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Reactive Arthritis in Children: A Literature Review with Analysis of Uncommon Infectious Triggers and Clinical Presentations

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Abstract

Background. Reactive arthritis (ReA) is an aseptic, immune-mediated arthritis typically developing 1-4 weeks after infection, most often affecting lower limb joints, sometimes with enthesitis, dactylitis, mucocutaneous lesions, or systemic symptoms. HLA-B27 positivity is associated with axial involvement and higher risk of chronicity, while HLA-B27-negative patients often show peripheral joint involvement. Diagnosis is primarily clinical, supported by serology or molecular tests, and requires exclusion of septic arthritis. Case reports included in this review highlight rare triggers in children, such as Enterobius vermicularis, Giardia lamblia, Clostridioides difficile, SARS-CoV-2, tuberculosis, rotavirus, and enteroviral infections. These cases illustrate the broad etiological spectrum, reinforce the importance of careful differential diagnosis, and provide practical insights for clinicians managing atypical pediatric ReA. Awareness of rare triggers and early recognition are essential for optimal outcomes.

Aim. To provide a comprehensive review of pediatric reactive arthritis, including its epidemiology, pathogenesis, clinical features, diagnostic approach, management, and to illustrate atypical presentations through selected case reports.

Material and methods. A literature review was conducted to summarize evidence on the epidemiology, clinical features, etiology, pathogenesis, diagnosis, treatment, and prognosis of pediatric reactive arthritis. A structured search of PubMed and Embase was performed between December 2025 and February 2026. The terms “reactive arthritis,” “post-infectious arthritis,” and “Reiter’s syndrome” were systematically combined with “children” and/or “pediatric” using Boolean operators (AND, OR) to identify studies involving individuals under 18 years of age. Owing to the scarcity of recent pediatric-specific data, earlier relevant studies were also included. In total, 39 pediatric cases published since 2000 were identified, of which 7 were included in the analysis based on relevance and completeness of clinical data. Reference lists of selected articles were additionally screened to identify further eligible studies.

Results. Analysis of pediatric case reports revealed a wide spectrum of uncommon infectious triggers, including *Enterobius vermicularis*, *Giardia lamblia*, *Clostridioides difficile*, SARS-CoV-2, tuberculosis, rotavirus, and enteroviruses. These cases emphasize the heterogeneity of etiological factors and clinical presentations in children. Diagnosis is primarily clinical, supported by serological or molecular testing, with mandatory exclusion of septic arthritis. The reviewed cases highlight the importance of considering rare pathogens in atypical presentations.
Conclusions. Pediatric ReA demonstrates considerable etiological and clinical variability. Increased awareness of rare infectious triggers and a thorough differential diagnostic process are essential for accurate diagnosis. Early recognition facilitates appropriate management and may reduce the risk of complications and chronicity.
Keywords: Pediatric reactive arthritis, Post-infectious arthritis, Reiter's syndrome

1. Introduction

Defining reactive arthritis (ReA) is challenging due to historical and terminological inconsistencies. Classically, ReA is referred to arthritis occurring after gastrointestinal or urogenital infections. The discovery of its association with human leukocyte antigen B27 (HLA-B27) enabled further stratification into HLA-B27-positive and HLA-B27-negative forms, with the former showing closer clinical and pathogenetic overlap with spondyloarthropathies and therefore being classified within this spectrum. Over time, an increasing number of infectious triggers have been identified, including most recently severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), expanding the concept of ReA beyond its classical framework. As a result, the broader term "infection-related arthritis" has emerged, reflecting the evolving understanding and ongoing ambiguity surrounding its definition [1]. In light of these considerations, we propose expanding the definition of reactive arthritis to include any non-septic arthritis temporally associated with a preceding infection.

ReA is an aseptic, immune-mediated arthritis that typically occurs 1 - 4 weeks after an infection in genetically predisposed individuals and is usually non-destructive. Common in children, ReA is driven by immune mechanisms rather than direct joint invasion, and antibiotics do not alter

its course. Although no live pathogens are cultured from joints, bacterial antigens and Chlamydia trachomatis messenger RNA (mRNA) have been detected in synovial tissue, questioning the concept of complete sterility [1–4].

2. Material and methods

A literature review was conducted to summarize evidence on the epidemiology, clinical features, etiology, pathogenesis, diagnosis, treatment, and prognosis of pediatric reactive arthritis. A structured search of PubMed and Embase was performed between December 2025 and February 2026. The terms “reactive arthritis,” “post-infectious arthritis,” and “Reiter’s syndrome” were systematically combined with “children” and/or “pediatric” using Boolean operators (AND, OR) to identify studies involving individuals under 18 years of age. Owing to the scarcity of recent pediatric-specific data, earlier relevant studies were also included. In total, 39 pediatric cases published since 2000 were identified, of which 7 were included in the analysis based on relevance and completeness of clinical data. Reference lists of selected articles were additionally screened to identify further eligible studies.

3. Epidemiology

ReA is rare in the pediatric population and most commonly occurs in individuals aged 18 - 40 years. No significant sex predilection has been consistently demonstrated [5]. It is recognized that the clinical course of ReA in children may differ from that observed in adults and can be more complex [2]. Children are more likely to develop ReA following enterocolitic infections, whereas in adolescents and adults, the urogenital route is more commonly implicated [6]. There is a lack of current data on the incidence of ReA in children. The prevalence of ReA varies worldwide. These differences may be attributed to variation in the distribution of causative pathogens, genetic susceptibility within specific populations, differences in gut microbiota composition, as well as heterogeneity in diagnostic approaches and criteria for ReA [2]. Enterocolitic infections are more prevalent in developing countries, which may explain the higher observed frequency of ReA in these regions.

A meta-analysis conducted by Pogreba-Brown et al. (2021) demonstrated that approximately 2.6% of individuals infected with the most common enteric pathogens (Campylobacter, Salmonella, Shigella, and Yersinia) subsequently develop ReA. The highest risk was associated with Salmonella infection [7].

4. Clinical features

Reiter's syndrome, now rarely observed, is the classic form of ReA characterized by a triad of arthritis, ocular involvement, and urethritis or cervicitis [6]. Affected joints are typically painful with reduced range of motion and tenderness on both active and passive movement, while joint temperature remains normal and the overlying skin appears unchanged [8]. ReA usually presents as monoarthritis or asymmetric oligoarthritis, most commonly involving large lower limb joints, particularly the knee and ankle. Less frequently, it affects the foot joints, small upper limb joints (such as the wrists and interphalangeal joints), the sacroiliac joints, and the spine.

In recent years, dermatologic manifestations have been increasingly recognized in ReA and may include keratoderma blennorrhagicum, circinate balanitis, ulcerative vulvitis, nail changes, and oral lesions. Patients may experience any number of features, including generalized symptoms such as fever, fatigue, and weight loss, and extra-articular manifestations (Table 1) [6].

Table 1. Extra-articular manifestations of reactive arthritis in children

System	**Main manifestations**
Genitourinary	Urethritis, cystitis, cervicitis/vaginitis, orchitis, epididymitis, prostatitis
Gastrointestinal	Abdominal pain, diarrhea, anal itching
Ocular	Conjunctivitis, uveitis, keratitis
Respiratory	Bronchitis/pneumonia, pulmonary tuberculosis, sinusitis, tonsillitis
Skin and mucosa	Cirinate balanitis, hyperkeratosis, acne, vesicular lesions (hands/feet)
Cardiovascular	Myocarditis, aortitis, arrhythmias

5. Etiology

Traditionally, ReA has been linked to infections of the genitourinary or gastrointestinal tract, particularly Chlamydia and Salmonella [6,9]. However, recent studies implicate respiratory infections as potential triggers, broadening the recognized etiological spectrum (Table 2) [10]. Although rarely reported, parasitic infections should also be considered as a cause of ReA, particularly in children, and included in the differential diagnosis [8]. In approximately 25% of cases, the etiological factor remains unidentified [11].

Earlier reports linked human immunodeficiency virus (HIV) to ReA, but current evidence indicates that in children with HIV, ReA is more likely triggered by associated genitourinary, gastrointestinal, or opportunistic infections rather than by HIV itself. [12,13].

Respiratory infections are an uncommon trigger of ReA, though cases linked to Chlamydia pneumoniae have been reported. Chlamydia-associated ReA typically involves one or a few large lower-limb joints, whereas non-chlamydial forms often affect more joints, including the upper limbs [14]. Rare cases of coronavirus disease 2019 (COVID-19) related ReA have been described in children, including a single neonatal case likely associated with transplacentally acquired maternal antibodies [15,16].

Table 2. Infectious agents associated with reactive arthritis in children

Bacteria	**Parasites**	**Viruses**	**Fungi**
Salmonella sp. Shigella sp. Yersinia sp. Campylobacter Chlamydia trachomatis Ureaplasma urealyticum Chlamydia pneumoniae Mycoplasma pneumoniae Clostridium difficile	Giardia lamblia Toxocara Cryptosporidium parvum Enterobius vermicularis Strongyloides stercoralis	Rubella virus Varicella zoster virus Adenovirus 7 HBV EBV Parvovirus B19 SARS-CoV-2 Rotavirus Ross River virus (RRV)	Candida Blastomycosis Histoplasmosis Coccidioidomycosis

Helicobacter pylori Borrelia sp. Bartonella sp. Mycobacterium tuberculosis Leptospira sp.		Coxsackie virus Enterovirus 71	

HBV - hepatitis B virus, EBV - Ebstein-Barr virus, SARS-CoV-2 - Severe Acute Respiratory Syndrome Coronavirus 2

6. Pathogenesis and mechanisms

The exact mechanisms driving inflammation in this condition remain unclear, though genetic, infectious, and environmental factors are thought to play a role.

6.1. Genetics

The HLA-B27 allele increases susceptibility to spondyloarthritis, including ankylosing spondylitis, psoriatic arthritis, and arthritis associated with inflammatory bowel disease. HLA-B27 prevalence varies geographically, being higher in Northern countries. In the United States, the estimated prevalence of HLA-B27 is 6 - 8% [17]. Ding et al. (2022) found that HLA-B27-positive patients more often had axial involvement resembling spondyloarthritis, whereas HLA-B27-negative patients more commonly showed upper-extremity and small-joint involvement [18]. HLA-B27 positive arthritis is associated with a worse prognosis and a higher risk of progression to chronic arthritis [19]. Assessment of HLA-B27 is not required to establish the diagnosis; however, it may support the diagnosis and provide prognostic information [20].

6.2. Cytokines

Cytokine dysregulation is a key factor in ReA. Studies show that patients often have reduced interleukin-2 (IL-2) (impairing cellular immune response) and elevated pro-inflammatory mediators like tumor necrosis factor alpha (TNFα), IL6, IL8, and chemokines in serum and synovial fluid [21,22]. IL17 from Th17 cells is also increased, sustaining joint inflammation [23]. Although patterns vary between studies, this highlights that immune imbalance drives

ReA, and further research on cytokine gene polymorphisms is needed to clarify individual susceptibility and disease mechanisms.

6.3. Etiologic factor

In ReA, infection triggers immune disturbances, causing joint inflammation from immune complex deposition and the infiltration of bacterial components and microbial genetic material into the synovium. This process involves immune cells - such as lymphocytes, monocytes, and macrophages - pro-inflammatory cytokines, and adhesion molecules, including integrins and selectins [24,25].

6.4. Gut microbiota

Research on the gut microbiome in ReA, mainly in adults, shows altered microbial profiles - enrichment of Erwinia, Pseudomonas, Dialister, Escherichia coli, and Staphylococcus aureus, with reductions in beneficial microbes like Faecalibacterium. HLA variants also influence microbiota, suggesting that genetic and microbial factors together drive ReA [10,26]. Pediatric studies are still limited.

7. Diagnosis and diagnostic tools

7.1.Diagnostic criteria

ReA is primarily a clinical diagnosis, and tests for detecting infection serve a supportive role. While exclusion of microorganisms in synovial fluid supports the diagnosis of sterile arthritis, the procedure is not obligatory in all patients. If the synovial fluid culture is positive, the patient should be managed as having septic arthritis and treated accordingly [27].

The diagnosis of ReA requires documentation of a preceding infection. In a study conducted by Fendler et al. (2001), a causative pathogen was identified in only 56% of 52 cases of reactive arthritis following intestinal or urogenital infection [28]. A thorough medical history including travel history and vaccinations is crucial, as mild or seemingly trivial infections may be overlooked or underestimated by parents. In cases where arthritis lasts ≥6 weeks without evidence of an infectious trigger, juvenile idiopathic arthritis should be diagnosed instead.

The American College of Rheumatology proposed diagnostic criteria for ReA in 1999, which are still widely referenced today (Table 3). For a definite diagnosis of ReA, both major criteria plus ≥1 minor criterion are necessary. Probable ReA is diagnosed when either both major criteria are present without minor criteria, or one major and ≥1 minor criteria are fulfilled [29] .

Table 3. Reactive arthritis diagnostic criteria

Major criteria	**Minor criteria**
Assymetric mono- or oligoarthritis of lower extremities	Evidence of triggering infection (culture positive)
Enteritis or urethritis preceding arthritis onset by 3 days to 6 weeks	Persistent synovial involvement

7.2. Clinical Evaluation

A detailed history is key to identifying preceding infections, even mild or subclinical. ReA often presents with mono- or oligoarthritis of the lower limbs and may include enthesitis, dactylitis, conjunctivitis, or mucocutaneous lesions, making careful timing correlation essential.

7.3. Laboratory Investigations

Laboratory tests generally reveal moderate systemic inflammation, with elevated erythrocyte sedimentation rate (ESR) or C-reactive protein (CRP), while blood cultures are typically negative. Serologic or molecular evidence of prior infection - such as antibodies against Chlamydia trachomatis, Salmonella, Yersinia, or other relevant pathogens - can support the diagnosis. In many cases, however, serologic confirmation is not possible due to the transient nature of the immune response or asymptomatic infections [2,3].

7.4. Synovial Fluid Analysis

Synovial fluid analysis in ReA usually shows an inflammatory profile with leukocytosis, predominantly neutrophilic or mixed, but cultures are negative for pathogens. Polymerase chain reaction (PCR) may detect microbial DNA or antigens in rare cases, but the absence of viable organisms in the joint distinguishes these conditions from infectious arthritis [8,27].

7.5. Imaging Studies

Ultrasound, radiography, or magnetic resonance imaging (MRI) can assist in evaluating joint inflammation and ruling out other causes of arthritis. Imaging findings are often non-specific, showing effusions or mild synovial thickening, without the destructive changes commonly seen in septic arthritis [27].

7.6. Differential Diagnosis

Differential diagnosis of pediatric joint pain includes infectious arthritis, ReA, spondyloarthritis, other autoimmune arthritides, and rheumatic fever. Infectious arthritis presents with acute onset and positive synovial fluid cultures, whereas ReA is typically post-infectious, often HLA-B27-associated, and some consider it a subset of post-infectious arthritides. Juvenile spondyloarthritis (including ankylosing spondylitis, psoriatic, or enteropathic arthritis) lacks a documented preceding infection but may involve HLA-B27. Other autoimmune arthritides, such as early rheumatoid arthritis, can mimic ReA. Post-streptococcal ReA differs from rheumatic fever by absent carditis, poorer nonsteroidal anti-inflammatory drugs (NSAID) response, and possible extra-articular features like tenosynovitis or renal involvement. Table 4 summarizes features differentiating ReA from septic arthritis [2,3,30].

Table 4. Comparison of reactive arthritis and septic arthritis

Category	**Reactive arthritis**	**Septic arthritis**
Cause	Post-infectious autoimmune reaction	Direct bacterial infection of the joint
Bacteria in joint fluid	Absent (sterile synovial fluid)	Present (positive Gram stain/culture)
Typical trigger	GI or GU infection (e.g., *Chlamydia*, *Salmonella*)	Most commonly *Staphylococcus aureus*
Onset	1-4 weeks after infection	Sudden (hours-days)
Joint involvement	Asymmetric, usually lower limbs	Usually monoarticular (one joint)
Common joints	Knee, ankle	Knee most common

Systemic symptoms	Mild fever possible	High fever, toxic appearance
ESR/CRP	Elevated	Markedly elevated
Synovial fluid WBC	Inflammatory range	Very high, purulent
Extra-articular features	Conjunctivitis, urethritis, enthesitis	Usually none (unless sepsis)
Genetic association	Associated with HLA-B27	No genetic association
Treatment	NSAIDs, steroids, DMARDs if chronic	Urgent IV antibiotics + joint drainage
Urgency	Not usually emergent	Medical emergency

GI - gastrointestinal, GU - genitourinary, ESR - Erythrocyte Sedimentation Rate, CRP - C-Reactive Protein, WBC - White Blood Cell Count, NSAID - nonsteroidal anti-inflammatory drugs, DMARD - disease-modifying antirheumatic drugs, IV - intravenous

8. Treatment

More than 50% of patients with ReA experience a self-limited course, with symptom resolution within 2 to 6 months without specific treatment. Approximately 30% develop recurrent disease, and 10-20% progress to a chronic form [31]. Early initiation of therapy is recommended to relieve symptoms and reduce the risk of chronicity.

In the acute phase, NSAIDs are the first-line treatment [27]. ReA may be considered refractory when at least two different NSAIDs, administered at maximal doses for a minimum of two weeks each, fail to produce a clinically meaningful response [32]. In cases of persistent monoarthritis or inadequate response to NSAIDs, local or intra-articular glucocorticosteroids may be used. Systemic glucocorticosteroids are reserved for polyarthritis or significant extra-articular manifestations.

Management depends on infection status: active pathogens warrant targeted antibiotics, while resolved infections offer no benefit from antimicrobial therapy [27]. Notably, antibiotic treatment of traveler's diarrhea does not reduce the risk of subsequent ReA, and antibiotics are ineffective in post-enteric ReA. In contrast, antibiotic therapy appears beneficial in chlamydia-triggered arthritis [1].

In chronic or refractory cases, disease-modifying antirheumatic drugs (DMARDs) are used, reflecting the clinical overlap between ReA and spondyloarthropathies. Methotrexate is the most commonly prescribed agent, although its efficacy has not been conclusively confirmed in clinical trials [32]. Recent studies demonstrate that biological agents, including anti-TNF, anti–IL-17, and anti–IL-6 therapies, are effective in refractory disease [33]. Rehabilitation remains a cornerstone of management, and patients should be encouraged to engage in regular physical exercise to prevent muscle weakness and preserve function [32].

9. Prognosis and long-term outcomes

The course of ReA in the pediatric population is generally milder than in adults. Most children experience a complete resolution of symptoms, often within weeks to a few months [34–36]. However, in some cases, the disease may follow a recurrent pattern or evolve into a chronic spondyloarthropathy later in life [29,36]. Factors influencing the long-term prognosis include the severity of initial symptoms, HLA-B27 status, and the presence of extra-articular manifestations such as uveitis or enthesitis [27,37].

10. Case series

We encourage readers to consult the original case reports for detailed descriptions and insights.

Case 1 - Clostridioides difficile

A 6-year-old boy developed acute right hip arthritis four weeks after mesenteric adenitis treated with ceftriaxone. Imaging revealed joint effusion; synovial fluid was inflammatory and sterile. Gastrointestinal PCR detected Clostridioides difficile (C. difficile). Metronidazole and symptomatic therapy led to clinical improvement [38].

Prior antibiotic use - especially third-generation cephalosporins like ceftriaxone - is a major risk factor for C. difficile infection. Although more common in adults, it is increasingly seen in children. ReA secondary to C. difficile is rare but should be considered in children with acute arthritis and recent antibiotic-associated diarrhea.

Case 2 - Tuberculosis (Poncet’s disease)

A 9-year-old boy presented with migratory arthritis and systemic inflammation; synovial fluid was sterile. Imaging revealed bilateral hilar lymphadenopathy, three hypodense splenic

nodules, and multiple mesenteric lymph nodes, raising suspicion of tuberculosis. Interferon-gamma release assay was positive, and the tuberculin skin test measured 18 mm. Active tuberculosis was confirmed by imaging, immunologic tests, and a positive gastric lavage culture for Mycobacterium tuberculosis [39].

The arthritis was diagnosed as tuberculous reactive arthritis (Poncet's disease), which should be distinguished from septic tuberculosis arthritis. Since symptoms may precede apparent tuberculosis signs, this diagnosis should be considered in children with unexplained inflammatory arthritis, particularly in regions with high tuberculosis prevalence or low vaccination coverage.

Case 3 - SARS-CoV-2

A 14-year-old boy presented with acute seronegative polyarthritis (elbow, knees, ankles) three weeks after mild COVID-19. Inflammatory markers were mildly elevated; autoantibodies and HLA-B27 were negative. Ultrasound showed knee effusions. Intravenous followed by oral glucocorticoids resulted in full recovery [16].

Post-COVID reactive arthritis in children is rare, typically seronegative, involves the knees and ankles 2-4 weeks after infection, and responds well to steroids or cyclooxygenase-2 (COX-2) inhibitors. Some children may also develop post-COVID multisystem inflammatory syndrome, which can include arthritis among systemic features.

Case 4 – Rotavirus

An 18-month-old girl developed bilateral knee arthritis three days after resolution of rotavirus gastroenteritis. Laboratory tests were unremarkable, and symptoms resolved within five days of NSAID therapy [40].

This case demonstrates that reactive arthritis may follow rotavirus gastroenteritis in infants, even when laboratory tests are normal. Awareness of this rare post-infectious complication can help clinicians avoid unnecessary investigations and provide appropriate supportive care.

Case 5 - Hand-foot-and-mouth disease

A 3-year-old boy developed acute knee arthritis shortly after hand-foot-and-mouth disease. Laboratory findings were normal, and symptoms resolved within one week of NSAID therapy [41]

.This case highlights that reactive arthritis can occur even after common viral infections such as hand-foot-and-mouth disease. Clinicians should consider post-viral reactive arthritis in children presenting with acute monoarthritis, even when laboratory findings are unremarkable.

Case 6 - Enterobius vermicularis

A 12-year-old boy presented with recurrent right hip arthritis and perianal pruritus. Inflammatory markers were low; rheumatologic and infectious workup was negative except for stool microscopy positive for Enterobius vermicularis. Treatment with pyrantel and NSAIDs led to resolution [8].

This rare case highlights parasitic infection as a potential trigger. ReA due to Enterobius vermicularis is extremely uncommon, with single pediatric case reported. Given the parasite's prevalence and mild course, this link may be underrecognized. Parasitic causes should be considered in children with unexplained arthritis, especially if gastrointestinal symptoms or perianal itching are present.

Case 7 - Giardia lamblia

An 18-month-old girl experienced recurrent bilateral hip arthritis initially diagnosed as transient synovitis. Laboratory tests were negative, but stool examination revealed Giardia lamblia. Antiparasitic treatment resulted in complete remission, with no recurrence during follow-up [42].

Giardia lamblia is the most common parasitic infection worldwide, mainly affecting children in developing countries. Gastrointestinal symptoms are typical, while reactive arthritis is rare and likely immune-mediated, occurring more often in children and adolescents than adults.

11. Conclusions

Pediatric reactive arthritis is a rare, post-infectious, immune-mediated arthritis with generally good prognosis. While traditionally linked to gastrointestinal and genitourinary infections, recent evidence shows triggers including viruses (e.g., SARS-CoV-2) and parasites. HLA-B27 positivity and immune dysregulation influence severity and risk of chronicity. Diagnosis is primarily clinical, supported by infection history, inflammatory markers, and exclusion of septic arthritis. Treatment is usually symptomatic, with steroids or DMARDs reserved for refractory cases, and most children recover within weeks.

Infection-related arthritis in children represents a heterogeneous and evolving clinical entity. Given ongoing terminological ambiguity and expanding etiological associations, we advocate for an inclusive definition of ReA encompassing all non-septic arthritides temporally related to infection. Greater awareness and standardized diagnostic approaches are needed to avoid underdiagnosis and improve long-term outcomes.

Disclosure

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