AB0891

HYPERURICEMIA AND GOUT IN PATIENTS WITH PAGET'S DISEASE OF BONE

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Background: Hyperuricemia and gout has been described as metabolic complications in patients with more extensive Paget's disease, with a very variable prevalence. However, there is no data about fluctuations of uric acid levels associated with its treatment and its correlation with alkaline phosphatase variations during gout attacks.

Objectives: To analyze the frequency of hyperuricemia and gout in patients with a diagnosis of Paget's disease of bone (PD) and to establish the correlation between the activity of Paget's disease and its treatment in the fluctuations of uric acid levels.

Methods: Patients with Paget's disease and with scintigraphic uptake suggestive of activity were included. A database was created including demographic information, the presence of symptoms associated with PD, its type (monostotic or polyostotic), alkaline phosphatase (AP) and uric acid (UA) levels at diagnosis and the treatment received. AP and UA levels after treatment were also collected as well as the presence of possible hyperuricemic risk factors (HRF), history of gout and levels of AP and UA during gout attacks, if any. Finally data was analyzed using SPSS v21.

Results: A total of 95 patients were included with a mean age of 70.59 years (range 45-89), with 56% of them being women. The mean values of AP and UA at diagnosis were 178.05 ± 82.81 mg/dL and 5.84 ± 1.74 mg/dL respectively. 58.9% of the patients had monostotic involvement (56/95) and only 53.12% had associated symptoms (51/95), whereas in the rest of the patients PD was an incidental finding. 70.83% did not present HRF and 56 patients (58.9%) had received treatment for their PD (41% of them with zoledronic acid). Pearson correlation between PA and UA levels was found moderately positive (0.710 with $p < 0.0005$) and 50.52% of the patients (48/95) had UA levels above 6mg/dL at the time of diagnosis, with a mean of 7.10 mg/dL ± 1.74 . In 93.5% of the cases there was a decrease in UA levels after the treatment for PD, with a mean decrease of 0.78 ± 1.3 mg/dL over the baseline value at one year of follow-up. Only 11.57% of the patients in our cohort presented gout symptoms during their follow-up, with an increase in AP levels at the time of the attack in 63.63% of the cases.

Conclusion: In our cohort, there was a moderate positive correlation between the elevation of AP and UA in patients with active Paget's disease. Treatment for PD produced a decrease in UA levels from baseline in most patients. Similarly, during gout attacks, an increase in AP levels could be.

REFERENCE

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AB0892

ACUTE POLYARTICULAR GOUT WITH SYSTEMIC INFLAMMATORY RESPONSE – A SINGLE TERTIARY CENTRE COHORT

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Background: Gout is a common, inflammatory, crystal-induced disease with increasing prevalence and incidence. Typical monoarticular involvement can be quite easily and effectively treated. In recent years, we have observed more frequently gout cases with severe polyarticular manifestation and with systemic inflammatory response syndrome (SIRS). In clinical practice such a complicated gout attack used to be identified with delay and therapy is not adequate.

Objectives: To assess clinical and laboratory parameters of systemic inflammatory polyarticular gout and to identify risk factors and convenient therapeutic strategy.

Methods: Retrospective analysis of 133 consecutive new patients (14 females) with gout (according to the classification criteria ACR/EULAR 2015) on the presence of acute polyarticular involvement (≥ 4 joints) and fulfilling criteria for SIRS.

Results: Seventeen (12.8%) of 133 gout patients had polyarticular manifestation and met SIRS criteria; 2 females and 15 males, average age 73.2 ± 11.3 years, BMI 29.9 ± 3.8 . All these patients had to be admitted to inpatient department because of gout severity. Mean CRP was 204 ± 113 mg/l, leucocytes $15.2 \pm 6.8 \cdot 10^9/l$, serum uric acid 401 ± 140 $\mu\text{mol/l}$. Procalcitonin was slightly positive in 2 patients (with concurrent respiratory infection). Mean 6 joints were affected, 13 (76.5%) patients had involvement of both upper and lower limbs, tophi were present in 7 of 17 patients. Synovial fluid analysis was performed with proving urate crystals and leucocyte count $24.4 \pm 17.3 \cdot 10^9/l$. Typical gout features were found in 70.6% patients on joint X-ray and in 52.9% ones on musculoskeletal ultrasound. Risk factors for systemic inflammatory polyarticular manifestation were preceding renal function impairment, long-term diuretic therapy and alcohol above-average consumption. Monotherapy was not effective in any patients, combination of NSAID with colchicine or systemic glucocorticoids was used with success.

Conclusion: Acute polyarticular gout with systemic inflammatory response mimics septic status and mostly requires hospitalization. Complex management with combination therapy should be guided by an experienced rheumatologist.

REFERENCE

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AB0893

WHAT IS THE CHANCE IN AN INDIVIDUAL GOUTY PATIENT THAT DISEASE WILL BE STILL ACTIVE IN A YEAR? PREDICTIVE MODELLING MIGHT COME TO AID

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Background: The treat-to-target concept is currently applied to gout [1] and a composite outcome to measure disease activity may serve as a target in clinical practice. Strategies for achieving the target are still not individualized and the ability to adjust the treatment on an individual basis using prognostic information from predictive models might help practitioners to identify patients with a high or low probability of remission or disease inactivity.

Objectives: To develop a prediction model for disease inactivity in gouty patients.

Methods: Data were retrieved from the Kick-off of the Italian Network for Gout (KING) observational cohort multicentre study on behalf of the Italian Society for Rheumatology. The gout activity score (GAS) [2,3] was used as validated outcome to define inactive disease (cut off < 2.5) at 12 months in gouty patients with active disease at baseline and receiving the standard of care in two visits 6 months apart. Logistic regression analyses and backwards elimination of baseline predictors with bootstrap resampling were used to develop parsimonious models. Performance and correction for optimism were assessed. Missing data were handled through multiple imputation by chained equations (50 completed datasets).

Results: Out of 446 patients enrolled from 30 rheumatology centres, 265 (59%) had active disease at baseline (mean age 63 ± 11 years, male 91%, median disease duration 7 years, tophi 23%, urate lowering therapy 82%, mean serum urate [sUA] levels 6.7 ± 1.6 mg/dl) and a 12-month visit. In 34% (65/206) gout was inactive at 12 months. From 20 candidate predictors (complete cases $n=150$) disease duration, use of nonsteroidal anti-inflammatory drugs (NSAIDs), number of flares in the previous 12 months, presence of tophi, sUA < 6 mg/dl, visual analogue scale (VAS) global patient, and baseline GAS were selected. The performance of the four developed models is shown in table. In the completed case analysis ($n = 258$) the c-statistics (95% confidence interval [CI]) of