ORIGINAL RESEARCH PAPER

Volume-7 | Issue-2 | February-2018 | PRINT ISSN No 2277 - 8179

INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

REACTIVE ARTHRITIS FOLLOWING HEV INFECTION: A RARE CASE REPORT

Medicine

Dr Rimi Som Sengupta	Associate Professor, department Of Medicine, ESIC PGIMSR & medical College, Joka
Dr Samir Chakraborty*	Assistant Professor, Department Of Medicine,ESIC PGIMSR & medical College, Joka. *Corresponding Author
Dr Snehasish Ghosh	Senior Resident, , Department Of Medicine, ESIC Pgimsr & medical College, Joka
Dr Anirban Ghosh	Assistant Professor, Department Of Medicine, ESIC PGIMSR & medical College, Joka
Dr Anirban Sarkar	Assistant Professor, Department Of Medicine, ESIC PGIMSR & medical College, Joka
(Dr) Tapas Kumar Mondal	Professor Head Of The Department, Department Of Medicine, ESIC PGIMSR & medical College,Joka

ABSTRACT

Reactive arthritis is a sterile oligo-arthritis associated with infection. Chlamydia, Salmonella, Shigella have been postulated as most common organisms associated with Reactive arthritis. Genetic susceptibility, mainly HLA-B27 positivity, plays a key role in the pathogenesis. It is thought that antigen from infectious organisms trigger inflammatory response in synovium in a genetically susceptible host. Two infectious syndromes-genitourinary and gastrointestinal infections have been classically depicted with Reactive arthritis. However, in literature, other sorts of infections have occasionally been described with this form of arthritis. Here we report a case of reactive arthritis associated with HEV infection.

KEYWORDS

Introduction

Reactive arthritis(ReA) is a form of spondyloarthritis triggered by a distant infection, usually gastrointestinal or genitourinary. There is a latency period of few weeks between the infection and mounting of arthritic response. Prevalence of ReA varies according to region and endemicity of the causative microorganism. 20-40 yrs age-group is more frequently affected with no gender predilection[1]. Presentation of ReA is oligoarticular with lower limb predilection along with enthesitis and often extra-articular manifestation in the form of ocular and cutaneous manifestation. Despite knowing the initial event (gastrointestinal or genitourinary infection), pathogenesis of ReA is incompletely understood. Genetic factors like HLA-B27 positivity plays an important role. Environmental factors, the causative microbes of gastrointestinal and genitourinary infections namely Chlamydia, Shigella, Salmonella,Yersinia, Campylobacter are said to trigger the arthritic response, although precise mechanism is largely unknown. Few recent studies have shown persistence of these microbes or their antigen, after subsidence of initial distant infective process, in various body tissues including involved joints, lymph nodes, spleen, liver, peripheral blood cell[2,3,4], whereas some others have shown an abnormal cytokine response involving TNF and IL-10 linked with ReA[5,6].

Although large extent of literature till date have been dedicated to association of ReA with genitourinary or gastrointestinal infections, only sparse to suggest other form of infection to trigger ReA. Here we present a case of ReA following HEV associated acute hepatitis.

Case Report

A 25yrs old male was admitted in ESIC-PGIMSR Hospital with fever, nausea, vomiting, abdominal pain followed by jaundice and was diagnosed as acute viral hepatitis with following investigations- Total bilirubin 5.6, Conjugated bilirubin 3.4, SGPT 4211, SGOT 3938, ALP 275, GGT 80, HEV IgM Reactive. With conservative management patient improved gradually and was in dischargeable condition in next 10 days with following blood parameters- Total bilirubin 6.7, conjugated bilirubin 4.2, SGPT 87, SGOT 138, ALP263. After 1 week patient visited medicine OPD with complaint of pain and swelling of both ankles, knees and left elbow. On examination, asymmetric joint involvement with gross effusion and marked tenderness was noted. Small joints were spared. There were no evidence of enthesitis, uveitis or axial skeletal involvement. Balanitis was present. History of diarrhea, dysuria in last few weeks were absent. No personal or family history of psoriasis or IBD was there. On further enquiry, a second degree relative was found to suffer from spondyloarthritis since young age. Blood parameters were as follows- Hb 12.9, TLC 9300, Platelet 2.8 lac/cmm, Total bilirubin 2.1, SGPT 55, SGOT 50, ALP 274. Urine ME showed 15-20 pus cell/hpf, 10-12 RBC/hpf; Urine C/S- nogrowth; USG knee showed synovial thickening with fluid in suprapatellar bursa. Joint fluid study revealed- pale yellow, viscous, hazy fluid with mucoid coagulum, TLC 7500, PMN 80%, lymphocyte 15%, Macrophage 5%, Protein 4.6, LDH 1004, ADA 13, Gram stain and ZN stain negative, Culture nonproductive. HLA B27 was positive. RF, Anti CCP, Serum ACE and Ceruloplasmin were within normal limit. Considering the presentation and investigation reports we proposed a diagnosis of Reactive arthritis and put him on NSAID with only modest benefit in subsequent days. Then we added prednisolone 40 mg per day. Patient responded well this occasion.

Discussion

First case of Reactive arthritis was reported by Hans Conrad Reiter. ReA is a sterile synovitis predisposed by HLA B27 positivity and triggered by distant infection preceded by few weeks. According to American Rheumatology Association criteria patients with Reactive arthritis presents with asymmetric arthritis which lasts for more than 1 month associated with 1 or more of the following features: urethritis, inflammatory ocular disease, balanitis, mouth ulcer, or radiographic evidence of sacroilitis, periostitis, heel spurs[7].

This patient developed asymmetric arthritis with balanitis and sterile urethritis. No feature suggestive of renal, cardiovascular pulmonary or neurological involvement. Diagnosis of ReA is clinical with no specific confirmatory laboratory marker. HLA B27 positivity only increases likelihood; HLA-B27 is positive in this case. Elevated ESR and CRP are inflammatory markers. Synovial fluid analysis is compatible with inflammatory synovitis. RF, Anti-CCP, SACE, Ceruloplasmin negativity rule out close differential diagnoses.

The etiology of ReA is incompletely understood. Various GI and urinary pathogens have been described to trigger this inflammatory arthropathy through sharing of antigen epitope with that of synovial components. Chlamydia, Shigrella, Salmonella have been reported as the culprit pathogens on numerous occasions in literature. Virus as a group have only rarely been reported. In this case, having no other identifiable culprit organism, HEV cab justifiably be ascribed as the triggering agent of Reactive arthritis.

Treatment of ReA employs NSAIDs and sulfasalazine. Corticosteroids are used in cases not responding to NSAIDs. Immunomodulators like Azathioprine, Methotrexate, Cyclosporine can be used as steroid sparing agents. In severe cases biologicals like TNF-a antagonists are drug of choice.

Course of ReA is variable and is probably dependent upon triggering pathogen, gender of patient and genetic background. In most of the cases remission occurs within 6 months. Only a small fraction develops chronic persistent arthritis lasting more than 6 months.

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