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

INTERSTITIAL NEPHRITIS

/Plane of exercise /

A/ Introduction

Frequency

The frequency of tubulointerstitial diseases affecting the kidneys is difficult to be determined. Their importance, however, can be appreciated with the fact, that 20 to 40 % of all patients undergoing treatment for end-stage renal disease have been estimated to have a primary tubulointerstitial disease as the cause of their renal failure.

ACUTE INTERSTITIAL NEPHRITIS

Definition

The association of acute renal failure and infiltration of the renal interstitium by inflammatory cells characterize acute interstitial nephritis (AIN).

The definition, however, needs further comments, since the clinical and pathologic picture may be more complex. Indeed AIN may occasionally occur in patients with preexistent renal disea-ses and may therefore result in an acute exacerbation of pre-existing renal failure.

Frequency

The real incidence of AIN is unknown. Renal biopsy or autopsy findings are necessary to document the diagnosis; the incidence of AIN in patients with acute renal failure undergoing renal biopsy ranged from 8 to 14 %.

Etiology

In etiologic aspect we can divide AIN in two main events or parts:

A/ Drug-induced AIN

B/ AIN associated with systemic infections

1. *In the first group are included:*

- Antibiotics
- Diuretics
- Sulfonamides

- Anticonvulsive drugs
- Non-Steroidal Antiinflammatory Drugs /NSAID/.

Systemic infections associated with AIN, more recently are:

- Septicemia
- Leptospirosis.

Pathomorphology

- Analysis of the renal cell infiltrate may provide an inside into the pathogenetic mechanism involved in AIN and may afford schematic classification. AIN due to bacterial infection is characterized by massive infiltration of polymorphonuclear neutrophils, whereas in drug-induced AIN, the infiltrate is composed of:
  - either mononuclear cells
  - lymphocytes
  - plasma cells
  - eosinophils

However, in AIN related to viral infection, the interstitial infiltrate consists of mononuclear cells; eosinophils may predominate in some cases of idiopathic AIN. Thus, characterization of the interstitial cells affords useful but not definitive information on the cause of AIN. More precise typing of lymphocytes with monoclonal antibody may provide additional clues to the pathogenesis.

Except for extrarenal symptoms characteristic for certain causes, the clinical renal features of AIN are nonspecific and may mimic those of acute tubular necrosis. The patients may be oliguric or nonoliguric. Kidneys are of normal size or enlarged if cell infiltration is diffuse and profuse.

Lab Studies:

- *Eosinophils* in AIN can be activated and lose some of their typical granules, including the major basic protein. Increa- sed urinary levels of this protein were found in AIN.
- *Proteinuria* is usually not detected. It is not uncommon, however, to find moderate proteinuria ranging from 0,5 to 2 g per day.
- *Hematuria.* The proteinuria is frequently associated with microscopic hematuria, more rarely with gross hematuria. It was thought that gross hematuria and the nephrotic syndrome were mainly restricted to drug-induced AIN.
-

B/ Students' work with a patient.

C/ **Discussion of the case.**

Diagnosis.

Excluding typical cases, it is sometimes difficult to put right diagnosis.

About it **is typical:**

1/ The nephritis develops several days or weeks after the initiation of therapy.

2/ The disease is not dose-dependent.

3/ It is associated with:

- fever
- skin rash
- arthralgias
- sometimes liver involvement
- gross hematuria
- blood eosinophilia.

4/ Often it is associated with acute renal failure.

Only early renal biopsy shows the true diagnosis!!!

Treatment

- *Medical care:*

All the patients with acute interstitial nephritis should be treated in a hospital. It is observed diuresis and generally condition of patient.

- *Medication:*

However, to stop preceding therapy is sufficiently.

In the difficult cases it includes corticosteroid medicaments, in various doses - 1 to 10mg/kg per day:

1. Methylprednisolon – 5 to 10mg/kg per day, for 3 to 5 days – intravenous way.
2. Prednisolon. – 1 to 2mg/kg per day- single dose- morning, for 7 to 15 days.

Often, the cases with acute renal failure /ARF/ are treated with *hemodialysis*.

Prognosis

The prognosis *is good as whole*, with almost 100% survival. The rehabilitation of renal function depends on the therapy, preceding state, and the age of patients.

CHRONIC INTERSTITIAL NEPHRITIS

The chronic interstitial nephritis in etiologic aspect also may to divide in two parts:

1/ Drug-induced

2/ Different causes

In second group is examined many factors as:

- immunologic diseases
- metabolic disorders
- infection
- hemopoetic diseases
- endemic diseases
- idiopathic diseases, etc.

Pathologic morphology:

In the chronic form, *interstitial fibrosis* and *tubular atrophy* are present, and *the cellular infiltrate is uniformly mononuclear*.

Etiologic agent affects different parts of the kidney and the nephron at the beginning. For example:

for Balkan nephropathy are typical the *cortical lesions*
for analgesic nephropathy *medullar lesions* are typical
the heavy metals damaged the *proximal tubule*
the hypercalcemia- *distal tubule.*

B/ Student's work with a patient.

C/ Discussion of the case.

Diagnosis.

For chronic interstitial nephritis **is typical**:

1/ Symmetrical damages.

2/ Early reduction of concentration capacity.

3/ Renal tubular acidosis.

4/ Low rate of proteinuria.

5/ Earlier anemic syndrome.

Treatment

In the most of cases, the treatment is limited to a termination of the exposition of the agent.

- *Symptomatic*

1. The symptomatic treatment with a diet and vitamins is sufficiently until Chronic Renal Failure.

2. Diuretics:

- *Furosemid /Furanthril*/ - tabl. 40mg/ amp. 20mg / - 40 to 200mg per day.
- *Chlortalidone/ Saluretin/* - tabl.100mg – 25 to 100mg per day.

3. Antihypertensive drugs:

- ACE-inhibitors*:*

Captopril – tabl. 25 and 50 mg; 25 to 50mg – three or four times a day.

Enalapril maleas – tabl. 5 and 10 mg; maximal dose-40 mg a day.

- Blockers of calcium canals:

Nifedipine – tabl. 10 and 20 mg /retard form/ - 30 to 80mg a day.

Diltiazem – tabl. 30, 60 and 90 mg; dose - 240 mg a day.

Up to the moment, there is a certain lack of a special therapy that probably would improve the prognosis of the disease.

As a whole, the prognosis of the disease is relatively optimal one, considering the survival.

The ills come to Chronic Renal Failure more slowly and it slowly progresses in cases of chronic tubulointerstitial nephro-pathies.

Exceptions are the cases of an existing cancer or serious fundamental disease.