

Prospective 12-month ultrasound evaluation of carotid atherosclerosis, urate deposits, and musculoskeletal inflammation in newly diagnosed gout

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Abstract

Objective

To assess the longitudinal association between carotid atherosclerosis and ultrasound signs of urate crystal deposition and musculoskeletal inflammation in gout.

Methods

We carried out a single-centre, observational, prospective study including consecutive patients with crystal-proven gout from a rheumatology unit. Ultrasound assessments were performed at baseline (M0) and six months (M6) and 12 months (M12) after initiating urate-lowering therapy. Six joints and four tendons were scanned to detect urate crystal deposits (double contour, aggregates, and tophi); local inflammation was identified based on the power Doppler (PD) signal. The carotid arteries were scanned for increased intima-media thickness and atheromatous plaques according to the Mannheim consensus. The association between changes in carotid arteries, deposits, and PD signal in the M0-M6 and M6-M12 periods was studied using the chi-square and Fisher exact tests.

Results

Of the 103 patients included, 91 (88.3%) and 78 (75.7%) underwent the M6 and M12 evaluations, respectively. At M0, the mean number of locations with deposits and a positive PD signal was 9.9 (SD 4.1) and 1.1 (SD 1.1). Carotid atheromatous plaque was identified in 59%. During study, near of 97% received ULT and almost half of patients used lipid-lowering drugs. At M6, no association was found between changes in carotid and musculoskeletal variables. However, at M12, plaque regression was more common in those with an absent PD signal than in those with a persistent PD signal (64.0% vs. 28.6%, $p=0.017$). No association was noted with changes in crystal deposits.

Conclusion

After 12 months of urate-lowering treatment, the absence of musculoskeletal inflammation at ultrasound in gout patients was closely linked to improvements in carotid atheromatous plaques.

Key words

gout, ultrasonography, atherosclerosis, inflammation

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Introduction

Gout is characterised by inflammation of varying intensity. Such inflammation, along with oxidative stress driven by hyperuricaemia (1), probably impairs endothelial cell function. This in turn results in atherosclerosis and cardiovascular (CV) events, with associated morbidity and mortality. Indeed, gout patients are characterised by a higher prevalence of carotid atherosclerosis than in non-gouty population (2, 3) and greater all-cause and CV mortality (4, 5).

Previous reports demonstrated an association between the presence of carotid atheromatous plaques and the extent of sonographic urate crystal load (mainly tophi) and inflammatory load expressed as a positive power Doppler (PD) signal (3, 6). However, the studies were cross-sectional, and the association could not be firmly established.

This study aims to assess the relationship between changes in carotid atherosclerosis and changes in sonographic urate deposits and musculoskeletal inflammation after urate-lowering therapy (ULT) and flare prophylaxis in clinical practice. We hypothesised that improvements in urate deposits and PD signal would be associated with a reduction in atheromatous plaques.

Methods

Study design and population

This was a single-centre, observational, prospective study, conducted in the rheumatology unit of a tertiary university hospital with a catchment population of 295,197 inhabitants (2024). The participants were consecutively selected from incident cases in primary care, hospital admissions and emergency department at the time of a gout flare or after its subsidence. Patients with crystal-proven gout confirmed in synovial fluid or tophi were eligible for the study regardless of whether gout was newly diagnosed or previously established. No exclusion criteria applied. The first evaluation (M0) was performed without ULT, although previous use of ULT and flare prophylaxis was allowed. Patients' characteristics and baseline associations had been previously published (3). Patients were treat-

ed in parallel by their rheumatologists, who were aware of the scan results. During follow-up, two additional ultrasound assessments during intercritical periods were scheduled at 6 months (M6) and 12 months (M12) with the same sonographer (AMS or MA) that performed at baseline. Repeated musculoskeletal assessment was restricted to anatomical sites with deposits or inflammation at the previous evaluation. Carotid arteries were evaluated in all patients at each time point.

According to the Declaration of Helsinki, all participants signed a written informed consent. The local ethics committee approved the protocol (PI2018/027).

Ultrasound assessment

The two sonographers (AMS and MA) had broad ultrasound experience. Their intra- and interobserver reliability ranges from 0.65–0.75 ($p=0.001$) and 0.66 ($p=0.001$) (3), indicating good agreement. Both sonographers were blinded to clinical data. Musculoskeletal and carotid scans were performed at the same appointment. The ultrasound equipment was the Mindray DC-70 device, with a high-frequency (6–14 MHz) linear probe (L14-6NE model) and grey-scale and PD modalities. Frequencies of at least 12 MHz were used to scan for elementary lesions.

For the musculoskeletal assessment, the regions evaluated, adapted from Naredo *et al.* (7), were wrists, second metacarpophalangeal and first metatarsophalangeal joints, triceps and patellar tendons and their entheses. In each location, the domains evaluated were lesions of urate deposits: double contour [DC] sign (only for joints), aggregates, and tophi, following the 2015 OMER-ACT definitions (8), and inflammation (presence of a local PD signal graded 0–3 (9–11)).

The carotid arteries were bilaterally scanned for increased intima-media thickness (IMT) and the presence of atheromatous plaques, according to the Mannheim consensus, considering increased IMT to be above 0.9 mm (12). The evolution of plaques and IMT was evaluated separately.

At the follow-up visits, the sonographer

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Table I. Clinical variables by study period.

Variables	M0-M6 [n=91]	M6-M12 [n=78]
ULT, n (%)	88/90 (97.8)	75/77 (97.4)
Allopurinol, n (%)	82/88 (93.2)	68/77 (88.3)
Febuxostat, n (%)	6/88 (6.8)	7/77 (9.1)
Colchicine, n (%)	87/91 (95.6)	58/78 (74.4)
Cumulative dose of colchicine (mg), mean (SD)	0.56 (0.22) [n=88]	0.42 (0.3) [n=75]
Prednisone, n (%)	2/91 (2.2)	2/78 (2.6)
Cumulative dose of prednisone (mg), median (IQR P25-P75)	0 (0-0) [n=47]	0 (0-0) [n=43]
Serum urate (mg/dL), mean (SD)	6.14 (1.9) [n=86]	5 (1.5) [n=67]
Serum urate level <6 mg/dL, n (%)	47/86 (54.7)	51/66 (77.3)
CRP (mg/dL), mean (SD)	0.6 (0.96) [n=85]	0.5 (1.0) [n=64]
eGFR (mL/min/1.73 m ²), mean (SD)	72.3 (22.6) [n=86]	72.8 (20.6) [n=69]
CKD-EPI (eGFR <60 mL/min/1.73 m ²), n (%)	20/86 (23.3)	20/69 (29.0)
Stage 4-5 CKD-EPI (eGFR <30 mL/min/1.73 m ²), n (%)	4/86 (4.7)	0/69 (0.0)
Total cholesterol (mg/dL), mean (SD)	175.6 (40.6) [n=85]	167.2 (34.2) [n=66]
Non-HDL cholesterol (mg/dL), mean (SD)	132.1 (39.5) [n=79]	119.4 (35.6) [n=58]
LDL cholesterol (mg/dL), mean (SD)	113.2 (39) [n=76]	97.7 (31.2) [n=54]
HDL cholesterol (mg/dL), mean (SD)	45.8 (10.7) [n=79]	46.5 (10.9) [n=58]
Triglycerides (mg/dL), mean (SD)	157.6 (77.6) [n=84]	157.8 (77.3) [n=62]
Use of statins, n (%)	45/91 (49.5)	42/78 (53.8)
Use of high-potency statins*, n (%)	14/91 (15.4)	13/78 (16.7)

*High-potency statins were defined as at least 20 mg rosuvastatin and at least 40 mg atorvastatin.

M0: month 0 (baseline); M6: month 6; M12: month 12; ULT: urate-lowering therapy; SD: standard deviation; IQR: interquartile range; CRP: C-reactive protein; eGFR: estimated glomerular filtration rate; CKD-EPI: chronic kidney disease epidemiology collaboration; HDL: high-density lipoprotein; LDL: low-density lipoprotein.

For continuous variables, values in brackets indicate the number of valid observations used for the calculation. Categorical variables are presented over the number of valid cases. Missing data are excluded from the analysis.

classified the evolution of deposits, carotid plaques and PD signal as ‘disappearance’, ‘reduction’, ‘persistence’, and ‘progression’ compared to the previous evaluations (M6 vs. M0, M12 vs. M6). The maximum grade in PD signal in the whole assessment was taken as the reference at M0. Absolute results for IMT thickness were also compared between periods.

Variables

The outcome variables were changes in IMT and atheromatous plaques by period. The primary explanatory variables were variations in individual urate deposits and in PD signal by period.

Secondary explanatory variables included ULT (allopurinol or febuxostat), prophylactic agents (colchicine or corticosteroids), cumulative dose of corticosteroids, estimated glomerular filtration rate (eGFR), C-reactive protein (CRP), and serum urate (SU) and lipid levels by period. We recorded the use of high-potency statins (20–40 mg rosuvastatin and 40–80 mg atorvastatin) because these agents can lead to plaque stabilisation/reduction (13). The clinical and laboratory variables were collected from participants’ case files.

Statistical analysis

Descriptive statistics are presented as frequencies (n) and percentages (%) for qualitative variables and as mean (standard deviation) and median (interquartile range) for quantitative variables. Changes in plaques, urate deposits and PD signal between periods were dichotomised into ‘disappearance/reduction’ or ‘persistence/progression’ for the statistical analyses and were analysed using the chi-square and Fisher exact tests. Changes in IMT were evaluated by comparing period-specific means with the paired - test. To assess the impact of secondary variables on changes in carotid plaques, we built a bivariate logistic regression model calculating odds ratio (OR) with its 95% confidence interval (CI). No specific approaches were followed to deal with missing data. The analysis was performed using IBM SPSS Statistics, v. 25.0. Statistical significance was set at $p < 0.050$.

Results

Clinical characteristics

The study population comprised 103 patients included between December 2017 and June 2020. Patients were of moderate age (62.3 years) and predomi-

nantly men (94%) with significant multimorbidity, such as cardiovascular disease (22.3%), chronic kidney disease (25.2%), diabetes mellitus (22.3%), dyslipidaemia (55.3%) or hypertension (60.2%). The median time from the first flare to enrolment was 4 years (IQR 0.0–10.0). Tophi were present in 16.7%, and 43.7% experienced oligo- or poly-articular flares. At baseline, 38.8% were on diuretics and 35.9% on lipid-lowering therapy (3).

At M6 and M12, 91 (88.3%) and 78 (75.7%) were evaluated respectively. Table I presents their treatment and laboratory data by period. Patients dropping at M12 were found similar to those attending M12 according to their baseline characteristics, besides being the attendants more obese (54.7% vs. 26.1%, $p = 0.016$).

Approximately 97% received ULT, mainly allopurinol. Colchicine was the most common flare prophylaxis, less frequent during the M6-M12 period. At follow-up, more patients used lipid-lowering drugs and nearly 16% were high-potency statins. The laboratory data revealed a progressive reduction in SU and low-density lipoprotein (LDL) cholesterol levels.

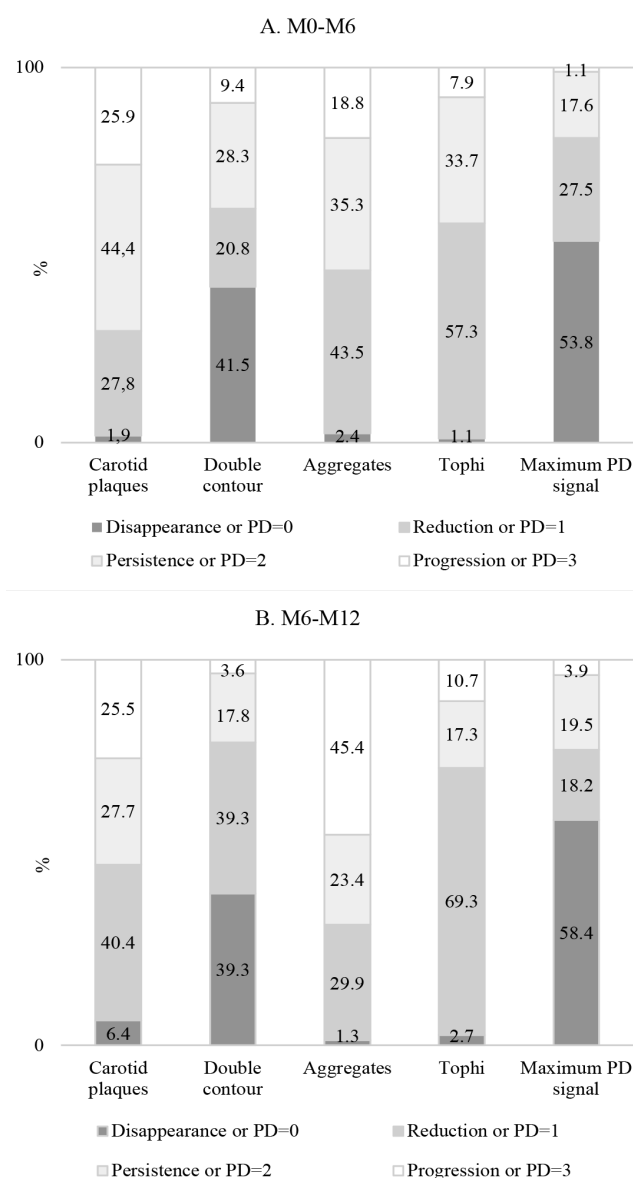


Fig. 1. Evolution of carotid plaque, urate deposits, and power Doppler (PD) signal by period. M0: month 0 (baseline); M6: month 6; M12: month 12; PD: power Doppler.

Ultrasound assessment

At baseline, ultrasound revealed urate deposits in all participants, with a mean sum of 9.9 (SD 1.1): DC sign, 0.9 (SD 1.1), aggregates, 4.1 (SD 2.8), and tophi, 4.9 (SD 2.3). A positive PD signal was observed in 67.0%, and the mean number of locations with a positive PD signal was 1.1 (SD 1.1) (0.4 [SD 0.7] with PD signal 2–3). Fifty-nine percent (n=61) had carotid plaques. The mean IMT was 0.82 mm (SD 0.21), and this increased in 33 participants (32%). Figure 1 shows the evolution of carotid plaques, urate deposits, and PD signal by period. At M6, the DC sign and PD signal had disappeared in nearly half

of the patients. Plaques and tophi decreased, more pronounced at M12. Aggregates tended to increase at M12. IMT remained unchanged at M6 (mean 0.81 mm, SD 0.2) and M12 (mean 0.84 mm, SD 0.2) compared to M0 ($p=0.80$ and $p=0.82$).

Association analysis

Table II presents the changes in sonographic deposits and PD signal in plaques by period after dichotomisation. At M6, no association was found between carotid and musculoskeletal variables. However, at M12, reduction or disappearance of plaques was more common in those with an absent

PD signal (64.0% vs. 28.6%, $p=0.017$). Improvement in carotid plaques was statistically unrelated to improvements in the DC sign (62.5% vs. 0%, $p=0.087$), aggregates (57.9% vs. 37%, $p=0.162$), and tophi (48.4% vs. 42.9%, $p=0.731$).

The use of high-intensity statins was also associated with the reduction/disappearance of atheroma plaques at 12 months (OR 0.17; 95%CI 0.03–0.97; $p=0.046$). No relationship was observed with other variables (Supplementary Table S1).

Discussion

This study revealed that reducing sonographic musculoskeletal inflammation in gout patients after one year of ULT was associated with an improvement in carotid plaques. Although improvements in urate deposit were also observed, these changes were not directly associated with improvements in atherosclerosis. The use of high-intensity statins was also associated with carotid plaque regression, which, interestingly, was not mediated by LDL cholesterol levels. This finding is consistent with the pleiotropic effects of statins, including anti-inflammatory action and plaque stabilisation (13).

Our findings are supported by prior imaging studies in gout (3), which demonstrated that crystal and inflammatory burdens are associated with carotid atherosclerosis. A similar pattern was observed in rheumatoid arthritis (14), where active joint inflammation detected by 18F-PET-CT and MRI exhibited increased metabolic activity in carotid plaques.

Clinical trials CANTOS (15), with canakinumab, and COLCOT (16), with colchicine have proven able to prevent CV events in patients with subclinical inflammation. Specifically in gout, Solomon *et al.* (17) reported lower rates of CV events and mortality in gout patients receiving colchicine. Cipolletta *et al.* (18) observed a reduction in CV events among patients initiating ULT with prophylactic colchicine, compared to those not receiving colchicine.

Accordingly, it seems crucial to conduct an early articular and vascular assessment for newly gout patients and to con-

Table II. Association between changes in sonographic deposits and PD signal and changes in carotid atheromatous plaque.

			Atheromatous plaque		p
			Disappearance/reduction, n (%)	Persistence/progression, n (%)	
Double contour	M6 - M0	Disappearance/Reduction, n (%)	7 (38.9)	11 (61.1)	0.237
		Persistence/Progression, n (%)	2 (15.4)	11 (84.6)	
	M12 - M6	Disappearance/Reduction, n (%)	10 (62.5)	6 (37.5)	0.087
		Persistence/Progression, n (%)	0 (0.0)	4 (100.0)	
Aggregates	M6 - M0	Disappearance/Reduction, n (%)	6 (25.0)	18 (75.0)	0.459
		Persistence/Progression, n (%)	9 (34.6)	17 (65.4)	
	M12 - M6	Disappearance/Reduction, n (%)	11 (57.9)	8 (42.1)	0.162
		Persistence/Progression, n (%)	10 (37.0)	17 (63.0)	
Tophi	M6 - M0	Disappearance/Reduction, n (%)	10 (31.3)	22 (68.8)	0.556
		Persistence/Progression, n (%)	5 (23.8)	16 (76.2)	
	M12 - M6	Disappearance/Reduction, n (%)	15 (48.4)	16 (51.6)	0.731
		Persistence/Progression, n (%)	6 (42.9)	8 (57.1)	
PD signal	M6 - M0	Disappearance, n (%)	9 (34.6)	17 (65.4)	0.439
		Persistence, n (%)	7 (25.0)	21 (75.0)	
	M12 - M6	Disappearance, n (%)	16 (64.0)	9 (36.0)	0.015
		Persistence, n (%)	6 (28.6)	15 (71.4)	

p-values were considered significant below 0.050 (bold).

M0: month 0 (baseline); M6: month 6; M12: month 12; PD: power Doppler.

sider intervention in at-risk populations. The main strengths of our study are its prospective design, the use of validated imaging modalities, and the blinding of the observers.

Some limitations should be noted. Baseline evaluations included patients not receiving ULT, although prior ULT exposure and flare prophylaxis were allowed, introducing heterogeneity in disease and vascular status. Only 75.7% of participants completed 12-month follow-up, which might have affected power and introduced bias, as the reason for dropouts was not systematically obtained. Concomitant medications, including diuretics, statins, and other therapies, could have influenced urate, lipid levels, and vascular outcomes. The sample size was based on the baseline cross-sectional study, and participants from specialised care had more severe disease and comorbidities, limiting generalisability. Adherence to ULT was not formally assessed, although decreasing serum urate was reassuring. An additional limitation concerns the ultrasound scoring systems used. We used 2015 OMERACT definitions for gout-specific lesions,

available when the study was designed; however, more recent semiquantitative scoring systems (19) might allow a more accurate estimation of deposit extent and improved characterisation of participants' evolution. Finally, the study design allows us to identify associations, but not to establish definitive causality. Although we observed a relationship between certain variables and the outcomes, it is possible that unmeasured factors or confounding factors contributed to these findings. Use of high-intensity statins, for example, was an associated variable, and its anti-inflammatory effects could be a significant confounder.

Conclusion

Ours is the first study to prospectively demonstrate that the improvement in musculoskeletal inflammation under ULT among patients with gout may also have a positive impact on reducing CV risk, as evidenced by regression in atheromatous plaques. This finding provides additional evidence for the link between crystal-driven inflammation and atherosclerosis in patients with gout.

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