Case Report

Shigella flexneri associated reactive arthritis - GI transmitted or sexually transmitted?

Nidhi Kaeley[1], Manish Kumar[2], Bharat B. Bhardwaj[1], Vempalli Nagasubramanyam[1]

Departments of [1]Emergency Medicine and [2]General Medicine, All India Institute of Medical Sciences, Rishikesh, Uttarakhand, India

Abstract

The pathogenic association of reactive arthritis with human immunodeficiency virus (HIV) needs more attention. In this case report we described a case of 22 year old male patient suffering from severe HIV infection. He presented with the complaints of left knee joint pain associated with swelling and tenderness. He also developed keratotic papules on palms and soles and polycyclic erosions on the glans penis. He was diagnosed as a case of reactive arthritis with HIV infection.The patient was treated with sulfasalazine and anti retroviral therapy. We, hereby discuss the underlying pathogenesis and treatment modalities in patients of reactive arthritis with underlying HIV infection. The treatment of reactive arthritis with HIV is a challenge due to limited options of immunosupressive agents.

Keywords: HIV, reactive arthritis, shigella flexneri

Introduction

Multiple musculoskeletal manifestations of human immunodeficiency virus (HIV) have been described. They are HIV-associated arthropathy, seronegative spondyloarthropathies (SPA), which includes reactive arthritis, psoriatic arthritis (PsA), undifferentiated arthritis, and painful articular syndrome.[1] Management of musculoskeletal disorders in HIV positive patients is challenging in view of safety profile of immunosuppressive drugs. We present a case of reactive arthritis in a patient suffering from HIV infection secondary to Shigella flexneri infection and its management.

Case Report

A 22-year-old male patient was presented with the complaints of right toe dactylitis and redness of bilateral eyes since the last six months. After four months he developed pain and swelling of left knee and right heel with morning stiffness. History revealed to be positive for MSM. Two months later he developed bilaterally symmetrical erythematous keratotic papules on palms and soles suggestive of keratoderma blenorrhagicum [Figures 1 and 2]. He also developed lesions on glans penis which were polycyclic erosions suggestive of circinate balanitis. The patient was diagnosed as a case of HIV positive one year back for which started on zidovudine.

At time of admission his CD4 count was 85 cells/mm^3. He gave history of loose stools six months back which lasted for five days. Stools were watery in consistency and blood tinged. Stool cultures were positive for Shigella flexneri. There was no family history or past history psoriasis and arthritis.

On examination, his vitals were normal except for tachypnea. The respiratory system examination was suggestive of right sided infrascapular crepitation. Rest of systemic examination was normal.

Address for correspondence: Dr. Manish Kumar, Department of General Medicine, All India Institute of Medical Sciences, Rishikesh - 249 203, Uttarakhand, India.
E-mail: drmanishkumar123@gmail.com

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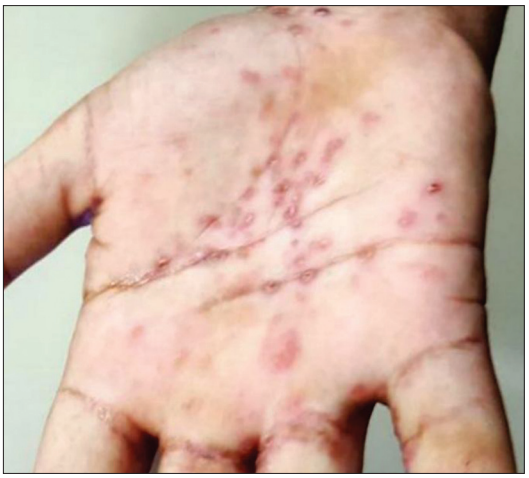

Figure 1: Keratoderma blenorrhagicum lesions over palm

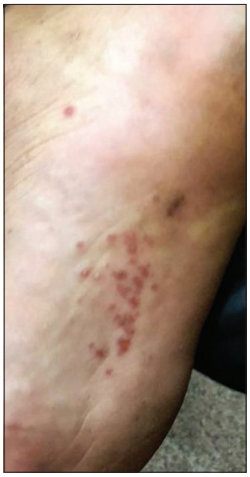

Figure 2: Keratoderma blenorrhagicum lesions over sole

Complete hemogram revealed mild anaemia and leucocytosis. Kidney function and liver function test were within normal range. Inflammatory markers ESR and CRP were significantly raised. HLA - B27 was positive. Tests for hepatitis B and C were negative. Rheumatoid factor (RF) was negative. The sputum test was positive for AFB (acid fast bacilli). Screening for chlamydia, gonorrhoea, and syphilis was negative. Urethral, rectal and pharyngeal swab cultures were negative for neisseria gonorrhoea. Chest x-ray showed left lower zone consolidation. Left knee x-ray was normal. Left knee synovial fluid aspiration was suggestive of leucocytosis with no organisms and growth in cultures. Skin biopsy from palm showed regular acanthosis, parakeratosis, neutrophilic abscesses in stratum corneum, supra papillary thinning of epidermis and dilated capillaries in papillary dermis, which was consistent with reactive arthritis.

Diagnosis of reactive arthritis with AIDS with pulmonary tuberculosis was made. Intra-articular corticosteroid (triamcinolone 20 mg) was started. The patient's joint pain did not show any improvement. Hence, he was started on non-steroidal anti-inflammatory drugs (NSAIDS) and oral sulfasalazine (one gram twice daily). The patient's joint pains and skin lesions gradually improved. Since CD4 count was low anti-tubercular therapy was initiated and anti-retroviral therapy was discontinued for eight weeks.

Discussion

This patient fulfilled the criteria of reactive arthritis (oligoarthritis, enthesitis, circinate balanitis and keratoderma blenorrhagicum). HIV infection is associated with multiple opportunistic infections. Thus, use of immunosuppressive drugs is controversial in these patients. There are varied manifestations of HIV associated arthritis. They range from asymmetrical oligoarthritis to symmetrical polyarthritis. Oligoarthritis mainly involves knees and ankles.[2,3] Spondyloarthropathies can present as reactive arthritis, psoriatic arthritis, undifferentiated arthropathies, and painful articular syndrome. Other articular manifestations of HIV are avascular necrosis of bone, hypertrophic pulmonary osteoarthropathy, osteopenia and osteoporosis.[4]

Reactive arthritis was first described by Hans Reiter in 1916. It was called as Reiter's syndrome and constituted of arthritis, urethritis and conjunctivitis. It was later termed as reactive arthritis in 2003 when Hans Reiter was held responsible for war crimes in Second World War.[5] HLA B-27 is strongly associated with reactive arthritis. When reactive arthritis is seen in children and adolescents less than 16 years of age, termed as juvenile idiopathic arthritis (JIA).[6]

It is common in age group of 20-40 years and usually occurs in few days to six weeks after preceding urogenital or enteric tract infection.[7] Most probable causative organisms in patients with reactive arthritis are Chlamydia trachomatis, Shigella flexneri, Salmonella enteritidis, Salmonella typhimurium, Yersinia enterocolitica, Yersinia pseudo typhimurium, and Campylobacter jejuni. Very rarely caused by haemophilus parainluenza.[8] Various components of these organisms have been observed in synovium of the patients with reactive arthritis. HLA-B27 positivity in these patients is directly related to increased severity of reactive arthritis and increased probability of developing various extra-articular manifestations.[9] Apart from HLA B-27, IL-10 and IL-12 also play a pivotal role in predisposing inflammatory response in the synovial fluid.[10] Another theory proposed is role of IFN-Ý in clearing pathogenic bacteria causing reactive arthritis.[11] HIV positive patients having reactive arthritis have increased probability of developing extra-articular manifestations such as dactylitis, achilles tendonitis and plantar fasciitis. They have more severe mucocutaneous manifestations in form of keratoderma blennorrhagica and circinate balanitis. Various inflammatory dermatoses also manifested with HIV.[12] Treatment of reactive arthritis in HIV positive patients is same as that of HIV negative patients. NSAIDS and sulfasalazine are mainstay of treatment.[13] However, TNF-α blockers should be used with caution in these patients.[14] It has been proposed that patients with resistant arthritis respond better with highly active retroviral therapy (HAART).[15]

In HIV related infection, exposure to opportunistic infections is increased. This leads to inflammatory reaction but there is sparing of CD8-T cells. These multiple biochemical factors lead to localisation of primary disease.[16] As per previous studies, association of HIV and reactive arthritis is rare. Berman *et al.* described reactive arthritis in 10% cases of HIV.[17] Shigella flexneri is a known micro-organism to cause reactive arthritis. It is usually transmitted by contaminated food and water. However, it is also transmitted by sexual route in 3 to 8.1% cases of reactive arthritis.[18]

Conclusion

HIV infection is associated with multiple complex rheumatological manifestations. Early identification and treatment can prevent comorbidities and improve their disability index. Family physicians will be frequently encountering HIV associated with arthritis as prevalence of STD steadily growing. Steroids use in this group is highly discouraged and NSAID and sulfasalazine are highly beneficial.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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