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CASE BASED REVIEW

Acute onset of psoriatic spondyloarthritis as a new manifestation of post-streptococcal reactive arthritis: a case series

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Abstract

Streptococcus is well associated with a myriad of inflammatory diseases. Among others, this bacterium is linked to the triggering of psoriasis and to post-streptococcal reactive arthritis (PSRA), an arthritis which is typically confined to peripheral joints. Three patients who developed acute psoriatic spondyloarthritis (SpA) following a recent streptococcal infection are described in this article. We searched the existing literature for cases of axial involvement in PSRA and reviewed the association between streptococcal infection and psoriasis or psoriatic arthritis)PsA). In all patients, psoriatic SpA occurred within 7–10 days of a confirmed streptococcal infection. The main presenting syndrome was inflammatory back pain with evidence of acute axial spondyloarthritis on magnetic resonance imaging. One patient had guttate psoriasis, the second patient developed pustular psoriasis, and the third patient had exacerbation of pustular palmoplantar psoriasis. Two patients required treatment with tumor necrosis factor alpha (TNF-α) blockers. Axial involvement in PSRA is very rare. A potential association of streptococcal infection and development of PsA has been explored in several articles. However, to the best of our knowledge, acute psoriatic SpA as a manifestation of PSRA has yet to be described. Acute psoriatic SpA should be considered in the differential diagnosis of new-onset inflammatory back pain followed by psoriasis in young adults who had a recent throat infection.

Key Points

- *Our case series describes three cases of acute psoriatic spondyloarthritis that occurred within 7–-10 days of a confirmed streptococcal infection and progressed to full blown chronic disease.*
- *Acute psoriatic spondyloarthritis as a manifestation of post streptococcal reactive arthritis should be considered in the differential diagnosis of new onset inflammatory back pain followed by psoriasis in young adults who had a recent throat infection.*

Keywords Group A streptococcus · Post-streptococcal reactive arthritis · Psoriasis · Reactive arthritis · Spondyloarthritis

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Introduction

Group A streptococcus (GAS), e.g., *Streptococcus pyogenes*, is an aerobic gram-positive bacterium that is associated with a diverse spectrum of syndromes that usually occur 1–3 weeks after a throat or skin infection [1]. Among the post-streptococcal rheumatic diseases, acute rheumatic fever (ARF) and post-streptococcal reactive arthritis (PSRA) are considered the most common clinical entities, each with a distinct pattern of clinical manifestations [2–10]. ARF is mainly characterized by migratory, non-erosive, self-limited polyarthritis with predominant involvement of the large joints and also by carditis, chorea, erythema marginatum, and subcutaneous nodules (major Jones criteria) [2]. In contrast, PSRA is usually manifested by acute non-migratory peripheral arthritis that may affect any joint, and has a protracted course and low responsiveness to NSAIDs [4]. In a subset of patients with PSRA, arthritis of the lower extremities and/or the involvement of the axial spine (rarely) resemble the articular disease in patients with classic reactive arthritis [6]. However, unlike PSRA, reactive arthritis is usually triggered by enteric and genitourinary pathogens (e.g., *Salmonella*, *Shigella*, *Chlamydia*, and *Yersinia*) and may be characterized by a wide spectrum of extra-articular symptoms, including ocular, cardiac, gastrointestinal, or genitourinary, as well as mucocutaneous lesions [11]. A triad of symptoms (conjunctivitis, arthritis, and urethritis) is found in only one-third of reactive arthritis patients. Among the dermatological manifestations of reactive arthritis is keratoderma blennorrhagicum, a hyperkeratotic erythematous dermatitis that is clinically and histologically comparable with pustular psoriasis [12]. Similarly, streptococcal infection may induce guttate psoriasis, presented by a sudden onset of diffuse red and scaling papules, that commonly resolves after several months or may progress to plaque psoriasis [13, 14].

Additionally, it has been reported that streptococcal throat infection led to a worsening of chronic plaque psoriasis [15]. To the best of our knowledge, acute psoriatic spondyloarthritis (SpA) as a manifestation of PSRA has yet to be reported, despite the known relationship between streptococcal infection and inflammatory arthropathy and the known relationship between such an infection and psoriasis.

In the current case series, we present three patients who developed psoriatic SpA following a recent streptococcal infection. Two of them experienced new-onset pustular and guttate psoriasis, and in the remaining patient, pustular palmoplantar psoriasis exacerbated after 15 years of full remission.

We searched for cases of axial spondyloarthritis as a manifestation of PSRA in the published literature. Additionally, the association between streptococcal infection and psoriasis or psoriatic arthritis (PsA) was reviewed.

Case 1

A 37-year-old woman presented to the emergency department with a severe inflammatory low back pain for the past seven days followed by a pustular rash located on her shins, groins, and lower abdomen. Two weeks prior to the admission, the patient was diagnosed with tonsillopharyngitis that was supported by a positive throat culture for GAS, and treated accordingly with oral penicillin V. She had no previous history of arthritis, back pain, enthesopathy, uveitis, psoriasis, or other extra-articular symptoms of axial spondyloarthritis. She denied a recent urinary, gastrointestinal, or ocular infection, or the presence of a sexually transmitted disease (STD). Family medical history was unremarkable.

The examination revealed chest wall and thoracic spine tenderness, bilateral positive FABER (Patrick) test, positive sacroiliac distraction test, and a pustular psoriasis on her lower limbs sparing the palms of her feet.

Blood analysis revealed elevated C-reactive protein (CRP) of 94 mg/L (normal < 5 mg/L). Anti-nuclear antibodies (ANA), rheumatoid factor (RF), anti-cyclic citrullinated peptide (anti-CCP), human leucocyte antigen-B27 (HLA-B27), and viral hepatitis serologies were negative. A radionuclide bone scan demonstrated increased uptake in several joints including the right sternoclavicular joint, left costovertebral joint, and both sacroiliac joints.

Magnetic resonance imaging (MRI) of the lumbar spine and sacroiliac joints showed acute bilateral sacroiliitis, along with active corner inflammatory lesions at L2–L4 (Fig. 1). The patient was diagnosed as having PSRA with axial involvement followed by pustular psoriasis and was started on intravenous (IV) methylprednisolone therapy for 3 days. She was discharged on naproxen 500 mg twice daily which was later changed to etoricoxib 90 mg/day with partial improvement. One month later, in light of a Bath Ankylosing Spondylitis Disease Activity Index (BASDAI) score of 6.9 and severe pustular psoriasis, biological treatment with adalimumab (40 mg every two weeks subcutaneously) was initiated. Following this treatment, the psoriasis resolved completely within one year and BASDAI decreased to < 3. One year later, while being treated with adalimumab, the patient had another episode of tonsillitis with a new episode of inflammatory back pain. The back pain improved with the continuation of adalimumab and etoricoxib.

Case 2

A 35-year-old woman was referred to a rheumatologist due to inflammatory back pain and pain in the buttocks for two months that was concomitant with diffuse non-pruritic red and scaling papules. During the last year, she had several episodes of tonsillitis and high fever that required treatment with antibiotics. The last episode occurred 10 days prior to the

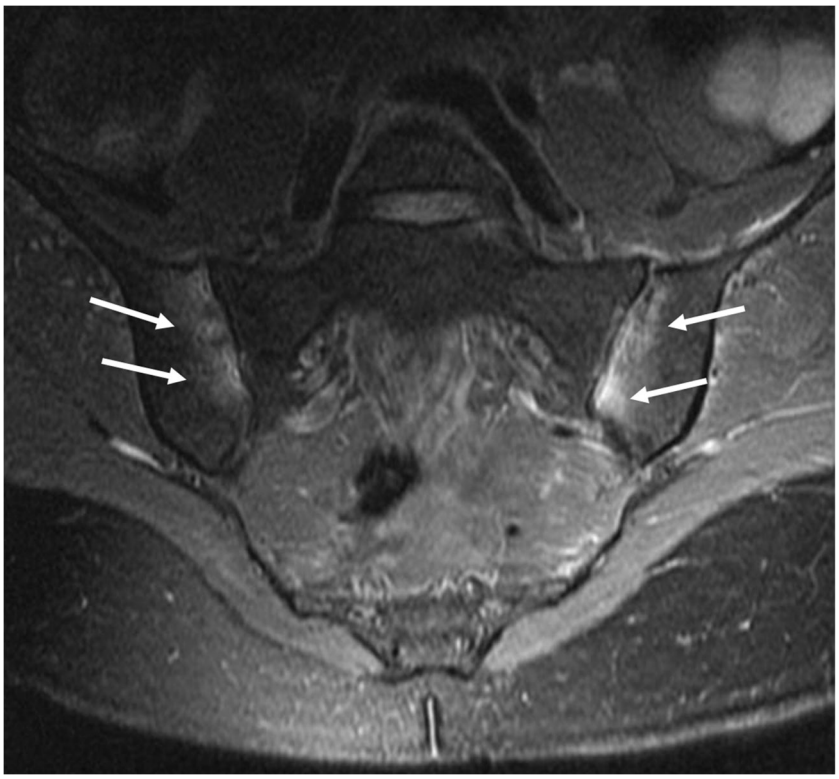

Fig. 1 Case 1. Pelvic magnetic resonance imaging of the sacroiliac joints with gadolinium enhancement and fat suppression performed 10 days after admission. High signal intensity is seen bilaterally at the subcortical bone marrow as well as inside the sacroiliac joint (arrows). Compatible with sacroiliitis

onset of back pain and rash, and she was treated with amoxicillin/clavulanate for 10 days.

She had no previous history of articular or extra-articular manifestations of axial spondyloarthritis, but had a family history of psoriasis in a second-degree relative.

Physical examination revealed arthritis of the left sternoclavicular joint, tenderness of the left tibialis posterior tendon, and a positive bilateral FABER (Patrick) test. Skin examination revealed teardrop-shaped rash on both legs and on the right forearm that were consistent with guttate psoriasis.

Blood analysis showed increased CRP level (40 mg/L), while HLA-B27, RF, ANA, and viral hepatitis serologies were negative. The serum anti-streptolysin O (ASO) titer was high (510 IU, normal < 200 IU) and confirmed the preceding streptococcal infection.

MRI of the lumbar spine and sacroiliac joints showed acute bilateral sacroiliitis. MRI of the spine revealed active corner inflammatory lesions on the anterior vertebral edge of L1, L2, L3, C5, and D1.

She was diagnosed with PSRA with axial involvement followed by guttate psoriasis and treated with NSAIDs including etoricoxib (90 mg/day for ten days) followed by etodolac (800 mg/day), with only a partial improvement. Afterwards, she was treated for four months with oral corticosteroids (prednisone at a dosage of 40 mg/day with gradual tapering) in combination with oral sulfasalazine (1.5 g/day), with a resolution of the rash and peripheral arthritis but without alleviation of inflammatory back pain. After 4 months of treatment, she had a BASDAI score of 5 and started treatment with infliximab (5 mg/kg every eight weeks). One year after the commencement of infliximab, her BASDAI reduced to 2.5, she had no rash, and her CRP level was within normal limits.

Case 3

A 36-year-old woman was admitted to the Department of Internal Medicine due to severe inflammatory low back pain that lasted for a week. Additionally, she had pustular rash that covered the forearms, right hip, left ankle, and the palms of both hands. Two weeks before the hospitalization, she was diagnosed with follicular tonsillitis. At that time, since the patient had allergy to penicillin, treatment with first-generation cephalosporins was initiated for one week with a full resolution of symptoms. Her previous medical history included pustular psoriasis that was successfully treated with phototherapy and topical corticosteroids without recurrence of psoriasis for 15 years. She had no previous history of articular or extra-articular manifestations of axial spondyloarthritis. Prior to the hospitalization, she was treated with ibuprofen (1200 mg/day) and with injections of diclofenac sodium (75 mg) intramuscularly for 5 days.

Physical examination revealed pustular psoriasis on the arms and palms, as well as bilateral tenderness of the sacroiliac joints with a positive FABER test. No peripheral arthritis,

enthesitis, tendinitis, or limitation of spinal movement was observed.

Blood analysis revealed elevated C-reactive protein (CRP) of 9 mg/L (normal < 5 mg/L), while HLA-B27, RF, ANA, EBV, CMV, HIV, and viral hepatitis serologies were negative. The first ASO test that was performed at the same day of hospitalization was invalid due to technical issues, but a repeated test that was done 5 weeks after the acute infection was positive (250 IU). MRI revealed acute bilateral sacroiliitis.

She was diagnosed with PSRA with axial involvement (bilateral sacroiliitis) followed by pustular palmoplantar psoriasis. Parenteral corticosteroids, particularly methylprednisolone at a dosage of 250 mg/day, were given for 4 days. Afterwards, she was treated for two weeks with oral corticosteroids at a dosage of 20 mg/day with a rapid tapering. In addition, etoricoxib (90 mg/day) was administered for three months with clinical improvement. The patient continued to receive topical treatment for psoriasis and remains in follow-up in our outpatient clinic.

The demographic and clinical characteristics of all three patients with psoriatic SpA are summarized in Table 1.

Discussion

PSRA appears to be a rare heterogeneous clinical entity that may present as axial spondyloarthritis in a subset of patients [6]. In our experience, while psoriatic SpA is relatively common, its presentation as a part of PSRA has not been described previously. We report three cases of HLA-B27-negative patients who developed psoriatic SpA following a recent episode of tonsillopharyngitis that occurred, on average, 8 days beforehand. A streptococcal infection was confirmed by a positive throat culture or ASO test. The main presenting syndrome was inflammatory back pain with evidence of acute bilateral sacroiliitis and inflammatory spinal lesions on MRI. One patient had a family history of psoriasis and developed guttate psoriasis. In the remaining two patients, the GAS infection was followed by the occurrence of pustular psoriasis or its exacerbation after prolonged remission. At presentation, all patients had clinical characteristics of PSRA and fulfilled the classification criteria for psoriatic arthritis (CASPAR). All our patients had clinical features of psoriatic SpA that progressed to full-blown chronic disease with low-moderate response to NSAIDs and corticosteroids. Two patients required initiation of tumor necrosis factor alpha (TNF-α) blockers.

There is limited data regarding axial spondyloarthritis as a part of PSRA [3, 5–10]. A few case reports and case series reported the development of axial involvement in both HLA-B27-positive and HLA-B27-negative patients with PSRA [3, 6]. One of the first reports of PSRA with axial involvement was made in 1982 and presented a HLA-B27-positive woman with peripheral arthritis and sacroiliac pain following a streptococcal throat infection [16]. More recently, an interesting study including 25 PSRA patients reported that 6 of them had axial disease and 3 of them were positive for HLA-B27. It has been suggested that HLA-B27-positive patients have a higher susceptibility to developing sacroiliitis [3]. Additional features of spondyloarthritis such as enthesitis and tenosynovitis have also been reported in the setting of PSRA, either with or without the presence of arthralgias or arthritis [7, 10]. Furthermore, concomitant anterior uveitis in HLA-B27-negative patients with

Table 1 Characteristics of patients affected by psoriatic SpA

Feature	Patient 1	Patient 2	Patient 3
Gender (M/F)	F	F	F
Age at diagnosis (years)	37	35	36
Evidence of GAS infection	Positive throat culture	ASO—510 IU	ASO—250 IU
Onset of arthritis after infection (days)	7	10	7
Axial involvement	Bilateral SI/lumbar spine	Bilateral SI/lumbar spine	Bilateral SI
Peripheral involvement	Arthritis—right SCJ	Arthritis—left SCJ, tendinitis—TP	No
Type of psoriasis	Pustular	Guttate	Pustular palmoplantar
Family history of psoriasis	No	Yes	No
Previous psoriasis	No	No	Yes, pustular
CRP (mg/L) (normal < 5 mg/L)	94	40	9
HLA-B27	Negative	Negative	Negative
Response to NSAIDs	Low	Low	Moderate
Treatment with TNF-α blockers	Adalimumab	Infliximab	No
Duration of follow-up (months)	12	12	3

ASO serum anti-streptolysin O, *CRP* C-reactive protein, *GAS* group A streptococcus, *IU* international units, *NSAIDs* nonsteroidal anti-inflammatory drug, *PSRA* post-streptococcal reactive arthritis, *psoriatic SpA* psoriatic spondyloarthritis, *SCJ* sternoclavicular joint, *SI* sacroiliitis, *TNF-α* tumor necrosis factor alpha, *TP* tibialis posterior

PSRA had been also described, and this complication should be identified early and treated accordingly [17].

Several lines of evidence support the idea that psoriasis may be a sequela of streptococcal infection [13–15]. For example, the reported incidence of streptococcal infections preceding guttate psoriasis ranges between 56 and 97% [13]. Likewise, the potential association of this type of infections and the development of PsA has been also explored [18]. Thus, in one study, the prevalence of anti-DNase-B antibodies was 51% in patients with PsA, in comparison with 10% in healthy controls [19]. An additional study found an association between PsA and the presence of *Streptococcus pyogenes* itself [20]. In particular, higher levels of 16S rRNA of *Streptococcus pyogenes* were observed in the blood of 19 patients with PsA, in comparison with patients with rheumatoid arthritis or patients with other types of arthritis. In contrast, another study has not found increased incidence of a preceding streptococcal infection in patients with PsA [21].

The pathogenic mechanisms underlying the development of PSRA are not fully understood. While various factors are involved in the pathogenesis of classic reactive arthritis, including genetic factors (i.e., HLA-B27), bacterial antigenicity, and the type of host response (i.e., Th1/Th2 imbalance), it is unknown whether these factors are involved in the pathogenesis of PSRA [11]. Regarding the genetic aspect, while HLA-B27 is positive in 50 to 80% of patients with reactive arthritis [11] and in 20–35% of patients with PsA [22], its prevalence in patients with PSRA is similar to the prevalence in the general population [5]. However, additional genetic factors associated with psoriasis and PsA, such as HLA-Cw6, may be possibly involved in the pathogenesis of acute psoriatic SpA triggered by GAS infection [23]. In our case series, all patients were HLA-B27 negative, but one of them had a family history of psoriasis.

Since classic reactive arthritis, PSRA, and psoriasis all represent post-infectious entities, molecular mimicry should be considered to be one of the pathogenetic mechanisms in these disorders. Similarly, this mechanism may be possibly involved in acute psoriatic SpA. It has been postulated that the extensive homology between streptococcal M proteins and human epidermal keratin may play a role in the pathogenesis of psoriasis [24]. Thus, the disease may be initiated by CD8 T cells that recognize the streptococcal M protein in the palatine tonsils and the keratin in the skin. Subsequently, skin-homing CD4 T cells, along with γδ T cells, could play the role of an amplifier of inflammation and contribute to a self-sustaining inflammatory loop in the dermis [25, 26]. In the case of PsA, the pathogenic link between inflammatory T cell responses arising in the skin and the potential development of this disease has been recently investigated. Thus, markedly increased levels of IL17-producing CD8 T cells were found in synovial fluid of patients with PsA, and they were correlated with the disease severity [27].

One of the clinical features of PSRA is a prolonged course with low-to-moderate response to NSAIDs [3]. Similarly, two of our patients were refractory to NSAIDs and corticosteroids, and one of them had no response to sulfasalazine. There are no guidelines regarding further treatment in non-responders, and these patients are usually treated with corticosteroids and non-biological DMARDs, mainly sulfasalazine. Based on the efficacy of TNF-α blockers in both PsA and reactive arthritis, two of our acute psoriatic SpA patients were commenced on TNF-α blockers (adalimumab or infliximab), with a good response. To the best of our knowledge, only one case report described successful treatment of refractory PSRA with a TNF-α blocker (adalimumab) [28].

In our case series, all patients received short-term antibiotics. Currently, there is no consensus about the necessity and the duration of antibiotic prophylaxis for adults with PSRA. In two cases series, no increased risk of carditis was found in patients with PSRA who did not receive antibiotic prophylaxis during long-term follow-up [29, 30]. Similarly, there are no specific recommendations regarding prophylactic antimicrobial therapy or tonsillectomy for the treatment or prevention of flares in patients with psoriasis or PsA.

In conclusion, we present an interesting case series of three HLA-B27-negative patients who presented with acute psoriatic SpA triggered by GAS infection. Our observation suggests that this clinical entity should be considered in the differential diagnosis of new-onset inflammatory back pain followed by psoriasis in young adults who had a recent throat infection.

Compliance with ethical standards

Ethics We have been officially waived from the IRB committee approval, as this is a case series.

Disclosures None.

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