LESSON 16

Glomerulonephritis in children. Dysmetabolic nephropathies in children

1. SIGNIFICANCE

Glomerulonephritis is the name given to a range of conditions that can affect the kidney, specifically the glomeruli of the kidney. The glomeruli become damaged, commonly because of a problem with the body's immune system. Many people with glomerulonephritis may not notice any symptoms initially. However, salt and excess fluid can build up in the body if the glomeruli and kidneys are not working normally. This can lead to complications such as high blood pressure and, in some cases, chronic kidney disease, which may lead to end-stage kidney failure. Treatment will depend on the underlying cause as well as the severity of symptoms.

Dysmetabolic nephropathies are kidney disease due to metabolic disorders in the child's body. There are primary and secondary nephropathy. Basically dysmetabolic nephropathy arise from disorders of calcium metabolism, excess oxalic acid, phosphates, oxalates or urates. There are mixed nephropathy.

2. PREREQUISITES

The skills listed below will not be taught in this lesson but are necessary to perform physical examination of the patient at nephrology department and the intensive care unit during practical training. Therefore, before beginning this lesson, one has to be sure of the ability to:

- Identify abdominal or flank mass;
- Examine skin for pitting edema;
- Measure blood pressure using a standard technique;
- Percuss and auscultate the heart to recognize arrhythmia or cardiac hypertrophy, stemmed from electrolyte disturbances and systemic hypertension;
- Elicit Pasternatsky sign in older children;
- Perform gross examination of urine;
- Read and comment the results on the routine urinanalysis;
- Read and comment the results on the routine blood biochemistry.

3. EDUCATIONAL OBJECTIVES

Student should know:
- etiology, pathology, classification, clinical presentation, differential diagnosis, treatment and prognosis for in children.

Student should be able:
- to identify the child with glomerulonephritis and dysmetabolic nephropathies, make correct decisions during physical examination of the patient with given conditions, take appropriate actions based on those decisions, demonstrate skills to develop management and follow up measures.

4. INTERDISCIPLINARY INTEGRATION

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### 5. ABSTRACT FOR PRE-WORKSHOP SELF-EDUCATION

**5.1. Glomerulonephritis (GN) in children.**

Glomerulonephritis it is heterogenic group of diseases with primary glomerular localization of pathological changes. It is immunologically mediated diffuse inflammatory disease which involves both kidneys symmetrically affecting mainly the glomerulus and associated with changes in tubules, interstitial tissue and vessels.

**Etiology of Glomerulonephritis:**

- Beta-hemolytic streptococci of group A (strains 1, 4, 8, 12, 49). The streptococcal infection is most often in the respiratory tract (pharyngitis, sinusitis, tonsilitis), but infections of other sites (skin and middle ear) may also proceed nephritis (acute GN)
- infections – immunologic factors (infections hepatitis H B3 Ag positive, viruses, rickettsias, mycoplasms)
- noninfectious – immunologic factors

**Pathogenesis of Glomerulonephritis:**

- The two main processes are involved in the pathogenesis of glomerulonephritis.
- **Autoimmune**: antibodies (antiglomerular basement membrane) react with an antigen in the glomerular basement membrane and produce glomerulonephritis (5% cases).
- **Immune complex theory.**

Streptococcal or other antigens provoke antibody response, and the subsequent antigenantibody complexes in the circulation are deposited in the glomerular cappillary walls. These complexes activate the complement pathway with the
liberation of chemotactic factors causing polymorpho-leucocytic infiltration the release of lysosomal enzymes from neutrophils and the direct effect of the complement system lead to damage of the capillary wall including the glomerular basal lamina.

**Clinical features of AGN (nephritic syndrome).**

Typical clinical picture is presented now rarely.

- A latent period of from 5 days to 4-6 weeks occurs between the streptococcal infectious and the abrupt or acute onset of nephritis
- Sings of intoxication (fatigue decreased appetite)
- Edema (periorbital, leg or sacral edema or generalized due to salt and water retention)
- Mild or severe hypertension (headaches, visual disturbances secondary to hypertension, rarely hypertensive encephalopathy may be the presenting complains of AGn)
- Sings of left ventricular failure (orthopnoe, breathlessness, 3achycardia)
- Renal impairment manifesting as oliguria or acute renal failure
- Dark urine (cola-colored urine)
- Changes on the retina (spasm of arteries, dilatation of veins, hemorrhages)
- Eclampsia due to cerebral edema and hypertension
- Sometimes the onset of the disease may be insidious with weakness, fatigue and malaise or mild edema as the most prominent symptoms after the history of a sore throat, respiratory disease or other
- In such situation urinalysis should be prescribed
- A history of streptococcal or other infection 1-4 weeks prior to onset of erythrocytorea, proteinurea with development of edema or hypertension are patogonomic of Acute glomerulonephritis.

**Syndromes in GN:**

- **Urinary syndrome** (proteinuria (less than 3 gm day), RBCs and casts in the urinary sediment)
- **Nephritic syndrome** (abrupt onset of hematuria, proteinuria (usually associated with non-nephrogenic range), casturia, oliguria, hypertension)
- **Nephrotic syndrome** (proteinuria more than 3.5 gm/day, hypoproteinemia and hypoalbuminemia, severe adema, hyperlipidemia)
- **Edema** (mostly is locaried on the face (periorbital), is pale, warm, appears in the morning than decreased, in the second part of the day develops on the legs)
- **Hypertensive syndrome** (hypertension is hyperkinetyc and not severe):
  - Hypertensive stage: complains on headache, disturbances of vision, insomnia; objective examination reveals high blood pressure, hypertrophy of left ventricular, signs of heart failure, cerebral and cardiac complications
  - Stage of chronic renal failure signs and symptoms according to the stage (I - IV).

**Nephrotic syndrome (NS)** - clinical and laboratory syndrome which includes:

- **Proteinuria** more than 1 g/m2 24 hours (3.5-4 g/ 24 hours ),
- Hypoproteinemia with hypoalbuminemia less than 25 g/l, hyper-alfa-2-globulinemia,
- hyperlipiduri, lipiduria,
- edema.

**Complications of NS**
- **nephrotic crises**
  - severe pain in abdomen, associated with peritoneal symptoms,
  - fever,
  - oliguria and look like thrombosis of mesenterial arteries and need urgent consultation of surgeon),
  - skin symptoms migrate erythema

**Laboratory findings**

- Urine analysis
  - the urine may be scanty, brown, smoky of frankly bloody.
• From 0.5 to 30 gr/day of protein excreted
• The urinary sediment contains RBCs, RBC cast (are the pathognomic of glomerulitis from any etiology)
• WBC, renal tubular cells, WBC cast and granular (protein droplets) casts are also may be common
• Urinanalysis Nechyporenko (more than 50 000 RBCs in 1 ml of urine is named as hematuric component)
• Blood analysis (mild anemia (due to hypervolemia), mild leucocytosis, lymocytesis, increased ESR)

**Laboratory findings biochemical blood analysis:**
• (hypoproteinemia and hypoalbuminemia, hyperlipidemia (hypoalbuminemia triggers increased synthesis of all forms of plasma proteins including lipoproteins resulting in hyperlipidemia), elevated level of antistrepolysin-titre (more than 1:3000);
• serum complements levels (C3, C4 and the total hemolytic activity) are usually diminished during the active phase of the disease (returns to normal at 6-12 weeks);
• serum urea, creatinine may be elevated due to digurea and creatinine clearance reduced;
• hypercoagubility may result from
  – increase urinary loss of antitrombin III
  – altered levels and/or activity of protein C
  – hyperfibrunogenemia due to increased hepatic synthesis
  – impaired fibrinolysis
  – increased platelet aggregability

**Instrumental investigation**
• Renal biopsy usually required for diagnosis in adults
• Ultrasound examination may show enlarged kidneys

**Pareculiaritis** of clinical and laboratory signs of CGn according to morphologic changes in kidneys
• Mesangiproliferative Gn (Ig A nephropathy) – isolated urinary syndrome, nephritic syndrome, hematuria in adults
• Mesangiocapillary Gn – nephrotic syndrome, urinary syndrome with hematuric component, hypertension
• Membranosus Gn – nephrotic syndrome (80%) in age 40-50
• Focal and segmental Gn - nephrotic syndrome, hypertension in Afro-Americans
• Minimal change disease - nephrotic syndrome in children
• Fibroplastic Gn - nephrotic syndrome (50%), hypertension, chronic renal failure

**Differential diagnosis**

**Urinary syndrome**
• Acute pyelonepritis
• Activation of primary chronic glomerulonephritis
• Toxic nephritis
• Goodpasture’s Syndrome
• Hereditary nephritis (Alport’s Syndrome)

**Nephrotic syndrome**
• Amyloidosis
• Diabetic nephropathy
• Colagenic nephptopathy (SLE scleroderma)

**Hematuric component**
• Malignancy associated nephritis
• Urotuberculosis
• Renal stones

An example of diagnosis
Acute glomerulonephritis, urinary syndrome, hematuric component.
Acute glomerulonephritis, nephrotic syndrome.
Chronical glomerulonephritis, urinary syndrome, hypertensive stage, phase of activation.

Duration of acute glomerulonephritis
• Recovering during first 2-4 weeks or 2-3 month
• Prolonged duration (duration more than 4 month, full recovering is 2-3 times rare)
• Negative prognostic feature is nephrotic syndrome, associated with severe hypertension
• Development of chronic glomerulonephritis (urinary syndrome, edema or hypertension are present more than 12 month)

Complications of acute glomerulonepherts
• Eclampsia (angiospastic encephalopathy)
• Acute heart (left ventricular) failure
• Acute renal failure

Treatment of acute glomerulonephritis
Acute glomerulonephritis have to be treated only in specialised nephrologic department
– Regimen: bed-rest during 2-4-6 weeks until desappearing of edema and normalizing of blood pressure
– Diet № 7a
– Daily record of fluid intake and output
– Restriction of dietary protein if azotemia and metabolic acidosis are present
– Salt free diet (Sodium intake is restricted only when circulation overload, edema, or severe hypertension is present)

The aim of drug therapy is recovering of the patient
• Anti microbial drug
• Symptomatic therapy
• Membranenostabilizative therapy
• Pathogenetic therapy

Antimicrobial therapy
• If a bacterial infection is still present when nephritis is discovered, it should be treated with an appropriated antimicrobial drug
• Semisynthetic penicillins in middle therapeutic doses have to be prescripted

Symptomatic therapy
Edema
• Loop diuretics such as furosemide or lasix (40-400 mg/day or 1-2 gr/day) help in the management of the expanded extracellular fluid volume (side effects: hypocholremic alkalosis, decreasing K, Na level in blood)
• In patients with decreased of furosemide should be prescribed uregit (50-200-500 mg/day orally) or the combination with the thiazides (hypothiazide 25-100 mgm/day)
• 2.4 % solution euphylline 10 ml i/v
• Albumin may help in the management of hypoproteinemia
• Daily weighting to check change in the body fluid status and record of fluid intake and output have to be made in patients which receive diuretics

Hypertensive syndrome
Antihypertensive drug therapy is usually started with single drug, but if there is incomplete response a second drug is added.
One of the following drugs as a single drug treatment can be used:
• ACE inhibitors (or angiotensine II receptors blockers)
• (loop) diuretics
• calcium channel blockers (non dihydropiridine agents)

If single drug treatment is unsuccessful then the combination therapy may be given as two-drugs or three – drugs therapy

Two – drug therapy:
• calcium channel blocker + diuretic
• ACE inhibitor + diuretic

Triple – drug therapy is used very rare
• calcium channel blocker + diuretic + ACE inhibitor

Such drugs as adelfan or trirezide (which contained fixed doses of several hypotensive drugs) are not good in therapy of hypertensive syndrome

Hematuric component
• Dicinon (etamsilate) 2 ml 12.5% solution twice a day (7-10 days) i/m, then 0.25-0.5 three times a day orally
• Kvarcetin 1.0 in a half of glass of water three times a day.
• Ascorbinic acide 500 mg a day.
• Ascorutine 1 tabl. three times a day
• Rutine and other.

Membranostabilizative therapy
• Have to be prescribed in patients with AGn, urinary syndrome, hematuric component, after prescription of symptomatic therapy.
• Unitiol (5 ml 5% solution i/m during 1 month)
• Dimephosphon (100 mg/kg/day 1 month)
• Aminochoinolytic drugs (delagil – 0.25 two times a day orally 1 month, then 0.25 a day during 5-12 month)
• (side effects: leucopenia, degeneration of retina, allergy, dyspepsia)
• á-tocoferol (50 mgm/day – 5-12 month)

Pathogenetic therapy have to be used in patients with:
• Gnn, nephrotic syndrome after 3-4 weeks from the beginning of the disease, when symptomatic and membranostabilisativetherapy is unsuccessful

Pathogenetic therapy includes:
• Glucocorticoids
• Cytostatics
• Anticoagulants and antiagregative drugs

Glucocorticosteroids
– Prednisolone 1 mg/kg/day for 4-6 weeks followed by decreasing of dosage on 2.5 mg each 5-7 days
– In patients with high activity of patogenetic process pulse-therapy with metyprednisolone (metipred, soly-pred, solu-medrol) (1000 mg/d three days) can be used and then therapy in previous doses
– (Side effects: obesity, hirsutism, disturbances of menstrual function, acne, Cushing syndrome, ulcers of alimentary tract, hyperglycemia, hemorrhagic, pancreatitis, psychiatric disturbances.
– After abrupt discontinuiong of the drug usage can be worsening of the duration of the main disease).

Cytotoxic drugs
• Cytotoxic drugs should be given in refractory cases or if glucocorticosteroids are contraindicated.
• Cyclophosphamide (1.5-2 mg/kg/day), imuran (2-3 mg/kg/day), leukeran, chlorbutin (0.2 mg/kg/day), cyclosporn A (sandimun) or others are given for 4-6 weeks at
the nephrologic department and then 4-6 months at home under the control of blood analysis each 7 days.

- Pulse-therapy of cytotoxic drugs (1000-1200 mg of cyclophosphane i/v once a month 5-6 times) at specialized nephologic department can be used
- (Side effects: cytopenia, dyspepsia, hemorrhagic cystitis, toxic hepatitis, sexual dysfunction, infertility)

**Anticoagulants and antiagregants**

- Direct antiocoagulants (fraxiparine 0.3-0.6 ml/day subcutaneous 10-14 days, heparin 5000-10000 subcutaneous 4 times a day 1-1.5 month (under the control of time of blood coagulation or time of bleeding) then gradual decreasing of the dose during 1 week)
- Side effects: hemorrhages, allergy.

- Non – direct antiocoagulants (pheniline 0,045 – 0,06/d 1 – 2 month)
- Antiagregative therapy (curantyl 200 – 400 mg/d, trental 600 mg/d 2 – 6 month)

In patients with high activity of pathologic process 4-component therapy have to be used (glucocorticosteroids, cytotoxic drugs, antiocoagulants and antiagregants simultaneously)

**Treatment of eclampsia**

- i/m: 25% solution of magnesium sulfates 10 ml 2-4 times a day;
- 1 ml of 25% solution of aminazine;
- 10 ml of 2,4% solution of euphylin;
- 80-120 mg of furosemide;
- 30 ml of 40% glucose solution.

**Instrumental methods of treatment.**

*Indications*: side effects or nonefficasy of pathogenetic therapy

*Contraindications*: level of the Hb less than 80 gm/l, hypotension, leucocytopenia, thrombocytopenia, allergy on protein preparations, hemorrhagic complications, ulcer disease.

**Types:**

- Plasmapheresis (may be safer and more effective when high titers of anti – GBL antibodies are present in the case of fulminant immune complex disease)
- Hemosorbtion
- Lymphosorbtion

5.2. Nephropathy kidneys in children (dysmetabolic nephropathy).

It is a kidney disease due to metabolic disorders in the child's body. There are primary and secondary nephropathy.

Primary nephropathy is caused by hereditary factors. In this case there is a progressive course, early development of urolithiasis and chronic renal failure. This form of kidney disease is quite rare.

Secondary dysmetabolic nephropathy associated with the intake of certain substances, in this case violated their exchange due to dysfunction of some organs and systems. Violation of the functions of the digestive system, for example, may be related to certain medications.

Basically dysmetabolic nephropathy arise from disorders of calcium metabolism, excess oxalic acid, phosphates, oxalates or urates. There are mixed nephropathy.

**Features of dismetabolic nephropathy**

**Calcium oxalate nephropathy**

Is most often occurs in children. The reason - in violation of calcium metabolism and oxalic acid salts. Oxalate enter the body with food, or synthesized in the organism.

oxalates formed as a result of the impact of the following factors:

- Elevated levels of oxalates in the diet.
- bowel diseases, including hereditary (Crohn's disease), ulcerative colitis, and others.
- increased oxalate synthesis by the body.

**Hyperoxaluria**
In this case, apart from the genetic factor plays an important role environmental influences, stress, nutrition, physical stress. The disease can occur at any age, even in infants. The general condition and development of children with this form of kidney disease do not usually changed, but they are characterized, as a rule, such metabolic disorders as obesity, allergies, IRR, and headaches. Aggravation of the disease can occur at puberty and hormonal changes of the body. Progressing, renal oxalate nephropathy in children can lead to the formation of kidney stones, the accession of infection and the appearance of inflammation of the kidneys.

**Phosphate nephropathy**
The main reason for this form of kidney nephropathy in children is a violation of the calcium-phosphorus metabolism and chronic infectious process in the kidneys.

**Urate nephropathy**
It causes disturbances exchange urate (uric acid). Crystals of uric acid salts are deposited and accumulate in kidney tissue, impairing their function. Urine in the accumulation of urate becomes saturated brick color.

**Cystine nephropathy**
This form of kidney nephropathy in children is associated with a genetic defect. Cystine is an amino acid metabolite methionines. It is noted an excess accumulation of cystine and violation of reabsorption in the renal tubules processes.

Progresses, the disease leads to kidney stones and inflammation of the kidneys due to the accession of the infectious process.

**Diagnosis and treatment of nephropathy**
For detection of the disease is carried out:
1. ultrasound of the kidneys;
2. urine clinical in identifying salt crystals;
3. Testing AMCEN, calciphylaxis.

Treatment should be comprehensive and aimed at the implementation of the following rules:
- Healthy lifestyle.
- Nutrition and drinking regimen.
- Diet.
- Specific therapy.

Increasing fluid intake, thereby reducing the concentration of solutes in the urine. In addition, the targeted volume increase nocturnal urination due to fluid intake before bedtime. In this case it is best to drink mineral or just pure water. Reduced salt load on the kidneys as a result of the appointment of diet.

When oxalate nephropathy kidney in children excluded meat broth, sorrel, carrots, cocoa, chocolate, cranberries, beets. It is recommended to use a potato, cabbage, dried apricots, prunes, pears. Assign reception of mineral waters (Slavyanovskaya, Smirnoff). Assign vitamins A, E, B6, magnesium preparations.

Assign the preferred dairy and vegetable diet. Limited consumption of meat products, beans, nuts, cocoa. The important point is sufficient fluid intake (up to two liters per day). Mineral water should be drunk slightly alkaline. Preferably drink teas horsetail, dill, knotweed, birch buds. Good help from the broth of oats. Under the strict supervision of prescribed allopurinol and nicotinamide, Phytolysinum.

When renal phosphate nephropathy in children prescribed mineral water Narzan, Dzau-Soir. From preparations: tsiistinal, vitamin C, methionine. Limit the consumption of cheese, biscuits, chocolate, caviar.

Cystine nephropathy treated with diet, including the limitation or exclusion from the diet of fish, eggs, meat, cheese. Mineral water is desirable to consume alkaline. It is important to consume at least two liters of fluid. It is useful to drink a lot before bedtime.
Long penicillamine treatment is carried out, as well as its less toxic analogue kuprenilom. Assign vitamins A, E. In addition, when cystinosis successfully used kidney transplantation, which should be carried out at a young age, preferably before 18-19 years. However, there is a risk of the transplanted kidney as well.

Prognosis of kidney nephropathy in children is usually favorable in compliance with all standards of treatment and prevention of exacerbations of the disease. Preventive measures are directed generally to maintain and respect a strict diet, routine checkups and laboratory and instrumental methods of investigation of kidney function, conducting urine tests. Carry out the spa treatment and rehabilitation of children. It is advisable to visit the mineral springs resorts in Morshyn, Truskavets, etc.

6. MATERIALS FOR METHODOLOGICAL BACKGROUND OF THE WORKSHOP

6.1. Quiz

1. Definition of acute glomerulonephritis.
2. What is the most common etiology for acute glomerulonephritis?
3. What is epidemiology of an acute post-streptococcal glomerulonephritis?
4. Define acute post-streptococcal glomerulonephritis?
5. List symptoms and signs of an acute post-streptococcal glomerulonephritis.
6. What are investigations in acute post-streptococcal glomerulonephritis?
7. When is renal biopsy indicated in acute post-streptococcal glomerulonephritis?
8. What are complications of acute post-streptococcal glomerulonephritis?
9. What diet and lifestyle should be followed by patient with acute post-streptococcal glomerulonephritis?
10. List the treatment aims for acute post-streptococcal glomerulonephritis?
11. What is pharmacologic treatment for an acute post-streptococcal glomerulonephritis?
12. What is the normal cause of an acute post-streptococcal glomerulonephritis?
13. What do you know over the prognosis of acute post-streptococcal glomerulonephritis?
14. What are follow-up measures for acute post-streptococcal glomerulonephritis?
15. Definition of acute dysmetabolic nephropathy.
16. What is the most common etiology for dysmetabolic nephropathy?
17. What is epidemiology of dysmetabolic nephropathy?
18. List symptoms and signs of dysmetabolic nephropaty.
19. What are investigations in dysmetabolic nephropaty?
20. What are complications of dysmetabolic nephropaty?
21. What diet and lifestyle should be followed by patient with dysmetabolic nephropaty?
22. List the treatment aims for dysmetabolic nephropaty?
23. What is the normal cause of dysmetabolic nephropaty?
24. What do you know over the prognosis of dysmetabolic nephropaty?
25. What are follow-up measures for dysmetabolic nephropaty?

6.2. Multi-choice questions

A 17-year old girl comes to hospital with his mother with complaints of pain in loin region, appearance of blood in urine. Her mother tells that he was suffering from viral pharyngitis 2 weeks ago. A physical examination reveals mild edema in lower extremities, blood pressure is 125/75 mm Hg, pallor and slightly lethargic. Laboratory investigations reveals positive antistreptolysin-O titer, urinalysis shows hematuria, proteinuria and leukocytes. Microscopic examination reveals dysmorphic RBCs and RBC casts. What is the diagnosis?

A. Acute post-streptococcal glomerulonephritis*
B. IgA nephropathy
C. Alport disease
D. Hemolytic uremic syndrome
E. Urinary tract infection

6.3. Sample case report
A 8 years old boy 2 weeks ago was admitted to hospital due to palpable purpuric rash, mild periorbital edema and “cola like” color of urine and BP 120/75 mmHg. His GFR was 85 ml/min. In anamnesis: 3 weeks ago appeared rash on posterior surface of legs and buttocks. Today patient complains on severe headache, vomiting, nose bleeding and weakness. On physical examination: patient is pale, has severe periorbital and peripheral edema, BP is 130/90 mmHg. In laboratory investigation: Blood analysis show leucocytosis with mild anemia and elevated ESR. BUN and creatinine levels are markedly elevated. Urinalysis shows modest proteinuria, microscopic hematuria, RBCs, and RBC and WBC casts. GFR is 30 ml/min. A renal biopsy specimens show a diffuse, proliferative, necrotizing glomerulonephritis with crescent formation.

1. What is the diagnosis?
2. What is the differential diagnosis?
3. What is the follow-up?

Suggested reading

Additional reading