International Journal of Advances in Medicine
*Yousaf I et al. Int J Adv Med. 2026 Mar;13(2):105-107*
http://www.ijmedicine.com
pISSN 2349-3925 | eISSN 2349-3933
DOI: https://dx.doi.org/10.18203/2349-3933.ijam20260391

**Case Report**

# Reactive arthritis linked to *Abiotrophia defectiva*: expanding the spectrum of post-infectious arthritis

**Imran Yousaf, Deepak Gupta*, Yasmin Bilal**

Division of Rheumatology, Department of Medicine, TidalHealth, Salisbury, MD, USA

**Received:** 18 December 2025
**Accepted:** 19 January 2026

***Correspondence:**
Dr. Deepak Gupta,
E-mail: deepak.gupta@tidalhealth.org



## ABSTRACT

Reactive arthritis is a form of sterile inflammatory oligoarthritis that occurs days to weeks following a genitourinary or gastrointestinal infection presenting with joint pain and swelling. Extra-articular manifestations include uveitis, oral ulcers, urethritis, and skin rashes. It is commonly due to gram negative bacteria such as *Chlamdyia, Campylobacter, Salmonella, Shigella*, and *Yersinia*. Recently, rare pathogens such as *Streptococci, Clostridium difficile, Escherichia coli, Mycoplasma pneumoniae* have also been implicated. However, *Abiotrophia defectiva*, a gram-positive bacterium, is not listed among them. We present to the best of our knowledge the only case of *Abiotrophia defectiva* as a causative agent of reactive arthritis.

**Keywords:** Reactive arthritis, *Abiotrophia defectiva*, HLA B27, Diarrhea, Knee swelling

## INTRODUCTION

Reactive arthritis is a form of sterile inflammatory mono- or oligoarthritis occurring days to weeks following a genitourinary or gastrointestinal infection with symptoms of joint pain, swelling, and stiffness. Extra-articular manifestations can include uveitis, oral ulcers, urethritis, skin rashes, and nail changes.[1] The presence of human leukocyte antigen B27 (HLA B27) may also increase the risk by altering various immune responses to bacterial infections.[2]

The etiology is commonly associated with gram negative bacteria such as *Chlamdyia, Campylobacter, Salmonella, Shigella*, and *Yersinia* species.[3] A growing list of rare pathogens have also been identified including streptococci, *Clostridium difficile, Escherichia coli, Mycoplasma pneumoniae*, among others.[4]

However, *Abiotrophia defectiva* (a gram positive bacteria) is not listed among them. We present, to the best of our knowledge, the first known case of *Abiotrophia defectiva* as a causative agent of reactive arthritis.

## CASE REPORT

A 55-year-old Caucasian male presented to the hospital with right knee and left ankle pain for 3 days. He reported several bouts of watery diarrhea with low grade fever two to three weeks prior to admission. The diarrhea resolved, but he soon developed painful swelling in his right knee. The arthrocentesis of his right knee showed elevated white blood cells (WBC, 17,000/ul) negative for crystals or organisms. He was prescribed Naproxen but soon developed left ankle and foot swelling. A faint rash also appeared on his chest, abdomen, and legs. He denied any difficulty breathing or tongue swelling. He discontinued naproxen, but his right knee pain worsened, and he was unable to bear weight on right leg. He visited his primary care physician where he was noticed to have an elevated heart rate. He was sent to the hospital where vital signs were significant for sinus tachycardia (139 bpm). Examination revealed right lower leg edema, reduced active and passive range of motion (ROM) in his right knee, and a faint maculopapular rash on his right hand and knee. Initial workup revealed leukocytosis (12,000/ul), thrombocytosis (547,000/ul), elevated D-dimer (2.40 ug/ml), lactic acidosis (3.2 mmol/l), elevated C-reactive

protein (CRP, 69 mg/l), and elevated erythrocyte sedimentation rate (ESR, 88 mm/hr). Urinalysis showed mild pyuria with few bacteria, and urine cultures were negative. Computed tomography angiography (CTA) of his chest was negative for pulmonary embolism. CT of his abdomen and pelvis revealed no acute intrabdominal pathology. Venous duplex ultrasound of the right lower extremity was negative for deep vein thrombosis. Radiographs of his right knee showed no fractures or effusions with preserved joint spaces. A knee arthrocentesis revealed elevated leukocyte count (3,946/uL) with neutrophilic predominance, without crystals or organisms, thus ruling out septic arthritis. A tickborne panel was collected, and he was started on doxycycline empirically. A CT of the right knee revealed lateral suprapatellar effusions, osteophytes, and minimal soft tissue swelling. Indomethacin was started due to a concern for reactive arthritis, along with famotidine for gastric protection. He was screened for antinuclear antibody (ANA), rheumatoid factor, anti-cyclic citrullinated (Anti-CCP) antibody, anti-proteinase 3 (PR3), and anti-myeloperoxidase (MPO) antibodies, which were all normal. However, his HLAB27 was positive. Radiographs of the sacroiliac (SI) joints showed mild degenerative changes in SI and hip joints without sclerosis, narrowing, or ankylosis. On day 2, blood cultures revealed *Abiotrophia defectiva.* A transthoracic echocardiogram (TTE) showed no valvular vegetations. Intravenous (IV) vancomycin was added to doxycycline. On day 3, the patient's right knee pain improved, and he was able to ambulate with the assistance of a walker. Tick-borne antibodies panel returned negative. On Day 7, repeat blood cultures were negative, and he was discharged to a skilled nursing facility to complete the course of IV vancomycin. The patient also continued indomethacin and was seen in outpatient follow-up two weeks later. Although symptomatically better, he still required a rolling walker to ambulate. Examination of his right knee revealed warmth with minimal fluid and tenderness along the inferomedial patella. Active ROM was more limited in extension; however, passive ROM was intact in both flexion and extension. An intraarticular corticosteroid injection was administered to his right knee joint. Indomethacin was replaced with high dose ibuprofen, and famotidine was replaced with pantoprazole. At four weeks of follow-up, his right knee pain and swelling had significantly improved, and he was able to ambulate without a walker. The active and passive ROM of his right knee was intact in all planes of motion. Repeat labs revealed an improved ESR (52 mm/hr) and normal CRP. He remained well on follow up.

## DISCUSSION

The estimated incidence of reactive arthritis is 1 to 30 in 100,000 persons, and commonly affects young adults between the ages of 20 to 40.[1] The frequency of cases is also reportedly higher in patients with a positive HLAB27 allele, presumably due to its role in manipulating various immune responses to bacterial infections.[2] Selmi et al have proposed major and minor criteria to diagnose reactive arthritis (Figure 1).[1]

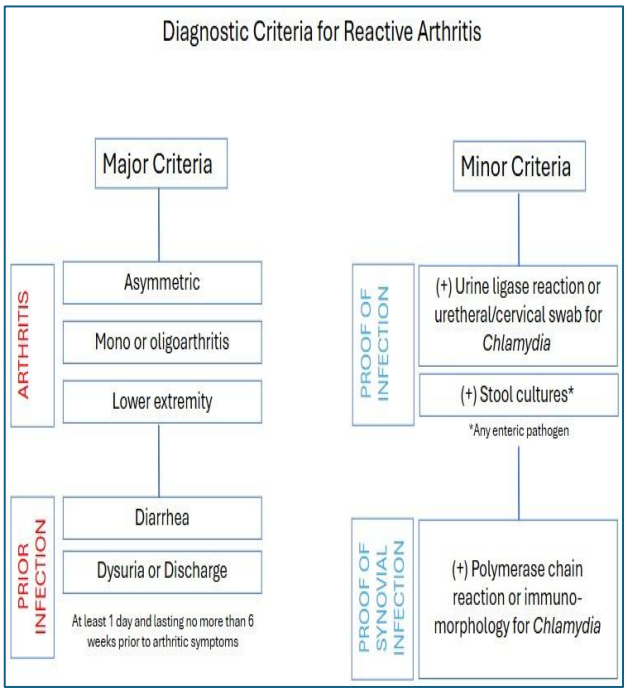


**Figure 1: Diagnostic criteria for reactive arthritis adapted and modified from Selmi et al.[1] Definitive diagnosis is made with both major and at least 1 minor criteria. Probable diagnosis is made with 1 major and 1 or more minor criteria.**

While commonly associated with gram negative bacteria, *Abiotrophia* has not been previously described as a causative agent. This gram-positive pleomorphic bacterium comprises the normal flora of the oral cavity and has been associated with high morbidity infections including infective endocarditis, spondylodiscitis and brain abscess, prosthetic joint infections, and liver abscess.[5-10] Due to its complex nutritional requirements, it can often lead to negative blood cultures and delays in diagnosis.[6-8,11] The pathophysiology of reactive arthritis involves several mechanisms including the ability of microbial antigens and their DNA to access synovial fluid and drive joint inflammation;[12] HLAB27 carriers displaying specific inflammatory peptides to T cells; imbalance of Th1 and Th17 pathways with an excess production of proinflammatory cytokines;[13] and changes in the gut microbiome.[14] Treatment primarily consists of NSAIDs and/or glucocorticoids. Antimicrobial therapies are tailored to the underlying infection; azithromycin or tetracyclines are often used to treat *Chlamydia*-induced arthritis.[1]

## CONCLUSION

In this case, septic arthritis was ruled out when knee arthrocentesis revealed inflammatory arthritis that was culture negative on two separate occasions. Tickborne illness was also ruled out due to the absence of history of

tick bites, rashes and negative tickborne antibodies. Although he was initially started on doxycycline, it was discontinued when urine cultures were negative for *Chlamydia*. Viral arthritides were initially considered but were thought to be less likely as these cases present acutely with symmetric involvement of small and large joints. Moreover, they often follow a self-limited course, none of which was observed. Eventually, the patient met both major and minor criteria for reactive arthritis as evidenced by subacute post-diarrheal migratory polyarthritis, evidence of infection, and positive HLAB27. This was further supported when symptoms improved with NSAIDs and intraarticular steroids. This case underscores the importance of maintaining a high suspicion in patients with a recent history of gastrointestinal or genitourinary infection who shortly develop painful migratory polyarthritis, especially in the context of a positive HLAB27. Prompt recognition and treatment is critical for mitigating joint destruction, preserving joint mobility, and improving patient outcomes.

*Funding: No funding sources*
*Conflict of interest: None declared*
*Ethical approval: Not required*

## REFERENCES

1. Selmi C, Gershwin ME. Diagnosis and classification of reactive arthritis. Autoimmun Rev. 2014;13(4-5):546-9.
2. Sahlberg AS, Granfors K, Penttinen MA. HLA-B27 and host-pathogen interaction. Adv Exp Med Biol. 2009;649:235-44.
3. Shafiee D, Salpynov Z, Gusmanov A. Enteric Infection-Associated Reactive Arthritis: A Systematic Review and Meta-Analysis. J Clin Med. 2024;13(12):3433.
4. Zeidler H, Hudson AP. Reactive Arthritis Update: Spotlight on New and Rare Infectious Agents Implicated as Pathogens. Curr Rheumatol Rep. 2021;23(7):53.
5. Christensen JJ, Facklam RR. *Granulicatella* and *Abiotrophia* species from human clinical specimens. J Clin Microbiol. 2001;39(10):3520-3.
6. Washington ER, Carius BM, Dougherty C. *Abiotrophia defectiva* triple threat: A rare case of infective spondylodiscitis, endocarditis, and brain abscess. Am J Emerg Med. 2024;75:199:e1-4.
7. Seguiti C, Piacentini E, Fraghì A. *Abiotrophia defectiva* and *Granulicatella*: A Literature Review on Prosthetic Joint Infection and a Case Report on *A. defectiva* PJI and Concurrent Native Valve Endocarditis. Microorganisms. 2025;13(5):1113.
8. Carleo MA, Del Giudice A, Viglietti R. Aortic Valve Endocarditis Caused by *Abiotrophia defectiva*: Case Report and Literature Overview. *In Vivo*. 2015;29(5):515-8.
9. Li J, Zhou L, Gong X. *Abiotrophia Defectiva* as a Rare Cause of Mitral Valve Infective Endocarditis with Mesenteric Arterial Branch Pseudoaneurysm, Splenic Infarction, and Renal Infarction: A Case Report. Front Med (Lausanne). 2022;9:780828.
10. Rasic P, Bosnic S, Vasiljevic ZV. *Abiotrophia defectiva* liver abscess in a teenage boy after a supposedly mild blunt abdominal trauma: a case report. BMC Gastroenterol. 2020;20(1):267.
11. Christensen JJ, Facklam RR. *Granulicatella* and *Abiotrophia* species from human clinical specimens. J Clin Microbiol. 2001;39(10):3520-3.
12. El-Gabalawy HS, Duray P, Goldbach-Mansky R. Evaluating Patients with Arthritis of Recent Onset: Studies in Pathogenesis and Prognosis. JAMA. 2000;284(18):2368-73.
13. Eliçabe RJ, Cargnelutti E, Serer MI. Lack of TNFR p55 results in heightened expression of IFN-γ and IL-17 during the development of reactive arthritis. J Immunol. 2010;185(7):4485-95.
14. Manasson J, Shen N, Garcia Ferrer HR. Gut Microbiota Perturbations in Reactive Arthritis and Postinfectious Spondyloarthritis. Arthritis Rheumatol. 2018;70(2):242-54.

**Cite this article as:** Yousaf I, Gupta D, Bilal Y. Reactive arthritis linked to *Abiotrophia defectiva*: expanding the spectrum of post-infectious arthritis. Int J Adv Med 2026;13:105-7.