with administration of IVGC. **Use of steroid sparing drugs** Six patients used methotrexate for other diseases before their GCA diagnosis. Among the remaining patients, 6 out of 56 patients with documented LV-GCA received a steroid sparing drug within the first 30 days after diagnosis, while this was only true for 3 out of 194 patients without documented LV-GCA (p=0.005). All nine patients who received a steroid sparing agent received methotrexate. One of them also received tocilizumab.

Conclusion: In our study visual symptoms seem to most strongly influence the initial treatment decisions regarding GC. Paradoxically, scalp tenderness, also a cranial symptom, was the only factor associated with a lower initial oral GC-dose. The presence or absence of documented LV-GCA showed no statistical association with GC-treatment, although the number of patients were rather low. However, a higher proportion of patients with LV-GCA received a steroid sparing drug within the first 30 days after diagnosis.

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Table 1 Final predictor models for initial oral GC dose and the use of intravenous GC

	N(%)[1]	Predictors for initial oral steroid dose		Predictors of IV glucocorticoids	
Patient characteristic	N = 255	**Beta (95% CI)**	**p-value**	**OR (95% CI)**	**p-value**
Age at diagnosis (years)		-0.06 (-0.26 to 0.14)	0.55	0.96 (0.89 to 1.03)	0.29
Male sex	76 (30%)	-1.6 (-5.3 to 2.2)	0.41	1.19 (0.33 to 4.00)	0.78
Time from first symptom to diagnosis (days)		-0.01 (-0.03 to 0.01)	0.36	1.00 (1.00 to 1.01)	0.13
Administration of intravenous glucocorticoids	21 (8.3%)	3.3 (-2.9 to 9.4)	0.3		
Positive temporal artery biopsy	166 (71%)	4.9 (0.82 to 9.0)	0.019	4.58 (0.85 to 40.4)	0.079
Large vessel involvement	58 (23%)	4.0 (-1.1 to 9.1)	0.13	3.39 (0.45 to 25.0)	0.23
Localized headache	200 (78%)			0.27 (0.07 to 0.99)	0.049
Scalp tenderness	101 (40%)	-4.4 (-7.8 to -0.98)	0.012		
Constitutional symptoms	176 (69%)			0.27 (0.07 to 0.95)	0.041
Reduced pulse in temporal artery	67 (26%)	2.5 (-1.3 to 6.3)	0.19	2.77 (0.75 to 10.2)	0.12
Visual disturbances incl. blindness	77 (30%)	12 (7.9 to 16)	<0.001	18.6 (4.81 to 103)	<0.001
Erythrocyte sedimentation rate (mm/hour)		0.03 (-0.04 to 0.10)	0.35	1.00 (0.97 to 1.03)	0.88
C-reactive protein (mg/L)		-0.02 (-0.05 to 0.01)	0.25	1.01 (1.00 to 1.02)	0.042
Platelet count (x10^9/L)				0.99 (0.99 to 1.00)	0.03

Beta = Linear regression coefficient, CI = Confidence Interval, OR = Odds Ratio

[1]N(%) shown for all categorical variables shows the number (%) of patients with presence of a given characteristic, with absence of the characteristic as comparator. For sex female sex is comparator. For continuous variables N(%) is empty.

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POS0777

WHAT IS THE PROFILE OF AXIAL SPONDYLOARTHRITIS PATIENTS TREATED WITH IXEKIZUMAB AND ITS EFFECTIVENESS IN CLINICAL PRACTICE RESULTS FROM THE ESPADA STUDY

Keywords: biological DMARD, Observational studies/ registry, Real-world evidence

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Background: Interleukin-17A inhibitors (IL-17Ai) have emerged as an additional therapeutic option for the management of axial spondyloarthritis (axSpA) and, together with tumor necrosis factor inhibitors, are currently positioned as first-line treatment in patients who have an inadequate response to nonsteroidal anti-inflammatory drugs, according to the latest ASAS-EULAR recommendations. However, evidence on the use of IL-17Ai in real life is still limited.

Objectives: The ESPADA study evaluated the use of ixekizumab (IXE) for the treatment of axSpA in routine clinical practice in Spain.

Methods: A retrospective observational study was carried out in ten Spanish hospitals. Patients aged ≥18 years diagnosed with axSpA [radiographic (r-axSpA) or non-radiographic (nr-axSpA)] according to their rheumatologist who also met the ASAS criteria were included. Patients must have started IXE between June 2020 and August 2023, having a minimum follow-up of 12 weeks and at least one follow-up visit available, regardless of whether they continued with IXE. Demographic and clinical characteristics, treatment patterns, total and subgroup persistence assessed using Kaplan-Meier, as well as effectiveness at 12, 24, and 52 weeks according to BASDAI and ASDAS were evaluated.

Results: A total of 106 patients were included, mostly men (58.5%) and with a mean (SD) age of 51.9 (12.1 years). 69.8% presented r-axSpA; nearly one-third had psoriasis, and more than half had peripheral arthritis. The median (IQR) time from diagnosis to IXE initiation was 9.4 (3.2 - 20.3) years (Table 1). At IXE initiation, 31.1% of patients also exhibited peripheral involvement. Mean (SD) BASDAI score (n=86) was 6.4 (2.0), and 95.1% of patients showed high/very high disease activity according to ASDAS (n=81). Most patients (98.1%) had previously received a biological (b) or targeted synthetic (ts) DMARD (b/tsDMARD); with a mean (SD) of 2.3 (1.4) previous b/tsDMARDs. One-third of patients were receiving concomitant treatment with conventional synthetic DMARDs (csDMARDs) at the start of IXE treatment. Among the patients with effectiveness data available at week 52, 18.9% had improved their BASDAI score ≥50% (n=37) and 38.2% had low/inactive disease according to ASDAS criteria (n=34). The Kaplan-Meier analysis showed a treatment persistence of 56.3% at week 52 (Figure 1), with the median survival not having been reached. On the other hand, no statistically significant differences in persistence were observed in the analyzed subgroups: baseline C-reactive protein levels (normal: 52.8%; elevated: 47.0%), HLA-B27 status (negative: 53.9%; positive: 50.1%), number of previous b/tsDMARDs (1: 65.6%; ≥2: 51.8%), previous secukinumab treatment (no: 56.1%; yes: 56.2%). Among those patients who discontinued IXE, the most frequently reported reasons for discontinuation were lack of effectiveness (72.5%, n=29) and adverse events (22.5%, n=9).

Conclusion: Most axSpA patients treated with IXE in the study cohort had long-standing, highly active disease, involving multiple domains and a history of prior b/tsDMARD treatment. Despite these characteristics, more than half of the patients maintained one-year treatment persistence with IXE, highlighting IXE as a viable therapeutic option.

Table 1. Demographic and clinical characteristics at IXE initiation (n=106).

Variable	Value
Age (years), mean (SD)	51.9 (12.1)
Sex, n (%)	
Male	62 (58.5)
Time since diagnosis (years), median (IQR)	9.4 (3.2-20.3)
Type of axSpA, n (%)	
nr-axSpA	30 (28.3)
r-axSpA	74 (69.8)
Unknown	2 (1.9)
Predominant involvement, n (%)	
Axial	73 (68.9)
Peripheral	12 (11.3)
Mixed (Peripheral and axial)	21 (19.8)
axSpA characteristics, n (%)	
Arthritis	57 (53.8)
Enthesitis	26 (24.5)
Dactylitis	9 (8.5)
Uveitis	14 (13.2)
Psoriasis	30 (28.3)
Inflammatory bowel disease	3 (2.8)
Family history of axSpA, n (%)	22 (20.8)
HLA-B27 positive, n (%)	55 (51.9)
Normal C-reactive protein levels (<5 mg/L), n (%)	52 (52.5)
Previous treatments, n (%)	
csDMARDs	41 (38.7)
b/tsDMARDs	104 (98.1)
TNFi	100 (94.3)
IL-17Ai (secukinumab)	42 (39.6)
JAKi	5 (4.7)
Number of previous b/tsDMARDs, n (%)	
0	2 (1.9)
1	36 (34.0)
≥2	68 (64.2)

IL-17Ai: Interleukin-17A inhibitors, IQR: interquartile range, JAKi: Janus kinase inhibitors, TNFi: Tumor necrosis factor inhibitors.

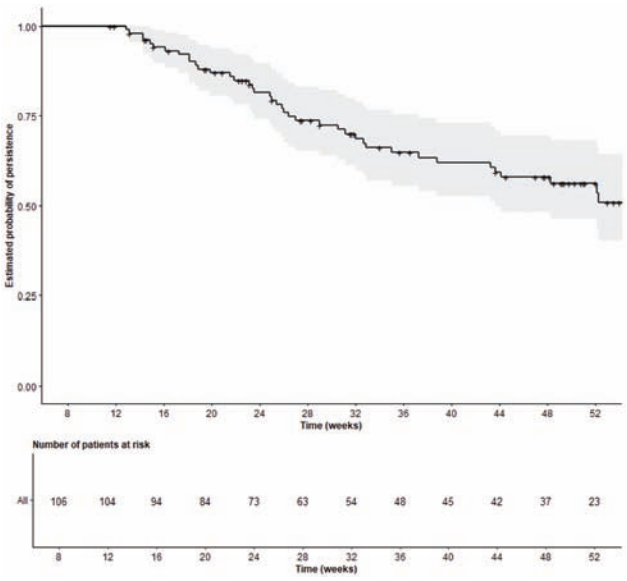

Figure 1. Time to discontinuation: Kaplan-Meier curve.

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POS0778

INTERFERON-γ RELEASE ASSAY AS AN EMERGENT POWERFUL BIOMARKER IN SYSTEMIC LUPUS ERYTHEMATOSUS

Keywords: Biomarkers, Descriptive Studies, Prognostic factors

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Background: The major role of type I IFN in systemic lupus erythematosus (SLE) pathophysiology has been extensively studied and resulted in the marketing of anifrolumab. However, the pre-SLE stages are also characterized by alterations in type II IFN-γ activity followed by auto-antibodies (autoAb) detection [1], and accumulation and fluctuations in type II IFN-γ signatures are associated with disease activity and autoAb production [2–4]. As such, the whole blood IFN-γ release assay (IGRA) can facilitate the monitoring of type II IFN-γ levels among SLE patients by providing insights into both spontaneous IFN-γ release through the IGRA-nil tube result used as a negative control; and from phytohemagglutinin (PHA)-stimulated T cells through the IGRA-PHA tube used as a positive control [5]. It is unclear whether data established among Asian patients are also valid for French, primarily Caucasian, patients; whether IGRA is independent from other biomarkers of SLE (i.e., anti-dsDNA, complement…), and whether it could be a predictor of achieving remission on treatment.

Objectives: To investigate the *ex vivo* IFN-γ release assay (IGRA) as a biomarker of SLE activity and disease outcome.

Methods: A retrospective study conducted between 2008 and 2024 at a single French tertiary care center, included 145 SLE patients at various disease stages (diagnosis, relapse, and remission) and 145 age-/sex-matched healthcare workers as controls. Data were collected on spontaneous IFN-γ release (IGRA-nil) and on phytohemagglutinin-induced IFN-γ release (IGRA-PHA, after subtracting IGRA-nil result). In clinically active patients, the test was performed at initiation or intensification of treatment. All the SLE patients met the 2019 ACR/EULAR classification criteria. Clinically active patients had a cSLEDAI-2k score > 0. Disease remission status was adapted from the 2021 definition of remission in SLE (DORIS) criteria as follows: (i) cSLEDAI-2k score = 0; (ii) prednisone prescribed at 5 mg/day or less, with stable antimalarials, immunosuppressive drugs and biologics; and (iii) the global physician assessment was considered to be <0.5 when the disease was described as 'clinically inactive' or 'in remission' or 'asymptomatic'. Patients were categorized according to their glucocorticoid (GC) intake at the time of the sampling into three prednisone groups: negative/low (≤5mg/day, n=88 including 49 in the active group), medium (6-49 mg/day, n=44), or high (≥50mg/day, n=13). Patients were followed up as part of their routine care, at the rate of a visit every 1, 3, 6 or 12 months depending on their disease activity, and at the time of relapse.

Results: The *ex vivo* spontaneous IFN-γ release was increased (p=0.004), and the PHA-induced IFN-γ release (IGRA-PHA) was decreased in active SLE patients (p<0.01) compared to inactive SLE patients and controls (Figure 1). In multivariate logistic regression analysis cSLEDAI2K score influence on IFN-γ production (OR= 1.5 [1.29;1.75]) was independent from GC prescribed doses (OR 0.954 [0.392;2.32] for 5-50 mg/d prednisone; OR 1.37 [0.17;11.06] if prednisone > 50 mg/d, prednisone ≤5 mg/d being the reference). A diminished response in IGRA-PHA testing was associated with an elevated median cSLEDAI score and more constitutional ($p<10^{-4}$), mucocutaneous (p=0.004), renal (p=0.04), and serositis manifestations (p=0.0003). IGRA-PHA predicted clinically inactive SLE better than IGRA-nil (ROC AUC= 0.853 versus 0.722), 8 IU/ml corresponding to the optimal Youden index and level <1.6 IU/ml indicating 100% specificity for active disease. IGRA-PHA ≥ 8 IU/ml was an independent predictor of clinically inactive SLE (OR= 10.6 [95% CI: 3.72-30.17]; $p=9.7x10^{-6}$) in multivariate regression analysis including anti-DNA dosage, low CH50, albumin < 40 g/L and urinary protein/creatinine ratio as co-variables (Table 1). Based on the IGRA-PHA responses, patients not in remission at inclusion were categorized into low responders (IGRA-PHA < 1.6 IU/mL, n=50), medium responders (1.6 ≤ IGRA-PHA < 8 IU/mL, n=25), and high responders (IGRA-PHA ≥ 8 IU/mL, n=21). Low responders had a median time to achieve remission of 1252 days, compared to 711 days for medium responders, and 582 days for high responders (log-rank test: $p=2.4x10^{-6}$).

Conclusion: In primarily Caucasian patients, IGRA-PHA appears as a powerful, not influenced by GC prescription, independent biomarker of SLE clinical activity, and as a predictor of treatment-induced remission when performed at the initiation or intensification of therapy.

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Table 1. Multivariate logistic regression modeling the relation between various biomarkers and clinical disease inactivity (cSLEDAI2K= 0).

variable	odds ratio	p-value
Model 1 (relation between SLE remission and covariables including IGRA-nil)		
Intercept	0.000337 [2.8x10^{-6};0.0406]	0.0011
IGRA-nil	1.1x10^{-9} [7.3x10^{-17};0.0168]	0.0145
Anti-dsDNA (<10 IU/mL)	1.06 [0.38;2.97]	0.907
CH50 (%)	1.04 [1.01;1.08]	0.026
Albumin (≥40 g/L)	1.17 [1.06;1.3]	0.003
Urinary PCR (<0.5 g/g)	1.07 [0.271;4.25]	0.920
Model 2 (relation between SLE remission and covariables including IGRA-PHA)		
Intercept	0.000557 [4.1x10^{-6};0.076]	0.0028
IGRA-PHA (≥8.0 IU/mL)	10.6 [3.72; 30.17]	9.7x10^{-6}
Anti-dsDNA (<10 IU/mL)	0.81, [0.29; 2.29]	0.697
CH50 (%)	1.01 [0.98; 1.04]	0.459
Albumin (≥40 g/L)	1.14 [1.02; 1.28]	0.023
Urinary PCR (<0.5 g/g)	0.69 [0.17; 2.77]	0.605