Conclusion: In this study, the presence of synovial hypertrophy, erosions, DC signal and intra-articular deposits were the most frequent US findings in patients with gout. Moreover, these findings at the MTP1 joint allowed to distinguish between gout patients and matched control subjects. US seems to be useful to demonstrate evocative signs of crystal accumulation, inflammation or joint damage, even in the absence of overt arthritides.

REFERENCES


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AB0889 NEURAL NETWORKS IN PREDICTION OF KIDNEY DISEASE IN GOUT PATIENTS WITH OBESITY
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Background: Nowadays, in addition to correctly verified diagnosis and effective therapy of gouty nephropathy in obese patients, there is a problem to predict the development of kidney disease in this cohort of patients. Gouty nephropathy may have subclinical course for years, that’s why, with what probability it can occur? To solve such kind of problem we used neural networks or “artificial intellect”.

Objectives: To predict gouty nephropathy.

Methods: A one-center cohort prospective study was conducted that included 117 patients with gouty arthritis, who were on scheduled inpatient treatment at the Rheumatologic Department of Ternopil University Hospital during 2015-2018 years. All patients had no history of any kidney disease. Statistical analysis was performed with STATISTICA software.

Results: To create a neural network, as input information, it is necessary to select data that have the greatest correlation with gouty nephropathy. Significant correlations between gouty nephropathy and hyperuricemia (r=0,85), uricosaemia (r=0,79), hypercholesterolemia (r=0,44), hypertriglyceridemia (r=0,84), an increase of low density lipoproteins (r=0,80), concomitant arterial hypertension (r=0,81), diabetes mellitus (r = 0.59) were established. Age of the patients, the number of involved joints and the duration of the disease were also included. Then the probability of development of gouty nephropathy can be calculated by the formula:

\[ P(I) = \frac{1}{1+e^{-5D+W}} \]

\( P(I) \) — probability (III) of having gouty nephropathy in a patient.
Accepts values from 0 to 1. 0 — absolutely (100%) healthy patient, 1 — absolutely (100%) ill patient;
\( W \) — weight, vector of weights of synaptic arches of artificial neuron;
\( D \) — patient’s data scaled at intervals of 0 to 1, in such a way that the minimum possible value corresponds to 0, and to the maximum possible — 1;
e — Euler’s number.

To facilitate mathematical calculations, a peculiar “calculator” for predicting kidney damage in obese gout patients has been developed. This formula was written in Microsoft Office Excel and automatically calculates the probability of development of gouty nephropathy in%. Approval of this method indicated that 15.6% of the patients without gouty nephropathy may have development of kidney disease in the range of 55.1 - 98.8%.

Conclusion: Consequently, having input data (levels of uric acid in the blood, uric acid in the urine, cholesterol, triglycerides, low density lipoproteins, the presence of concomitant hypertension, diabetes mellitus, the number of affected joints and the duration of the disease), it is easy to predict the probability of kidney disease in patients with gout and obesity using the proposed formula.

REFERENCES


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AB0890 EFFECT OF DIFFERENT TYPES OF URIC ACID-LOWERING DRUGS ON GOUT ATTACK
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Background: Gout is an inflammatory arthritis associated with hyperuricemia which is caused by purine metabolism disorders. General uric acid lowering treatment sometimes causes the onset of gout, but the specific cause is not clear. Our group has found that adenine nucleoside triphosphate (ATP) plays an important role in the pathogenesis of gout. It synergizes with MSU to stimulate the secretion of IL-1β, leading to the onset of gout.1-3 The xanthine oxidase inhibitors promote the production of ATP, while the drugs that promote the excretion of uric acid does not affect ATP production. Therefore, we speculate that gout attacks induced by allopurinol and febuxostat are related to the role that ATP plays in the pathogenesis of gout.

Objectives: To compare the effects of drugs that inhibit uric acid production and promote uric acid excretion on gout.

Methods: A case-control study was used to compare the changes in serum uric acid concentration and observe whether the patients would suffer gouty arthritis within one month. The gout patients complied with the standard that the number of gout attacks did not exceed three times, as well as more than one time within the past six months, were selected. And patients with other chronic inflammatory and infectious diseases were excluded.

Results: A total of 148 patients with gout in the Department of Rheumatology and Immunology of Anhui Provincial Hospital were collected, all of whom were male. According to the patients taking uric acid-lowering drugs, they were divided into four groups: group A: control group (no uric acid-lowering treatment, n=35); group B: febuxostat group (40 mg Qd, n=66); group C: allopurinol group (0.1 g Bid, n=11); group D: benzen- bromarone group (50mg Qd, n=36). The statistical results are as follows: (1) The levels of serum uric acid in group B, C and D were significantly decreased after uric acid-lowering treatment compared with those before treatment (B: 389.6±88.9 vs 547.5±93.0, C:363.3±28.6 vs 504.0±38.3, D: 376.2±108.5 vs 557.3±101.8) within one month, and there was no significant difference in the level of uric acid before uric acid-lowering treatment among the three groups. (2) Comparison of gouty arthritis recurrence rates: The recurrence rate of gout patients in group B was 33.3%, and was 18.2% in group C, which was significantly higher than that in group D (11.1%), the difference was statistically significant (P<0.05); the recurrence rate of gout patients in group A was 14.3%, and there was no significant difference compared with group D (P=0.05); the recurrence rate of gout patients treated by drugs that inhibited uric acid production (B+C group) was 31.2%, which was significantly higher than patients treated by drugs that promoted uric acid excretion (D group) 11.1%, the difference was statistically significant (P<0.05).

Conclusion: Under the same uric acid-lowering intensity, the drugs that inhibit uric acid production can induce gout attacks during the process of uric acid lowering, while the drugs that promote uric acid excretion has less effect on the recurrence of gouty arthritis.

REFERENCES


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