the two groups are as follows: (1) 6 pairs of normal cholesterol group and elevated group were screened according to age, course and uric acid level. There is significant difference in the frequency of gout attacks between the two groups within six months and one year (1.17 ± 0.75 vs 2.83 ± 2.23 , $Z = -2.060$, $P = 0.039$; 1.33 ± 0.82 vs 4.17 ± 3.43 , $Z = -2.032$, $P = 0.042$). (2) 11 pairs of normal triglyceride group and elevated group were screened as above. There is significant difference in the frequency of gout attacks between the two groups within one year (1.45 ± 0.82 vs 3.45 ± 2.58 ; $Z = -2.328$, $P = 0.020$).

Conclusion: Cholesterol and triglyceride levels can affect the frequency of gout attacks. The phenomenon mentioned in background can be explained by the increase of cholesterol and triglyceride levels as the course prolongs.

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SAT0450 SENSITIVITY OF DUAL-ENERGY CT SCANNING, ULTRASOUND, AND X-RAY FOR CRYSTAL-PROVEN PSEUDOGOUT

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Background: Advanced imaging modalities such as ultrasound (US) and dual-energy CT (DECT) can help diagnose crystalline arthritis. DECT is highly sensitive and specific in gout and has not been well studied in pseudogout.

Objectives: To compare the sensitivity of DECT, US, and x-ray (XR) in pseudogout.

Methods: We prospectively enrolled patients with crystal-proven pseudogout at a tertiary care center, 3/2018-11/2018. We searched the electronic medical record for synovial fluid crystal lab orders and reviewed the record to identify candidates. Eligible patients were 18 years old with acute monoarthritis, joint aspiration, and synovial fluid calcium pyrophosphate (CPP) crystals on polarized microscopy. Patients with both monosodium urate and CPP crystals were excluded. Subjects provided informed consent and underwent DECT, US, and XR of the aspirated joint and standardized joint (right wrist). All images were interpreted by a musculoskeletal radiologist and a rheumatologist trained in US; consensus was reached on each image. DECT images were post-processed using Siemens Syngo.via software, applying color-coded overlay indicating volume and location of CPP deposits. We considered two volume thresholds (cm^3) for a positive DECT scan. Imaging abnormalities defining a positive scan were: color-coded changes consistent with CPP (DECT); hyperechoic deposits in hyaline- or fibro-cartilage, or tendon (US); and chondrocalcinosis in hyaline- or fibro-cartilage (XR). We calculated sensitivity and 95% confidence interval (CI) of positive scans in the aspirated joint and prevalence (95% CI) in the standardized joint.

Results: Ten of 27 eligible patients enrolled. Mean (SD) age was 73 (9.7) years; 40% were female. Eight knees and two wrists were aspirated a mean (SD) of 17 (9) days before enrollment. Six subjects received intra-articular steroids before enrollment. Imaging results are shown in Table 1. In the aspirated joint, sensitivity (95% CI) was 90% (71-100%) for DECT volume $>0.40 \text{ cm}^3$, and 100% (100-100%) for DECT volume $>0.01 \text{ cm}^3$; 100% (100-100%) for US; and 70% (42-98%) for XR. In the standardized joint, DECT was positive in 20% (0-45%) for volume $>0.40 \text{ cm}^3$, and 90% (71-100%) for volume $>0.01 \text{ cm}^3$. XR chondrocalcinosis was present in 30% (2-58%) and US was positive in 80% (55-100%) of wrists.

Conclusion: DECT and US had high sensitivity for pseudogout using synovial fluid CPP crystals as the gold standard. Larger studies testing DECT sensitivity and specificity in pseudogout vs. other arthritis and establishing a volume threshold are needed.

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none

Table 1. Presence and location of imaging abnormalities in 10 pseudogout subjects

Subject	Aspirated joint			Standardized joint (right wrist)		
	DECT color-coded changes (cm^3)	US hyperechoic deposits	XR chondrocalcinosis	DECT color-coded changes (cm^3)	US hyperechoic deposits	XR chondrocalcinosis
1	PF, PSFC, M, TF, (0.48)	M	M	IC, IP1, MCP3, RC, TFCC (0.07)	TFCC	TFCC
2	ICN, M (0.73)	FC, M	M	CMC1 (0.08)	TFCC	none
3	ICN, M, PF, TF (2.85)	FC	ICN, M, PF	CMC1, CMC3, IC, IP1, MCP2, RC, TFCC (0.51)	TFCC	not done
4	ICN, M, PF, PSFC, Pop, Q (1.05)	FC	M	IC (0.10)	none	none
5	M, PSFC, Q (0.50)	FC	M, PF	IC, TFCC (0.04)	TFCC	none
6	M, PF (0.67)	FC	none	CMC1, IC (0.03)	TFCC	CMC1
7	M, PF, PSFC (1.33)	FC, M	M, PF	none (0.01)	TFCC	none
8*	IC (0.02)	IC, TFCC	none	IC (0.02)	IC, TFCC	none
9	M (0.45)	M	none	IC (0.05)	TFCC	none
10	CMC1, IC, MCP1-5, TFCC (0.82)	TFCC	IC, MCP1-2, RC, TFCC	CMC1, IC, MCP1-5, TFCC (0.84)	none	IC, MCP1-4, TFCC

*Right wrist was the aspirated and standardized joint. CMC: carpometacarpal. FC: femoral cartilage. ICN: intercondylar notch. IP: interphalangeal. M: meniscus. MCP: metacarpophalangeal. PF: patellofemoral. Pop: popliteus tendon insertion. PSFC: posterior superior femoral condyle. Q: quadriceps tendon. RC: radiocarpal. TF: tibiofibular. TFCC: triangular fibrocartilage complex.

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SAT0451 GOUT FLARES BECOME INFREQUENT DURING A TREAT-TO-TARGET STRATEGY OVER ONE YEAR: DATA FROM THE NOR-GOUT STUDY

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Background: Urate lowering therapy (ULT) is expected to prevent new gout flares. Treat-to-target ULT is however often not performed, and we need more evidence on how often patient become flare-free during ULT.

Objectives: Urate lowering therapy (ULT) is expected to prevent new gout flares. Treat-to-target ULT is however often not performed, and we need more evidence on how often patient become flare-free during ULT.

Methods: In a prospective observational study, patients with crystal-proven gout with a recent gout attack and insufficiently treated serum urate (sUA) level ($>360 \mu\text{mol/L}$ / $>6 \text{ mg/dl}$) were included. They received ULT with drug escalation during monthly follow-up until the target sUA level was met (sUA $<360 \mu\text{mol/L}$, or $<300 \mu\text{mol/L}$ if clinical tophi). Assessment in this ongoing data collection included demographic and clinical variables, serum urate levels, previous medication with allopurinol, colchicine and NSAIDs, co-morbidities, and health related quality of life (SF-36). Flares during the last six months of one year with "treat-to-target" were recorded. Bivariate analyses and logistic regression analyses examined factors associated with and prediction (odds ratio with 95% confidence intervals) of a flare-free period during months 6-12.

Results: 202 patients were included, and 140 completed a 12-month-follow-up: 94.0% males, 90.3% Caucasian, mean (SD) age 56.7 (14.1) years, disease duration 8.0 (7.7) years, sUA level 494 (87) $\mu\text{mol/L}$, eGFR 78.0 (18.8) ml/min. 58.6% had college education, 76.9% were married/cohabiting, 18.3% had tophi.

Allopurinol had at baseline previously been used by 30.0% (54/180), colchicine by 51.8% (100/193) and NSAID by 78.6% (154/196).

70.0% (98/140) of the patients were flare-free between 6 and 12 months, and among patients with flares only 10.2% (4/39) were not at treatment target. 80.9% (106/131) were at treatment target at month 12 (sUA<360 or 300 $\mu\text{mol/l}$).

A number of variables at baseline were bivariate ($p<0.05$) related to a flare-free outcome: male gender, low BMI, no previous use of colchicine or NSAIDs, better physical function summary score (SF-36), low level joint pain during strongest attack ever, and high number (>5) of previous gout attacks.

In multivariate logistic regression analyses, also adjusting for age, and gender in the final model, a lower BMI (OR 0.90 per unit, 95% CI 0.83-0.99), low baseline serum urate (OR 0.99 per unit, 95% CI 0.99-1.00) and no previous use of colchicine (OR 4.0, 95% CI 1.6-10.2) predicted a flare-free period between months 6 and 12. When substituted for colchicine, also lack of NSAID use (OR 6.9, 95%CI 1.8-26.1) was a predictor of a flare-free period during this period.

Conclusion: Most patients (70%) did not experience flares during the second half of the first treat-to-target year during ULT. High baseline level of sUA, and high BMI decreased the chance of a flare free period, as did experience with colchicine or NSAID.

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SAT0452 VITAMIN D INSUFFICIENCY AND DEFICIENCY ARE ASSOCIATED WITH A HIGHER LEVEL OF SERUM URIC ACID: A SYSTEMATIC REVIEW AND META-ANALYSIS

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Background: Several studies have revealed a relationship between vitamin D status and a number of non-skeletal health conditions such as insulin resistance, metabolic syndrome, cardiovascular diseases, inflammatory diseases, infection and malignancy. The interaction between serum uric acid and vitamin D has also been observed. A study found that uric acid can suppress 1- α -hydroxylase in vitro and in vivo, leading to the reduction in the conversion of the storage form of 25-hydroxyvitamin D into the active form of 1,25-dihydroxyvitamin D [1]. In fact, several epidemiological studies have suggested that patients with vitamin D insufficiency and deficiency tend to have a higher level of serum uric acid compared to those with adequate vitamin D level although the results were inconsistent across the studies.

Objectives: To compare serum uric level between individuals with normal vitamin D level versus patients with vitamin D deficiency and vitamin D insufficiency.

Methods: A systematic review was conducted using MEDLINE and EMBASE database from inception to October 2018 to identify all studies that compared the level of serum uric acid between individuals with normal vitamin D level and patients with vitamin D insufficiency/deficiency. Eligible studies must be cohort or cross-sectional studies that consisted of two groups of adult participants, one with normal level of vitamin D (vitamin D level >30 ng/ml) and one with vitamin D insufficiency (vitamin D level 20-30 ng/ml) or vitamin D deficiency (vitamin D level of <20 ng/ml). Mean serum uric acid level and standard deviation of participants in each group were extracted from each study to calculate mean difference

(MD) between the groups. Pooled MD was then calculated by combining MDs of each study using random-effects model.

Results: A total of seven cross-sectional studies were eligible for the meta-analyses. Individuals with normal vitamin D level had a significantly lower serum uric acid level than patients with vitamin D insufficiency with the pooled MD of -0.33 mg/dL (95% CI, -0.61, -0.04) (figure 1) and also had a significantly lower serum uric acid level than patients with vitamin D deficiency with the pooled MD of -0.45 mg/dL (95% CI, -0.82, -0.08) (figure 2). The statistical heterogeneity was high with the I^2 of 78% and 89%, respectively. Funnel plots of both meta-analyses were fairly symmetric which did not suggest the presence of publication bias.

Conclusion: Both patients with vitamin D insufficiency and patients with vitamin D deficiency had a significantly higher level of serum uric acid compared to individuals with normal vitamin D level.

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Figure 1. Forest plot of the meta-analysis of vitamin D insufficiency versus normal vitamin D level

Forest plot of the meta-analysis of vitamin D deficiency versus normal vitamin D level

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Infection-related rheumatic diseases

SAT0453 JOINT MANIFESTATIONS OF WHIPPLE'S DISEASE: CLINICAL AND RADIOLOGICAL PRESENTATION OF 19 PATIENTS

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Background: Whipple's disease (WD) is a rare chronic systemic bacterial infection caused by *Tropheryma whippelii*. It affects middle-aged men with diarrhea, weight loss, fever and joint manifestations. It usually develops as oligoarthritis or polyarthritis of large joints. WD may mimic chronic inflammatory rheumatisms. Some studies and case reports show that immunosuppressive treatment can worsen WD¹.

Objectives: Clinical and radiological (radiograph, ultrasound (US), MRI) presentation of joint manifestations of WD.

Methods: This retrospective monocentric observational study was led in Strasbourg University Hospital, Reference Center for Rare Diseases and included patients with WD diagnosed between 2006 and 2017. Patients were included if they had joint manifestations, positive *T.whippelii* polymerase chain reaction in stools or/and saliva, positive *T.whippelii* polymerase chain reaction on duodenal biopsy and/or positive periodic acid-Schiff performed on duodenal biopsy and good evolution with antibiotics.

Results: Nineteen patients including fifteen men were included. The median age at diagnosis was 57. The median time from first symptoms to diagnosis was 60 months. Before the diagnosis of WD, 13 patients had immunosuppressive therapy.

Clinical presentation was: 11 polyarthritis, 3 oligoarthritis, 1 monoarthritis and 4 inflammatory polyarthralgia including 3 with axial pain. Wrists (15/19), ankles (11/19) and knees (10/19) were the most affected. The metacarpophalangeal joints were affected in 8 patients. Eight patients had diarrhoea and weight loss.