

Hidradenitis Suppurativa, Spondyloarthritis, and Demyelinating Lesions: A Case Report of a Complex Clinical Association

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Running title: Complex Triad: HS, SpA, and Demyelinating Lesions

Abstract

Background: Hidradenitis suppurativa (HS) is an inflammatory skin disease primarily affecting large folds, which can be associated with spondyloarthritis (SpA). However, the rare coexistence of HS, SpA, and demyelinating lesions presents significant challenges in therapeutic decision-making, especially when resistant to first-line therapies. **Case Presentation:** We report the case of a 17-year-old boy with a three-year history of stage III hidradenitis suppurativa (HS) who presented with chronic inflammatory back pain for three months and a two-month history of a waddling gait. Initial treatments, including topical and oral antibiotics, failed to control his skin lesions. Physical examination revealed lumbar stiffness, positive sacroiliac tests, and brisk lower limb reflexes. Pelvic X-ray revealed bilateral stage III sacroiliitis, and brain MRI showed T2 and FLAIR hyperintensities suggestive of demyelinating lesions. Extensive workup, including cerebrospinal fluid analysis and normal levels of ANA, anti-DNA, anti-NMO, anti-MOG antibodies, syphilitic and HIV serologies, vitamin B12, and folate, concluded to non-specific demyelinating lesions. A diagnosis of radiographic axial SpA and peripheral enthesitis associated with HS was

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established. Worsening skin condition prompted initiation of anti-TNF- α therapy (adalimumab) after multidisciplinary consultation. His gait disturbances improved with physiotherapy, with neurological evaluation favoring a functional origin. Conclusion: Managing concurrent HS and SpA with demyelinating lesions is challenging. Although anti-TNF- α therapy is effective for HS and SpA, its link to demyelinating diseases demands caution. Further research is needed to refine treatment strategies and improve patient outcomes.

Keywords: hidradenitis suppurativa, spondyloarthritis, demyelinating lesions, anti-TNF- α therapy

1. Introduction

Hidradenitis suppurativa (HS), also known as acne inversa or Verneuil's disease, is a chronic inflammatory skin condition that occurs in flare-ups. It primarily affects large skin folds, such as the axilla, submammary, inguinal, and perianal regions [1]. HS has a prevalence of 1% to 4% and predominantly affects young women [2,3]. HS can be associated with other inflammatory diseases, either cutaneous such as psoriasis, acne conglobata, and skin cancers [4–6], or extra-cutaneous such as chronic inflammatory bowel diseases and spondyloarthropathies [7–9]. Spondyloarthritis (SpA) is a chronic inflammatory rheumatic disease that affects the entheses and primarily involves the axial skeleton, potentially progressing to ankylosis. The association of HS and SpA has been reported in the literature [8–11], presenting a therapeutic challenge requiring multidisciplinary management. We report the case of a 17-year-old boy with severe hidradenitis suppurativa (HS) associated with axial SpA, resistant to antibiotics and requiring initiation of anti-Tumor necrosis factor- α (anti-TNF- α) biologic therapy. The patient also presented with gait disturbances and demyelinating lesions on MRI, complicating therapeutic decision-making. A thorough evaluation and multidisciplinary consultation were conducted to guide management.

2. Case Presentation

We report the case of a 17-year-old boy who presented to our rheumatology department in 2023 with chronic inflammatory low back pain persisting for three months before admission. The pain was associated with morning stiffness lasting 20 minutes, improving with activity. The patient had a three-year history of HS classified as stage III according to the Hurley classification [4], initially treated with topical and oral antibiotics, including

tetracyclines, clindamycin-rifampicin, and acitretin with no improvement and worsening of the skin lesions over time. Subsequently, he experienced gait disturbances characterized by a waddling gait for 2 months. Physical examination revealed lumbar stiffness with positive sacroiliac tests and brisk osteotendinous reflexes in the lower limbs with no motor deficits. His skin lesions were characterized by painful nodules, abscesses which progressed to pus discharge, pustules, and subsequent healing with hypertrophic scars mainly located in major folds such as the armpits, buttocks, and intergluteal fold. The pelvic X-ray revealed bilateral stage 3 sacroiliitis. In the presence of neurologic abnormalities, brain MRI was performed and revealed T2 and FLAIR hypersignals in the white matter and in the subcortical occipital region bilaterally, as well as adjacent to the right and left frontal horns, suggestive of demyelinating lesions. There was no evidence of spinal myelitis. Additionally, inflammatory sacroiliitis with involvement of the manubriosternal joint and vertebral enthesis at T10 and T11 was observed. Laboratory tests revealed a CRP level of 53 mg/L and a normal complete blood count. A diagnosis of radiographic axial spondyloarthritis and peripheral enthesitis associated with Hidradenitis Suppurativa was made. To investigate the gait disturbances and demyelinating lesions on brain MRI, a lumbar puncture was performed. Chemical, cytological, and bacteriological analysis of the cerebrospinal fluid (CSF) revealed no abnormalities. Isoelectric focusing of CSF and serum showed a normal profile with a IgG index of 0.42. Levels of ANA, anti-DNA, anti-NMO, anti-MOG antibodies, syphilitic and HIV serologies, as well as vitamin B12 and folate levels, were all within normal ranges. The diagnosis of demyelinating disease was ruled out and the lesions were considered nonspecific. The patient was started on NSAIDs with improvement in rheumatic symptoms. However, he continued to be bothered by his skin disease, for which anti-TNF- α therapy was indicated. Adalimumab is the biologic therapy of choice for resistant HS [12–14]. After a multidisciplinary discussion involving rheumatologists, neurologists and dermatologists, the benefit-risk balance favored initiation of anti-TNF- α therapy, despite the presence of demyelinating lesions. Under adalimumab therapy, the patient showed a marked improvement in articular symptoms, with the BASDAI decreasing from 6.3 to 2. However, no improvement in cutaneous lesions was observed after 12 weeks of treatment, and C-reactive protein (CRP) remained elevated at 30 mg/L.

In view of the persistent dermatological lesions, an intra-class switch to infliximab was performed. This resulted in a favorable clinico-biological response, with significant improvement in both cutaneous and articular

manifestations. At 8 weeks of treatment, CRP had decreased to 3 mg/L and the BASDAI to 1.9, with sustained clinical remission thereafter.

Regarding the gait disorder, the neurological assessment favored considering it a functional gait disorder due to the absence of an organic cause. The patient began physiotherapy sessions, leading to a gradual recovery of normal walking. Summary of the patient’s history is provided in Table 1.

Table 1. Summary of Clinical, Radiological, and Therapeutic Features of Hidradenitis Suppurativa, Spondyloarthritis, and Demyelinating Lesions

	Hidradenitis suppurativa	Spondyloarthritis	Demyelinating lesions
Duration at presentation	3 years	3 months	2 months (gait disturbance)
Symptoms	Painful nodules and abscesses Pus discharge, pustules Hypertrophic scarring Axillae, buttocks, intergluteal fold	Chronic inflammatory low back pain Morning stiffness (~20 min), improving with activity Lumbar stiffness, positive sacroiliac tests Peripheral enthesitis BASDAI: 6.3 at baseline	Waddling gait (2 months) Brisk lower limb reflexes No motor deficit
Key investigations	Clinical diagnosis — Hurley stage III	Pelvic X-ray: bilateral sacroiliitis stage III MRI: sacroiliitis, manubriosternal joint involvement, enthesitis T10–T11 CRP: 53 mg/L	MRI: T2/FLAIR hyperintensities — subcortical occipital (bilateral) and adjacent to frontal horns; no spinal myelitis CSF: normal (chemistry, cytology, bacteriology) IgG index: 0.42 (normal) ANA, anti-DNA, anti-NMO, anti-MOG: normal HIV, syphilis: negative; B12, folate: normal
Diagnosis retained	Hurley stage III HS — refractory to systemic antibiotics and acitretin	Radiographic axial SpA + peripheral enthesitis	Non specific demyelinating lesions — demyelinating disease ruled out

Prior treatments & response	Topical antibiotics — failure Tetracyclines — failure Clindamycin-rifampicin — failure Acitretin — failure, worsening	NSAIDs — improvement of rheumatic symptoms	
1st biologic: Adalimumab	No improvement in skin lesions at 12 weeks	Marked improvement — BASDAI decreased from 6.3 to 2.0	Multidisciplinary discussion: Rheumatology / Neurology / Dermatology
Intra-class switch: Infliximab	Significant improvement of cutaneous lesions; sustained remission	Sustained remission — BASDAI: 1.9; CRP: 3 mg/L at week 8	Benefit-risk balance favored anti-TNF- α initiation despite demyelinating lesions No worsening of neurological status under anti-TNF- α treatment
Gait disorder outcome	Neurological assessment: functional gait disorder (no organic cause identified) — Progressive recovery of normal walking after physiotherapy		

Abbreviations: BASDAI, Bath Ankylosing Spondylitis Disease Activity Index; MRI, magnetic resonance imaging; CRP, C-reactive protein; T2, T2-weighted; FLAIR, fluid-attenuated inversion recovery; CSF, cerebrospinal fluid; IgG, immunoglobulin G; ANA, antinuclear antibodies; anti-DNA, anti-deoxyribonucleic acid antibodies; anti-NMO, anti-neuromyelitis optica (anti-aquaporin-4) antibodies; anti-MOG, anti-myelin oligodendrocyte glycoprotein antibodies; HIV, human immunodeficiency virus; HS, hidradenitis suppurativa; SpA, spondyloarthritis; NSAIDs, non-steroidal anti-inflammatory drugs; TNF- α , tumor necrosis factor alpha.

3. Discussion

We report a case of an association of HS and SpA treated with anti-TNF- α therapy evolving in a context of demyelinating lesions. The association between SpA and HS has been documented in several studies. In a prospective study involving 640 cases of HS, 3.7% of patients had an associated SpA, with skin involvement preceding joint involvement in 90% of cases [8]. A monocentric cross-sectional study with 39 HS patients reported an association with SpA in 28.2% of cases (11 patients) [9]. Additionally, in a study of 449 patients with axial SpA, HS was diagnosed in 41 patients (9.1%), with those having HS showing higher ASDAS scores

[10]. In a retrospective study reevaluating pelvic MRIs of 46 patients, performed as part of the surgical excision preparation for HS, 56.5% of these MRIs showed typical changes indicative of SpA [15]. The pathogenesis of HS remains insufficiently clear [16]. An aberrant innate and adaptive immune response plays a key role in the disease. Notably, both these two diseases share commonalities in their pathogenesis, such as the involvement of TNF- α and interleukins (IL)-23/IL-17 pathways. This shared mechanism explains why anti-TNF- α agents are effective for both conditions [13,16,17]. International guidelines recommend biologic therapy for moderate to severe HS in cases of resistance to systemic antibiotics. Adalimumab is the only approved biologic in adolescents for HS and serves as the first-line biologic treatment in all guidelines [14]. Its efficacy and safety in Hurley stage II/III HS have been demonstrated in randomized controlled trials [18–20]. It is administered subcutaneously, starting with a 160 mg loading dose at week 0, followed by 80 mg at week 2, and then 40 mg weekly [21]. Infliximab is generally considered a second-line biologic therapy, administered using the same dosing regimen as in SpA: 5 mg/kg intravenously at weeks 0, 2, and 6, followed by infusions every 8 weeks.

If anti-TNF- α therapy fails, anakinra and ustekinumab may be alternative options, though supporting evidence for their efficacy in HS remains limited [14]. For SpA, biologic therapy is recommended when at least two classes of NSAIDs fail to provide improvement after four weeks of treatment. Available options include anti-TNF- α , IL-17 inhibitors (IL-17i), and JAK inhibitors, with no clear evidence favoring one class over another in axial SpA [22]. For our patient, the resistance of HS to local and oral antibiotics and acitretin, as well as the recurrence and severity of flare-ups, indicated the need for anti-TNF- α biologic therapy, despite his SpA responding well to NSAIDs. However, the presence of T2 and FLAIR hypersignals in the white matter on cerebral MRI, frequently found in older subjects [23,24], required further investigation into demyelinating diseases such as multiple sclerosis (MS) or neuromyelitis optica.

Few studies have investigated the prevalence and clinical relevance of white matter hyperintensities (WMH) in young individuals.

A study involving individuals younger than 45 years reported a WMH prevalence of 25.9% (324/1249), excluding participants with a history of stroke, traumatic brain injury, neoplasm, psychiatric disorders, demyelinating diseases, metabolic disorders, or substance abuse. In this study, WMH were associated with a history of cardiovascular disease, including hypertension, diabetes mellitus, and hyperlipidemia [25].

Other risk factors for WMH in young adults reported in the literature include female sex, headaches and syncope, sleep disturbances[26], migraine[27], and bipolar disorder [28].

Furthermore, WMH in young adults have been associated with poorer working memory performance[29].

The association between demyelination and SpA has been reported. However, it remains unclear whether these events are coincidental, driven by a shared pathophysiological mechanism, or linked to anti-TNF therapy. A longitudinal observational study of 2,268 SpA cases identified 26 cases (1.14%) of demyelinating disease, including eight patients who were anti-TNF-naïve. This corresponds to an incidence of 31 demyelinating events per 100,000 person-years, which, although low, remains higher than the general population's incidence of multiple sclerosis (2.1 per 100,000 person-years) [30]. Demyelination of the central nervous system following anti-TNF- α treatment is a rare event [31]. However, cases of demyelinating diseases, including MS and neuromyelitis optica, have been observed in patients treated with these agents [32–35]. Exposure to anti-TNF- α has been associated with an increased risk of inflammatory central nervous system events, with a similar risk for both demyelinating and non-demyelinating events [36]. In MS, anti-TNF- α therapy has been associated with the potential to exacerbate demyelination and worsen symptoms [37,38]. Genetic susceptibility may predispose certain patients to demyelination when treated with these agents [39]. However, It remains unclear whether these events are coincidental or represent a treatment side effect [35,40]. Given the severity of his skin disease and the absence of an absolute contraindication to anti-TNF- α therapy, this treatment was indicated after a multidisciplinary consultation meeting. The patient was initially treated with adalimumab and experienced a marked improvement in joint symptoms; however, no improvement was observed in the cutaneous lesions. Although adalimumab is considered the first-line biologic therapy for HS, suboptimal responses have been reported, with an overall therapeutic response rate of approximately 40% [41]. Given the absence of cutaneous improvement, treatment was switched to infliximab, a second-line biologic option for HS. This therapeutic change resulted in a favorable clinical and biological outcome, with significant improvement in both the articular and cutaneous manifestations.

4. Conclusions

This case highlights the challenges of managing concomitant HS and SpA in the presence of demyelinating lesions. Anti-TNF- α agents are effective for HS and SpA but require caution due to their possible neurological risks. Awareness of these associations is crucial to guide therapeutic decisions.

Declarations

Author Contributions

Conceptualization: Yasmine Makhoulf, Wassim Tekaya.; Methodology: Yasmine Makhoulf, Wassim Tekaya.; Formal analysis: Yasmine Makhoulf, Wassim Tekaya.; Investigation: Yasmine Makhoulf, Wassim Tekaya.; Writing—original draft: Wassim Tekaya; Writing—review & editing: all authors.

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Data Availability Statement

The data presented in this study are available on reasonable request from the corresponding author.

Conflicts of Interest

The authors declare no conflict of interest.

Artificial Intelligence Statement

During the preparation of this manuscript, artificial intelligence tools were used solely for language editing and grammatical correction. The authors take full responsibility for the content of the manuscript.

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