

Problems within the SpA group – diagnostic versus classification criteria; differential diagnosis within the SpA group; the issue of axial psoriatic arthritis

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Abstract

Diagnostic delay in spondyloarthritis (SpA) results in the occurrence and progression of structural damage, which is irreversible and presented by a unique combination of inflammation-induced bone loss (erosions, periarticular and systemic osteoporosis), together with new bone formation and extraosseous ossification (ligaments/entheses). The most significant opponent of diagnostic delay is misdiagnosis, leading to incorrect treatment with its complications, together with the consequences of an untreated but present disease. In the second part of the review, the problems of early diagnosis in axial spondyloarthritis (axSpA) and psoriatic arthritis (PsA) will be considered - related to the available criteria, the accessible diagnostic methods, or arising from other factors (related to the patient or the systems for specialized medical care).

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1. Introduction

In the context of early diagnosis, it is important to bear in mind that the ASAS criteria for axSpA [1], pSpA, and the combined set (axSpA + pSpA) - shown in Figure 1 [2] and including the revised ASAS-SPARTAN 2025 classification criteria [3] (Table 1). They are applicable in patients with an already established diagnosis and are intended to define homogeneous groups of patients with the same pathology, necessary for the development of strategies for monitoring and management of this pathology, as well as for contemporary clinical studies [4].

In the diagnostic approach, the physician is faced with a patient without an established diagnosis, presenting with complaints (symptoms) suggestive of a particular disease (or group of diseases), known as “main symptoms.” Each main symptom is associated with a certain probability, the so-called “pre-test probability” (pre-test probability /P-pre/) for the presence of the respective disease in a patient presenting with the given main symptom. For example, in the general population of patients with chronic back pain (CBP), the P-pre for axSpA is 5% [5]. The use of the definitions (Calin/Berlin/ASAS) [5-7] for inflammatory back pain (IBP) increases the P-pre from 5% to 30–40%, depending on the definition used [8]. In the course of the diagnostic process, the physician identifies objective findings (signs) using various diagnostic tests (physical, laboratory, imaging). The different tests applied in this process have varying sensitivity and specificity, which determine their diagnostic value [9] (Table 1).

Table 1. Classification table of main definitions.

Test results	Population groups		Total (sum)	
	Disease /Condition present	Disease /Condition absent		
Test is positive	(True Positive) (A)	(False Positive) (B)	Total positive tests (A + B)	Positive Predictive Value (PPV) = A/(A + B)
Test is negative	(False Negative) (C)	(True Negative) (D)	Total negative tests (C + D)	Negative Predictive Value (NPV) =D /(C + D)
Total (sum)	Total number of people with disease / condition	Total number of people without disease /condition	Total number of people (A + B + C + D)	Accuracy = (A+D)/(A+B+C+D)

	(A + C)	(B + D)		
	Sensitivity A /(A+C)	Specificity D /(B+D)		
	Prevalence /Pretest Probability (P-pre) (A + C) /(A + B + C + D)			
Sensitivity = True Positive Rate (TPR) = A / (A + C) i.e., the proportion of patients with disease who will have positive test results. The high-sensitivity tests provide us with a high TPR while minimizing false negative rates (FNR), making it useful for ruling out the disease when the result is negative.				
Specificity = True Negative Rate (TNR) = D /(D + B) i.e., the proportion of people without the disease who will have a negative result. The high-specificity tests have very few false positives, making it useful for ruling in the disease when the result is positive.				
Positive predictive value (PPV) = Precision = A /(A + B) i.e., the proportion of people with a positive test result who actually have the disease. PPV is, sometimes, also referred to as the 'post-test probability of disease given a positive test.' PPV = sensitivity x prevalence / sensitivity x prevalence + (1 – specificity) x (1 – prevalence)				
Negative predictive value (NPV) = D /(C+D) i.e. the proportion of people with a negative test result who do not have the disease, or 'the probability of not having disease given a negative test'. NPV =Specificity x (1 – Prevalence) / (1 – Sensitivity) x Prevalence + Specificity x (1 – Prevalence).				
Prevalence = Pretest probability (P-pre) = (A+C)/(A+B+C+D) i.e. proportion of population with specific disease at given point in time, calculated by dividing the total number of cases (A+C) by the total population (A+B+C+D). The Prevalence (P-pre) of the same disease is different in different populations!				
Positive Likelihood Ratio (LR+) = TPR / FPR = Sensitivity / (1-Specificity). The higher the value of LR+, the higher the probability of diagnosis if the parameter is positive.				
Negative Likelihood Ratio (LR-) = FNR / TNR = (1-Sensitivity) / Specificity. The lower the value of LR-, the lower the probability of diagnosis if the parameter is negative.				
Diagnostic Odds Ratio (DOR) = LR+ / LR-				
False Positive Rate (FPR) = (1-Specificity) = B/(B+D)				
False Negative Rate (FNR) = (1-Sensitivity) = C/(A+C)				
False Omission Rate (FOR) = C/(C+D)				
False Discovery Rate (FDR) = B/(A+B)				
Proportion Correctly Classified = Accuracy = (A+D)/(A+B+C+D)				
Proportion Incorrectly Classified = (B+C)/(A+B+C+D).				

Sensitivity reflects the ability of a test to identify individuals who have a specific disease. By definition (Table 9), Sensitivity = True Positive Rate (TPR) = $A / (A + C)$. Tests with high sensitivity provide a high proportion of true positive results (TPR), while at the same time minimizing false negative results (FNR), thereby reducing missed diagnoses [10-15].

Specificity reflects the ability of a test to identify individuals who do not have the disease, that is, to yield a true negative result (TNR) for those who are healthy or without the respective disease. By definition (Table 1), Specificity = TNR = $D / (B + D)$. Tests with high specificity have very few

false positive results (FPR), which makes them useful for confirming disease when the result is positive [10-15].

The **predictive value** of each test is determined by its sensitivity and specificity, as well as by the prevalence of the disease (for which the test is used) in the respective population. The prevalence of the disease in a given population is equivalent to the pre-test probability (P-pre) that a given symptom (or finding) is associated with the presence of the specific disease [10-15].

Each test additionally has its own likelihood ratio (LR), which summarizes how many times it is more likely (positive likelihood ratio /LR+/) or less likely (negative likelihood ratio /LR-/) for a patient with a given disease to have the respective result (a positive test in the case of LR+ / a negative test in the case of LR-), compared to a patient without the disease [10-15]. For example, the parameter IBP, according to Rudwaleit and colleagues [16-18], has a positive likelihood ratio (LR+) of 3.1 (when present as a symptom) and a negative likelihood ratio (LR-) of 0.33 (when absent as a symptom). This means that the presence of the symptom IBP increases the probability of the presence of axSpA by 31%, whereas the absence of the symptom IBP decreases the probability of axSpA by 33% (the calculations are presented below). The positive (LR+) and negative (LR-) likelihood ratios can be obtained from the sensitivity and specificity of each test using the following formulas: $(LR+) = \text{Sensitivity} / (1 - \text{Specificity})$; $(LR-) = (1 - \text{Sensitivity}) / \text{Specificity}$. These coefficients (LR+ / LR-) represent probability ratios and are treated in the same way as risk ratios in the calculation of confidence intervals (Odds Ratio /OR/): OR = 1 indicates that there is no association between exposure (risk); a result OR > 1 indicates a higher probability of the outcome in relation to the exposure, whereas OR < 1 indicates a lower probability. In the same way, LR > 1 indicates that the test result is associated with the presence of the disease, whereas LR < 1 indicates that the test result is associated with the absence of the disease, regardless of whether it is a positive test (presence of the symptom, LR+) or a negative test (absence of the symptom, LR-). Accordingly, the higher the value of LR, the greater the probability of the presence of a diagnosis if the parameter is positive, and correspondingly, the lower the value of LR-, the lower the probability of a diagnosis if the parameter is negative [10-15]. The probability of the presence of disease after a positive or negative diagnostic test is referred to as the post-test probability (post-test probability /P-post/) and is calculated using Bayes' theorem [18], by applying the "post-test odds," the "pre-test odds," and the likelihood ratio (LR) of the respective test, in combination with the prevalence of the disease in the population (P-

pre) [16-18]: P_{pre} (pre-test probability) = disease prevalence (for the specific population of interest); Pre-test odds = $P_{\text{pre}} / (1 - P_{\text{pre}})$; For a single test, Post-test odds = pre-test odds $\times$ LR; For multiple tests, Post-test odds = pre-test odds $\times$ likelihood ratios (product), where the likelihood ratios (product) /LRP/ = $LR_1 \times LR_2 \times LR_3 \times \dots \times LR_n$; Post-test probability (P_{post}) = post-test odds / (post-test odds + 1).

The use of probabilities (LRs – likelihood ratios), rather than risks (OR), makes the calculation somewhat more complex, but already developed nomograms by Rudwaleit et al. [16-18] - Table 2 and Figure 1; the nomograms of Fagan T.J., which are freely available [19,20]; or the summation of the logarithms of the likelihood ratios (LR+ / LR-) using the method proposed by Feldtkeller E and colleagues [21], can be applied.

Table 2. Representative values of sensitivity and specificity for SpA features along with the resulting LR+ and LR- [16].

Parameter	Sensitivity (%)	Specificity (%)	Likelihood Ratio	
			LR+	LR-
Inflammatory back pain	75	76	3.1	0.33
Heel pain (enthesitis)	37	89	3.4	0.71†
Peripheral arthritis	40	90	4.0	0.67†
Dactylitis	18	96	4.5	0.85†
Iritis or AAU	22	97	7.3	0.80†
Psoriasis	10	96	2.5	0.94†
IBD	4	99	4.0	0.97†
Positive family history for axial SpA, reactive arthritis, psoriasis, IBD or anterior uveitis	32	95	6.4	0.72
Good response to NSAIDs	77	85	5.1	0.27
Raised acute-phase reactants (CRP/ESR)	50	80	2.5	0.63
HLA-B27†	90	90	9.0	0.11
Sacroiliitis shown by magnetic resonance imaging	90	90	9.0	0.11

Legend: $LR+ = \text{sensitivity} / (1 - \text{specificity})$; $LR- = (1 - \text{sensitivity}) / \text{specificity}$; CRP, C-reactive protein; ESR- erythrocyte sedimentation rate; HLA- human leucocyte antigen; IBD, inflammatory bowel disease; NSAIDs- non-steroidal anti-inflammatory drugs. † As enthesitis, dactylitis, uveitis, peripheral arthritis, psoriasis and IBD may not be present at disease onset but may develop later, it is recommended to ignore a negative test result of these tests in an early state of possible axial SpA. ‡The figures for sensitivity and specificity of HLA-B27 refer to a European Caucasian population. In European Caucasian patients with psoriasis or IBD, a sensitivity of 50%, a specificity of 90%, an $LR+$ of 5.0 and an $LR-$ of 0.56 for HLA-B27 should be applied. In other ethnic populations, sensitivity and specificity of HLA-B27 may be different, resulting in different $LR+/-$.

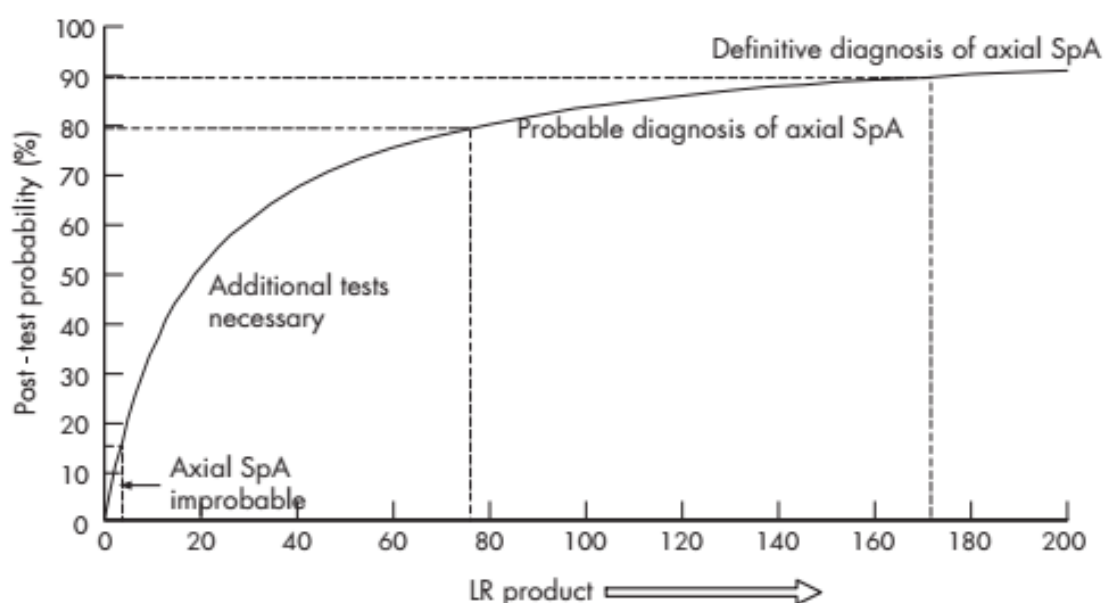


Figure 1. Dependency of the post-test probability of axial spondyloarthritis (SpA) on the resulting likelihood ratio (LR) product for an assumed pretest probability of 5%. This probability curve is meant to be applied in patients with chronic back pain suspected to have axial SpA. Image taken with permission from [18].

The diagnosis can be considered definite if the post-test probability (P-post) is $>90\%$; the diagnosis is probable if P-post is between 80 and 90%, and unlikely if P-post $<20\%$ [15–20]. Based on this principle (a system combining the positive and negative likelihoods of individual signs and symptoms to calculate P-post), diagnostic algorithms for axSpA have also been developed, for example - the Berlin 2004 algorithm and its modification - ASAS 2013 (sensitivity 78.5%; specificity 79.6%) [22]. However, regardless of the method chosen for determining P-post, it should be remembered that the diagnostic value of SpA characteristics varies among different patient populations (i.e., Caucasian vs. Asian vs. African-American) [23]. Furthermore, the specificity and positive likelihood ratio for diagnosis ($LR+$) of these tests vary depending on the clinical setting (primary versus secondary/tertiary care) in which they are applied, as well as on whether they have already served as selection criteria for referral to

a rheumatologist. This phenomenon is clearly demonstrated by the findings of the DIVERS study, discussed in the first part of this review [24].

2. Problems related to the use of the CASPAR criteria

The separation of psoriatic arthritis (PsA) from other arthritides began with the fundamental works of Verna Wright (1956–1959 – “Psoriasis and arthritis” [25-27]) and Baker H. (1963 – “Psoriasis and arthritis” [28]), which led to the inclusion of PsA as a distinct clinical entity in the classification of rheumatic diseases of the ARA (American Rheumatism Association) [28]. The first developed criteria (Moll & Wright 1973) [29] included: (1) presence or personal history of psoriasis or nail involvement; (2) presence of inflammatory involvement of the skeleton (peripheral arthritis and/or sacroiliitis or spondylitis); (3) absence of positive serological tests for rheumatoid arthritis (RA). These criteria, although not validated, are maximally simplified and objective, and form the basis of all subsequent definitions and attempts at classification of PsA (Table 3).

Table 3. Different criteria used to define psoriatic arthritis, with their sensitivity and specificity according to their authors [30-36] and the comparisons made [37,39,40].

Authors; Conditions for defining PsA; Sensitivity and Specificity	Criteria Sets
Moll & Wright 1973†‡ [28] DDg = (1) + (2) + one of (3) Sensitivity -91%; Specificity -94%	(1) Current psoriasis, history of psoriasis or nail disease. (2) NOT Rheumatoid Factor positive. (3) Plus one of 5 distinct clinical subset: 3.1. Oligoarticular arthritis; 3.2. Polyarticular arthritis; 3.3. DIP predominant; 3.4. Spondylitis predominant; 3.5. Arthritis Mutilans.
Bennett et al 1979 ‡ [29]; DDg = Mandatory criteria (2 of 2) + 6 of Supportive criteria (not of these in italic); Sensitivity -69%; Specificity -98%	Mandatory criteria: Clinically apparent psoriasis (skin or nails); Pain and soft tissue swelling and/or limitation of motion in at least one joint observed by a physician for six weeks or longer; Supportive criteria: Pain and soft tissue swelling and/or limitation of motion in one or more other joints observed by a physician; Presence of an inflammatory arthritis in a distal interphalangeal joint. Specific exclusions: Bouchard’s or Heberden’s nodes; Presence of ‘sausage’ fingers or toes; An asymmetrical distribution of arthritis in the hands and feet;

	Absence of subcutaneous nodules; A negative test for rheumatoid factor in the serum; An inflammatory synovial fluid with a normal or increased C3 or C4 level and an absence of infection (including acid fast bacilli) and crystals of monosodium urate or pyrophosphate A synovial biopsy showing hypertrophy of the synovial lining with a predominantly mononuclear cell infiltration and an absence of granuloma or tumour Peripheral radiographs showing erosive arthritis of small joints with a relative lack of osteoporosis. Specific exclusion: erosive osteoarthritis; Axial radiographs showing any of the following: sacroiliitis, syndesmo-phytes, paravertebral ossification
Vasey & Espinosa 1984 † [30]; DDg = Criterion I + one from either –Criterion II or III Sensitivity -97%; Specificity -96%	Criterion I: Psoriatic skin or nail involvement; Criterion II: Peripheral pattern (1) Pain and soft tissue swelling with or without limitation of movement of the distal interphalangeal joint for over four weeks; (2) Pain and soft tissue swelling with or without limitation of motion of the peripheral joints involved in an asymmetrical peripheral pattern for over four weeks. This includes a sausage digit; (3) Symmetrical peripheral arthritis for over four weeks, in the absence of rheumatoid factor or subcutaneous nodules; (4) Pencil in cup deformity, whittling of terminal phalanges, fluffy periostitis and bony ankylosis; Criterion III: Central pattern (1) Spinal pain and stiffness with the restriction of motion present for over four weeks; (2) Grade 2 symmetric sacroiliitis according to the New York criteria (3) Grade 3 or 4 unilateral sacroiliitis;
Gladman et al 1987 ‡ [31]; DDg = ‘A’ + one of ‘B’ + <u>absence of</u>: rheumatoid nodules, grade 4-osteoarthritis, reactive arthritis, enteropathic arthritis, gout, SLE; Sensitivity -91%; Specificity -98%	Mandatory criterion - history of or current psoriasis, psoriatic onychopathy; Supportive criteria: Arthritis; Inflammatory spinal disease; Sacroiliitis;
ESSEG 1991† [32]; DDG = One criterion from – ‘A’ and One or Two criteria from –‘B’; Sensitivity -74%; Specificity -98%	IBP or Synovitis (either asymmetrical or predominantly lower limb); One or more of the following: Positive family history of psoriasis; Psoriasis;
McGonagle et al 1999 ‡ [33]; DDG = ‘A’ + one of ‘B’; Sensitivity -98%;	Mandatory criterion –psoriasis or family history of psoriasis; Supportive criteria: Enthesitis (clinical or radiographic); DIP arthritis;

Specificity -97%	Sacroiliitis /Spinal inflammation; Dactylitis; Monoarthritis; Oligoarthritis (≤ 4 swollen joints); SAPHO; Arthritis mutilans; Onycho-pachydermo-periostitis; Chronic multifocal recurrent osteomyelitis.
Fournie' et al 1999*‡ [34]; DDg ≥ 11 points; Sensitivity -99%; Specificity -88%	Psoriasis antedating or concomitant with joint symptom onset – 6 points; Family history of psoriasis (if criterion 1 negative) or psoriasis post dating joint symptom onset – 3 points; Arthritis of a distal interphalangeal joint – 3 points; Asymmetrical monarthritis or oligoarthritis – 1 points; Buttock pain, heel pain, spontaneous anterior chest wall pain, or diffuse inflammatory pain in the entheses – 2 points; Radiological criterion (if any one criterion present) – 5 points: Erosion of distal interphalangeal joint; Osteolysis; Ankylosis; Juxta-articular periostitis; Phalangeal tuft resorption; Human leucocyte antigen (HLA)-B16 (38, 39) or B17 – 6 points; Negative rheumatoid factor – 4 points
CASPAR*† criteria 2006 [36]; Differential diagnosis – patient must have inflammatory articular disease (joint, spine, or enthesal) as a mandatory criterion and a total score of 3 or more points from the 5 categories listed in opposite column: Sensitivity and Specificity: For established disease [106]: Sensitivity - 91.4%; Specificity - 98.7% For early PsA [107, 108]: Sensitivity - 87.4%; Specificity - 99%	(1) Evidence of current psoriasis, a personal history of psoriasis, or a family history of psoriasis: 1.1. Current psoriasis is defined as psoriatic skin or scalp disease present today as judged by a rheumatologist or dermatologist (Score = 2) 1.2. A personal history of psoriasis is defined as a history of psoriasis that may be obtained from a patient, family physician, dermatologist, rheumatologist, or other qualified health care provider (Score = 1) 1.3. A family history of psoriasis is defined as a history of psoriasis in a first- or second-degree relative according to patient report (Score = 1) (2) Typical psoriatic nail dystrophy including onycholysis, pitting, and hyperkeratosis observed on current physical examination (Score = 1). (3) A negative test result for the presence of rheumatoid factor by any method except latex but preferably by enzyme-linked immunosorbent assay or nephelometry, according to the local laboratory reference range (Score = 1). (4) Either current dactylitis, defined as swelling of an entire digit, or a history of dactylitis recorded by a rheumatologist (Score = 1). (5) Radiographic evidence of juxtaarticular new bone formation, appearing as ill-defined ossification near joint

	margins (but excluding osteophyte formation) on plain radiographs of the hand or foot (Score = 1).
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Legend: *, validated definition; †, easy to use definition; ‡, difficult to use definition; ‡, presence of implied but undeclared characteristics/methods; DD, definite diagnosis.

It is worth noting that among all subsequent definitions [31-36], only that of Fournie and colleagues [36] has been validated. The sensitivity and specificity of all of them have been compared and evaluated [37]. The most sensitive for defining PsA ($\geq 97\%$) were the criteria of Vasey & Espinosa [32], McGonagle [35], and Fournie [36], whereas the others had lower sensitivity, ranging between 56% and 94%. The specificity of all criteria was significant and statistically similar, ranging between 88% and 98%. The criteria of Bennett and Fournie were the most difficult to use, whereas those of Vasey & Espinosa and Moll & Wright were the easiest to use [37].

The CASPAR criteria (CLASSification of Psoriatic ARthritis /CASPAR/ study group) [38] have also been validated and tested, both in patients with established PsA [39-41] and in patients with early PsA [42,43]. The criteria have been compared with all existing ones [39-43], with consistent conclusions-CASPAR criteria have the highest sensitivity among all previous ones (due to the inclusion of family history of PSO), while their specificity (98.7%) is equal to or higher than that of the criteria with the highest specificity ($\geq 98\%$) [39-41].

The reported limitations (by the authors themselves [44]) and by other groups [45,47] are:

The CASPAR criteria are classification, not diagnostic criteria. That is, they are applicable to patients with an established diagnosis of PsA, who have undergone a differential diagnostic process addressing the key disease characteristics (skin/nail psoriasis/dactylitis) - see differential diagnosis of the main features.

The placement of the data on “current PSO, personal or family history of PSO, and/or nail involvement” after the mandatory condition “presence of inflammatory involvement of the skeleton (spinal, peripheral, or enthesal)” makes it possible to classify a patient with axSpA presenting with dactylitis (1 point) and juxta-articular new bone formation (1 point) and negative RF (1 point) as PsA (3 points), in the absence of any evidence of PSO at present, including personal or family history of PSO.

The problem of the possibility of classifying axSpA as PsA in the absence of PSO (PsA sine PSO), as well as personal and family history of PSO, was

discussed as early as 2003 (Scarpa R, et al. [44]), and subsequently in 2007–2009 by the creators of the criteria [44] and other authors [45,47]. However, none of the proposed solutions: (a) inclusion of HLA-Cw6+ as a condition [46,47]; (b) inclusion of imaging biomarkers (MSUS/MRI) as a condition - was not approved [45].

The inclusion of the genetic marker HLA-Cw6 as a condition [46,47] is associated, on the one hand, with 100% development of PSO within 10 years, but on the other hand, only 36–76% of patients with PSO are positive for it, that is, there is a real possibility of missing approximately one-third of patients with PSO (those who are negative for HLA-Cw6 but at the same time develop cutaneous and/or nail PSO) [44]. Thus, the introduction of HLA-Cw6 as a mandatory criterion in the absence of PSO/history of PSO would significantly reduce the sensitivity of the criteria, with a non-significant (1.3%) increase in specificity [44].

The inclusion of a mandatory imaging biomarker (MSUS/MRI - mini-enthesitis and paratenonitis) [45] as a condition in addition to the presence of inflammatory involvement of the skeleton, although highly specific for PsA, would also lead to missing approximately one-third of patients (those with purely axial disease, for which enthesitis of the axial skeleton is characteristic). Thus, we would again significantly reduce sensitivity at the expense of a non-significant increase in specificity [44].

On the other hand, allowing the classification of axSpA as PsA in the absence of personal and family history of PSO is associated with a real probability of developing PSO, similar to that of the axSpA group (≈10% at 5 years; ≈20% at 10 years and ≈35% at 20 years) [48]. This fact, together with the low prevalence of the condition (Table 4), combined with the more destructive nature of arthritis in rPsA compared to that in axSpA, makes the significance of misclassification (axSpA as PsA) much lower compared to the alternative misclassification (PsA as axSpA) [48].

Table 4. Peripheral spondyloarthritis versus Psoriatic arthritis sine psoriasis [46]

Variables	PsA sine PSO†	PsA sine PSO‡
Prevalence	5.5% of all SpA-continuum (studies without overlapping SpA features –COMPOSA; PerSpA); 15% of all cases of PsA / 0.15% of population	2.5% of all SpA-continuum (studies without overlapping SpA features – COMPOSA; PerSpA); 5% of all cases of PsA /0.05% of population
Dactylitis	60-70%	36-40%
Enthesitis	36-40%	60-70%

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Arthritis pattern	DIP 50-70%	Large joints of lower limbs 50-70%
HLA-B27 (+)	18%	36-66%
HLA-Cw6 (+)	36-76%	<1%

Legend: † – PsA with family history of PSO or HLA-Cw6 (+), included in CASPAR criteria as a separate entity (15±5%), 100% progression toward PSO within 2-12 years; ‡ – PsA without any features of PSO (family history, HLA-Cw6+) = pSpA with dactylitis, enthesitis and juxtra-articular new-bone formation on plain radiographs. The probability of developing PSO in this condition (10% over 5 years, 20% over 10 years, and 35% over 20 years of follow-up) is the same as in pSpA.

Differential diagnosis (DD) represents another way of transforming classification criteria into diagnostic criteria, through assessment of the probability that other conditions may be the cause of the respective symptom or finding and their justified, stepwise exclusion.

The problem with DD in PsA within the SpA continuum has two directions: a) Misinterpretation of a rash - the greatest risk here concerns mycosis fungoides vs. PSO. Fortunately, 99% of the problem is avoided by combining dermatological examination with histopathological examination [49,50]; b) Misinterpretation of nail changes (onychomycosis/onychodystrophy vs. PSO) - again, dermatological consultation, staining and fungal culture, and histology solve the problem [51].

DD of the key characteristics for axSpA/PsA - dactylitis and enthesitis (Table 5 and Figure 2) [52-55].

2.1. Dactylitis

Dactylitis is detected clinically in up to 50% of patients with PsA, and as an asymptomatic finding on MSUS (Musculoskeletal Ultrasound) in up to 66% of patients. Dactylitis is a frequent finding at the onset of the disease (as an initial symptom), and recurrent dactylitis (very often in the same digit) may be the only manifestation of PsA for months to years. Dactylitis is usually asymmetrical, with more frequent involvement of the dominant compared to the non-dominant side, and with more frequent involvement of the feet - 66% (fourth toe, usually bilaterally), than of the hands - 44% (second and third fingers, usually unilaterally), while several digits may be affected simultaneously. When dactylitis occurs in a digit with synovial sheaths communicating with the ulnar palmar carpal sheaths, the swelling may extend to the palm (ulnar and/or radial bursae) [52]. Dactylitis may present as acute dactylitis (tender, tense, painful, often erythematous and warm digit) or as chronic (swollen but asymptomatic digit). Whether a chronically swollen but asymptomatic digit represents low-grade acute dactylitis or a resolving one can be determined only by MRI or MSUS, when performed in

combination using gray scale and power Doppler (GSUS/PDUS) [53,54]. MSUS examination, in addition to clinical assessment, is strongly recommended during the initial screening of a SpA patient, precisely because of the risk of missing subclinical dactylitis and enthesitis. The most recent validated MSUS index for the assessment of dactylitis is GLOUDAS (Global OMERACT Ultrasound Dactylitis Score), the validation of which also included participation of our experts. The instructions for scanning, definitions of primary lesions, and the scoring methodology are presented, together with detailed illustration, turning the article into a guide for the assessment of dactylitis with MSUS [55]. Early diagnosis of dactylitis in the course of PsO is essential, both for establishing the diagnosis of PsA and in the context of its role as an unfavorable prognostic factor - acute, "warm" (PDUS+) dactylitis is associated with active disease and predisposes to the development of local erosions/acral osteolysis and radiographic progression (Figure 2). It is therefore not surprising that dactylitis is a component of composite indices for the assessment of PsA, such as the Composite Psoriatic Disease Activity Index (CPDAI) and the Psoriatic Arthritis Disease Activity Score (PASDAS) [56]. The direct relationship between the nail unit and the DIP joint through the enthesis of the DIP extensor supports the link between enthesitis, dactylitis, and psoriatic nail disease, and their typical co-occurrence. This co-occurrence (dactylitis, enthesitis, DIP arthritis, and nail involvement) also shows a genetic association with specific "B" and "C" alleles of the HLA system: the allele B27:05:02-C02:02:02 is associated with all of them and additionally with the presence of symmetrical sacroiliitis; B08:01:01-C07:01:01 with dactylitis, enthesitis, asymmetrical sacroiliitis, nail involvement, and DIP arthritis; B37:01:01-C06:02:01 with enthesitis, dactylitis, nail involvement, and DIP arthritis [57]. The differential diagnosis of dactylitis is presented in Table 5 and illustrated for the rare cases (sarcoid/tuberculous dactylitis) in Figure 3 [56].

2.2 Enthesitis

Enthesitis in peripheral arthritis - is a hallmark of the SpA group and is only sporadically encountered in the course of the other destructive peripheral polyarthritis. That is, MSUS findings of mini-enthesitis/paratenonitis practically resolve the differential diagnosis - RA vs. pSpA/pPsA [51-53] (Figure 2). In the same way, the presence of dactylitis, combined with dermatological consultation and histological findings from a punch biopsy, resolves the differential diagnosis pSpA/pPsA, usually without the need for HLA-Cw6 genotyping.



Figure 2. ‘Hot dactylitis’ –swollen and tender (sausage digit), as a result of soft-tissue oedema, related to the inflammation of the digital enthesial structures, including the fibrous skeleton of the digit (pulleys) also known as ‘pseudotenosynovitis’ or ‘paratenonitis. The flexor tenosynovitis is also present, while the ‘true’ synovitis of MCPs /PIPs /DIPs are usually absent, but may be found in the subacute stage when the ‘paratenonitis’ begins to resolve.

Table 5. Differential diagnosis of dactylitis [50, 56].

Type of dactylitis	Clinical picture	Structures involved	Diagnostic finding	Imaging findings
PsA	Generally asymmetrical swelling of entire digit that is discernibly different than adjacent digits; tenderness; pain, mostly along flexor tendons. May be presenting and only feature for some time	Few fingers and/or toes; sometimes simultaneous involvement of most fingers. May extend below digit to ulnar bursa	US features of ‘hot dactylitis’ (PDUS+); paratenonitis and flexor tenosynovitis	MRI: subcutaneous soft-tissue edema, tenosynovitis, synovitis, osteitis, and bone marrow edema
Sarcoid (figure 3-a)	Focal or diffuse spongy soft-tissue swelling over affected digit from granuloma formation; pain may be absent; overlying skin may be erythematous. Mostly observed in chronic sarcoidosis. May be presenting and only feature for some time	Phalanges and adjacent soft tissue. May involve single or multiple digits. Distal and middle phalanges involved more than proximal phalanges, metacarpals, and meta-tarsals	HRCT- Noncaseating granulomas	Radiographic: incidental bone lesions (diffuse trabecular changes leading to a honeycomb or lattice-like configuration)
Syphilitic	Mostly hands.	Mostly hands	Osteomyelitis	Radiographic: mimics

	Bone changes – osteomyelitis.		(X-Ray; CT; MRI; microbiology)	tuberculous dactylitis, but involvement is bilateral and symmetric; periostitis is more exuberant; soft-tissue swelling less prominent.
Tuberculous (figure 3-b)	Painful soft-tissue swelling around diaphysis of involved bone. Trauma may precede disease onset	Tuberculous dactylitis affects the short tubular bone of the hands and feet. Osteomyelitis extending to digit soft tissue. Hand bones involved more than feet bones. Phalanges affected more often than metacarpals. Proximal phalanges of index and middle fingers most common sites of infection.	Specific granulomatous inflammation with caseous necrosis and positive PPD skin test.	Radiographic: spina ventosa; periostitis. MRI: sinus tract (caused by lesion extending to soft tissue through cortical defects)
Blistering Distal Dactylitis	Tense bullae; Typically, no constitutional symptoms	Anterior fat pad of distal portion of finger or thumb; blister may extend dorsally and involve proximal and lateral nail folds	Infection of anterior fat pad of volar surface of distal part of digit. Mostly caused by group A β -hemolytic streptococci	Not applicable
Sickle cell disease	Sudden onset of very painful, warm, tender global swelling of hands and/or feet lasting 1–3 weeks; fever and leukocytosis may be present.	Carpal and tarsal bones and phalanges	Bone marrow infarction	Radiographic: soft-tissue swelling (first few days); radiolucent areas of the shaft, with surrounding periostitis intermingled with lytic areas and focal osteo-

				sclerosis giving a moth-eaten appearance (within weeks; resembles osteomyelitis)
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Legend: MRI, magnetic resonance imaging; US, ultrasound imaging; PPD, purified protein derivate.



Figure 3. Differential diagnosis of dactylitis: (A) Image of sarcoid pseudodactylitis (left image) and plain x-ray of the right hand (right image), showing intra-osseous bone lesions –the 4th metacarpal displays lattice changes typical of osseous sarcoidosis. Image taken with permission of courtesy of Dr Gurjit Kaeley. (B) Image of osteoarticular tuberculosis dactylitis affecting the right thumb (left image), index and fourth fingers (right image). The X-rays of the affected thumb and fingers showing osteolytic lesions of the second phalanges. Reproduced with permission of [56].

2.3. DD of axSpA from imitators of chronic back pain

The present DD in the context of fibromyalgia with concomitant degenerative disc disease (DDD) and/or osteoporosis, and/or biomechanical problems (scoliosis). This DD, as well as the use of the above-

mentioned (P-pre / P-post / LR±) in the Dg/DD process, is discussed in detail by Philip Mease and Atul Deodar. [57].

Table 6. Differentiating axSpA from mimickers presenting with chronic back pain [57].

Condition and its characteristics	Differentiating clinical features	Key DD methodologies and findings
Fibromyalgia (central sensitization): CBP not IBP; Tenderness mimicking enthesitis; Possibly co-occurring;	CBP has no inflammatory features (better with rest and worse with activity, no / minimal relief with NSAIDs); No objective inflammatory disease in the musculoskeletal system or in the eyes, gut or skin; If observed as a comorbidity - very high patient-reported disease activity after treatment of nr-axSpA is reported	MRI –No objective inflammatory disease [109-114]; MSUS (GSUS and PDUS) examination of enthesal sites [107,108]
Degenerative disc disease: Symptoms of nerve root compression (NRC) in the corresponding dermatome; Possibly co-occurring in younger adults	CBP has no inflammatory features (better with rest and worse with activity); Characteristic of pain – sharp or burning pain, numbness that spreads along a corresponding dermatome; Symptoms of NRC along a corresponding dermatome;	Plain radiographs -disc space narrowing; subchondral sclerosis; CTE -changes in the context of DDD (disc height loss; vacuum phenomenon; bulging /herniation; spondylolisthesis; foraminal or central spinal stenosis. MRI –No sacroiliitis [109-114]; Changes are concentrated in one segment, typically in the lumbar spine, rather than distributed across the whole spine; Spinal bone marrow edema and fatty lesions across the whole endplate rather than just the anterior or posterior corners of the vertebrae on MRI
Spinal osteoarthritis/ Vertebral fractures Older patients; Possibly co-occurring in older patients	CBP has no inflammatory features (better with rest and worse with activity);	Plain radiographs – Degenerative changes; Osteoporosis; Vertebral fractures; MRI – No inflammatory changes; No sacroiliitis [109-114]

DISH (diffuse idiopathic skeletal hyperostosis)/ Forestier disaese	Older patients; Metabolic syndrome; CBP with stiffness but no inflammatory features, weather-related – worsens in damp or cold weather	Plain radiographs/CTE – preserved disc height; pronounced bony bridging over at least four vertebral bodies; right-sided predilection with cervical and lumbar symmetry; pronounced pelvic enthesopathy without sacroiliitis; pronounced peripheral enthesophytes (bony spurs) and ossification of ALL; Osteophytes are largely paraspinal with a linear lucency often seen between the anterior vertebral body cortex and the osteophyte. MRI - fatty metaplasia in the vertebral corners, without or to a lesser extent bone marrow edema [109-114].
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Legend: MRI, magnetic resonance imaging; MSUS, musculoskeletal ultrasound; GSUS, grey scale ultrasound; PDUS, power doppler ultrasound; CTE, computer tomography examination.

3. The problem of axial involvement in PsA (axPsA)

The questions asked here are: Is it present and what is it – a combination of r/nr-axSpA with PSO or a different phenotype of PsA? Where, when and how should we look for it? How should we treat it (role of IL-23 inhibitors in the two types of axial involvement during the course of axPsA)?

A categorical answer to the first part of the first question is given by Dafna D. Gladman [58] – ‘Axial involvement is present in 20 up to 45% of patients with PsA. The variation in reported rates is due to the different tools used (definitions; patient cohorts; diagnostic screening tools; imaging modalities, i.e. MRI vs. X-ray) and the disease duration – because the axial involvement develops and progresses over time.’ The answer to the second part of the question comes several years later, with the definition of the 2 types of axial involvement in PsA, based on many analyses including the follow-up of PsA cohorts, analyses of radiographic / MRI changes, genotyping and the phenotypes in PsA [59-65].

3.1 AxPsA with symmetric involvement of the axis

AxPsA with symmetric involvement of the axis (AxPsA-SAI) is directly associated with carriage of HLA-B27 and its distribution in populations

(different even within one continent – for example, Norwegians 16% [66] and Greeks 6% [67]), explaining the different frequency of this phenotype, even between European countries. The involvement of the axis here is analogous to that in r-axSpA (former AS): 99% presence of IBP /IBP (Inflammatory Back or Buttock Pain) as an initial symptom; the presence of symmetric sacroiliitis in $\geq 90\%$ of cases and symmetric involvement of the spine by marginal syndesmophytes. It is observed approximately in 26–33% of axPsA cases. In addition, in this phenotype there are the highest associations within the PsA group with AAU (25%) and IBD (6%) [68, 69].

3.2 AxPsA with asymmetric involvement of the axis

AxPsA with asymmetric involvement of the axis (axPsA-AAI), observed $\approx 66\%$ of axPsA, is not associated with HLA-B27 (+); IBP/IB*P are present in no more than 50% of cases, and their presence in another 50% of cases is not associated with the presence of sacroiliitis /spondylitis on imaging studies (including MRI). Conversely, $\approx 50\%$ of imaging findings of sacroiliitis /spondylitis (X-ray /MRI) identified in patients with axPsA are in asymptomatic patients [58–64]. The characteristic findings here are asymmetric and/or unilateral sacroiliitis, uneven (zonal or ‘patchy’) involvement of the spine (skipping individual vertebrae or even entire segments), preference for the cervical spine, as well as asymmetric, coarse and non-marginal (not growing from the edge of the vertebra) osteophytes [58–64].

4. Where, when and how should we look for axPsA?

In early PsA (E-PsA), covering the transition from subclinical PsA to classifiable PsA and the first 12 months of the development of classifiable PsA (review part one) [70, 71], – 3 large studies attempt to define the presence of the individual phenotypes in E-PsA [72–74]. Unfortunately, however – they only show us the influence of selection bias of the patients on the final result. All three studies use dermatological cohorts with PSO of similar size (384 [72] vs. 355 [73] vs. 365 [74]). The dermatological patients were screened for the presence of symptoms of subclinical /classifiable PsA by dermatologists, however using different DCS (dermatologist-centred screening tool). After the screening, the ‘suspected’ patients were referred to a rheumatologist for the decision – PsA present or absent. The rheumatologist, for making this decision, performed an examination and could order extended investigations (including HLA typing, MSUS /MRI). Zabotti A, and colleagues [72], use a DCS – centred on the search for arthralgia /synovitis – ‘Have you recently had pain in the joints of your arms (from your shoulders to your fingers) or legs (from your hips to your toes)

for no apparent reason?' and obtain a result that 56 of 384 dermatological patients (15%) have classifiable PsA, of which 82% had peripheral arthritis versus 3.6% with axial disease and 12.5% with enthesal (axial + peripheral) involvement or, in total – 16% for axis involvement [72]. A German study [73], uses the same methodology, but with a DCS – centred on the search for IBP /IB*P – 'Have you recently had back or buttock pain after resting or that wakes you up in the morning and makes you move or stretch to make it go away?' with a result – 19 /350 (6%) presence of classifiable PsA among patients with PSO. Of these: 5/19 (26%) have isolated peripheral PsA; 3/19 (16%) have mixed (peripheral + axial involvement) and 14/19 (74%) have axial involvement. An Italian study [74], uses a DCS with a combined questionnaire – PURE-4 [75] (covering both the peripheral – arthralgia /synovitis and the axial – IBP/IB*P, inflammatory pain). Of 365 screened with DCS, 265 were referred to a rheumatologist (73%), of whom 124 (47%) were assessed as subclinical PsA and were subjected to the extended investigations mentioned above. In 21/124 (17%) isolated pPsA was established; in 36/124 (29.0%) mixed involvement (axial + peripheral) was present, and in 43/124 (46%), PsA remained subclinical (non-classifiable) [74]. What do these numbers mean?

Centred DCS (directed only towards IBP /IB*P or only towards arthralgia /synovitis) might miss real patients with PsA among dermatological cohorts with PSO, generating a lower percentage of the PSO – PsA transition: 15% [72] vs. 6% [73] vs. 46% [74]. On the other hand, axPsA exists already at the E-PsA stage, and it is reasonable to accept a frequency among E-PsA of 20-24% (intermediate relative to the data of Proft F, and colleagues – 64% [73] and Zabotti A, and colleagues – 16% [72]) with a pPsA/axPsA ratio (in E-PsA) $\approx$ 4-5/1. Of this percentage in E-axPsA, 2/3 are HLA-B27 (+) patients (axPsA-SAI phenotype), in whom the axial symptoms start early, may precede the skin manifestations in up to 1/4 of cases and are clinically expressed. In the stage of established axial psoriatic arthritis (axPsA), more and more patients with the axPsA-AAI phenotype are added to the axPsA-SAI phenotype over time. These patients are HLA-B27 negative (-), and their axial symptoms develop slowly [58]. This gradual progression results in approximately 45% axial disease involvement in long-standing PsA, which includes the combined "axial + peripheral" phenotype. Accordingly, the pPsA to axPsA ratio in long-standing established disease is 3:2 (58% vs. 42%) [58]. This 40–45% prevalence correlates excellently with the findings reported by Xabier Michelena and colleagues, who identified 20% "pure axial" and 25% "axial + peripheral" phenotypes in patients with long-duration PsA [76].

The answer to the question – when should we look for axPsA? (which we have just given) is directly related to the question – where and how should we look for axPsA?

Here the answer is screening and we can propose the one used by us: 1) Personal and family history – with great weight in making a decision for extended MSUS/genetic screening/MRI investigations, in young men with PSO and ‘unclear’ clinical findings; 2) Precise physical examination directed towards the enthesal involvement (table 7), on the principle – ‘the more entheses we assess – the better’: MASES – created and validated for the assessment of enthesitis in r-axSpA (AS) – assesses mainly axial entheses and only one peripheral; LEI – created and validated for the assessment of enthesitis in PsA, assesses 6 peripheral entheses; SPARCC and its modification – SPARCC plus One (including all zones from LEI) are created for axSpA [77]. In our practice, the SPARCC plus One index is used due to the inclusion of a greater number of peripheral entheses.

Table 7. Enthesal sites assessed by the most commonly used enthesitis indices [77]

Enthesal sites assessed	MASES	LEI	SPARCC*
First costochondral	R, L		
Seventh costochondral	R, L		
Supraspinatus insertion			
Lateral epicondyle humerus		R, L	R, L
Medial epicondyle humerus		R, L	R, L
Posterior superior iliac spine	R, L		
Anterior superior iliac spine	R, L		
Iliac crest	R, L		
Fifth lumbar spinous process	X		
Achilles tendon	R, L	R, L	R, L
Greater trochanter			R, L
Medial condyle femur			SPARCC plus One‡
Insertion plantar fascia			R, L
Quadriceps insertion patella			R, L
Inferior pole patella			(R, L)*
Tibial tubercle			(R, L)*

Legend: MASES, Maastricht Ankylosing Spondylitis Enthesis Score, developed and validated in patients with AS [148]; SPARC, Spondyloarthritis Research Consortium of Canada, developed and validated in patients with AS [149]; ‡, include one more site (medial femoral condyle – SPARCC plus One, which then

allows calculation of both the SPARCC and Leeds indices, because the other four sites of the LEI are included in the SPARCC); LEI, Leeds Enthesitis Index, developed and validated in patients with PsA [150]; X, single site present, not bilateral; *, The total score of SPARCC is 16, because the distal patella and tibial tuberosity, in close proximity, are considered as one site; R, right; L, left.

Genetic screening (extended screening for alleles associated with axial involvement: B27:05:02–C02:02:02; B08:01:01–C07:01:01; B37:01:01–C06:02:01; HLA-B38; HLA-B39; HLA-Cw07:02 [54, 78]) has a slightly lower cost than the combination: MRI of the whole spine (WS - whole spine) with MRI of the sacroiliac joints (SIJ-MRI). In our setting, genetic screening is usually performed after extended MSUS screening and before WS accompanied by SIJ-MRI.

MSUS assessment of entheses is mandatory, because relying only on history and physical examination (using the enthesitis indices mentioned in table 7), we miss 16–20% of existing subclinical enthesitis [79]. In addition to the assessed peripheral entheses (Table 8) [80–85], an assessment is also performed of all accessible axial and axially-related entheses (A /AR-E – axial /axially related entheses). Three groups of entheses are assessed (Figures 3 and 4) [86–106].

Table 8. Comparison of existed ultrasound (US) enthesal indices –GUESS [80], MASEI [81], OMERACT US-working group (WG) [82], GRAPPA US-WG [83, 84], DEUS Enthesitis Index (DEI) [85].

Enthesal Site	GUESS	EI	OMERACT	MASEI	GRAPPA
Achilles enthesitis (superior pole of the calcaneus)	+	+	+	+	(+)
Plantar fascia (inferior pole of the calcaneus)	+	+	–	+	(+)
Tibialis posterior insertion on navicular bone tubercle	–	–	–		+
Distal patellar ligament attachment (tibial tuberosity)	–	–	–	+	(+)
Proximal patellar ligament attachment (inferior pole of the patella)	+	+	+	+	(+)
Quadriceps insertion (superior pole of the patella)	+	+	+	+	+
Triceps insertion (olecranon tuberosity)	–	–	–	+	+
Lateral epicondyle (common extensor tendon attachment)	–	–	–	–	(+)
Medial epicondyle (common flexor tendon attachment)	–	–	–	–	+
Deltoid muscle insertion on the deltoid tuberosity of the humerus	–	–	–	–	+

Supraspinatus tendon attachment (superior facet of the greater tubercle of the humerus)	–	–	–	–	(+)
Doppler within 2 mm of the cortex of the enthesis	–	+	+	+	+
Doppler >2 mm from the cortex of the enthesis	–	+	–	+	+
Doppler at bursa	–	+	–	+	+

Legend: GUESS, Glasgow Ultrasound Enthesitis Score; DEI, Defining enthesitis on Ultrasound in Spondyloarthritis (DEUS) Enthesitis Index; OMERACT, proposed enthesal sites and scores by the OMERACT Ultrasound Enthesitis Working Group; MASEI, Madrid Sonography Enthesitis Index; GRAPPA, proposed enthesal sites and scores by the GRAPPA Ultrasound Working Group; (+), selected enthesal sites in the GRAPPA-DUET project to distinguish PSA from age- and sex-matched healthy individuals.

4.1 Entheses of the anterior chest wall

Entheses of the anterior chest wall (ACWE) are often involved in axSpA and are accessible to musculoskeletal ultrasound (MSUS). These encompass the entheses of the sternoclavicular joint (SCJ), the costochondral joints and entheses (CCJ) - where the sites at which the ribs meet the cartilage frequently exhibit inflammatory changes - and the manubriosternal joint and its entheses.

4.2 Spinal entheses

Among the spinal ligaments suitable for MSUS evaluation is the iliolumbar ligament (ILL). Notably, one of the first publications regarding this structure originated from a Bulgarian research group [92], and it was subsequently included in the protocol for the MSUS scanning of spinal entheses, again developed with Bulgarian participation (Figure 4) [93]. Other evaluable structures include the lateral costotransverse ligaments (LCTL), which connect the costal tubercle on the posterior aspect of the rib to the lateral border of the transverse process of the corresponding thoracic vertebra (Figure 4) [93], and the supraspinous ligaments (SSL) (Figure 4). Furthermore, the erector spinae muscles, which originate from the common tendon of the thoracolumbar fascia, course upward to attach to the posterior part of the ribs and the spinous processes of the thoracic and cervical vertebrae. These insertions are typical sites of enthesitis in axSpA that are accessible for MSUS evaluation and have already been incorporated into established assessment protocols [93].

4.3 Pelvic and hip joint entheses (Axial/Axial-related)

Pelvic and hip joint entheses represent significant sites of axial and axial-related involvement. In the lumbosacral region, accessible structures include the insertions of the iliocostalis lumborum muscle (which originates from the pelvis and sacrum and primarily inserts into the lower ribs and the transverse processes of the lumbar vertebrae) and the longissimus thoracis muscle (originating from the sacrum, iliac crest, and lumbar vertebrae, and inserting into the thoracic vertebrae and lower ribs) onto the posterior and lateral iliac crests [88]. This region also encompasses the insertions of the thoracolumbar fascia, where the erector spinae muscles converge into a common tendon - the thoracolumbar aponeurosis - that attaches to the iliac crests, posterior sacrum, and spinous processes of the lumbar vertebrae, serving as a key site for the accumulation of mechanical stress and inflammation in axSpA [88]. Other relevant sites involve the greater trochanter, specifically the attachment sites of the gluteus medius and gluteus minimus tendons, which are frequently affected in axSpA [94–96], and the iliac crest at the attachment sites of the abdominal muscles and fascia [97, 98]. Additionally, at the sacroiliac joints (SIJ), the attachments of the posterior longitudinal ligament along the posterior wall and the joint itself are accessible for evaluation by an experienced operator [99–106]. A methodological issue arises here, as the ASAS–MRI definitions of sacroiliitis, which require the presence of osteitis with bone marrow edema and/or erosions [109–114], cannot be visualized by MSUS. Conversely, the pathologies visualizable by MSUS (such as enthesitis, synovitis, and ligamentitis) do not meet the ASAS (MRI) criteria for sacroiliitis. Consequently, the two imaging modalities rely on different pathologies to define the presence of inflammatory changes in the SIJ [99–106] vs. [109–114]. Nevertheless, this discrepancy inherently supports their complementarity, suggesting a diagnostic approach where MSUS screening precedes SIJ and whole-spine (WS) MRI, culminating in a comprehensive assessment of the combined findings.

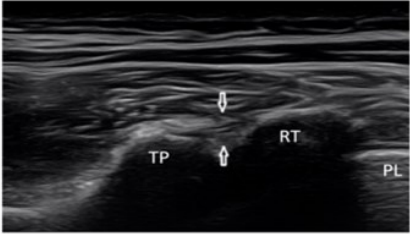
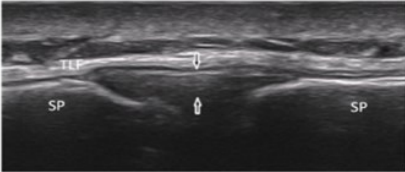
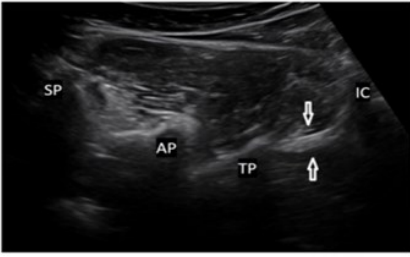
Structure	Patient position	Transducer position	Landmarks	US image
Lateral costotransverse ligament	Seated position with slight anterior flexion of the spine		Vertebral transverse process Rib tubercle	
Supraspinous ligament	Seated position with anterior flexion of the spine		Vertebral spinous process	
Iliolumbar ligament	Lateral decubitus		Transverse process of L5 Posterior Iliac crest	

Figure 3. Available for MSUS examination axially related entheses in axSpA [88]

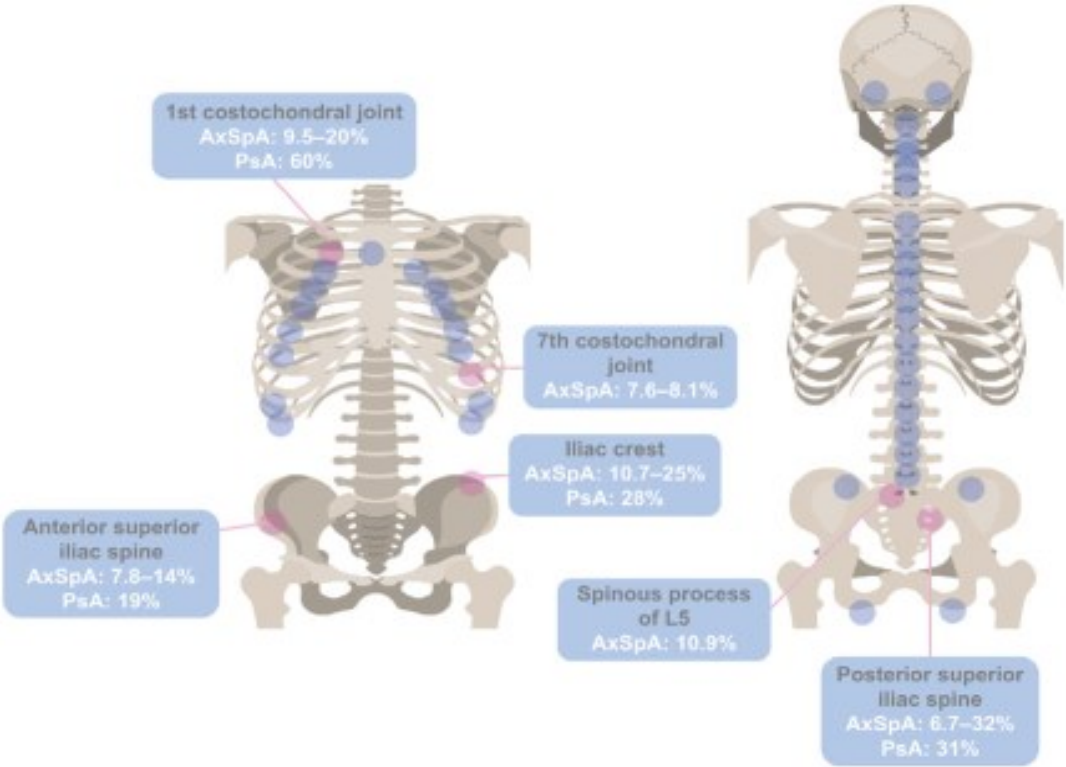


Figure 4. US scanning protocol of the three ligaments studied developed in healthy controls. Arrow: Ligament; AP, articular process; IC, iliac crest; PL, pleural line; RT, rib tubercle; SP, spinous process; TLF, thoracolumbar fascia; TP, transverse process; MSUS, Musculoskeletal Ultrasound.

As can be seen from Table 8, all ultrasound indices for enthesitis assessment cover the key entheses relevant to axSpA (plantar fascia and Achilles tendon insertion to the calcaneus; superior and inferior poles of the patella (quadriceps tendon and patellar tendon)). However, only one of them – GRAPPA-DUET (diagnostic ultrasound enthesitis tool) – was developed specifically for PsA, which is reflected in the greater number of peripheral entheses included in it, with similar US definitions of the primary lesions detected at enthesitis sites (GRAPPA-DUET vs. MASEI vs. DEI vs. GUESS vs. OMERACT) [80–85]. Here again, the rule applies – “the more, the better”, especially when it comes to entheses in axPsA – peripheral [80–85] + axial or axial-related [88–106]. In our practice, MASEI [81] is used for the assessment of axSpA, and GRAPPA-DUET [83, 84] – for the assessment of PsA. Available data (history, enthesitis indices) are an indication for performing MSUS – A/AR, as mentioned above [88–106]. The role of MSUS/PDUS in evaluation and decision-making (biomechanical vs. inflammation-driven lesions) [87] is crucial in differentiating enthesitis from mimickers associated with chronic back pain. Such conditions include those mentioned in Table 6, fibromyalgia [105], and others, especially considering that all of them may also coexist with true axSpA.

In contrast to r-axSpA (100% involvement of the sacroiliac joints (SIJ)) and nr-axSpA ($\geq 75\%$ involvement of the SIJ), where a diagnostic MRI-SIJ is sufficient, in axPsA – in order to capture the focal involvement of the spine, a WS-MRI (whole spine) in addition to SIJ-MRI is required [109–114]. This combination (WS-MRI + SIJ-MRI), as a method for objectifying early (non-radiographic) axial involvement in axPsA, has been used in clinical studies since 2017 [115–120].

However, the examination is expensive and burdensome for the patient (long duration of the examination), therefore the decision to perform it is made after careful assessment of the risk of its presence during the course of PsA (in our practice, after clinical and MSUS evaluations, before or after extended genetic screening).

4. Conclusions

Having classification rather than diagnostic criteria, our path to an accurate, early diagnosis is thorny. None of the widely adopted diagnostic modalities

– (MSUS, including PDUS); MRI (including the new criteria for assessing the nature of lesions), as well as genetic typing – are capable on their own of providing a categorical and final answer (definitive diagnosis). For example, the typically described MRI bone marrow edema of the SIJ was identified in 3131 (30.8%) of 10,165 patients with a mean age of 53 years, during whole-body MRI (WB-MRI) performed in the German NAKO cohort – established to investigate the causes and risk factors for common chronic diseases in Germany [120]. This percentage is ≈ 50 times higher than the reported 0.6% prevalence of axSpA in Germany [120]. That is, each of the new modalities has its strengths and weaknesses, and their results must be carefully weighed when making decisions. This weighing is still only within the capabilities of an experienced physician, because it involves the assessment of many factors: (1) Anamnestic data (ethnicity, sex, age, family history), characteristics of the main symptoms (inflammation vs. biomechanics), their course over time, and the influence of environmental factors (climate, lifestyle, work) or prior interventions (physical or pharmacological) on them. All of these help us calculate the pre-test and post-test probabilities and the likelihood ratio ($LR_{\pm}$) of the main symptoms in the specific patient; (2) Proper selection of tools for physical examination (so that key findings for the group are not missed (enthesitis indices)); (3) Proper selection of laboratory investigations: a) biochemical (lower $LR_{\pm}$ values and respectively Dg/DD values of acute-phase proteins /CRP/ in the nr-axSpA /PsA group), b) serological (the presence of RF /ACPA antibodies in p-PsA with a corresponding impact on $LR_{\pm}$ and Dg/DD value of the findings), c) genetic investigations (HLA-B27, etc.), which also have their $LR_{\pm}$ and corresponding Dg/DD value in decision-making; (4) Proper selection of imaging modalities (conventional X-ray vs. CT vs. MRI vs. MSUM) and assessment of their $LR_{\pm}$ (Dg/DD) values in the specific patient. Careful assessment of post-test probabilities and $LR_{\pm}$ values (the calculations, nomograms, diagnostic algorithms mentioned at the beginning), accompanied by precise DD of the present main characteristics (enthesitis, dactylitis, rashes, nail changes, presence of eye involvement – AAU and bowel involvement – IBD), is our path to the definitive diagnosis. The authors hope that recalling all these details will be useful for correct decision-making by rheumatologists in their daily practice.

Declarations

Author Contributions

Conceptualization: L.S.; Methodology: L.S., T.S.; Formal analysis: M.M & K.B.; Investigation: all authors; Writing - original draft: L.S. & M.M.; Writing – review & editing: all authors. The authors take full responsibility for the content of this manuscript, including all figures and tables.

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Conflicts of Interest

The authors declare no conflict of interest.

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