High serum uric acid concentration is a risk factor for radiographic spondyloarthritis: a cross-sectional study

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Abstract

Background: Cardiovascular disease is a kind of comorbidity of spondyloarthritis (SpA). Hyperuricaemia is a risk factor of cardiovascular disease. Previous SpA study concerning cardiovascular disease and hyperuricaemia found that hyperuricaemia is more prevalent in radiographic SpA. Thus, we evaluated the association of serum uric acid (SUA) concentration and radiographic SpA.

Methods: We made use of data from a cross-sectional study in Chinese Shenzhen Second People's Hospital from 2016 to 2018, which included 202 SpA patients diagnosed by rheumatologists. This study detected the prevalence of comorbidities and risk factors of SpA patients. Using data from this study, we evaluated the association between radiographic images of SpA and SUA concentration. We compared the SUA concentration between radiographic SpA group and non-radiographic SpA group, with logistic regression models. Stratified and interaction analyses were also performed to further confirm the consistence of the relationship. Multiple imputation was used to deal with missing data.

Results: This research studied 202 SpA patients' data including their SUA, sacroiliac images, and other relevant laboratory data. Elevated SUA concentration was found to be associated independently with the increased risk of radiographic SpA after adjusted for confounders. The risk for developing radiographic SpA increased 13% [adjusted odds ratio (OR) = 1.13, 95% confidence interval (CI):(1.05, 1.22), $p=0.0014$] for per 10umol/L SUA increased. Stratified analyses, interaction analyses and multiple imputation analyses also confirmed the consistence of the association.

Conclusions: Based on this Chinese population cross-sectional study, we identify that elevated SUA concentration is a risk factor for developing radiographic SpA.

Background

Spondyloarthritis(SpA) includes axial and peripheral SpA, according to the Assessment of SpondyloArthritis international Society(ASAS) classification criteria.[1, 2] Criteria for both axial and peripheral SpA include sacroiliitis. Sacroiliitis on imaging plays an important role in the assessment of SpA. Structural sacroiliitis on sacroiliac joint image is named radiographic SpA, while normal sacroiliac joint without structural sacroiliitis is named non-radiographic SpA.[3–5] It is controversial whether radiographic and non-radiographic SpA should be considered as two different diseases or as a continuous disease spectrum. The currently prevailing opinion is that SpA encompasses the disease spectrum in which patients with non-radiographic SpA may develop into radiographic SpA over time. However, not all patients with non-radiographic sacroiliitis will ultimately develop radiographic changes.[6] There may be some factors influence the development from non-radiographic SpA to radiographic SpA.

In the recent years, a number of studies have focused on the cardiovascular comorbidities and risk factors in SpA patients.[7] Hyperuricaemia has been considered as a risk factor of cardiovascular diseases.[8–10] Recently, this association has been confirmed pathologically by dual-energy computed

tomography (CT) which demonstrates cardiovascular monosodium urate (MSU) crystal deposits, with identification of polarized light microscopy.[11] Since elevated serum uric acid (SUA) increase the MSU crystal deposits which subsequently destruct the normal histological structure and lead to corresponding cardiovascular disease. MSU crystal deposits at sacroiliac joints may lead to sacroiliac destruction. Actually, in recent researches of structural sacroiliitis, MSU deposits in sacroiliac joints are detected by dual-energy CT and ascertained by polarized light microscopy in SpA patients.[12, 13] This pathologic discovery may lead to a hypothesis whether elevated serum uric acid (SUA) concentration is a risk factor of structural sacroiliitis.

Additionally, our previous published study, with respect to SpA comorbidities and risk factor, has confirmed that prevalence of hyperuricaemia in radiographic SpA subgroup was 40·4%, while in non-radiographic SpA subgroup was 27·9%.[14] Thus, we hypothesized that in SpA patients elevated SUA concentration may be associated with structural sacroiliitis, which is also named radiographic SpA. Detection of the association between SUA concentration and radiographic SpA in this Chinese population was conducted.

Methods

Study design

This is a cross-sectional study, with data extracted from hospitals information systems.

Patients

For this cross-sectional study, data were collected from SpA patients in Shenzhen Second People's Hospital inpatient and outpatient departments between January 1, 2016 and December 31, 2018. The study was approved by the Ethics Committee of Shenzhen Second People's Hospital, with approval ID 20200224001. What is more, written consent was not required for this retrospective study if individual privacy and commercial interests were not involved, according to the medical ethical law of China.

Data collection

During the initial consultation, patients were asked whether they suffered from one or more of the following symptoms including spinal manifestations, arthritis, enthesitis (heel), dactylitis, psoriasis, uveitis, and Crohn's/ulcerative colitis, when they consulted at inpatient and outpatient departments of Immunology and Rheumatology, Ophthalmology, or Gastroenterology. The medical files of the patients, who suffered from the manifestations discussed above, were extracted from the information systems with searching keyword of SpA, uveitis, and Crohn's/ulcerative colitis. Furthermore, additional information

about human leucocyte antigen B27 (HLA-B27) and sacroiliac joint imaging (including radiography, CT) were extracted from the clinical laboratory system and imaging system as well.

All the medical files were examined to see if the diagnosis of axial or peripheral SpA could be confirmed. Once the diagnosis of axial and peripheral SpA was confirmed, the following chapters of data were collected.

1. Demographics and disease characteristics were collected. Disease activity was detected by the Bath Ankylosing Spondylitis Disease Activity Index (BASDAI).[15] The Ankylosing Spondylitis Disease Activity Score was calculated with the CRP (ASDAS).[16,17] Disease severity was measured by the Bath Ankylosing Spondylitis Functional Index (BASFI).[18] Past and current medications including non-steroidal anti-inflammatory drug (NSAID), corticosteroids, conventional synthetic disease-modifying antirheumatic drugs (DMARDs)), and tumour necrosis factor inhibitor (TNFi) were also collected.
2. Extra-articular manifestations including uveitis, psoriasis, and IBD (Crohn's/ulcerative colitis) were examined.
3. Laboratory data included uric acid, creatinine, and HLA-B27. The unit of measurement for uric acid was $\mu\text{mol/L}$, which was relatively tiny for observing changes. Thus, we transform the unit of measurement for uric acid from $\mu\text{mol/L}$ into $10\mu\text{mol/L}$, after collecting the original data of uric acid.
4. We examined all the patients for sacroiliac joint image: Radiographic SpA is defined as grade II bilaterally or grade III–IV unilaterally, according to the Modification of the New York Criteria (mNYC) for radiography.[3] It can also be defined as an erosion score and/or joint space score of 2 or higher in any of the 24 regions of both joints for computed tomography (CT).[4] Non-radiographic SpA is defined as sacroiliac joint image without structural sacroiliitis described above.[5] Sacroiliac images were evaluated by a rheumatologist and a radiologist, separately, independently, and retrospectively. Discordant examinations of CT were discussed between the rheumatologist and the radiologist to establish a final consensus of structural sacroiliitis.

Sample size calculation

It is required a sample size of 130 patients to detect a difference, based on a power of 80% and a two-sided significance level of 0.05. The sample size collected in this study is 202, which meets the criterion of statistics.

Data analysis

First, descriptive data analysis and comparison of patients from non-radiographic SpA group and radiographic SpA group were conducted, in terms of the demographic and disease characteristics,

activity, and severity. Student t test (normal distribution) and Mann-Whitney (non-normal distribution) were performed for continuous variables and χ^2 tests were used for categorical variables. Second, risk factors for radiographic SpA group were analyzed by univariate and multivariate logistic regression with generalized estimating equations. Third, to evaluate the consistent association between serum uric acid and radiographic SpA, stratified analyses were performed with the individual covariates using stratified GAM models. Tests for effect of the modification by strata used interaction terms between strata indicators and serum uric acid, following the likelihood ratio test. Finally, Missing values were multiple imputed according to the Fully Conditional Specification method.[19] We used multiple multivariate imputations to handle the missing data of covariates (exposure and outcome excluded). The missing data analysis procedures used missing at random (MAR) assumptions. We created 5 imputed datasets, using a mice software package (based on chained equations). Therefore, we created complete data for analysis. In addition, we used sensitivity analysis to check whether imputed complete data were significantly different from pre-imputation data.

Data were analyzed, using the statistical packages R (The R Foundation; <http://www.r-project.org>; version 3.4.3) and EmpowerStats (www.empowerstats.com; X&Y Solutions Inc.).

Results

Between January 1 2016 and December 31 2018, 202 individuals were included in this study. Individuals who were not examined for renal function (n=111) were excluded. Individuals who were not examined for sacroiliac joint imaging (n=21) were excluded. The study profile of this study was published in the former article.[14]

Demographics and disease characteristics

Demographics and disease characteristics are listed in table 1. We divided our subjects into non-radiographic SpA group and radiographic SpA group. The percentages of males in the two groups were 57.38% and 79.43% ($p=0.001$), respectively. Compared with the non-radiographic SpA group, the radiographic SpA group was associated with higher level of disease duration ($p<0.001$), SUA ($p<0.001$), axial involvement(0.019), BASDAI($p=0.020$), ASDAS-CRP($p=0.017$), and the usage of [conventional synthetic DMARDs](#)($p=0.002$). No significant difference was found in the result between radiographic group and non-radiographic group, with respect to age, ever gout, peripheral involvement, enthesitis involvement, dactylitis, uveitis, psoriasis, IBD, ESR, CRP, creatinine, BASFI, and usage of NSAID.

Association between SUA and radiographic SpA

Univariate logistic regression analyses for crude association between clinical variables and radiographic SpA are presented in Supplementary Table S1. In the univariate analyses, we found that male gender,

disease duration (months), axial involvement (any), conventional synthetic DMARDs, BASDAI, ASDAS-CRP, HLA-B27, and SUA were associated with radiographic SpA ($p < 0.05$).

Multivariate regression analyses for associations between elevated SUA concentration (per 10 $\mu\text{mol/L}$) and radiographic SpA are demonstrated in Table 2. In the unadjusted model, SUA was found to be significantly associated with radiographic SpA [odds ratio (OR) = 1.06; 95% confidence interval (CI), 1.03–1.10; $p < 0.001$]. Elevated SUA was associated with increased risk of radiographic SpA independently even after adjusted the confounders. In the fully adjusted model, the risk for developing radiographic SpA increased 13% [adjusted odds ratio (OR) = 1.13, 95% confidence interval (CI): (1.05, 1.22), $p = 0.0014$] for per 10 $\mu\text{mol/L}$ SUA increased. Then we divided SUA (10 $\mu\text{mol/L}$) into 3 categories as low tertile (14.30–33.00), middle tertile (33.19–41.66) and high tertile (41.70–76.94). The low tertile category included 67 patients, with SUA (10 $\mu\text{mol/L}$) of 26.95 ± 4.61 . The middle tertile category included 67 patients, with SUA (10 $\mu\text{mol/L}$) of 37.50 ± 2.38 . The high tertile category included 68 patients, with SUA (10 $\mu\text{mol/L}$) of 49.44 ± 8.14 . When using SUA as categorical variables, multivariate logistic regression analyses were conducted again to further confirm the consistent association between SUA and radiographic SpA.

Stratified analyses and interaction analyses

The results of the stratified analyses of the association between the SUA and radiographic SpA are presented in Table 3. All the demographics and disease characteristics of patients were all stratified. When the SUA increased, the risk of radiographic SpA increased consistently and steadily for almost all the strata, though positive relations were not statistically significant. The association was also not significant between Radiographic SpA and difference of all the strata (p for interaction > 0.05).

The interaction analyses revealed that the association between the SUA and radiographic SpA was not interacted with other factors consistently.

Smooth curve fitting

Furthermore, in the smooth curve fitting model adjusted for potential confounders, the predictive risk of radiographic SpA generally increased with rising level of SUA (Fig. 1). A roughly positive non-linear correlation was observed between SUA and radiographic SpA. The smooth curve exhibited a three-stage change with two breakpoints which are 26.5 (10 $\mu\text{mol/L}$) and 54.29 (10 $\mu\text{mol/L}$). The risk of radiographic SpA increased slowly at the stage below SUA of 26.5 (10 $\mu\text{mol/L}$), increased faster at the stage of SUA between 26.5 (10 $\mu\text{mol/L}$) and 54.29 (10 $\mu\text{mol/L}$), and increased slowly again at the stage over SUA of 54.29 (10 $\mu\text{mol/L}$).

Sensitivity analysis

Sensitivity analysis was performed for the association of SUA and radiographic SpA. After missing data (including 7 for disease duration, 3 for HLA-B27, 16 for ESR, 4 for CRP, 80 for BASDAI, 84 for ASDAS-CRP, 80 for BASFI) were multiple imputed, logistic analysis was performed again on the imputed data. The result shows that the risk of developing radiographic SpA increased 7% [adjusted odds ratio (OR) = 1.07, 95% confidence interval (CI):(1.02, 1.12), $p=0.004$] for per 10umol/L SUA increased, which was consistent with that of the pre-imputation data, 13% increase [adjusted odds ratio (OR) = 1.13, 95% confidence interval (CI):(1.05, 1.22) , $p=0.0014$] for per 10umol/L SUA increased as reported in table 2.

Two cases

At the end of the result section, figure 2 demonstrated two separate patients' CT sacroiliac images comparison. Patient 1 (image A-D) was a 30 years old male, HLA-B27 positive, with disease duration of 120 months and SUA 489.2umol/L. Patient 2 (image E-H) was a 32 years old male, HLA-B27 positive, with disease duration of 120 months and SUA 361.4umol/L. Patient 1 presented radiographic SpA, while patient 2 presented non radiographic SpA.

Discussion

Few population-based studies to date have examined the relation between SUA and radiographic SpA, our study offers some new insights on this area. Our study reported that elevated SUA concentration was a risk factor for developing radiographic SpA among patients with SpA. The risk of developing radiographic SpA increased 13% [adjusted odds ratio (OR) = 1.13, 95% confidence interval (CI): (1.05, 1.22), $p=0.0014$] for per 10umol/L SUA increased.

Based on our results, we hypothesize that the potential biologic mechanism, by which SUA interacts with radiographic SpA, is that high level SUA leads to deposits of monosodium urate (MSU) and MSU deposits destroy the structure of sacroiliac joints.

In the past, reports already confirmed that high serum uric acid increased the burden of monosodium urate (MSU) deposits.[20,21] Previous studies reported that MSU deposits would lead to structural damage in corresponding organs where the MSU deposits were found. A nephrological research found that high SUA led to uric acid super saturated in the urine, and supersaturation in the urine increased

monosodium urate (MSU) deposits in the kidney formed uric acid stones.[22] In a research of coronary heart disease, high SUA led to MSU deposits in aorta and coronary arteries, which was assessed by dual-energy computed tomography (DECT) and verified by polarizing microscope.[11] Arthritic study also reported that MSU deposits could be detected in different joints of other rheumatic arthritis diseases except gout.[23] It had been ascertained by a number of surgeries for axial arthritis, that MSU deposits were found at the axial joints (including sacroiliac joints), when pathological examination was performed. [24-27] In the recent years, more and more rheumatologists and radiologists started to focus on the similarities and differences between classical SpA and the damage caused by the MSU crystal deposition at the sacroiliac joint.[28,29] In a rheumatology research for SpA, the MSU crystal deposition at the sacroiliac joints, detected by Dual-energy computed tomography, was associated with the progress of radiographic grade at sacroiliac joints in those patients.[30] The outcome of that rheumatology research supported our result.

Thus, our study proved that hyperuricaemia is a risk factor for developing radiographic SpA, built on the former studies which confirmed that hyperuricaemia leads to MSU deposits at sacroiliac joints and structural sacroiliitis.

Our study has several strengths. First, this research included patients who met the classification criteria of either axial spondyloarthritis or peripheral spondyloarthritis. Classification criteria for both of those diseases include sacroiliitis. We evaluated the images of sacroiliac joints in both axial SpA and peripheral SpA. Unlike some previous studies which detected sacroiliac CT for radiographic sacroiliitis only in the axial SpA patients, the criteria of this study which included both axial and peripheral SpA diagnosis would reduce bias. This evaluating measurement offers some new insights on this area. Second, the medical files and laboratory data were obtained from hospital information system. All the data were measured according to a standard protocol. We considered and adjusted for many potentially confounding factors. Population of this study was based on a Chinese hospital, which minimized the confounding effect of lifestyle. Most importantly, the study results consistently demonstrate a strong relation between SUA and radiographic SpA, defining SUA either as a continuous variable or as a categorical variable. Sensitive analyses were also performed to confirm the consistence of outcomes between multiple imputed data and pre-imputation data.

There are also limitations for the study. The cross-sectional study could not define casual effect of serum uric acid and the development of radiographic SpA. Thus, we need cohort study to further demonstrate the relation between SUA and radiographic SpA. Furthermore, the sample size of this study was not large enough for stratified analyses. When stratified by the clinical variables, the sample size for each stratum

limited the statistic significance of the result. Thus, cohort study with larger sample size is needed to confirm the consistence of this result among all the patients of different characteristics.

In summary, this Chinese population-based study of SpA found a significant positive relation between SUA and radiographic SpA. The study findings, if further confirmed, will have important implications. Additional research is needed including an intervention study to evaluate whether decreasing SUA concentration leads to remission of clinical symptoms and stabilization of radiographic structural SpA. Further elucidation of how SUA interacts with radiographic SpA may provide us with its specific biologic mechanism, and a new therapy for radiographic SpA.

Declarations

Competing interests

None declared.

Ethics approval and consent to participate

The study was approved by the Ethics Committee of Shenzhen Second People's Hospital, with the approval ID 20200224001.

Consent for publication

Not applicable.

Availability of data and materials

Data are available upon reasonable request.

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Author's contribution

YL and YZ were co-first authors. JX, CX, and YL were joint corresponding authors. YL and CX designed the research protocol. YL, YZ, ZL, YH, and TN extracted data from medical files in the information system

and interpreted the data. YZ extracted data from the laboratory system. YL and PH extracted data from the image system and evaluated the sacroiliac CT images. YH, XL and PH formatted graphs and diagrams. YL, YZ, ZL, and XL performed the statistical analysis. YL, YZ, and JX wrote the manuscript. YL, YZ, JX and CX approved the version submitted. All authors contributed to conducting the study.

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Tables

Table 1: Demographics and disease characteristics of the patients

	Non-radiographic SpA	Radiographic SpA	p-value
Number of patients	61	141	NA
Age(years)	37.59 ± 12.68	38.45 ± 12.54	0.655
Gender(males)	35/61 (57.38%)	112/141(79.43%)	0.001
Current smoker	6/61 (9.84%)	29/141 (20.57%)	0.064
Alcohol	4/61 (6.56%)	23/141 (16.31%)	0.061
Ever gout	3/61 (4.92%)	11/141 (7.80%)	0.459
Disease duration ^a (months)	37.78 ± 53.23	107.07 ± 89.49	<0.001
Axial involvement (any)	50/61 (81.97%)	131/141 (92.91%)	0.019
Peripheral involvement (any)	29/61 (47.54%)	75/141 (53.19%)	0.461
Enthesitis involvement	9/61 (14.75%)	16/141 (11.35%)	0.500
Dactylitis	3/61 (4.92%)	6/141 (4.26%)	0.834
Uveitis	12/61 (19.67%)	23/141 (16.31%)	0.562
Psoriasis	2/61 (3.28%)	8/141 (5.67%)	0.471
IBD	0/61 (0.00%)	4/141 (2.84%)	0.184
ESR ^a	26.32 ± 30.70	27.26 ± 22.85	0.821
CRP ^a	17.02 ± 37.06	16.24 ± 21.22	0.852
Creatinine (umol/L)	67.35 ± 14.58	70.14 ± 16.57	0.257
SUA (10umol/L)	33.98 ± 9.98	39.77 ± 10.66	<0.001
HLA-B27 ^a	59/61 (96.72%)	118/138 (85.51%)	0.020
BASDAI ^a	2.62 ± 2.19	3.51 ± 1.69	0.020
ASDAS-CRP ^a	2.19 ± 1.44	2.76 ± 0.98	0.017
BASFI ^a	1.28 ± 1.95	2.04 ± 2.32	0.100
NSAID	32/61 (52.46%)	75/141 (53.19%)	0.924
conventional synthetic DMARDs	6/61 (9.84%)	43/141 (30.50%)	0.002
TNFi	3/61 (4.92%)	18/141 (12.77%)	0.093

ALL results are presented as mean±SD for continuous variables and presented as numbers (percentages) for categorical variables.

BASDAI: Bath Ankylosing Spondylitis Disease Activity Index; ASDAS: Ankylosing Spondylitis Disease Activity Score-CRP; BASFI: Bath Ankylosing Spondylitis Functional Index; IBD: inflammatory bowel disease; SUA: serum uric acid; HLA-B27: human leucocyte antigen B27; NSAID: non-steroidal anti-inflammatory drug; DMARDs: disease-modifying antirheumatic drugs; TNFi: tumour necrosis factor inhibitor.

^aNumbers that did not add up to 100% were attributable to missing data.

Table 2: Association of SUA and radiographic SpA

Unadjusted Model			Multivariate Adjusted Model 1		Multivariate Adjusted Model 2	
Variable	OR (95% CI)	p	OR (95% CI)	p	OR (95% CI)	p
SUA	1.06 (1.03, 1.10)	0.0006	1.05 (1.01, 1.09)	0.0096	1.13 (1.05, 1.22)	0.0014
SUA Tertile						
14.30-33.00	1.0	NA	1.0	NA	1.0	NA
33.19-41.66	2.38 (1.15, 4.95)	0.0198	2.04 (0.92, 4.52)	0.0791	4.56 (1.30, 16.01)	0.0178
41.70-76.94	3.13 (1.46, 6.69)	0.0033	2.60 (1.09, 6.24)	0.0316	13.28 (2.72, 64.77) 64.64.77)64.77)64.77)64.77)64.77)64.77)64.77)	0.0014

Multivariate logistic regression models evaluated the association of uric acid and radiographic SpA. Model 1 was adjusted for age, gender. Model 2 was adjusted for age, gender, current smoker, alcohol, ever gout, disease duration, axial involvement (any), peripheral involvement (any), enthesitis involvement, dactylitis, psoriasis, uveitis, IBD, HLA-B27, ESR, CRP, Creatinine, NSAID, conventional synthetic DMARDs, and TNFi. SUA=Serum uric acid.

Table 3: Stratified analyses of association between SUA and radiographic SpA

	n	OR	(95%CI)	p	p for interaction
Gender					0.9550
Females	55	1.04	(0.97, 1.12)	0.2255	NA
Males	147	1.05	(1.00, 1.09)	0.0452	NA
Current smoker					0.6361
No	167	1.06	(1.02, 1.09)	0.0028	NA
Yes	35	1.08	(0.97, 1.21)	0.1477	NA
Alcohol					0.2384
No	175	1.06	(1.03, 1.10)	0.0008	NA
Yes	27	0.97	(0.85, 1.12)	0.7125	NA
Ever gout					0.5372
No	188	1.06	(1.02, 1.10)	0.0023	NA
Yes	14	1.10	(0.97, 1.25)	0.1345	NA
Peripheral involvement (any)					0.2858
No	98	1.08	(1.03, 1.14)	0.0032	NA
Yes	104	1.04	(1.00, 1.09)	0.0498	NA
Axial involvement (any)					0.5756
No	21	1.09	(0.98, 1.21)	0.1186	NA
Yes	181	1.06	(1.02, 1.09)	0.0029	NA
Enthesitis involvement					0.7649
No	177	1.06	(1.02, 1.10)	0.0012	NA
Yes	25	1.05	(0.96, 1.14)	0.2705	NA
Dactylitis					0.6452
No	193	1.06	(1.02, 1.10)	0.0008	NA
Yes	9	1.13	(0.85, 1.51)	0.4035	NA
Uveitis					0.8004
No	167	1.06	(1.02, 1.10)	0.0037	NA
Yes	35	1.07	(0.99, 1.15)	0.0723	NA
Psoriasis					0.1345
No	192	1.07	(1.03, 1.11)	0.0003	NA
Yes	10	0.95	(0.83, 1.09)	0.4982	NA
IBD					0.9999

No	198	1.06	(1.03, 1.10)	0.0004	NA
Yes	4	1.00	(0.00, infinity)	1.0000	NA
HLA_B27				0.0998	
No	22	0.97	(0.89, 1.07)	0.5828	NA
Yes	177	1.07	(1.03, 1.11)	0.0002	NA
NSAID				0.3555	
No	95	1.04	(0.99, 1.09)	0.0797	NA
Yes	107	1.08	(1.03, 1.13)	0.0026	NA
conventional synthetic DMARDs				0.0689	
No	153	1.05	(1.01, 1.09)	0.0089	NA
Yes	49	1.16	(1.03, 1.31)	0.0119	NA
TNFi				0.9519	
No	181	1.06	(1.02, 1.10)	0.0011	NA
Yes	21	1.06	(0.91, 1.24)	0.4261	NA

The associations between SUA and radiographic SpA were stratified by baseline demographics and disease characteristics.

Figures

Figure 1

Odds ratio and 95% confidence intervals for radiographic SpA in relation to SUA The smooth curves for relation between Serum uric acid (SUA) and radiographic spondyloarthritis (SpA) were fully adjusted for age, gender, current smoker, alcohol, evergout, disease duration, axial involvement (any), peripheral involvement (any), enthesitis involvement, dactylitis, psoriasis, uveitis, IBD, HLA-B27, ESR, CRP, Creatinine, NSAID, conventional synthetic DMARDs and TNFi.

Figure 2

Two separate patients' CT sacroiliac images comparison Patient 1 (imageA-D) presented radiographic SpA, with sclerosis, erosions and joint space of ankylosis; patient 2 (imageE-H) presented non-radiographic SpA, with only sclerosis.

Supplementary Files

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