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Diagnostic Dilemma of Anemia and Oligoarthritis After Tumor Necrosis Factor Inhibitor Therapy in Spondyloarthritis: Flare, Infection, or Paradoxical Reaction?

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Abstract

In patients with axial spondyloarthritis (axSpA), inadequate disease control may necessitate escalation to immunosuppressive therapy. However, subsequent clinical worsening can create a diagnostic dilemma, particularly when disease activity, treatment-related paradoxical reactions, and infection present with overlapping features. We report a 33-year-old man with axSpA receiving targeted synthetic disease-modifying antirheumatic drug (DMARD) therapy who developed peripheral large-joint oligoarthritis. Suspected inadequate disease control led to treatment escalation with a tumor necrosis factor inhibitor (TNFi). He subsequently developed fever, anemia, and worsening arthritis. Infectious workup and synovial fluid analysis were inconclusive. Bone marrow examination showed pure red cell aplasia (PRCA), and polymerase chain reaction (PCR) testing confirmed a high parvovirus B19 viral load. He improved following treatment with intravenous immunoglobulin (IVIg). This case highlights the importance of considering parvovirus B19 infection in patients with inflammatory arthritis and unexplained cytopenias, as early diagnosis can prevent unnecessary escalation of immunosuppressive therapy.

Categories: Hematology, Rheumatology, Internal Medicine
Keywords: adalimumab, anemia, biologic therapy, intravenous immunoglobulin, oligoarthritis, parvovirus b19, pure red cell aplasia (prca), spondyloarthritis, tumor necrosis factor inhibitor (tnfi), viral arthritis

Introduction

Axial spondyloarthritis (axSpA) is a chronic inflammatory condition characterized predominantly by axial involvement, but extra-axial manifestations, including peripheral arthritis, enthesitis, and dactylitis, may also occur. Intercurrent infections in patients with underlying rheumatic diseases receiving immunosuppressive therapy may be misinterpreted as disease flares or treatment failure, potentially leading to inappropriate escalation of immunosuppression. This diagnostic challenge is particularly relevant in the era of biologic and targeted therapies, in which the risk of infection is increased, and clinical presentations may be atypical.

Tumor necrosis factor inhibitor (TNFi) may also induce paradoxical inflammatory reactions, including new-onset or exacerbation of psoriasis, lupus-like syndrome, and other autoimmune manifestations. Therefore, a careful diagnostic evaluation is essential to distinguish disease flare from treatment-related complications and alternative etiologies, including septic arthritis, crystal-induced arthritis, and viral infections.

Viral arthritis is an acute inflammatory joint condition triggered by viral infection, accounting for approximately 1% of acute arthritis cases. Parvovirus B19 infection is a well-known cause of inflammatory arthritis and can closely mimic autoimmune rheumatic diseases, thereby creating substantial diagnostic ambiguity in clinical practice. Although its articular manifestations classically resemble rheumatoid arthritis (RA), atypical presentations, including oligoarticular involvement, have also been described [1,2]. Most cases resolve spontaneously within weeks to months, without permanent joint damage. Viral infections may trigger transient, low-titer elevation in autoimmune serological markers, such as rheumatoid factor, further complicating the distinction between viral arthritis and primary rheumatic disease. Failure to recognize an infectious etiology may result in unnecessary immunosuppression and delay in appropriate treatment.

We report a case of parvovirus B19-associated pure red cell aplasia (PRCA) presenting with fever and inflammatory oligoarthritis soon after the initiation of TNFi therapy in a patient with axSpA. This case highlights the diagnostic difficulty arising from overlapping clinical features and underscores the importance of considering alternative etiologies, including viral infections, in patients with atypical presentations

before escalating immunosuppressive therapy.

Case Presentation

A 33-year-old man presented with a two-year history of inflammatory lower back and neck pain, characterized by insidious onset, morning stiffness lasting for more than one hour, and improvement with activity. During the initial 18 months, he sought alternative treatments, including herbal preparations and homeopathic medications, and intermittently used over-the-counter analgesics, with only limited relief. Due to progressive worsening of axial symptoms, further assessment was undertaken at an outside facility.

On examination, the flexion, abduction and external rotation (FABER) test was positive on both sides, and modified Schober's test measured 4 cm, raising clinical suspicion of sacroiliitis. There was no evidence of enthesitis or dactylitis, and no clinical features indicative of psoriasis were noted. Cervical spine movements were well-preserved.

Investigations revealed elevated inflammatory markers, with a C-reactive protein (CRP) level of 53.21 mg/L (reference value <5 mg/L) and an erythrocyte sedimentation rate (ESR) of 52 mm in the first hour. Initial investigations showed a hemoglobin level of 12.1 g/dL, a total leukocyte count of 11,200 cells/cumm, and a platelet count of 412 × 10^9/L. Plain radiography of the pelvis, including the sacroiliac joints, demonstrated bilateral sacroiliac joint-space narrowing. Magnetic resonance imaging of the sacroiliac joints confirmed chronic bilateral sacroiliitis with T2/STIR hyperintensity, more pronounced on the right than on the left. HLA-B27 was positive by flow cytometry. His Ankylosing Spondylitis Disease Activity Score (ASDAS) and Bath Ankylosing Spondylitis Disease Activity Index (BASDAI) were 4.4 and 4.9, respectively, indicating very high disease activity.

A diagnosis of axSpA was made, and treatment with tofacitinib 5 mg twice daily was initiated, along with a short tapering course of oral prednisolone and nonsteroidal anti-inflammatory drugs. However, after three months of treatment, he reported no significant clinical improvement. During treatment, he developed new-onset peripheral arthritis involving both knees, the left ankle, and the left wrist, with clinical evidence of active synovitis.

He was subsequently referred to our tertiary care center for further evaluation and management. The diagnosis of axSpA was reconfirmed based on clinical assessment, laboratory parameters, and imaging findings. On presentation, routine investigations revealed anemia with thrombocytosis (hemoglobin 10.9 g/dL, total leukocyte count 7.2 × 10^9/L, and platelet count 601 × 10^9/L), along with markedly elevated C-reactive protein of 160 mg/L and erythrocyte sedimentation rate of 10 mm/hr. He had high disease activity, with ASDAS and BASDAI values of 5.6 and 6.7, respectively. The trends in laboratory investigations are summarized in Table *1*.

Category	Investigation	Baseline (Day 0)	Post-admission (Day 7)	Reference range
Hematological parameters	Haemoglobin	10.9 g/dl	7.3 g/dl	12-15 g/dl
	Total leukocyte count	7,200 cells/mm³	11,500 cells/mm³	4000-10,000 cells/mm^3
	Platelet count	601 × 10^3/mm³	563 × 10^3/mm³	150-450 10^3/mm^3
	CRP	160 mg/L	161 mg/L	<5 mg/L
	ESR	10 mm/hr	29 mm/hr	0-15 mm/hr
Biochemical parameters	Creatinine	0.81 mg/dL	1.06 mg/dL	<0.90 mg/dL
	AST	34.3 U/L	43 U/L	<45 U/L
	ALT	68.7 U/L	42.4 U/L	<45 U/L

TABLE 1: Comparison of baseline (Day 0) and post-admission (Day 7) hematological and biochemical parameters

Abbreviations: ALT, alanine aminotransferase; AST, aspartate aminotransferase; CRP, C-reactive protein; ESR, erythrocyte sedimentation rate

In view of the persistently high disease activity, initiation of tumor necrosis factor inhibitor (TNFi) therapy was planned. Pre-biologic screening for latent tuberculosis (TB) with Interferon-gamma release assay, HIV-1/2 antibodies, hepatitis B surface antigen, and anti-hepatitis C virus antibodies was negative. Adalimumab

was initiated, with the first dose of 40 mg administered subcutaneously. In addition, an intra-articular corticosteroid injection was administered into the left knee for symptomatic relief.

Within four days of receiving adalimumab, the patient developed high-grade fever with chills, followed by worsening pain and swelling of the left knee and ankle over the subsequent two weeks. In the setting of combined immunosuppression with tofacitinib, corticosteroids, and adalimumab, several differential diagnoses were considered, including septic arthritis, opportunistic infection, a TNFi-related paradoxical inflammatory reaction, and hematological malignancy. The possibility of hemophagocytic lymphohistiocytosis (HLH), secondary to infection or underlying disease activity, was also considered.

In view of the persistent fever, worsening peripheral arthritis, and progressive anemia following the recent escalation of immunosuppression, we proceeded with a stepwise evaluation to identify an underlying infectious, inflammatory, or hematological cause.

Initial work-up was undertaken to screen for common and serious infections. Blood cultures, urinalysis, and chest imaging were inconclusive. Synovial fluid analysis from the affected joint showed non-purulent inflammatory fluid, with a negative Gram stain and cultures, making septic arthritis less likely. Considering the clinical setting, a targeted infectious workup, including *Brucella* serology, was also performed. Although *Brucella* IgG was borderline positive at a titer of 11.88 (positive >11), this finding was not considered clinically significant (Table *2*). Empirical antibiotics (intravenous ceftriaxone 1 g twice daily and oral doxycycline 100 mg twice daily) were initiated to cover possible atypical infections; however, there was no clinical improvement, prompting further evaluation.

Category	Investigation	Result	Reference range
Anaemia workup	Indirect and direct Coombs test	Negative	Negative
	Lactate dehydrogenase	273	0.6-1.3 mg/dl
	Ferritin	905 ng/ml	15-150 ng/mL
	Reticulocyte count	1.39%	0-45 U/L
	Vitamin B 12	2000 pg/ml	135-214 U/L
	Serum vitamin D	35.7 ng/mL	>30 ng/mL
	MCV	89.7 fl	83 – 101 fl
	MCHC	30.7 g/dL	31.5 - 34.5 g/dL
Synovial fluid analysis	Appearance	Pale yellow, clear	Pale yellow, clear
	Total leukocyte count	500 cells/mm³	<200 cells/mm^3
	Differential count	Neutrophils 75%, lymphocytes 25%	Neutrophil <25%
	Gram stain	No organisms seen	No organisms seen
	AFB stain/CBNAAT for TB	Negative	Negative
Infectious workup	Scrub typhus IgM ELISA	Negative	Negative
	Dengue NS1 and IgM	Negative	Negative
	VDRL	Negative	Negative
	Leptospira IgM ELISA	Negative	Negative
	Quantiferon TB Gold	Negative	Negative
	Blood/urine/stool culture	No growth	Negative
	Brucella IgG ELISA	Borderline	Negative

TABLE 2: Summary of investigation for evaluation of anaemia, synovial fluid, and infections

Abbreviations: AFB, acid-fast bacilli; CBNAAT, cartridge-based nucleic acid amplification test; ELISA, enzyme-linked immunosorbent assay; IgG, immunoglobulin G; IgM, immunoglobulin M; LDH, lactate dehydrogenase; MCV, mean corpuscular volume; MCHC, mean corpuscular hemoglobin concentration; PCR, polymerase chain reaction; TB, tuberculosis; VDRL, Venereal Disease Research Laboratory

As the fever persisted without an identifiable source, a more detailed evaluation for infection was undertaken. Contrast-enhanced computed tomography (CT) of the thorax and abdomen and transthoracic echocardiography were performed to rule out occult infection, malignancy, and endocarditis; these investigations were inconclusive. An 18F-fluorodeoxyglucose positron emission tomography (18F-FDG PET) scan showed metabolically active inflammation in the left knee, along with reactive lymphadenopathy and diffuse splenic uptake, suggesting systemic immune activation, but showed no evidence suggestive of malignancy.

At this stage, the differential diagnoses included opportunistic infection, a TNFi-induced paradoxical reaction, and hematological malignancy. A paradoxical reaction was considered less likely because the patient had high-grade fever and worsening anemia, which are not typical features. Infection was also considered less likely in view of negative microbiological investigations, nonpurulent synovial fluid, lack of response to antibiotics, and the absence of a clear focus of infection on imaging. Malignancy was considered but was not supported by the PET findings. HLH was also considered; however, negative markers made this diagnosis less likely.

A blood viral panel using qualitative real-time polymerase chain reaction (PCR) for enterovirus, human herpes virus, adenovirus, cytomegalovirus, parvovirus, and varicella-zoster virus was negative.

Given the unexplained anemia, we proceeded with bone marrow evaluation. Bone marrow aspirate and biopsy showed adequate cellularity for age (approximately 50-60%), but marked suppression of the erythroid lineage, with a markedly increased myeloid-to-erythroid ratio of 88:1. Plasma cells were mildly increased

(6%). Immunohistochemistry for E-cadherin confirmed a near absence of erythroid precursors, consistent with pure red cell aplasia (PRCA). Morphological examination revealed giant pronormoblasts with characteristic nuclear inclusions and cytoplasmic projections (a “dog-ear” appearance), strongly suggesting parvovirus infection (Figure *1*).

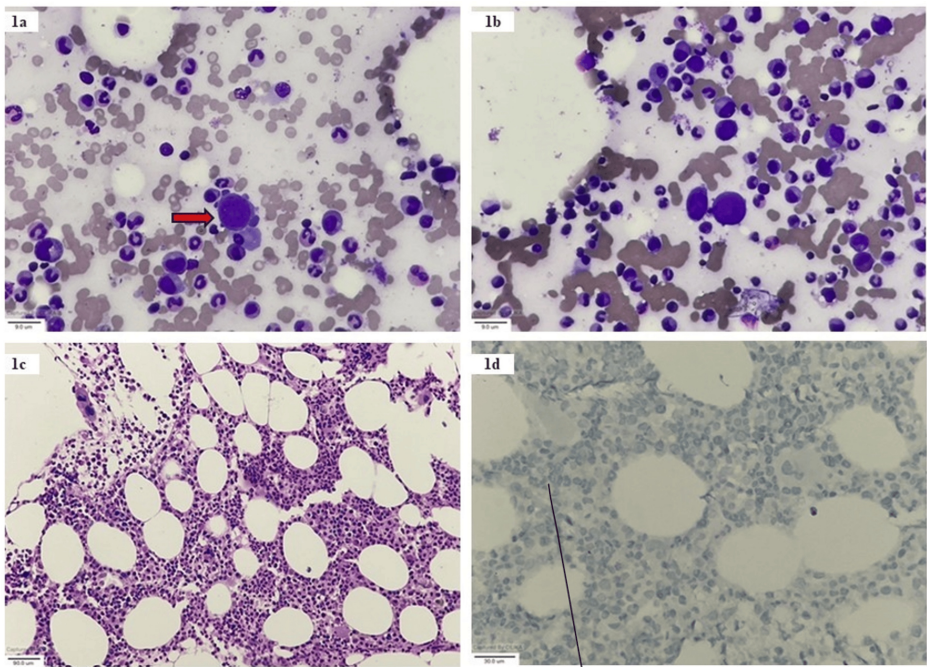


FIGURE 1: Bone marrow findings demonstrating pure red cell aplasia associated with parvovirus B19 infection.

(a) Giant pronormoblast with "dog-ear" cytoplasmic projections (red arrow) in the bone marrow (400x). (b) Absence of normal erythroid precursors in the bone marrow aspirate (400x). (c, d) Bone marrow biopsy shows absence of erythroid precursors, which is confirmed by E-cadherin immunohistochemistry (400x).

Based on these findings, specific testing for parvovirus B19 was performed. Although qualitative PCR was negative, quantitative PCR demonstrated a high viral load of 582,900 IU/mL, confirming active parvovirus B19 infection as the cause of PRCA and the associated systemic symptoms.

The patient was treated with intravenous immunoglobulin (IVIg) at a total dose of 2 g/kg administered over five days, following which he showed clear clinical improvement, with resolution of fever and gradual recovery of hemoglobin.

Discussion

Parvovirus B19 is a ubiquitous deoxyribonucleic acid (DNA) virus associated with a wide range of clinical manifestations, including erythema infectiosum in children, hydrops fetalis in pregnancy, aplastic crisis in patients with hemolytic disorders, and persistent PRCA in immunocompromised individuals [1].

Parvovirus B19 infection typically progresses through a viremic phase followed by an antibody-mediated phase [2]. The viremic phase may be asymptomatic or may present with a mild flu-like illness and transient reticulocytopenia approximately 10-12 days after infection. The subsequent immune phase, occurring around 15-18 days after infection, is characterized by rash, arthralgia, or arthritis in association with the development of anti-B19 IgM, by which time infectivity usually declines [3]. The clinical manifestations of parvovirus B19 infection are strongly influenced by host factors, such as age, hematologic background, and immune status. Acute parvovirus B19 infection has been implicated in various musculoskeletal manifestations that may resemble systemic autoimmune diseases, including myalgia, myositis, arthralgia, arthritis, and skin rash, often accompanied by laboratory abnormalities [3-5]. These overlapping features may mimic a disease flare, creating diagnostic uncertainty and increasing the risk of inappropriate escalation of immunosuppression in patients with underlying rheumatic disease.

Musculoskeletal involvement is more commonly observed in adults, with joint symptoms reported in up to 80% of cases and a higher prevalence among women [6,7]. Articular manifestations are thought to occur through immune complex-mediated mechanisms [8]. Classically, parvovirus B19-associated arthritis presents as an acute symmetric polyarthritis involving the small joints of the hands, wrists, knees, and feet,

often closely mimicking RA. Symptoms may progress in an additive pattern with prominent morning stiffness, further resembling RA. In some cases, persistent infection has also been associated with erosive changes and the development of rheumatoid factor positivity, further strengthening this resemblance [4]. Although polyarticular disease is most frequently described, oligoarticular involvement has also been reported [9]. Parvovirus B19-associated arthritis presenting with spinal involvement is rare. Solomon et al. reported a case of parvovirus B19 arthritis presenting as spinal disease in a 50-year-old man with fever, cervical spine pain, and stiffness [10]. In patients with underlying SpA, peripheral oligoarticular arthritis is a recognized clinical phenotype and may be difficult to distinguish from a disease flare.

In our patient, the development of asymmetric oligoarticular synovitis involving the knee, ankle, and wrist in the context of active axial disease initially suggested progression of SpA or a suboptimal response to therapy, prompting escalation of immunosuppression with the TNFi adalimumab. However, persistent fever accompanied by progressive anemia, together with the recent intra-articular corticosteroid injection, broadened the differential diagnosis to include septic arthritis, opportunistic infections, macrophage activation syndrome, a TNFi-related paradoxical reaction, and hematological malignancy in the setting of combined immunosuppression. Extensive infectious work-up and synovial fluid analysis were noncontributory, whereas bone marrow examination revealed PRCA, providing a crucial clue to parvovirus B19 infection. To our knowledge, parvovirus B19 infection presenting as oligoarticular arthritis mimicking a flare of spondyloarthritis has not been previously reported, highlighting an important diagnostic challenge in patients with established rheumatic disease.

In addition to parvovirus B19-associated disease, pharmacovigilance data have reported an increased frequency of various viral infections in patients receiving TNF inhibitors, suggesting a broader pattern of susceptibility to infection in this setting. Increased reporting has been observed for influenza and upper respiratory tract infections with etanercept, chikungunya virus infection with adalimumab and golimumab, and more severe viral illnesses, such as herpesvirus infections, cytomegalovirus disease, and infectious mononucleosis, particularly with monoclonal antibody TNFi agents [11]. These observations support the possibility that TNFi therapy may be associated with atypical or severe viral infections that can present with systemic manifestations and musculoskeletal involvement, thereby complicating the clinical assessment of patients with underlying rheumatic disease.

Parvovirus B19 infection can be diagnosed using serological assays that detect antibodies against viral proteins or through direct detection of viral DNA using PCR. In immunocompromised patients, antibody responses may be blunted, limiting the reliability of serological testing and making PCR a more reliable diagnostic modality in this setting, as demonstrated by the detection of a high parvovirus B19 viral load in our patient [12].

Parvovirus B19-associated arthritis is usually acute and self-limiting, and most patients can be managed with supportive therapy, such as nonsteroidal anti-inflammatory drugs or short courses of glucocorticoids [7,13,14]. However, in immunocompromised individuals, inadequate viral clearance may permit persistent replication within erythroid progenitor cells, resulting in chronic PRCA. Immune complex-mediated arthropathy may also contribute to the persistence of joint symptoms in this subgroup. Evidence of clinical improvement following IVIg, accompanied by a reduction in parvovirus B19-infected bone marrow cells, provides a rationale for the use of IVIg in immunocompromised patients with persistent infection, as observed in our case [15,16].

Conclusions

After common causes of worsening arthritis had been excluded, bone marrow examination, despite being invasive, proved pivotal by establishing the diagnosis of PRCA and directing further evaluation toward parvovirus B19 infection. Subsequent parvovirus B19-specific quantitative PCR demonstrated a high viral load despite an earlier qualitative viral PCR panel being negative. This finding highlights that when clinical suspicion remains high, a negative qualitative PCR panel should not necessarily exclude a specific viral infection, and targeted quantitative PCR testing for the suspected virus should be considered. Treatment with IVIg resulted in clear clinical improvement, with resolution of fever and gradual recovery of hemoglobin.

Parvovirus B19 infection, although often overlooked, should be actively considered in patients with inflammatory arthritis and unexplained anemia, particularly when bone marrow findings point toward PRCA. PCR-based detection of viral DNA remains an important diagnostic modality in this setting.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Disclosures

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