) is recommended.

2. Consummation of liquids - hydration with alkaline mineral waters (Hissar, Gornia bania) so that the diuresis could be increased above 2-2,5l/24h, thus the uric acid drops < 4 vol.% and it cannot form crystals.
3. Alkalization with Soluran so that urine pH is from 6,2 to 6,8, which results in urate crystals destruction.
4. Milurit, tabl. 0,1 – blocks uric acid production in doses 3 x 1 tabl./24h.
    - In chronic renal insufficiency I- II – doses 2 x 1 tabl./24h.
    - In chronic renal insufficiency III-IV – doses 1 x 1 tabl./24h.
5. Conservative therapy in chronic renal insufficiency II- III – with diuretics and infusions so that diuresis is stimulated 2500 ml/24 h.
6. Haemodialysis with good effect in chronic renal insufficiency - III.

❖ Writing a prescription