Methods: Audit criteria were derived from the latest BSR gout guideline (Hui et al; 2017). A randomised sample of adult patients with a read code for the diagnosis of gout from Jan 2006-Jan 2018 was chosen from six large general practices in Leicestershire County of the United Kingdom. The data collected included demographics, provision of patient information, management of acute attacks and prophylactic treatment, screening of appropriate co-morbidities, dosing of urate-lowering therapy (ULT) and titration of doses against measurement of uric acid levels.

Results: Data was obtained for 861 patients. The mean age was 60 years and 91% were male, 21.5% were recorded as being provided with written information about gout and 60.5% of patients were treated with NSAIDs and COXIBs for acute attacks of gout. When colchicine was prescribed to patients, 71% had no dose recorded in their clinical records. 323 (37.5%) of patients were prescribed a ULT and the recorded starting dose of allopurinol was 100mg daily for 73.8%. Titration of subsequent allopurinol doses was recorded in only 21% of patients. 539 patients (62.6%) had no record of a serum urate level check after starting ULT.

Conclusion: Clinical records indicate that the management of gout by UK General Practitioners in Primary Care is suboptimal in concordance with the BSR guidelines. It was clear that general practices did not employ the treat to target strategy. There is a clear need for increased GP awareness and adherence to the BSR guidelines in order to optimise deficient areas of care, particularly in patient education, initiation and titration of ULT and monitoring of serum urate levels in gout patients. Appropriate patient recording templates are needed so that key information is captured during a patient consultation in order to enable medicines optimisation for those with gout. Most aspects of gout management in primary care did not concord well with published BSR guidelines.

REFERENCES

- [1] Kuo, C., Grainge, M.J., Mallen, C., Zhang, W. and Doherty, M. (2014) Rising burden of gout in the UK but continuing suboptimal management: a nationwide population study. *Annals of the Rheumatic Diseases*, [online] 74(4) pp.661-667.
- [2] Hui, M., Carr, A., Cameron, S., Davenport, G., Doherty, M., Forrester, H., Jenkins, W., Jordan, K., Mallen, C., McDonald, T., Nuki, G., Pywell, A., Zhang, W. and Roddy, E. (2017) The British Society for Rheumatology Guideline for the Management of Gout. *Rheumatology*, [online] 56 (7) pp. e1-e20.

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SAT0448 ATP IS THE SECOND KEY SIGNAL OF GOUT FLARE BESIDES MSU

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Background: Gout is an inflammatory disease associated with hyperuricemia and characterized by recurrent arthritis. In previous study, MSU which generated by hyperuricemia was recognized by the toll-like receptor and NOD receptor of the intrinsic immune system, then activated the NALP3 inflammasome to induce the secretion of IL-1 β , causing gout. However, this mechanism cannot explain why most patients with hyperuricemia do not have gout attacks in clinical practice, suggesting that there may be other pathogenic signals in the flare of gout. Our team previously found that P2X7R might play a key regulatory role in the pathogenesis of gout^[1]. What's more, single nucleotide polymorphisms associated with P2X7R function regulate the onset of gouty arthritis^[2], suggesting that ATP, as the ligand of P2X7R, may be a second key signal in addition to MSU to stimulate gout attack.

Objectives: To understand whether ATP act as a key factor in gout development besides MSU.

Methods: The rat hyperuricemia model was prepared by intraperitoneal injection of the uratease inhibitor oxazinic acid. Followed by ATP injection into the tail vein to observe the incidence of arthritis and compared with the Coderre's method-established gouty arthritis^[3].

Results: Forty hyperuricemia rats were prepared and 0.5ml ATP (10mM) solution was injected into the tail vein of the rat respectively, about 95% (38 rats) developed spontaneous arthritis. This type of spontaneous arthritis is very different from the Coderre's method in the following aspects: (1) The onset time of spontaneous arthritis is faster. The time of joint swelling in spontaneous arthritis peaked at 6.0 $\pm$ 2.0 hours after ATP injection, while it took 2 to 3 days for arthritis caused by local injection of

MSU to reach its peak. (2) The affected joints were different: The Coderre's method joints swelling only at the MSU injection site. Spontaneous arthritis can involve multiple joints, in which 71% have one joint affected, 21% have two joints affected and 8% with three or more than three joints affected by redness and swelling. (3) Distinct histopathologic characteristic: In Coderre's method, lymphocytes caused by local injection account for about 60%, with fewer neutrophils; In spontaneous arthritis, neutrophils account for more than 90% and lymphocytes are less. (4) Different sources of MSU: there is local MSU in spontaneous arthritis, which is associated with hyperuricemia; the local presence of MSU in Coderre's arthritis was artificially injected.

Conclusion: The spontaneous arthritis due to the synergistic effect of ATP and MSU is consistent with the characteristics of human gout arthritis, suggesting that ATP is the key second signal besides MSU to stimulate gout attack.

REFERENCES

- [1] Tao JH, Zhang Y, Li XP. P2X7R: A potential key regulator of acute gouty arthritis. *Semin Arthritis Rheum*. 2013; 43(3): 376-380.
- [2] Tao JH, Cheng M, Tang JP, et al. Single nucleotide polymorphisms associated with P2X7R function regulate the onset of gouty arthritis. *PLoS One*. 2017 Aug 10;12(8):e0181685.
- [3] Coderre TJ, Wall PD. Ankle joint urate arthritis (AJUA) in rats: An alternative animal model of arthritis to that produced by Freund's adjuvant. *Pain*. 1987; 28(3):379-93.

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SAT0449 EFFECT OF CHOLESTEROL AND TRIGLYCERIDE ON THE FREQUENCY OF GOUT ATTACKS

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Background: Gout is an autoinflammatory disease characterized by hyperuricemia and recurrent arthritis. ATP and MSU synergistically activate NALP3 inflammasome to induce the secretion of IL-1 β , leading to the onset of gouty arthritis, and P2X7R plays a key role in gout^[1,2]. This mechanism above can explain the clinical phenomenon that some patients with hyperuricemia never suffer gouty arthritis, however, it cannot explain why the frequency of gout attacks increases as the course prolongs. Cholesterol or triglyceride can activate the innate immune and induce inflammatory response. It is speculated that Cholesterol and triglyceride levels can increase with the duration of gout, and they may reduce the threshold of gout attacks.

Objectives: To demonstrate the effect of elevated cholesterol and triglyceride on the onset of gout.

Methods: A cohort study was performed to observe the difference of arthritis episodes between the high cholesterol group and the normal, the high triglyceride group and the normal in gout patients. The frequency of gout attacks was compared using statistical methods of independent sample test and paired sample test between the two groups.

Results: A cohort study was performed to observe the difference of arthritis episodes between the high cholesterol group and the normal, the high triglyceride group and the normal in gout patients. The frequency of gout attacks was compared using statistical methods of independent sample test and paired sample test between the two groups.

RESULTS: A total of 68 patients with gout were observed. Among them, 21 were in normal cholesterol group, 13 in elevated group, 21 in normal triglyceride group and 13 in elevated group. The results of the independent sample test between the two groups are as follows: (1) The frequency of gout attacks between the normal cholesterol group and the elevated group is statistically significant within three months, six months and one year (0.81 $\pm$ 0.60 vs 1.77 $\pm$ 0.83, Z=3.200, P=0.001; 1.14 $\pm$ 0.73 vs 3.15 $\pm$ 2.15, Z=-3.430, P=0.001; 1.43 $\pm$ 0.81 vs 4.77 $\pm$ 3.44, Z=-3.199, P=0.001). (2) The frequency of gout attacks between the normal triglyceride group and the elevated group is statistically significant within three months, six months and one year (0.81 $\pm$ 0.60 vs 1.54 $\pm$ 0.97, Z=-2.359, P=0.018; 1.14 $\pm$ 0.73 vs 2.38 $\pm$ 1.66, Z=-2.417, P=0.016; 1.43 $\pm$ 0.81 vs 3.54 $\pm$ 2.50, Z=-3.005, P=0.003). The results of the paired sample test between

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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REFERENCES

- [1] Tao JH, et al. P2X7R: A potential key regulator of acute gouty arthritis. *Semin Arthritis Rheum.* 2013; 43(3): 376-380.
- [2] Tao JH, et al. Single nucleotide polymorphisms associated with P2X7R function regulate the onset of gouty arthritis. *PLoS One.* 2017 Aug 10;12 (8):e0181685.

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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.

REFERENCES

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.