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A case report of recurrent reactive arthritis caused by an occult asymptomatic infection by an unusual organism

Rintu Marak[1], Pooja Kumari[2*], Barnali Bhuyan[3], Ibandalin M Shangpliang[1]
[1] Consultant, Department of General Medicine, Nazareth Hospital, Shillong, Meghalaya, India
[2] Student, Department of General Medicine, Nazareth Hospital, Shillong, Meghalaya, India
[3] Senior Consultant, Department of General Medicine, Nazareth Hospital, Shillong, Meghalaya, India

Abstract
Reactive arthritis most commonly occurs following an episode of gastroenteritis by Yersinia, Salmonella, Shigella, Campylobacter, and Clostridium difficile or a genitourinary infection by Chlamydia trachomatis and Neisseria gonorrhea. Reactive arthritis due to other atypical organisms in HLA-B27 negative patients is often missed to lack of suspicion. This case report presents a case of recurrent reactive arthritis caused by an asymptomatic urinary tract infection by E.Coli in a female patient with HLA-B27 negative. The diagnosis was delayed due to atypical organism and presentation. This case report aims to highlight the need for a high degree of suspicion for reactive arthritis in cases of urinary tract infection by E.Coli.

Keywords: reactive arthritis, E.coli, urinary tract infection, HLA-B27, oligoarthritis

Introduction

Reactive arthritis is an inflammatory condition with multiorgan system disease potential. It is an old disease, initially coined in 1916 by Hans Reiter as Reiter syndrome. Because the standard constellation of symptoms in Reiter syndrome (arthritis, conjunctivitis, and urethritis) is not typically present in all patients, the disease can be easily overlooked if clinical suspicion is not high upon presentation [1].

Most patients will present with an oligoarthritis affecting a large lower extremity joint, typically the knee. However, patients may also have polyarthritis and other joints affected, including axial joints, cervical, thoracic, and lumbar spine. Furthermore, patients may have enthesitis involving the Achilles tendon insertion at the heel or even plantar fasciitis [3]. Skin manifestations include keratoderma blennorrhagica, circinate balanitis, palm and sole macular lesions, nail pitting, or onycholysis among others [1].

ReA most commonly occurs following an episode of gastroenteritis by Yersinia, Salmonella, Shigella, Campylobacter, and Clostridium difficile or a genitourinary infection by Chlamydia trachomatis and Neisseria gonorrhea [2]. UTI with E. coli, even if asymptomatic, can lead to ReA [2]. Hence, it is important that clinicians are aware of this rare cause of ReA. The diagnosis is comparatively easier when there is a history of recent UTI or active symptomatic UTI. Urine analysis and urine culture can be done to confirm the diagnosis in suspected cases [2]. Recent reports of ReA cover several rare causative microorganism such as Neisseria meningitides, Clostridium difficile, Escherichia coli, Hafnia alvei, Blastocytosis, Giardia lamblia, Cryptosporidium, Cyclospora cayetanensis, Entamoeba histolytica/dispar, Strongyloides stercoralis, β-haemolytic Streptococci, Mycobacterium tuberculosis, Mycoplasma pneumoniae, Mycobacterium bovis bacillus Calmette-Guerin, and Rickettsia rickettsii. The most prominent new infectious agents implicated as causative in ReA are Staphylococcus lugdunensis, placenta- and umbilical cord–derived Wharton's jelly, Rothia mucilaginosa, and most importantly the SARS-CoV-2 virus [3].

Mucocutaneous lesions are very specific to ReA. Keratoderma blennorragica and pustulosis palmo plantaris occur in 5–30% of patients. Circinate balanitis is found in 20–40% of cases. Mouth erosions located in the hard and soft palate, gingiva, the tongue and cheeks occur in 5–10% of patients. On the skin, psoriatic-like lesions could of- ten be present. Dystrophic nail lesions are found in 6–12% of patients. Erythema nodosum is characteristic only of Yersinia infections and occur in 15% of cases [4].

Currently there are no validated diagnostic criteria for reactive arthritis. The different criteria proposed share characteristics of a typical clinical picture (asymmetric oligoarthritis predominantly of the lower limb), exclusion of other rheumatic diseases and evidence of a previous or ongoing infection in the patient's history or laboratory examination and aseptic synovial fluid [5].

Reactive arthritis is an easily managed disease but easily missed particularly in young otherwise healthy patients who may not present with classic symptoms. Vigilance with regard to patients with vague seemingly unrelated complaints particularly with a history of gastrointestinal- or genitourinary-related illnesses deserves consideration for this disease process [1].

Case report

A 54-years-old female patient with a history of repeated asymptomatic urinary tract infections followed by polyarthritis and maculopapular rash. She presented to the hospital with a 10 days history of generalized weakness followed by arthritis, dactylitis, and worsening skin rash. Her presentation started with feeling unwell, generalized weakness, lower back pain, and loss of appetite. Two days later, she started to have multiple joint pain starting from the right knee then gradually involving other joints, elbows, shoulders, and ankles in addition to small joints of both hands. Joint involvement was asymmetric. The patient became dependent on others for daily activities within 7 days of the onset of symptoms. Skin rashes were maculopapular to start with and progressed to hyperpigmented and hypopigmented lesions. More on sun-exposed areas and not associated with itching. There was no history of preceding insect bite, recent vaccination, or medication use. She could not link her symptoms to any particular food. The patient had recurrent documented episodes of an asymptomatic urinary tract infection and asymmetric additive polyarthritis unresponsive to NSAIDS and skin rashes since 2015. On examination, there were hyperpigmented maculopapular skin rashes with hypopigmented lesions on sun-exposed areas (Images 1 and 2). She had arthritis with redness, hotness, and detectable effusion mainly in her right wrist (Image 3), right knee (Image 4), and both ankles. In addition to the tenderness of bilateral elbows and some of her metacarpophalangeal joints. Her liver and kidney function tests were normal. On Blood routine examination: Hemoglobin-7.4 gm %, total leucocyte count-14060/cumm, inflammatory markers were high with ESR of 107 mm/1st hour and c-reactive protein of 90 mg/L. Autoimmune workups, including rheumatoid factor, antinuclear antibody, and anti-CCP antibody were negative. Her HLA B27 was negative as well as her serology for hepatitis B, hepatitis C and HIV were negative. Serum protein electrophoresis had no M-spike. ANA was negative. Her iron profile showed evidence of anemia of chronic disease with serum iron-2 mcg/dl and total iron-binding capacity-220 mcg/dl. Urine analysis was suggestive of urine infection. Urine culture report revealed the growth of Escherichia coli > 1 lakh colony-forming units, ESBL producer. The patient was started on Inj Ertapenem 1 g once daily. Resolution of her symptoms started after 2 days of antibiotic and she started to be self-dependent for her daily activities by day 5 of antibiotic. She was discharged with antibiotic prophylaxis for recurrent bacteriuria.

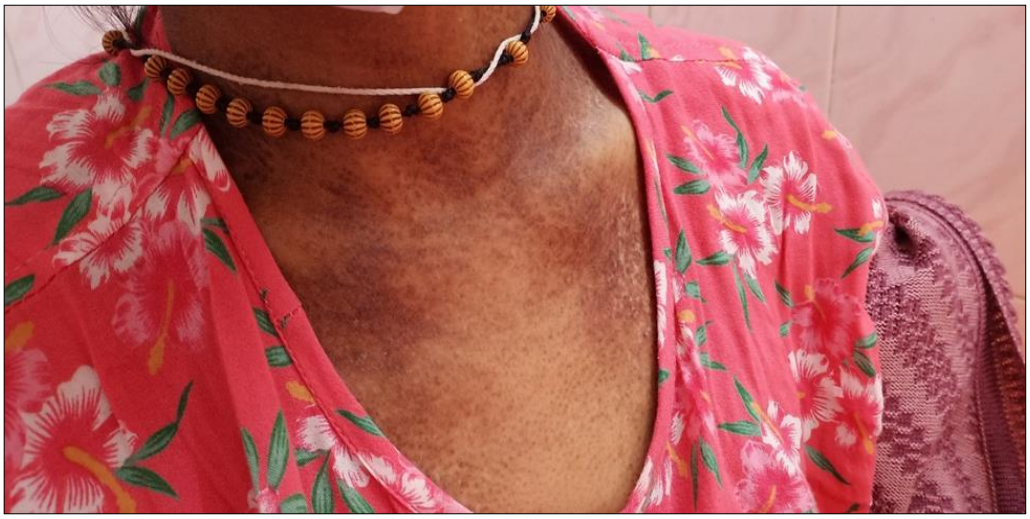

Fig 1: Hyperpigmented maculopapular skin rashes with hypopigmented areas in between over V area of neck

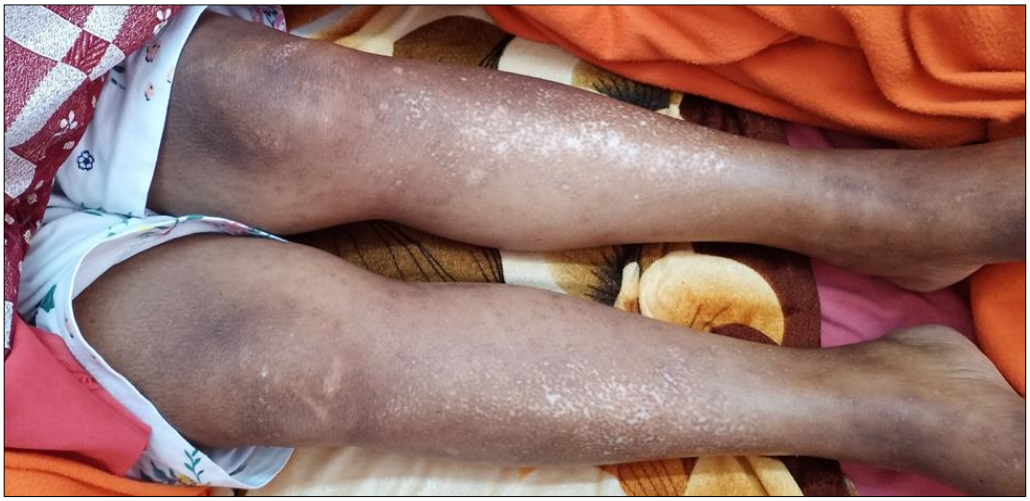

Fig 2: Hypopigmented lesions on bilateral shin

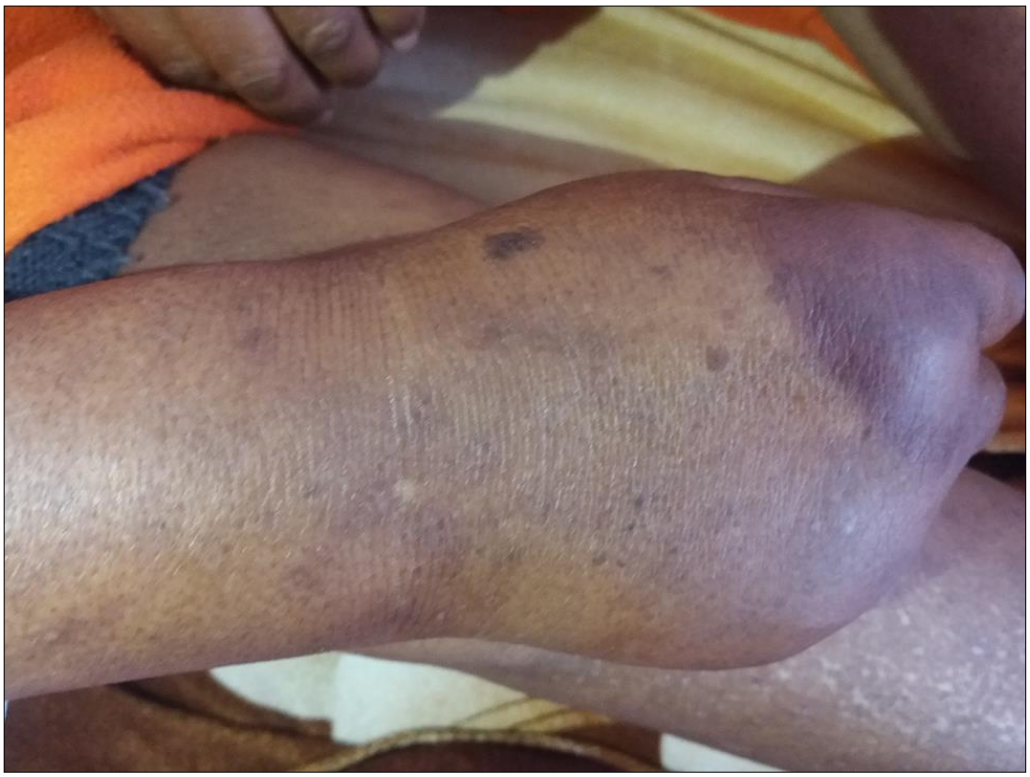

Fig 3: Arthritis with redness, hotness, and detectable effusion in right wrist

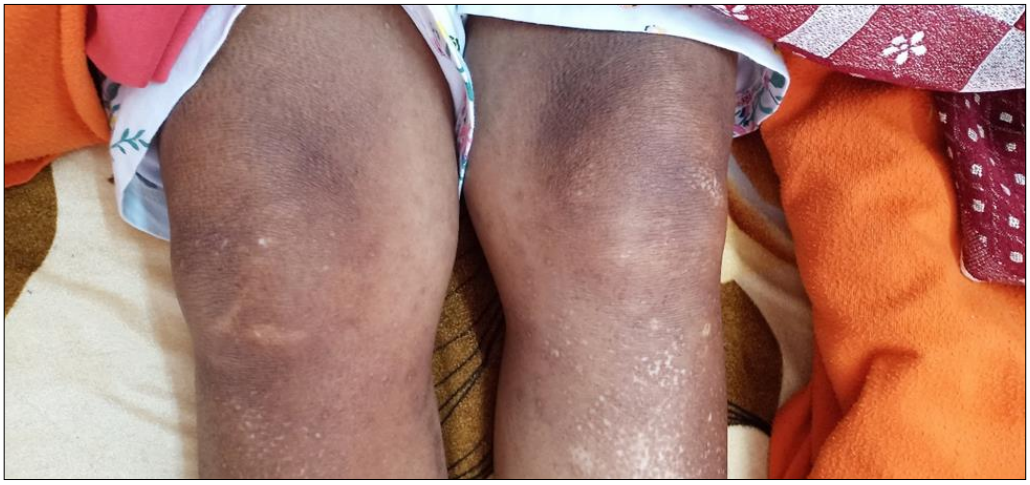

Fig 4: Arthritis with redness, hotness, and detectable effusion in right knee

Discussion

Recurrent, seronegative, asymmetrical oligoarthritis in a middle-aged female with underlying asymptomatic bacteriuria of E.coli was diagnosed as a case of reactive arthritis based on clinical presentation and suggestive laboratory investigations. We have ruled out rheumatoid arthritis, monoclonal gammopathy, gout, and SLE. Her arthritis was unresponsive to NSAIDs but improved dramatically after organism-sensitive antibiotic administration. This case is reported to make clinicians more aware of some rare organisms associated with reactive arthritis.

Reactive arthritis is most commonly seen in young males, but can also be seen in adult females. The diagnosis is comparatively easier when there is a history of recent UTI or active symptomatic UTI or asymptomatic bacteriuria. Urine analysis and urine culture can be done to confirm the diagnosis in suspected cases. There are only few reported cases of asymptomatic bacteriuria by E.coli as a cause of reactive arthritis. Reactive arthritis due to asymptomatic Escherichia coli bacteriuria in a young tuberculosis patient by Shahul HA *et al* [2]. The study conducted by Ali Lahu *et al*, on 100 patients concludes that: Out of 100 patients, 66% were males and 34% females. Among males we have noticed domination of post-urethritis and post-streptococcic reactive arthritis, whereas among females dominates reactive arthritis of enteral etiology. The study concludes that: urogenital tract was the source of infection with 66% of cases, nasopharyngeal tract with 19% of cases, and enteral tract with 15% of cases respectively. Predominantly presents bacteria are E. Coli with 21%, Staphylococcus aureus with 20%, Streptococcus B. hem. gr. A with 16% of cases respectively and other species [6]. Many rare

presentations of reactive arthritis have been reported such as a case report by Viviane de Carvalho *et al*: Reactive Arthritis Due to Subcutaneous Abscess by Streptococcus [7]. Individuals with the HLA-B27 allele or with a family history of spondyloarthritis apparently have an increased risk of developing reactive arthritis [8]. The prevalence of spondyloarthropathy and HLA-B27 gene positivity are correlated in a given population. The correlation is strongest in ankylosing spondylitis. Reactive arthritis following enteric or urogenital infection has a relationship that varies anywhere from below 50% to 85% [9]. In view of the large spectrum of causative agents, it is clear that diagnostic consideration must include the entire diversity of post-infectious arthritis termed ReA. The diagnostic procedure should not be restricted to the well-known HLA-B27- associated group of ReA, but must also cover the large number of more rare forms of arthritis following infections and vaccinations, as well as newly described members of the group of ReA summarized in this review [3]. Human leukocyte antigen B-27 is positive in 60-85% of patients with reactive arthritis but this may also occur in patients who are HLA-B27 negative [10]. Hence HLA-B27 negative patients should also be evaluated for reactive arthritis when suspected clinically.

Our patient had attended OPD multiple times with episodes of arthritis but the diagnosis was missed due to atypical presentation and low level of suspicion for reactive arthritis due to female gender and asymptomatic underlying infection. Hence it is important to always keep reactive arthritis as a differential diagnosis in cases of peripheral seronegative arthritis under evaluation even in HLA B-27 negative individuals.

Conclusion

To conclude, urinary tract infection with E. coli, even if asymptomatic, can lead to reactive arthritis. Hence, clinicians need to have a high degree of suspicion for reactive arthritis should consider the possibility of UTI as a cause in all cases of seronegative peripheral arthritis, and should judiciously employ urine analysis and urine culture for the diagnosis in indicated cases. Other than the commonly known organisms should also be considered as a source of reactive arthritis.

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