DIAGNOSIS AND TREATMENT OF GOUT NEPHROPATHY

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Abstract: Gouty nephropathy is a collective concept that includes the entire renal pathology observed in gout; tofuses in the kidney parenchyma, urate stones, interstitial nephritis, glomerulonephrosis and arteriosclerosis with the development of nephrosclerosis. Tubular tofuses are formed in 50%, and urate stones in the pelvis in 10-25% of patients. Both processes create .conditions for urinary tract infection. A distinctive feature of gout is kidney damage - interstitial nephritis (due to the widespread deposition of urates in the interstitial tissues of the kidneys). Kidney damage can occur many years before the joint syndrome!

Laboratory tests. The most important for the diagnosis and treatment of gout is the study of uric acid metabolism: the content of uric acid in blood serum, in daily urine and the determination of uric acid clearance.

During the attack, ESR increases in patients with gout (usually 25-40mm / h), moderate leukocytosis, a positive reaction to C-reactive protein and other indicators of the acute phase of inflammation may be observed. In the intercrime period, these indicators are normal, but in the presence of urate arthropathies, they may be weakly positive.

According to experts of the European Anti-Rheumatic League, the "gold standard" for the diagnosis of gout is the detection of sodium monaurate crystals in synovial fluid or in the contents of tofus, which reflects the pathogenetic essence of the disease. On ultrasound of the kidneys, it is possible to identify stones in the cup-pelvic system, but the deposition of uric acid in the pyramids is characteristic of the gouty kidney, which is manifested by their sclerosis.

In the presence of a classic picture of gout with a typical localization of the process in the I metatarsophalangeal joint, a rapid increase in symptoms of acute arthritis and its complete reverse development in a few days, suspicion of the possibility of developing this disease (especially in men) may arise already in the early period of the disease after 1-2 attacks. The diagnosis is confirmed by the detection of hyperuricemia, rapid relief of the attack with colchicine, especially the detection of sodium urate crystals in the synovial fluid.

In 1963, at the International Symposium on the Diagnosis of RB in Rome, the criteria for the diagnosis of gout were developed:

1. Increased uric acid content in blood serum (more than

0.42 mmol/l for men and 0.36 mmol/l for women),

2.Tofuses,

3.Sodium urate crystals in synovial fluid or in

tissues, detection by microscopic or chemical studies

4. Acute attacks of arthritis occur suddenly, with complete remission within 1-2 weeks.

A. Every patient with gout should be fully informed

about the pathophysiology of the disease, the availability of effective treatment methods, comorbid diseases, the principles of therapy for acute

arthritis attack and elimination of urate crystals by lifelong lowering of the serum uric acid level below the target level

B. Every patient suffering from gout should receive lifestyle recommendations: weight loss if necessary, refusal of the intake of alcohol (especially beer and strong alcoholic beverages) and sweetened beverages, the exclusion of overeating, excessive consumption of meat and seafood. Should be encouraged

consumption of low-fat dairy products. Regular physical exercises should be recommended

. Every patient with gout should be systematically screened for the detection of comorbid diseases and cardiovascular

risk factors, including renal failure, coronary

heart disease, heart failure, stroke, atherosclerosis

of peripheral arteries, obesity, hyperlipidemia, hypertension,

diabetes and smoking, the prevention and therapy of which should be considered as an integral part of the treatment of gout

The treatment has the following objectives:

I) stop the attack with anti-inflammatory drugs,

2} avert relapses of acute attacks (if they are too frequent with daily prophylactic intake of colchicine,

3) to prevent further complication of urate crystals and eliminate already existing topuses) that will give. by reducing the concentration of urates in the body fluids.

It is also necessary to prevent kidney damage and possible disability due to the erosion of bones and joints, cartilage.

Specific therapy is determined by the stage and severity of the disease. In addition, it is necessary to treat hypertension, hyperlipidemia and obesity associated with gout.

Drugs that reduce the level of uric acid in the blood should be prescribed only after the acute inflammation subsides, against the background of daily intake of colchicine. This is due to the fact that hypouricemic agents in the first weeks of their use can lead to acute attacks of gout, these attacks often occur during a decrease in the level of uric acid in the blood. With prolonged use of colchicine, nephropathy or myopathy may develop. Side effects: nausea, persistent diarrhea.

Contraindications: HF, CRF, YABZH, pregnancy.

With hypurekemic therapy, the level of uric acid should be periodically determined, monitoring the effectiveness of treatment. The choice of a specific drug and its dosage should ensure a significant reduction in the concentration of uric acid in the blood. It may take months or years for topuses to dissolve. From uricosuric agents, probenecid is prescribed (table 0.5 g) or sulfinpyrazone (table 100 mg). The optimal dose is selected so as to maintain the concentration of urate in the blood at a normal level. The starting dose of half a tablet 2 times a day is gradually increased to 4 tablets. in the day. Sulfinpyrazone has a stronger uricosuric effect than probenecid, but is more toxic. Salicylates counteract the uricosuric effect of both drugs, so they should be avoided; acetaminophen is used as an analgesic.

Inhibition of uric acid synthesis caused by allopurinol at a dose of 200-600 mg / day (several doses) also provides a decrease in its level in the blood. In addition to blocking the enzyme (xanthine oxidase), responsible for the synthesis of uric acid, allopurinol also suppresses the excessive synthesis of purines. This is of particular importance in urolithiasis (caused by the deposition of urates) and in significant violations of kidney function. With the help of allopurinol, it is possible to achieve the dissolution of already formed uric acid stones. Side effects may be minor disorders of the gastrointestinal tract, skin rash and leukopenia. In cases where this is achieved, extracorporeal ultrasound lithotripsy can be used. It is necessary to adhere to the following basic principles in allopurinol therapy:

1. It is not recommended to start therapy with allopurinol in the presence of an acute articular attack, it is necessary to completely stop the articular syndrome.

2. It should be remembered that during an attack of arthritis, the level of uric acid is usually lower than in the intercrime period, therefore, repeated studies of its level are required after the implementation of arthritis.

3. Start therapy with a small dose (more often 100 mg per day and up to 300 mg).A reflection of the correct selection of the dose of the drug is the rate of decrease in the level of hyperuricemia- no more than 0.6-0.8 mg% or 0.1-0.6 mg \ dl or 10% of the initial figures during 1 month of therapy.

4. It is generally accepted to adapt the dose according to creatinine clearance: at a level below 30 ml / min, it is advisable to reduce the dose of allopurinol to 50-100 mg per day.

5. To prevent an acute attack at the beginning of taking allopurinol, it is possible to use low doses of colchicine or NSAIDs.

6. When canceling allopurinol, the uric acid level increases rapidly (3-4 days

Auxiliary methods of treatment.

Additional fluid intake (3 liters per day) is useful for all patients with gout, especially with a tendency to form uric acid stones in the urinary tract. In these cases, it is also recommended to alkalize urine with sodium bicarbonate or trisodium citrate (5 g x 3 p. per day Hypouricemic agents so effectively reduce the level of uric acid in the blood that strict restriction of purines in the diet is not required. During the intercrime period, obese patients need to take measures

to reduce body weight. Large tofuses can be surgically removed, while others (with the exception of those whose shell has undergone significant fibrosis) usually gradually dissolve against the background of adequate preventive therapy.

Idiopathic hyperurcemia.

The question of specific treatment for asymptomatic hyperuricemia, not accompanied by gout, has been little studied. I believe that the daily use of probenecid or sulfinpyrazone is indicated only to persons under 40 years of age who have persistent hyperuricemia (> 9mg%), provided that the daily excretion of urates in urine is normal, if it is elevated, allopurinol should be prescribed.

In recent years, the use of calcium antagonists in the treatment of gout has been described in the literature. Of these, corinthar increases the excretion of uric acid in the urine, and the more the lower the tone of the kidney vessels.

Physiotherapy and spa factors in the treatment of gout.

An integral part of the comprehensive treatment of patients with gout is physical factors.

The purpose of physiotherapy is to help the dissolution of tissue uric acid compounds and their excretion by the kidneys and sweat glands, which leads to improved blood circulation and metabolic processes in the joints and periarticular tissues, the ability to eliminate pain, as well as an increase in the volume of movements in the affected joints.

Methods of physiotherapy are diverse:

1. Ultrasound has analgesic, desensitizing and anti-inflammatory effects.

2. Induction therapy, ultrasound and infrared irradiation, diadynamic (DDT) and sinusoidal modulated currents (SMT), electrophoresis of medicines, the use of ozokerite, paraffin, therapeutic mud also have anti-inflammatory, analgesic effects improve blood circulation, oxidative. regenerative processes, stimulate compensatory, adaptive, protective mechanisms.

Spa treatment is a powerful factor in primary and secondary prevention, treatment and rehabilitation of rheumatological patients, including patients with gout.

In the intercrime period, with intermittent and chronic gout, patients are prescribed sanatorium-resort treatment.

Radon and hydrogen sulfide baths, which have a general effect on the course of the disease and blood circulation, contribute to the improvement of metabolic processes, enhance the excretion of uric acid compounds by the kidneys and skin. Radon baths normalize purine metabolism, which is expressed in a decrease in the absorption and excretory function of liver parenchymal cells, favorably affect lipid metabolism, reducing the content of total lipids and cholesterol in the blood.

Hydrogen sulfide baths enhance microcirculation, as well as trophic processes in joint tissues, increase the secretory function of the synovial membrane, improve cartilage nutrition, have a stimulating effect on enzymatic systems, metabolic and immunological processes.

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