

Virus-induced sacroiliitis: parvovirus B19 expands the spectrum of spondyloarthritis-like phenotypes

Sirs,
Human parvovirus B19 (B19V) is a well-recognised cause of acute arthropathy in adults, typically involving wrists and small joints, and can occasionally mimic rheumatoid arthritis or systemic lupus erythematosus (1). Increasing evidence also supports its role as an immunological trigger capable of inducing cytokine activation and sustaining low-grade synovial inflammation beyond the acute infectious phase (2). By contrast, axial involvement remains distinctly uncommon, with only isolated reports of cervical bursitis or atlantoaxial arthritis (3, 4). We report three adults with MRI-confirmed sacroiliitis in whom acute B19V infection was supported by a consistent clinic-epidemiological and serological profile: symptom onset 5 days to 2 weeks after exposure, systemic viral features, strongly positive anti-B19V IgM with concomitant

IgG positivity, and uniformly negative RF, anti-CCP, ANA, and HLA-B27 (Table I); none had a previous history of inflammatory rheumatic disease. A 40-year-old man presented with fever and inflammatory back pain; MRI showed unilateral sacroiliitis with bone marrow oedema and erosive change, and B19V serology was consistent with recent infection (IgM 9.09; IgG >150). Although systemic inflammation improved, axial symptoms persisted beyond 24 weeks, and adalimumab was initiated for a chronic SpA-like course, with marked clinical benefit and complete resolution of bone marrow oedema on follow-up MRI. By contrast, two women had a more self-limited presentation: one developed fever, rash, lumbar stiffness, and proximal interphalangeal arthritis about 10 days after household exposure, with bilateral non-erosive sacroiliitis on MRI and complete remission after a short prednisone course (IgM 17.1; IgG 47.8); the other presented 5 days after familial exposure with fever, inflammatory back pain, and bilateral Achilles enthesitis, with bilateral non-erosive sacroiliitis on MRI and remission after NSAIDs

plus brief prednisone taper (IgM 12.2; IgG 36). Taken together, these observations raise the possibility that acute B19V infection might, in uncommon circumstances, be associated with axial inflammation closely resembling axial spondyloarthritis (axSpA). Although no causal inference can be drawn from a small case series, the concurrence of MRI-confirmed sacroiliitis, a close exposure-to-symptom interval, systemic viral manifestations, robust anti-B19V serological reactivity, and the consistent absence of autoimmune serological markers or HLA-B27 suggests that, in these patients, an infection-associated inflammatory process may be more likely than unequivocal idiopathic axSpA. The clinical course was not uniform: whereas two patients experienced spontaneous or rapidly treatment-responsive resolution, one developed more persistent and structurally relevant disease requiring TNF inhibition. All things considered, our findings may suggest that acute B19V infection might, albeit rarely, be associated with sacroiliitis and an axial SpA-like phenotype. Although causality cannot be established from a small case

Table I. Clinical characteristics, imaging, treatment, outcomes, and laboratory findings of three patients with parvovirus B19-associated axial involvement.

	Case 1	Case 2	Case 3
Demographics and timing			
Ethnicity; BMI (kg/m ²); comorbidities	Caucasian; 25; asthma	Caucasian; 33.2; obesity	Caucasian; 20.07
Exposure-onset interval	~2 weeks	~10 days	~5 days
Clinical course	Subacute onset; chronic course	Acute onset; self-limiting	Acute onset; delayed laboratory
Clinical features and examination			
Key clinical features	Fever; lumbar pain/stiffness; pleuritis; asthenia	Fever; lumbar pain/stiffness; rash; PIP arthritis	Fever; lumbar pain/stiffness; bilateral Achilles enthesitis; asthenia
Spinal examination	Lumbar tenderness; reduced range of motion; SI provocative tests positive	Lumbar tenderness; reduced range of motion; SI provocative tests positive	Lumbar tenderness; reduced range of motion; SI provocative tests positive
Cutaneous findings	None	Rash	None
Imaging			
MRI sacroiliac joints	Right sacroiliitis with bone marrow oedema and erosions	Bilateral sacroiliitis with bone marrow oedema; no erosions	Bilateral sacroiliitis with bone marrow oedema; no erosions
Treatment and outcome			
Treatment	NSAID (etoricoxib, intermittent)	Prednisone 25 mg for 1 week	NSAIDs plus prednisone 5 mg for 1 week
Hospitalisation	Yes (pain and fever)	No	No
Outcome and duration	Symptoms persisted >24 weeks; CRP normalised at ~7 months	Resolution within 2 weeks; CRP normalised at ~3 months	Clinical resolution within 2–3 weeks; ESR/CRP normalised at ~4 months
Laboratory findings			
Parvovirus B19 IgM	9.09	17.1	12.2
Parvovirus B19 IgG	>150	47.8	36
CRP (mg/L)	53.7	4.5	3.3
ESR (mm/h)	27	33	43
Haemoglobin (g/L)	127	130	125
White blood cells (×10 ⁹ /L)	4.93	8.25	7.2
Platelets (×10 ⁹ /L)	211	230	303
Rheumatoid factor	Negative	Negative	Negative
Anti-CCP	Negative	Negative	Negative
ANA	Negative	Negative	Negative
HLA-B27	Negative	Negative	Negative
Time to normalisation of inflammatory markers	CRP normalised after ~7 months	CRP normalised after ~3 months	ESR/CRP normalised after ~4 months

BMI: body-mass index; PIP: proximal interphalangeal; SI: sacroiliac; MRI: magnetic resonance imaging; NSAID: non-steroidal anti-inflammatory drug; CRP: C-reactive protein; ESR: erythrocyte sedimentation rate; Anti-CCP: anti-cyclic citrullinated peptide; ANA: antinuclear antibodies; BME: bone marrow oedema.

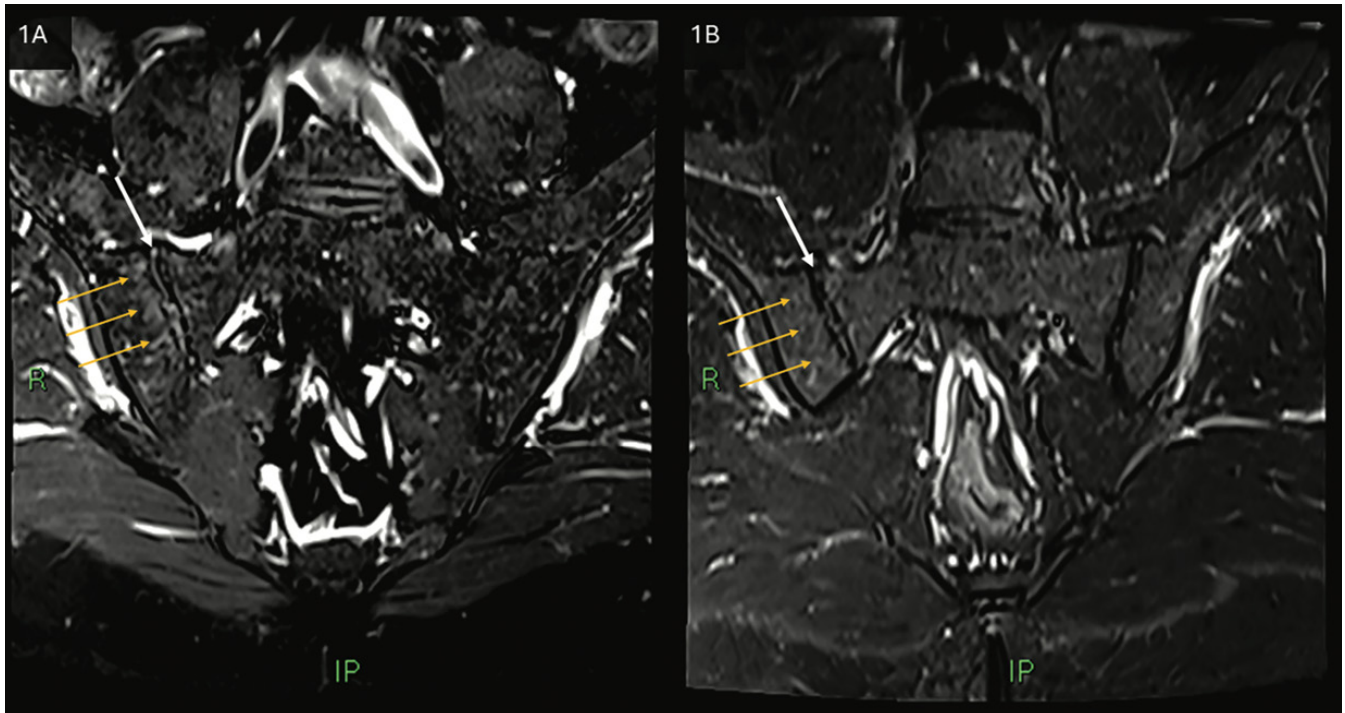


Fig. 1. Coronal STIR images of the pelvis obtained before (A) and after (B) TNF-inhibitor therapy in the same patient (Patient 1). Orange arrows indicate areas of subchondral bone marrow oedema (BME) at the right sacroiliac joint, prominent at baseline and clearly reduced after treatment. The white arrow marks reactive articular changes persisting despite partial regression of inflammatory oedema. Overall, the findings are consistent with active right-sided sacroiliitis evolving to partial remission following therapy, with residual structural alterations.

series, the convergence of MRI-confirmed sacroiliitis, a close temporal relationship between exposure and symptom onset, systemic viral manifestations, robust anti-B19V serological reactivity, and the consistent absence of HLA-B27 and autoimmune serological markers supports the possibility of an infection-associated inflammatory process rather than unequivocal idiopathic axSpA. Considered alongside previous reports of cervical axial involvement (3, 4), these cases cautiously broaden the recognised musculoskeletal spectrum of B19V infection to include the sacroiliac joints. This interpretation is biologically plausible in light of prior evidence showing B19V DNA and viral proteins in synovial tissue (5, 6), together with the capacity of viral persistence to sustain NF- κ B-driven and cytokine-mediated inflammatory pathways relevant to SpA pathobiology (1, 2). From a clinical perspective, all these observations may be relevant to the differential diagnostic assessment. During periods of community circulation, parvovirus-related musculoskeletal disease might transiently fulfil classificatory frameworks for early RA or axSpA, with potential consequences for therapeutic escalation (7-9). In this setting, acute B19V infection may merit consideration in HLA-B27-negative individuals presenting with new-onset inflammatory back pain or sacroiliitis, especially when accompanied by fever, rash, peripheral arthritis or enthesitis, and a compatible exposure history.

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