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Hyperuricemia, A Common Metabolic Condition with an Increased Cardiovascular Risk

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In recent years, the prevalence of gout has increased, especially in Western societies, with the clinical profile of this disease becoming more and more complex, due to the modern lifestyle, and the usual multiple comorbidity, which it presents.

Gout is a common metabolic disease characterized by the deposition of urinary monosodium crystals in the joints and extraarticular tissues. Uric acid is the end product of the metabolism of purine compounds. Purines are specific amino acids found in a variety of foods, especially foods of animal origin.

Purines are not dangerous or harmful to health, but when the body breaks them down, uric acid is produced. The metabolic disorder responsible for gout is hyperuricemia. There is no commonly accepted definition of hyperuricaemia. As a definition, we recommend any serum uric acid value greater than 6mg / dl. This value seems to be the limit above which we will probably have the appearance of clinical symptoms of Gout.

Gout is mainly expressed as

1. Asymptomatic hyperuricaemia
2. Gout
- Acute gout
- Interval of crisis chronic tuberculous arthritis

3. Kidney disease
Uric acid is insoluble in water, so hyperuricemia results in the deposition of monosodium uric acid crystals in various parts of the body.

Patients with gout suffer from multiple comorbidities

- Hypertension: 89%
- Metabolic Syndrome: 63-87% Obesity: increased risk of gout / Weight gain: increased risk of gout Chronic Kidney Disease: 47% Hyperuricemia is an independent risk factor for the onset of chronic kidney disease.
- Diabetes: 29-33%
- Hyperlipidemia: 63%

Coronary heart disease

37% Increased risk of myocardial infarction
Increased mortality due to coronary heart disease / Low doses of aspirin and use of diuretics induce an increase in uric acid and increase the risk of developing gout. A history of ischemic heart disease and congestive heart failure increase the risk of developing gout• Heart Failure: 12%

Urinary tract disease and inflammation

Hyperuricaemia is accompanied by systemic inflammation even in asymptomatic patients.

Recent studies report the presence of ultrasound findings that clearly indicate the presence of monosodium urea crystals in the joints or tendons of 30-50% of patients with long-term asymptomatic hyperuricemia.

IL-1β is located at the apex of the cataract of inflammation that causes systemic manifestations. IL-1β plays a key role in the pathophysiology of many concomitant diseases of gout. IL-1β may molecularly link hyperuricaemia to many of the comorbidities.

What about asymptomatic hyperuricemia

Initiation of drug treatment

- Blood uric acid value ≥ 6 mg / dL and in addition one of the following:
- Clinical or imaging presence of tombs

Frequent attacks of gout: ≥ 2 per year
Chronic renal failure stage ≥ 2
Urolithiasis from monosodium urinary stones

- For asymptomatic hyperuricaemia: very high blood uric acid levels> 12mg / dL in men and> 10mg in women.

AEO Therapeutic Protocol for Symptomatic Hyperuricemia
Pharmacological treatment of hyperuricaemia

- A'1. Administration of Allopurinol
- A'2. Urinary excretory drugs
- Probenecid
- Sulfinpyrazone
- Benzobromarone
- Fembuxostat:

On non-response to allopurinol in doses up to 300mg

Non-drug treatment

- Diet
- Reduction of purine intake
- Reduce fructose-containing beverages
- Increase low-fat dairy
- Increase in protein vegetables, cherries
- Reduction of alcohol - avoiding mainly beer
- Recovery of normal weight - avoidance of rapid loss (up to 1Kg / month)

In patients with nephrolithiasis

• Intake> 2 liters of water / day
• Alkalization of urine

Discontinuation / change of medications (aspirin, diuretics)
• Moderate daily exercise in conclusion

To date, allopurinol versus febuxostat has been used as a first-line treatment not because there are sufficient data from comparative studies but because the cost of both drugs and their efficacy at the appropriate dosage are taken into account.

At the usual allopurinol dosage regimen (300mg / die), 30-50% of patients with normal renal function do not achieve the therapeutic goal Patients with impaired renal function are advised to adjust the dose of allopurinol according to creatinine clearance, and if the goal is not achieved, the transition.

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