CASE REPORT

Navigating the Diagnostic Maze of Psoriatic Arthritis sine Psoriasis in Primary Care.

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ABSTRACT

Psoriatic arthritis, a seronegative spondyloarthropathy is an autoimmune inflammatory joint disease. However, the diagnosis is often delayed due to the absence of specific biomarkers and a lack of awareness among primary care providers, who may be unable to recognize the key features of the condition. We present a case of a 30-year-old woman with a 9-month history of lower back pain and multiple joint pain. Despite elevated inflammatory markers like C-reactive protein and erythrocyte sedimentation rate, other initial tests including rheumatoid factor and antinuclear antibody tests were all negative. The appearance of new skin lesions in the 10th month prompted further evaluation and resulted in a diagnosis of psoriatic arthritis. Treatment with Celecoxib and Methotrexate led to significant improvement in her condition. This case underscores the crucial role of primary care providers in the early detection and management of spondyloarthropathy, helping to prevent joint damage and enhance patient outcomes.

INTRODUCTION

Seronegative spondyloarthropathies (SpA) are a group of autoimmune, inflammatory arthritis conditions that include psoriatic arthritis (PsA), ankylosing spondylitis (AS), juvenile-onset SpA, enteropathic arthritis, and reactive arthritis. Common clinical features in SpA include asymmetrical peripheral arthritis, inflammatory back pain, dactylitis, enthesitis, and extra-articular manifestations. These conditions are characterized by a negative rheumatoid factor (RF) and pose significant challenges in early diagnosis. A delay in diagnosis of SpA with an average delay of 5 to 7 years, or even longer in women, can lead to structural joint damage.[1] Even a 6-month delay in diagnosis can increase the risk of joint erosion and negatively impact patient outcomes.[2] Early recognition and detection by primary care providers is crucial to mitigate the risk of joint erosion and improve long-term outcomes.

CASE REPORT

A 30-year-old woman presented with a 9-month history of intermittent lower back and joint pain. The lower back pain was characterized by an achy, stiff sensation, typically lasting for 30 minutes to an hour. The pain worsened with rest and improved with movement, frequently disrupting her sleep with severe pain, rated 8/10, which caused her to wake up in agony during the night. Despite multiple medical consultations, her symptoms persisted. Initial investigations, including anti-nuclear antibody (ANA), rheumatoid factor (RF) and radiographic yielded normal results. She was initially diagnosed with musculoskeletal pain and prescribed analgesics for symptom management.

As her symptoms worsened, the patient developed pain in both her heels, her right wrist, and the small joints in her right hand, which significantly impacted her daily activities and work performances, resulting in frequent medical leaves. Despite attempting various supplements and over-the-counter analgesics, her symptoms persisted. Throughout her illness, she denied experiencing any depressive episodes. After nine months of unresolved pain, she sought a second opinion at a tertiary hospital's primary care clinic.

On examination, onycholysis was noted on her right thumb (Figure 1), but there were no signs of nail pitting, transverse ridging, or tenderness and swelling in her hands. There was mild tenderness and fusiform swelling over the right fourth toe, consistent with dactylitis (Figure 2), along with mild swelling of the right heel, suggesting enthesitis. Otherwise, there were no visible skin lesions, unremarkable neurological and motor examinations and her spine examination revealed a full range of motion without deformity; Schober's test was negative and the sacroiliac joint was non-tender.

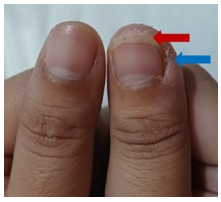

Figure 1: Onycholysis (red arrow) of right thumbnail with periungual involvement (blue arrow).

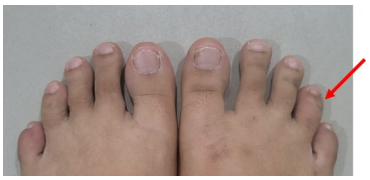

Figure 2: The presence of tender and fusiform swelling over the right 4th toe (dactylitis) with post-inflammatory hyperpigmentation.

Re-evaluation revealed elevated inflammatory markers, with CRP level of 36 mg/L and an ESR of 38 mm/hour, alongside negative RF, ANA and anti-cyclic citrullinated peptide (anti-CCP) tests. A pelvic x-ray showed irregularities and sclerosis in both iliac bones with a normal sacroiliac joint (Figure 3). Based on these findings, she was diagnosed with inflammatory back pain (IBP) with suspected diagnosis of seronegative rheumatoid arthritis, and referred to rheumatology for further evaluation.

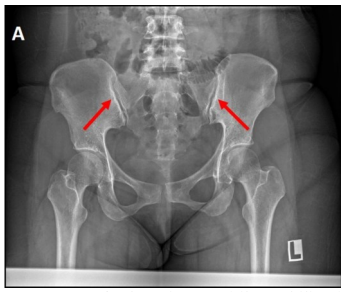

Figure 3: Presence of sclerosis over bilateral iliac bone and irregularities with normal sacroiliac joints

One month later, during the rheumatology assessment, new scaly skin lesions appeared on her scalp, elbow, knee and foot. She had no prior history of such lesions or family history of psoriasis. A dermatology referral confirmed the presence of plaque psoriasis affecting 1-2% of her body surface area, including the previously mentioned areas as well as the intergluteal cleft and bilateral postauricular regions (Figure 4). She was treated with topical corticosteroids and coal tar shampoo. Her human leukocyte antigen B27 (HLA-B27) testing was negative and magnetic resonance imaging (MRI) of the sacroiliac joints revealed bilateral iliac bone fat metaplasia, indicative of post-inflammatory changes consistent with axial spondyloarthritis (SpA).

The patient met the Classification Criteria for Psoriatic Arthritis (CASPAR) and was diagnosed with Psoriatic Arthritis (PsA). Due to persistent disease activity, she was initiated on a treatment regimen consisting of Celecoxib 200 mg twice daily, Methotrexate 15 mg once a week, and Folate 5 mg daily (excluding the day of methotrexate administration). Follow-up assessments revealed normalized ESR and CRP levels, significant pain relief, and clinical remission achievement which enable her to resume normal daily activities.

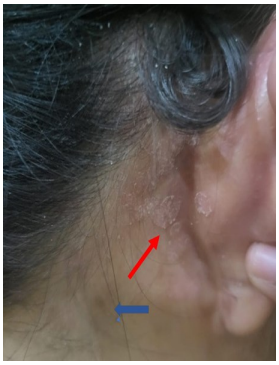

Figure 4: Post-inflammatory hyperpigmentation (blue arrow) with thin, mild psoriatic plaque with scaling (red arrow) were detected on her scalp and right posterior auricular.

DISCUSSION

Differentiating between inflammatory and mechanical low back pain is crucial as their treatments differ significantly. This patient's primary presentation was inflammatory back pain (IBP), a hallmark feature of axial SpA, which typically manifests before the age of 40. She met all five Assessment of Spondyloarthritis International Society (ASAS) criteria of IBP: age of onset less than 40 years old, insidious onset, nocturnal pain, lack of improvement with rest and improvement occurs with movement.

In this case, psoriasis developed 10 months after the onset of arthritis, illustrating the phenomenon of PsA sine psoriasis, which affects 13.5-24.6% of PsA cases globally.[3]

The prevalence of PsA sine psoriasis in Malaysia remains underreported. Typically, psoriasis appears 7–12 years prior to arthritis.[4] Thus, the delayed skin manifestation in this patient poses significant diagnostic and treatment challenges to the primary care. However, PsA can still be diagnosed in the absence of visible psoriasis using the CASPAR criteria, which offer high specificity (98.7%) and sensitivity (91.4%).[3]

PsA comprises of six key domains which include enthesitis, dactylitis, axial, nail, skin, and peripheral arthritis. Nail psoriasis, dactylitis, enthesitis, and distal interphalangeal (DIP) involvement are the key features that differentiate the diagnosis of PsA sine psoriasis from rheumatoid arthritis (RA) and other forms of SpA. While PsA typically begins asymmetrically and oligoarticularly, it may progress to a symmetric pattern resembling RA. Notably, PsA often affects the DIP joints, whereas RA predominantly involves the wrists and small hand joints symmetrically, sparing the DIP joints.[5]

Early detection of skin or nail lesions is crucial for diagnosing PsA. While psoriasis on the trunk or limbs is easily noticeable, lesions in areas such as the scalp, intergluteal region, umbilicus, elbows, knees and nails are often overlooked, as illustrated in this case.[6] Nail involvement is present in 40% of psoriasis patients but affects up to 80% of those with PsA, with fingernails being more commonly being affected than toenails.[7] Key signs of nail involvement include pitting, onycholysis and transverse ridging.

Enthesitis and dactylitis are common in PsA, affecting 30-50% and 40-50% of patients, respectively. Enthesitis typically involves the plantar fascia and Achilles' tendon, while dactylitis, is often typically seen in the feet, is associated with more severe disease.[5,6,8] Therefore, a comprehensive lower limb examination is essential to identify these key features in PsA.

Axial involvement occurs in 25-70% of PsA, predominantly affecting the spine and sacroiliac joints. Compared to AS, axial PsA generally presents with milder symptoms and radiographic findings. HLA-B27, a genetic marker for AS, has a low association with axial PsA, with positivity rate of 14-40% in PsA versus over 80% in AS.[9] While HLA-B27 testing is helpful, a positive result does not confirm axial SpA and a negative test does not exclude it.[1] Elevated ESR and CRP levels are common but not always present. For suspected axial involvement, an anterior-posterior pelvic x-ray should be performed and evaluated using the modified New York criteria. However, relying on plain x-ray alone may delay diagnosis by up to 10 years due to the late radiographic changes.[1] Therefore, MRI is preferred for early detection of active inflammatory changes such as subchondral bone edema, synovitis and capsulitis, as well as chronic lesions like fat metaplasia, sclerosis, erosions, joint space narrowing, syndesmophytes and ankylosis.[1,10]

Nonsteroidal anti-inflammatory drugs (NSAIDs) are the first-line treatment for axial PsA. In this case, the patient also had other PsA manifestations, including dactylitis, enthesitis, peripheral arthritis, and skin and nail involvement. Thus, she was started on methotrexate, a conventional Disease Modifying Anti-Rheumatic Drug (DMARD), which led to clinical improvement and remission. Although biologic DMARDs are recommended for treating axial PsA, they were not used in this case due to the absence of active sacroiliitis and the patient's positive response to methotrexate and NSAIDs.[9]

Primary care providers should be more vigilant in recognizing IBP and clinical features of PsA. This case highlights the diagnostic complexity of PsA and the importance of thorough history-taking, clinical examination, laboratory test and MRI to distinguish PsA from other forms of SpA.

CONCLUSION

Early detection of PsA in the absence of skin lesions is challenging and often leads to misdiagnosis. The ASAS IBP and CASPAR criteria can help primary care providers in identifying inflammatory back pain and diagnosis of PsA earlier. An early referral to rheumatologist and timely treatment can prevent joint damage and improve quality of life. Therefore, it is essential for primary care providers to be familiar with the clinical features of PsA to ensure early recognition and intervention.

CONFLICT OF INTEREST

The authors have no conflict of interest.

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