in the dorsal and palmar view at wrist, MCP, PIP and DIP 2-5 joint levels for synovitis and tenosynovitis.
Subsequently, a comparison of the findings in the affected joints was performed using US as the reference method. Furthermore, AUC was calculated to show the extent to which a new joint inflammation was associated with a change in diagnosis.
Results: Of the 60 patients initially examined (1), 30 patients (dropout rate 50%) were followed-up approximately 3 years later. The patients were newly divided into 3 groups: Diagnosed PsA (n=14, Group I), still suspected PsA, (n=6, Group II) and in-between diagnosed PsA (n=10, Group III). Patients with a change in the diagnosis from suspected to diagnosed PsA (Group III) showed a significantly increased prevalence of joints with pathological findings in FOI (46% at baseline, 88% at follow-up; p=0.046), with an unchanged joint distribution pattern, i.e. with a dominant involvement of the DIP joints. Compared to baseline, patients of group III were three times more common to show enrichment in p3 in FOI at follow-up (1.7% vs. 7.0%; p=n.s.). Newly detected pathologic joints by FOI (PVM, p2) and US at follow-up were positively associated with the change of diagnosis from suspected PsA to confirmed PsA (FOI: AUC 0.78; GSUS: AUC 0.77).Using US in greyscale as reference, inflammatory changes in the joints were diagnosed in all 3 cohorts by means of FOI in P1 and P3 with high specificity (Group III: 90.6%, Group II: 97.5%, Group I: 94.2%) and low sensitivity (Group III: 24.4%, Group II: 20.3%, Group I: 19.8%).
Conclusion: FOI appears to be helpful to differentiate between acute and chronic disease stages. Furthermore, it is specific for detecting inflammatory changes in the joints of the hands in PsA – in comparison to US. FOI could thereby become a helpful tool as a "dermatological-screening" method to select psoriasis patients with indication for further rheumatological evaluation.
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SAT0408

UTILITY OF CAROTID ULTRASOUND AND FRAMINGHAM RISK SCORE ON DISCRIMINATING CORONARY ARTERY DISEASE IN PATIENTS WITH PSORIATIC ARTHRITIS (PSA)

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Background: While carotid ultrasound (US) has been advocated for cardiovascular (CV) risk screening in patients with rheumatoid arthritis as various traditional scores underestimate CV risk, whether subclinical carotid atherosclerosis (SCA) is associated with coronary atherosclerosis on coronary computed tomography angiography (CCTA) in patients with psoriatic arthritis (PsA) remains uncertain.
Objectives: This study aimed to identify carotid US parameters which can discriminate PsA patients with coronary artery disease (CAD) and obstructive CAD (O-CAD), and determine the utility in combination with Framingham Risk Score (FRS).
Methods: Ninety-one PsA patients (56 males; age: 50±11years, disease duration: 9.4±9.2years) without overt CV diseases were recruited. Carotid intima-media thickness (cIMT), presence of plaque and total plaque area (TPA) were determined by high-resolution US. CAD was defined as the presence of any coronary plaque on CCTA. O-CAD was defined as >50% stenosis of the lumen. FRS <10% indicates low CV risk, 10-19% indicates intermediate risk while ≥20% indicates high risk (1).
Results: Thirty-five (38%) patient had carotid plaque. Fifty-five (60%) patients had CAD and 9 (10%) patients had O-CAD. 53 (58%), 25 (17%) and 13 (14%) were classified as low, moderate and high CV risk according to the FRS respectively. FRS underestimated the CV risk as only 11/55 (20%) of subjects with CAD were correctly identified as having high CV risk by FRS (Figure 1). Fifteen patients out of 53 (28%) with low CV risk based on FRS were reclassified as high CV risk by the presence of carotid plaque. Nine out of these 15 (60%) had CAD and 1/15 (6.7%) had O-CAD. Concerning the carotid ultrasound parameters, cIMT (mean and maximum) and TPA were increased in both the CAD+ and O-CAD+ group compared to those without CAD or O-CAD (Table 1). Multivariate logistic regression analysis revealed that mean cIMT (OR=1.06, 95% CI:1.01-1.11, *p*=0.013) was an independent explanatory variables associated with CAD. Meanwhile, mean cIMT (OR=1.06, 95%CI: 1.01-1.11, *p*=0.013) maximum cIMT (OR=1.06, 95%CI: 1.00-1.13, *p*=0.043), and TPA (OR=1.55, 95%CI: 1.01-2.36, *p*=0.043) were independent explanatory variables associated with O-CAD after adjusting for covariates. Based on Receiver Operating Curve (ROC) analysis, an optimal cut off for FRS at 5% and mean cIMT at 0.62mm yield 63% sensitivity and 73% specificity for the presence of CAD (AUC: 0.71, *p*=0.001).

Table 1. Relationship between carotid ultrasound parameters and the presence and extent of coronary artery disease on coronary computed tomography angiography.

	Coronary artery disease		
	No (n=37)	Yes (n=54)	*p*
Mean carotid IMT, mm	0.63 ± 0.12	0.69 ± 0.1	**0.017**
Maximum carotid IMT, mm	0.77 ± 0.17	0.84 ± 0.14	**0.040**
Carotid Plaque, n, %			
Absence	26 46.4%	30 53.6%	0.156
Presence	11 31.4%	24 68.6%	
Total plaque area, mm^2	0.0 [0,6]	0.0 [0, 10.8]	0.059
	Obstructive coronary artery disease		
	No (n=82)	Yes (n=9)	*p*
Mean carotid IMT, mm	0.65 ± 0.12	0.76 ± 0.07	**0.011**
Maximum carotid IMT, mm	0.80 ± 0.16	0.93 ± 0.14	**0.020**
Carotid Plaque, n, %			
Absence	53 93.0%	4 7.0%	0.235
Presence	29 85.3%	5 14.7%	
Total plaque area, mm^2	0.0 [0, 7.0]	6.0 [0, 15.3]	0.103

IMT-intima media thickness; coronary computed tomography angiography.

Conclusion: Increased cIMT and TPA were associated with CAD and O-CAD in PsA patients while the presence of carotid plaque alone was insufficient to discriminate patient with or without CAD. A combination of US parameters should be considered for CV risk stratification in patients with PsA.
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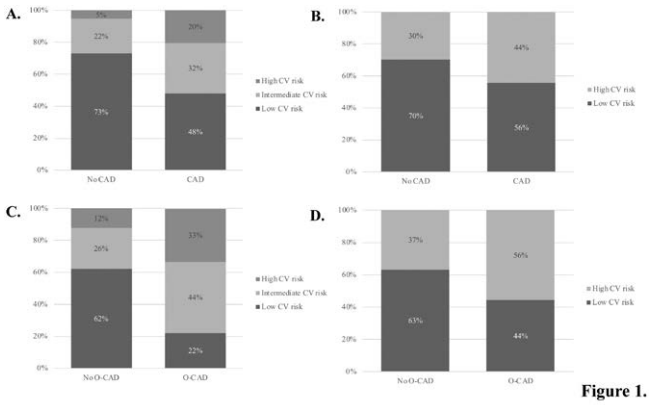

Figure 1.
CV Risk classification based on Framingham risk score (FRS) (A&C) (high CV risk: FRS ≥20%; intermediate CV risk: FRS 10-19%; low CV risk: FRS<10%) and CV Risk classification based on the presence of carotid plaque (high CV risk) and absence of carotid plaque (low CV risk) (B&D) in patient with or without CAD/.O-CAD

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SAT0409

BIOLOGIC TREATMENT IN PSORIATIC ARTHRITIS AND AXIAL SPONDYLOPATHY REDUCES SICKNOTES ISSUED BY GPS, DESPITE DELAYS IN DIAGNOSIS: A REAL-LIFE STUDY IN WALES.

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Background: Seronegative inflammatory arthritis including psoriatic arthritis (PsA) and axial spondyloarthropathies (AxS) are chronic inflammatory diseases associated with significant morbidities. The National Institute of Health and Clinical Excellence (NICE) has produced several pieces of guidance on disease management including the use of biologic therapies which have been shown to improve patient outcomes and quality of life. However, there are limited real-life data on patient journey from symptom onset to diagnosis and treatment including with biologics in the UK.

Objectives: The purpose of this study is to examine the real-life patient journey from symptom onset to diagnosis and treatment.

Methods: Data from the Secure Anonymised Information Linkage (SAIL) databank in Wales, which holds over a billion anonymised records, were used to assess the treatment of patients with PsA or AxS, aged 18 years or over with at least one READ code present for PsA or AxS in their primary care records. We examined the drug use of patients across primary, secondary care and specialist rheumatology clinics to explore the use of NSAIDs, DMARDs and biologics in the real-life setting while exploring demographics, comorbidities and surgical procedures of 1829 PsA and 908 AxS patients.

Results: The AxS patients were significantly younger at diagnosis and were predominantly male. Typical delays in diagnosis of 8-9 years from symptom onset were observed. The rate of stopping or switching a biologic medication was similar for AxS and PsA patients (Table 1). There was a significant reduction in sicknotes issued following biologic initiation for PsA (Difference: 14.6%, 95% CI: 8.7% to 20.4%) and AxS (Difference 16.9%, 95% CI: 10.5% to 23.3%)

Table 1. Characteristics of treatment of PsA and AxS patients

	PsA (n=1829)	AxS (n=908)	Difference (95% CI)
Female (%, n)	55.1% (1007)	29.1% (264)	26 (22.2 to 29.6)*
Mean age at diagnosis (years, SD)	46.9 (14)	43.5 (14.4)	3.4 (2.3 to 4.5)*
BMI (Index, SD)	30.3 (6.3)	28 (5.8)	2.3 (1.8 to 2.8)*
Time from symptom to diagnosis (years, SD)	8.9 (5.5)	8.0 (5.6)	0.9 (0.5 to 1.3)
Hypertension at diagnosis (%, n)	24.2% (442/1829)	19.4% (176/908)	4.8 (1.5 to 8.0)*
Time from diagnosis to biologic (years, SD)	6.3 (4.7)	6.1 (5.0)	0.2 (-0.9 to 0.5)
Used a Biologic (%, n)	23% (420/1829)	36.8% (334/908)	13.8 (10.2 to 17.5)*
NSAIDs used pre-biologic (SD)	11.3 (3.2)	11.6 (3.2)	0.3 (-0.2 to 0.8)
Number of DMARDs used pre-biologic (SD)	3.1 (1.5)	2.5 (1.7)	0.6 (0.4 to 0.8)*
Biologic treatment change/ failure (%, n)	21.6% (92/425)	22% (74/336)	0.4 (-6.4 to 5.5)
Sicknotes issued by GP‡ pre-biologic (SD)	33.9% (144/425)	33.6% (113/336)	0.3 (-6.5 to 7.0)
Sicknotes issued by GP ‡ post-biologic (SD) (%, n)	19.3% (82/425)	16.7% (56/336)	2.6 (-2.9 to 8.0)
Hospitalised for serious infection pre- diagnosis (%, n)	7.2% (131/1829)	6.3% (57/908)	0.9 (-1.2 to 2.8)
Hospitalised for serious infection post- diagnosis (%, n)	10.4% (190/1829)	11.6% (105/908)	1.2 (-1.3 to 3.8)
Hospitalised for serious infection post- biologic (%, n)	5.6% (24/425)	6.3% (21/336)	0.7 (-2.8 to 4.2)

* Significant at p<0.05

‡ General practioner/Primary care physician

Conclusion: Patients with PsA and AxS were treated with NSAIDs and DMARDs prior to receiving biologic medication in accordance with NICE guidelines. However, there was a long delay from symptom onset to diagnosis. Biologic treatment reduced sicknotes issued by GPs confirming the benefit of biologic treatment on work productivity observed in clinical trials.

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SAT0410 EFFICACY AND SAFETY OF IXEKIZUMAB IN PATIENTS WITH ACTIVE PSORIATIC ARTHRITIS BASED ON CONCOMITANT CONVENTIONAL DISEASE-MODIFYING ANTIRHEUMATIC DRUGS (CDMARD) USE: RESULTS FROM SPIRIT-P1 AND SPIRIT-P2

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Background: Biologic disease-modifying antirheumatic drugs such as ixekizumab (IXE), a high-affinity monoclonal antibody that selectively targets interleukin (IL)-17A, are commonly prescribed to patients with psoriatic arthritis (PsA) in combination with conventional synthetic disease-modifying antirheumatic drugs (cDMARDs). Previous studies have shown that, after 24 weeks of treatment, IXE is efficacious with or without concomitant cDMARD therapy in patients with active PsA.[1,2] However, there is limited evidence demonstrating efficacy and safety after 3 years of treatment.

Objectives: To evaluate the long-term (3-year) efficacy and safety of IXE in patients with active PsA from SPIRIT-P1 (NCT01695239) and SPIRIT-P2 (NCT02349295) based on concomitant cDMARD use.

Methods: Patients were subdivided into the following subgroups: 1) no cDMARD use for 3 years (ixekizumab monotherapy); 2) methotrexate (MTX) use without interruption (i.e., ≤14-day gap of not using MTX), but allowing a change of MTX dose; and 3) any cDMARD (MTX, sulfasalazine, leflunomide, ciclosporin, hydroxychloroquine) use during 3 years without interruption (i.e., ≤14-day gap of not using cDMARDs), but allowing a switch of cDMARD type and/or change of dose. The post-hoc integrated analysis assessed efficacy and safety up to 3 years by three subgroups. Efficacy outcomes included the American College of Rheumatology (ACR) 20/50/70, Psoriasis Area and Severity Index (PASI) 75/90/100, Health Assessment Questionnaire-Disability Index (HAQ-DI) ≥0.35-point improvement. Missing data were imputed using modified non-responder imputation. The IXE 80 mg every 4 weeks (IXEQ4W) dose data are reported here.

Results: Overall, IXE-treated patients showed improvement in all efficacy outcomes over 156 weeks, regardless of concomitant cDMARD use. ACR response rates by concomitant cDMARD use at 156 weeks are highlighted in Figure 1. Patients treated with IXEQ4W in the no cDMARD use, MTX, and any cDMARD use subgroups had similar ACR20 (59.1%, 67.0%, and 66.1%, respectively), ACR50 (46.2%, 47.4%, and 46.8%, respectively), and ACR70 (30.7%, 28.4%, and 28.1%, respectively) response rates at 156 weeks. Patients treated with IXEQ4W in the three subgroups also had similar PASI75 (65.5%, 60.8%, and 59.8%, respectively), PASI90 (53.6%, 49.7%, and 48.0%, respectively), and PASI100 (42.2%, 46.2%, and 42.4%, respectively) response rates at 156 weeks. The proportion of patients achieving HAQ-DI improvement ≥0.35 in the three subgroups (51.9%, 45.0%, and 47.5%, respectively) was comparable. The safety profile of IXEQ4W was consistent with that previously reported.[1,2] A similar proportion of IXEQ4W-treated patients in the three subgroups reported ≥1 treatment-emergent adverse events (TEAEs) regardless of the addition of MTX or other cDMARDs (91.0%, 84.1%, and 83.2%, respectively), and the majority of TEAEs were mild or moderate in all three subgroups.

Conclusion: IXEQ4W provided sustained improvements in the signs and symptoms of active PsA. While there are some numerical differences in ACR20/50/70 as well as PASI75/90/100, the overall responses with or without the addition of MTX or other cDMARDs were similar. In this post-hoc analysis, it appears that, for sustained responses over time, IXEQ4W does not require the addition of MTX or other cDMARDs. Addition of MTX or other cDMARDs to IXEQ4W did not negatively impact its favorable long-term safety profile.

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