

Inflammation or pain: What drives decision-making?

Dr Victoria Navarro-Compán
Hospital Universitario La Paz
Madrid, Spain

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Disclosures

Dr Victoria Navarro-Compán

University Hospital La Paz and
IdiPaz, Madrid, Spain

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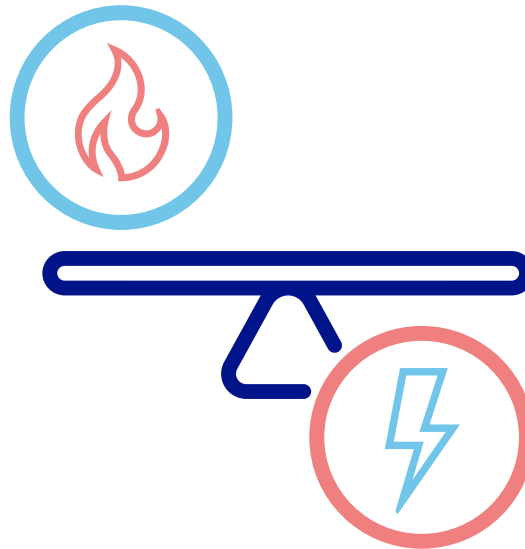
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What should drive decision-making in spondyloarthropathies?

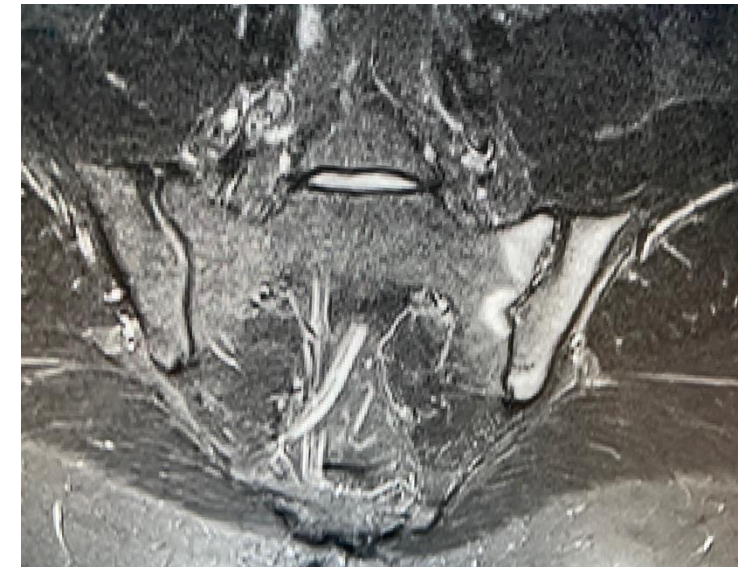
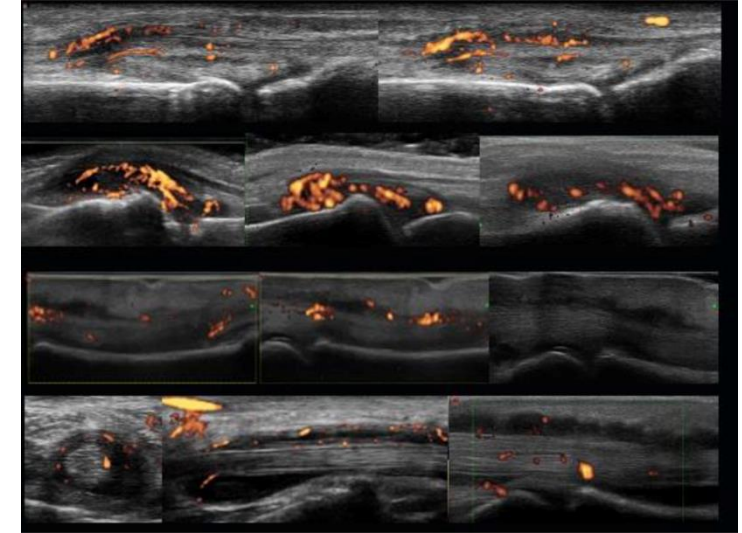
Objective, measurable
inflammation is the primary
determinant of disease progression
and should drive care decisions



While inflammation may drive
damage, **pain** drives the patient
and cannot be ignored

Why inflammation matters?

- axSpA and PsA are immune-mediated **inflammatory diseases**¹⁻³
- Inflammation is the key driver of **symptoms**^{1,2}
- Persistent inflammation leads to irreversible **damage**:
Syndesmophytes, erosions and deformities → **disability**²
- **Modern therapies** directly target inflammatory pathways²
- Inflammation **predicts therapeutic success**: Higher biologic response in patients with elevated CRP and positive MRI²



Images reproduced from (upper) Bonfiglioli KR, et al. 2024¹ and (lower) Navarro-Compán V, et al. Ann Rheum Dis. 2021.²
axSpA, axial spondyloarthritis; CRP, C-reactive protein; ESR, erythrocyte sedimentation rate; PsA, psoriatic arthritis.

1. Bonfiglioli KR, et al. J Pers Med. 2024;14(6):550. 2. Navarro-Compán V, et al. Ann Rheum Dis. 2021;80(12):1511-21.
3. Smolen JS, et al. Clin Exp Rheumatol. 2015;33(5 Suppl 93):S48-50.

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Recommendations to manage axSpA and PsA are inflammation-oriented



RECOMMENDATIONS

axSpA

- Beyond clinical diagnosis, eligibility/response **predictors for biologics** include objective inflammation¹
- EULAR axSpA recommendations suggest that **“objective signs of inflammation”** (CRP and/or MRI) be considered when selected b/tsDMARD therapy¹
- Treatment aims: **Remission or low disease activity**. Targets include CRP normalisation and imaging improvement¹

PsA

- EULAR PsA recommendations emphasize treat-to-target (remission/low disease activity) and **choosing therapy based on active domains** (peripheral arthritis, enthesitis, dactylitis, axial disease, skin/nail involvement) and severity/prognosis²
- Recommendations state **remission should be seen as an “abrogation of inflammation”** as the most favourable outcomes are achieved when inflammation is fully controlled²

Treatment targets are often not achieved in clinical practice^{1,2}



Remission



Low disease activity

Moreover, **40-50%** of patients with axSpA are classified as **primary non-responders**, with diminishing response rates observed upon subsequent biologic treatment^{2,3}

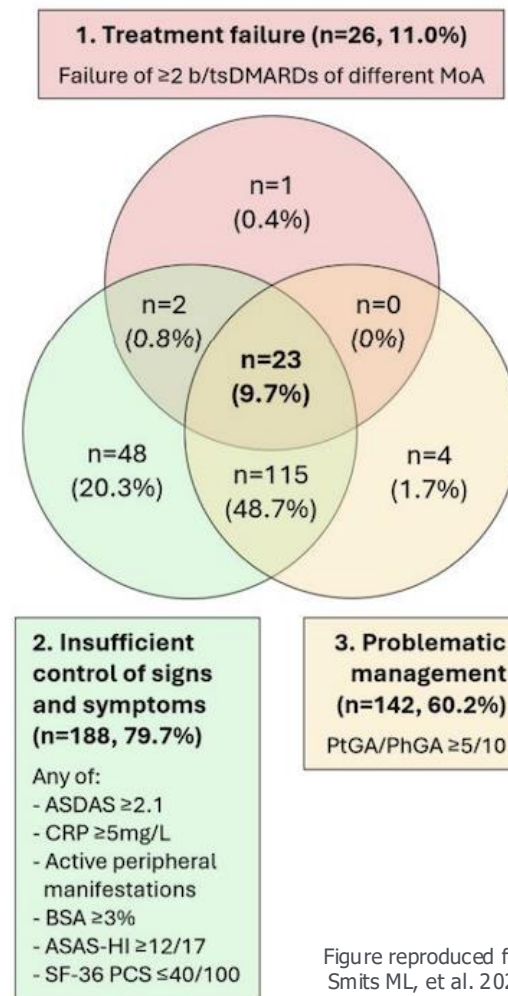
Why is remission not frequently achieved?

axSpA^{1*}

- **One in 10 patients** with axSpA have **D2M** disease by ASAS definition
- Only **2%** have **treatment-refractory** disease
- **Patient-reported outcomes** contribute importantly to the classification of D2M axSpA

PsA^{2†}

- **D2T-PsA** was detected in **19.3% of patients[‡]**
- **Comorbidities** such as fibromyalgia (FM) and diabetes mellitus (DM) and the median number of comorbidities are significantly **higher in D2T-PsA**



*Registry study (N=263 patients ≥1 year from diagnosis with SpA in the Netherlands).¹ †Retrospective observational study (N=171 patients with PsA in Turkey) to evaluate the proportion of patients with D2T-PsA.² ‡Defined as failure of ≥1 conventional DMARDs and ≥2 biological or targeted DMARDs with different mechanisms of action.²

axSpA, axial spondyloarthritis; ASAS, Assessment of SpondyloArthritis International Society; ASDAS, Axial Spondyloarthritis Disease Activity Score; BSA, body surface area; CRP, C-reactive protein; D2M, difficult-to-manage; D2T-PsA, difficult-to-treat PsA; DM, diabetes mellitus; DMARD, disease-modifying anti-rheumatic drug; FM, fibromyalgia; PsA, psoriatic arthritis; PhGA, physician global assessment; PtGA, patient global assessment; SF-36 PCS, 36-item short form health survey physical component summary; tsDMARD: targeted synthetic DMARD.

1. Smits ML, et al. Rheumatology (Oxford). 2025;64(6):3816-25. 2. Alp G, et al. Clin Exp Rheumatol. 2025;43(1)41-7.

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If it is not inflammation, then it must be...

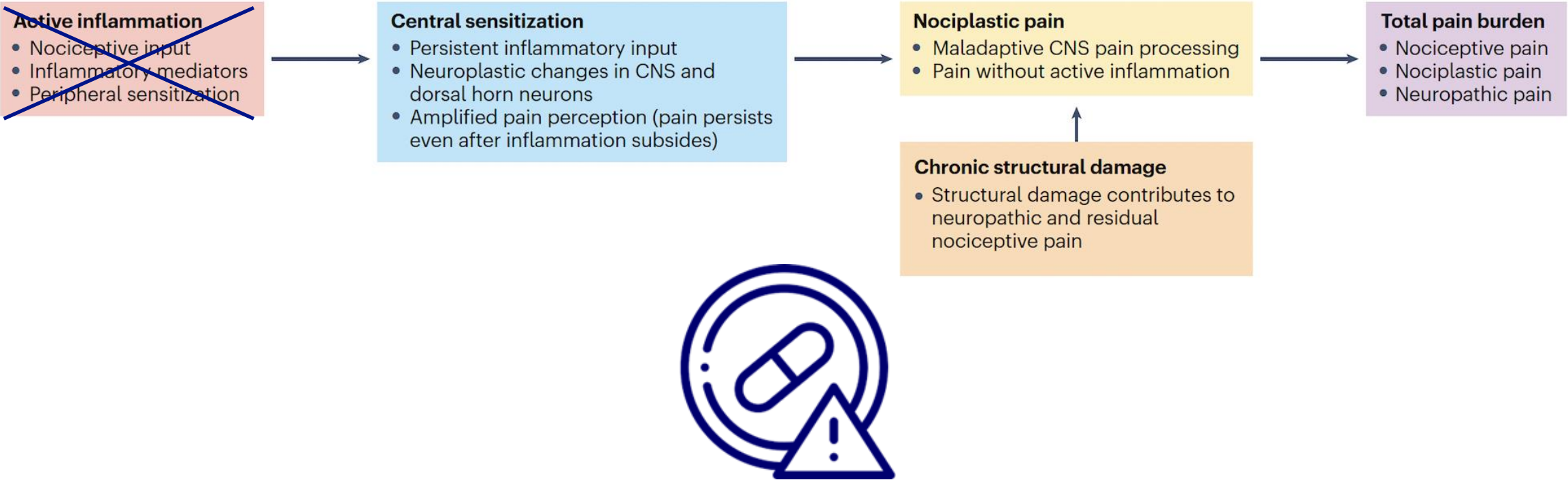
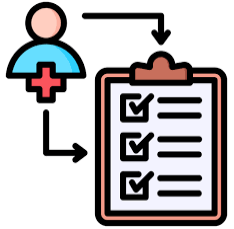


Figure reproduced from Baraliakos X, et al. 2026.¹
CNS, central nervous system.
Baraliakos X, et al. Nat Rev Rheumatol. 2026 [online ahead of print].
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Limitations of PROs for assessing disease activity and guiding treatment



- Inherently **subjective** and can be strongly **influenced by** mood, expectations, coping style, health literacy, and cultural/language factors
- **Recall bias** (e.g., “last week” symptoms) and **response shift** (patients recalibrate what “normal” feels like over time) can distort change scores
- Many PRO domains (pain, fatigue) are **not specific to inflammation** and can be driven by non-inflammatory factors
- Fibromyalgia/central sensitisation, osteoarthritis, mechanical back pain, depression/anxiety, sleep disorders, obesity, anaemia, and deconditioning can **elevate PROs independent of disease activity**
- May **under-capture peripheral or extra-musculoskeletal manifestations** (e.g., enthesitis, dactylitis, axial involvement, skin/nail disease, uveitis, IBD-related symptoms)
- High PRO scores driven by non-inflammatory pain may prompt **unnecessary escalation of immunomodulatory treatment**

Recommended strategy in patients with axSpA and PsA



High pain + high objective inflammation (CRP/MRI/ultrasound)¹⁻³

→ Escalation to biologic/targeted therapy is strongly supported

High pain + low objective inflammation¹⁻⁴

→ First reassess for fibromyalgia/central sensitisation, mechanical pain, sleep/mood issues or damage-related pain, rather than reflexively escalating immunosuppression

Summary: Why inflammation should drive treatment decisions in axSpA and PsA



- Predicts treatment response
- Associated with structural damage
- Measurable and modifiable
- Drives treatment decisions
- May reduce overtreatment

