periarticular tophi frequently seen in ultrasound appear as the likely source for these formations.

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AB1026 CLINICAL CHARACTERISTICS AND RISK FACTORS FOR GOUT ATTACK DURING THE POSTSURGICAL PERIOD

H. Jeong¹, Y.H. Eun², E.-J. Park³, H. Kim², J.Y. Chai⁴, J. Lee², C.H. Jeon¹.

¹Soonchunhyang University Hospital, Bucheon; ²Samsung Medical Center,

³National Medical Center, Seoul; ⁴Bundang Jesaeng Hospital, Seongnam, Korea, Republic of Ireland

Objectives: To evaluate the clinical features and risk factors for gout attack during the postsurgical period in patients with gout.

Methods: Seventy patients who had histories of gout and had been consulted to rheumatologic clinic before surgery under general anaesthesia at a single tertiary hospital were included. Clinical characteristics of patients who developed postsurgical gout attack were compared with patients who did not developed gout attack.

Results: Among 70 patients, 31 (44.3%) patients developed gout attack during postsurgical period. Mean time of gout attack after surgery was 3.7±4.9 days. Most of attacks involved lower extremity joints (83.9%) and tended to monoarticular involvement (61.3%). Knee joint (26%) and foot except 1st metatarsophalangeal (MTP) joint (26%) were more frequently involved than 1st MTP joint (13%). Uric acid levels before surgery (OR 1.46, 95% CI 1.13–1.88, p=0.004) and amount of uric acid changes between before and after surgery (OR 1.62, 95% CI 1.21–2.18, p=0.001) were risk factors for postsurgical gout attack. Taking medications for gout including uric acid lowering agents and/or colchicine reduced the risk of postsurgical gout attack (OR 0.11, 95% CI 0.04–0.32, p<0.001). Operation time, amount of blood loss during surgery, amount of fluid administration during surgery, and surgery site were not significantly associated with postsurgical gout attack.

Conclusions: Adequate uric acid control and taking medications for gout could prevent the postsurgical gout attack.

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AB1027 THE PREDICTIVE VALUE OF CYSTATIN C FOR GOUTY NEPHROPATHY

D. Rekalov, I. Daniuk, G. Medvedchuk. *Internal diseases-3, Zaporizhzhia State Medical University, Zaporizhzhia, Ukraine*

Background: Gout is the most common cause of an inflammatory arthritis in men elder than 30 years, varies up to 1,7% of the total morbidity. One of the most common complication of chronic gout is gouty nephropathy with the morphological signs of kidney damage, even in the early stages.¹

It's well known, that urine albumin and glomerular filtration rate (GFR) are the generally accepted markers of nephropathy. But, recent data has shown, that the informative and obtainable new biomarkers of the kidney function are still needed. Thus, evaluation of the pathogenic role in the renal failure of cystatin C could be a "clue". The dozen trials have shown that an increasing of ranges cystatin C in serum could be tested at the stage of subclinical reduction of GFR with normal level of creatinine.^{2;3}

Objectives: To determine the serum ranges of cystatin C and creatinine as the markers of nephropathy in patients with gout.

Methods: The main group was the 80 males with primary gout (average age – 53,4±8,2 years), with the disease duration from 3 up to 25 years. The second group – 20 healthy men (49,5±4,5). We determined the concentration of serum uric acid, serum cystatin C, creatinine, calculate the GFR using CKD-epi formula. For urine albumin evaluation we used the albumin/creatinine ratio in the morning urine portion.

Results: e determined that the main group identified by more significant violations of renal function. In the main group was found the statistically significant increase of the serum creatinine (97±18 µmol/l) and decrease of GFR (73^{64; 94} ml/min².

The serum cystatin C concentration in the main group was (1,7^{1,4; 1,9} mg/l) which was the significantly larger than in the comparison group (0,8 [0,7; 0,92] mg/l, p<0,001).

The albumin/creatinine ratio was statistically higher in the main group (26^{14; 56} mg/g) than in comparison (0,8 [0,7; 0,92] mg/l, p<0,001). Also the patients with gout are characterised by the larger concentration of uric acid in the serum (530±74) µmol/l than healthy men (347±75) µmol/l and negative correlation between uric

acid level and GFR (r=-0,4, p<0,05) which explain the increase of uric acid concentration in the serum.

We determined the correlation between level of urine alabumin and creatinine concentration (r=+0,5, p<0,05) in the main group, but concentration between albuminuria and cystatin C was stronger (r=+0,6, p<0,05). We detected that cystatin C level had a greater accuracy for the diagnosis of albuminuria than creatinine according to the ROC-analysis. These facts show that serum concentration of cystatin C more closely connected with the renal function than creatinine.

Conclusions: 1. The increase of serum cystatin C level can be identified before the clinical manifestation of renal dysfunction while the serum creatinine remain relatively normal.

2. Serum ranges of cystatin C more closely correlate with elevation of urine albumin than creatinine.

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AB1028 ACTH VS BETAMETHASONE FOR THE TREATMENT OF ACUTE GOUT IN HOSPITALISED PATIENTS

AN OPEN LABEL, COMPARATIVE STUDY

P. Kordas¹, I. Antonopoulos², D. Velissaris¹, D. Daoussis². ¹Internal Medicine; ²Rheumatology, University of Patras Medical School, Rio, Patras, Greece

Background: The management of gout can be problematic in the hospital setting; hospitalised patients usually have significant comorbidities and receive multiple medications which leads to a high frequency of contraindications to established gout therapies. We have previously shown the ACTH is a safe and fast acting therapeutic option for acute gout in hospitalised patients in a large scale retrospective study.¹

Objectives: To directly compare the efficacy of ACTH vs betamethasone for the treatment of acute gout in hospitalised patients in a prospective manner.

Methods: Hospitalised patients with acute gout, fulfilling the ACR criteria, were treated with an IM injection of either 100 IU of ACTH (Synacthen Depot) or 6 mg of betamethasone (Celestone Chronodose-the most widely use IM steroid formulation in our country) on an alternate 1/1 basis. Clinical efficacy was assessed at 24, 48, 72 hour and 5 days as follows: a) Intensity of pain using a Visual Analogue Scale (VAS 0–10), b) physician global assessment (0–10) and c) swelling, redness and warmth (0–3 scale). Pain VAS was also self reported by the patient at 6 and 12 hour. Comorbidities representing contraindications to established gout therapies were recorded. Primary outcome of the study was the change in pain VAS at 24 and 48 hour. Secondary outcomes were changes in physician global assessment and changes in objective signs of inflammation.

Results: This is a 6 month interim analysis of an ongoing investigator initiated clinical study. Twelve patients (8 male) with a mean ±SD of 66.9±12.3 years were recruited and treated with ACTH or betamethasone on an alternate basis (6 in each treatment group). In most cases (n=11) the attack was monoarticular. The majority of patients had multiple comorbidities with the commonest being hypertension (9/12). Both treatments were effective. ACTH led to a significant decline in pain VAS at 24 hour compared to baseline (mean ±SEM: 2.33±1.21 vs 7.66 ±0.81 respectively, p=0.0002) and at 48 hour (1.40±1.14, p=0.0011 compared to baseline). Betamethasone was also effective with an improvement in pain VAS at 24 hour compared to baseline (mean ±SEM: 1.83±0.98 vs 5.33±2.16 respectively, p=0.0024) and at 48 hour (0.75±0.95, p=0.02 compared to baseline). However, direct comparison between treatment arms showed that ACTH treated patients exhibited a higher change in pain VAS at 24 hour compared to betamethasone treated patients (mean ±SEM: 5.5±0.5 vs 3.5±0.61 respectively, p=0.03). At the 48 hour time point ACTH treated patients still showed a higher change in pain VAS (mean ±SEM: 6.4±0.6 vs 4±0.91 respectively, p=0.056). A trend favouring ACTH was already evident at the 12 hour time point; the change in pain VAS was 4±1.54 vs 3.1±1.47 for ACTH vs betamethasone, respectively, p=ns). No changes in physician global assessment and objective signs of