wasting by tubular reabsorption of phosphate (TRP) was calculated, PU resulted increased in all.

Tumor was localized in all cases (Fig.1) and were localized in bone and soft tissue, by using functional imaging, followed by anatomical techniques. Before the introduction in routinely practice of ^{68}Ga -DOTATATE-PET-CT in 2013, Octreoscan-SPECT/CT and ^{18}F FDG-PET were used as imaging modalities. Since 2013, diagnostic delay consistently reduced, from 8.6 ± 8.3 yrs (7 patients) to 4.5 ± 2.6 yrs (9 patients), confirming higher diagnostic accuracy of ^{68}Ga -DOTATATE-PET-CT.

Figure 1.

13 patients underwent surgery; in two cases surgery was not possible due to tumor location, so pharmacological support with phosphate supplements and calcitriol was started; a patient underwent to TC-guided radiofrequency ablation. After surgery, 7 patients experienced a complete remission, 3 had a persistence of the disease, and 3 an overtime relapse, even after a long-standing normalization of PS (6 years). After surgical tumor removal, PS significantly increased in few days (from 1.36 ± 0.39 to 2.9 ± 1.1 , $p=0.0001$), while IGF-23 levels tended to rapidly decreased (from 396.6 ± 707.3 to 62.8 ± 78.4). Before the introduction of ^{68}Ga -DOTATATE-PET-CT, 6 patients underwent to imaging-guided closed biopsy to confirm tumor localization; by using ^{68}Ga -DOTATATE-PET-CT only 2 subjects had closed biopsy. Furthermore, in our population only patients who had biopsy to detect the lesion (7 patients) had relapses compared to patients who did not.

Conclusion: To our knowledge, this is the widest European cohort of patients affected by TIO reported in the last two decades. We confirm an important delay between symptoms onset and diagnosis. To locate tumor, a stepwise approach is recommended, starting with a thorough medical history and physical examination, followed by functional imaging, preferring ^{68}Ga -DOTATATE-PET-CT. Tumor biopsy is not recommended due to the potential cell spilling. Surgery is considered the only definitive treatment, aiming to a wider excision. Active surveillance is always needed, due to the possible relapses, even after a long period of complete clinical and biochemical remission.

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THU0422

CALCIUM PYROPHOSPHATE CRYSTAL DEPOSITION WITHIN TOPHUS LOBULE: A FREQUENT ASSOCIATION OBSERVED IN LONG-TIME COURSE TOPHI

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Background: Calcium pyrophosphate (CPP) crystals and monosodium urate (MSU) crystals are frequently found in the same synovial fluids of gouty patients suggesting an interaction in crystal formation and deposition. This association has never been reported in tophus.

Objectives: we aimed to describe the prevalence of CPP crystal deposition in tophus and to determine the associated risk factors.

Methods: 22 tophi consecutively harvested were fixed in 4% paraformaldehyde and embedded in paraffin. 5- μm thick sections were analyzed by compensated polarized microscopy (CPM) after hematoxylin and eosin staining. Characterization of CPP crystals were performed by scan electronic microscopy (SEM) and Fourier transform infrared (FTIR) spectroscopy. Clinical characteristics were compared between patients having tophus with CPP deposition and patients having tophus without CPP crystals.

Results: All tophi appeared multi-lobulated depositions of MSU crystals separated by fibrous tissue and surrounded by a foreign giant cell reaction. CPP crystals were identified in few lobules of 5 tophi (22.7%) harvested from 3 great toes, 1 elbow bursa and 1 finger of 5 patients. CPP crystals formed aggregate deposition within the lobule of MSU crystals. Both monoclinic and triclinic CPP crystal phases were identified by CPM, SEM and FTIR. Tophi were harvested from 22 male gouty patients with a mean age of 50.8 (28-66) years and a mean BMI of 24.2kg/m² (18.9-29.4). Mean serum urate level (SUL) was $499 \pm 107 \mu\text{mol/L}$. 59% of patients had chronic renal disease stage 2 or 3, 40.9% dyslipidemia, 22.7% type 2 diabetes mellitus, 13.6% hypertension and 50% obesity. Patients with tophi containing CPP deposition were older ($61.2 [56-66]$ vs $47.8 [28-64]$ years, $p=0.009$) and had a longer gout duration ($19 [10-31]$ vs $9 [3-20]$ years, $p=0.007$) and tophus duration ($11.4 [8-16]$ vs $4.5 [1-9]$ years, $p<0.0001$) than patients with tophi alone. Tophi did not display calcification on radiographies performed before surgery. However, the density of tophi containing CPP crystal deposition was higher than the density of tophi without CPP crystals ($51 [25-100]$ vs $21.5\% [0-40]$, $p=0.009$). The proportion of bone erosion and gout arthropathy was similar between the two groups. Similarly, no difference was observed for SUL (467 ± 43 vs $509 \pm 109 \mu\text{mol/L}$), estimated glomerular filtration rate (76.6 ± 11.9 vs $74.9 \pm 15.7 \text{ ml/min/1.73m}^2$) and prevalence of comorbidities. Interestingly, no calcification was detected on knee and wrist radiographies of patients with tophi containing CPP deposition.

Conclusion: These results reported for the first time, in a small sample size, that CPP crystal deposition occurred within tophus lobules. They suggested that long-time course tophi might act as a facilitating agent of CPP nucleation. This hypothesis needs specific confirmation studies

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THU0423

ADHERENCE TO URATE-LOWERING THERAPY IN PATIENTS WITH SEVERE GOUT WHO RECEIVED CANAKINUMAB FOLLOWING A 5-YEAR RETROSPECTIVE ANALYSIS

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Background: The adherence to lowering therapy for gout is low, including in chronic severe gout. Whether interleukin 1 inhibitors may contribute to better adherence is unknown.

Objectives: To compare adherence to urate-lowering therapy in patients with severe gout who received canakinumab versus patients who received standard anti-inflammatory therapy (NSAIDs, glucocorticoids, colchicine).

Methods: Of the 513 patients with gout observed at the V.A. Nasonova Research Institute of Rheumatology, Moscow from 2013 to 2014 y 247 patients with the most severe gout, requiring regular symptomatic treatment, were selected. Of these, 25 patients (3 (12%) women and 22 (88%) men), the average age of 54.5 ± 12.7 g, received (at least 1) canakinumab injection of 150mg subcutaneously as a symptomatic therapy, the remaining 222 patients (men) mean age

51.9 ± 11.4g. received standard anti-inflammatory therapy (colchicine (55% of patients), glucocorticoids (5%), NSAIDs (40%), or a combination of these (3%). On average, after 4.8 ± 1.7 years, a comparative analysis of adherence to reducing therapy was carried out, as well as the need for anti-inflammatory therapy and assessment of adherence according to the Score compliance on the scale of the Moriscy-Green patients who received and did not receive canakinumab.

Results: Evaluation was available in 180 patients (16 who received canakinumab and 164 who received standard anti-inflammatory therapy) who were initially given reducible therapy. 11 patients died (2 patients (8%) who received canakinumab and 9 (4%) patients on standard anti-inflammatory therapy), 56 patients (7 (28%) and 49 (22%), respectively) were not available for observation. Adherence to urate-lowering therapy was better in patients who received canakinumab (see table 1).

Table 1. Score compliance on the scale of the Moriscy-Green

Adherence to therapy	Patients receiving canakinumab, (n %)	Patients receiving standard anti-inflammatory therapy, (n %)	p=
High (> 4 points)	14 (87)	83 (51)	0,07
Moderate (3 points)	2 (13)	46 (28)	0,13
Low (2 and <points)	0	35 (21)	0,03

The likelihood of maintaining the target uric acid level when taking urate-lowering drugs in patients who previously received canakinumab was higher (12 patients (75%) who received canakinumab and 32 (20%) patients received standard anti-inflammatory therapy (p = 0.005).

During the year preceding the analysis, there were no acute attacks of arthritis in 12 (75%) patients who received canakinumab and 46 (28%) patients received standard anti-inflammatory therapy (p = 0.002).

132 patients who previously received regular anti-inflammatory therapy and received standard anti-inflammatory therapy and 1 patient (1 attack) (p = 0.005) who previously received canakinumab took anti-inflammatory drugs over the past year due to the development of exacerbations (an average of 3 seizures per year): NSAIDs (54%) or colchicine (46%).

Conclusion: Therapy with interleukin 1 Kanakinumab may contribute to a better adherence to lowering therapy and is identified with a lesser need for symptomatic therapy with long-term follow-up.

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THU0424 SHORT-TERM ADHERENCE OF URATE-LOWERING AGENTS WILL NOT IMPROVE END-STAGE RENAL DISEASE AND MORTALITY

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Background: Gout is the most common inflammatory arthritis worldwide with a diverse spectrum of clinical manifestations. Many studies indicated that gout is associated with hypertension, metabolic syndrome, chronic kidney disease and ischaemic heart diseases.

Objectives: This study investigated the association between the compliance of urate-lowering agents (ULA) compliance and progression end-stage renal disease (ESRD) and all-cause mortality.

Methods: We identified 53,917 patients with incident gout from 2002 to 2012. We followed up these patients till 31DEC2015. The primary data source was the National Health Insurance database of Taiwan. Proportion of days covered (PDC) was used to measure medication adherence. Patients with PDC ≥ 80% more than two years defined adherence to ULA. Cox proportional hazards model was used to estimate difference and hazard ratios (HRs) for ESRD and all-cause mortality.

Results: A total of 53,917 patients were included (figure 1). Patient with urate-lowering agent PDC ≥ 80% more than 2 years were 2,371. After propensity score matched 1:4 with age, sex and comorbidities, PDC <80% were 9,484 matched cohort (Table 1). The mean follow-up duration is 12.30±2.45 years in PDC ≥ 80% group and 9.52±3.46 years in PDC <80%. During follow-up, ESRD was detected in 1,349 patients (306 with PDC ≥ 80% and 1,043 with PDC <80%) and all-cause mortality was found 3,430 patients (842 with PDC ≥ 80% and 2,588 with PDC <80%). This matching showed no difference in ESRD (HR, 0.91; 95% CI, 0.80-1.03) and all-cause mortality (HR, 0.96; 95% CI, 0.88-1.03) (Table 2).

Table 1. Baseline characteristic of adherence and non-adherence of urate-lowering agent among patients with gout in matched and unmatched cohort

Variable	Unmatched Cohort		P	Propensity-score Matched Cohort		P
	PDC ≥ 80% N=2371^	PDC <80% N=51546^		PDC ≥ 80% N=2371^	PDC <80% N=9484^	
Age (years) (mean ± standard deviation)	59.96±13.44	51.52±15.61	<0.0001	59.96±13.44	60.23±13.41	0.3755
follow-up duration (years) (mean ± standard deviation)				12.30±2.45	9.52±3.46	
Gender			0.3098			0.2855
Male	1929(81.36%)	42358(82.18%)		1929(81.36%)	7624(80.39%)	
Female	442(18.64%)	9188(17.82%)		442(18.64%)	1860(19.61%)	
Comorbidities						
Hypertension	406(17.12%)	3695(7.17%)	1.71E-71406(17.12%)	1571(16.56%)	0.5138	
Diabetes Mellitus	276(11.64%)	2367(4.59%)	1.78E-54276(11.64%)	1080(11.39%)	0.7291	
Ischemic heart disease	109(4.60%)	988(1.92%)	1.58E-19109(4.60%)	444(4.68%)	0.8617	
Cerebrovascular disease	113(4.77%)	891(1.73%)	1.05E-26113(4.77%)	481(5.07%)	0.5416	
Peripheral arterial disease	20(0.84%)	170(0.33%)	3.67E-0520(0.84%)	74(0.78%)	0.7561	
Congestive heart failure	81(3.42%)	606(1.18%)	1.88E-2181(3.42%)	286(3.02%)	0.3137	
Anemia	182(7.68%)	1559(3.02%)	5.22E-36182(7.68%)	769(8.11%)	0.4882	
CKD	266(11.22%)	1978(3.84%)	2.61E-69266(11.22%)	963(10.15%)	0.1281	

Table 2. hazard ratio of ESRD and all-cause mortality in patients with gout

Outcome	Hazard ratio (95% Confidence interval)
ESRD	
ULA adherence level	
PDC <80%	Reference
PDC ≥ 80%	0.91 (0.80-1.03)
All-cause mortality	
ULA adherence level	
PDC <80%	Reference
PDC ≥ 80%	0.96 (0.88-1.03)

PDC= Proportion of days covered

Figure 1. Flow chart for study design