Case Report

Phil. J. Internal Medicine, 47: 37-40, Jan.-Feb., 2009

REITER’S SYNDROME

Janice V. Villanueva, M.D. and Maria Jasmin J. Jamora, M.D.

ABSTRACT

Objective: To report a case of Reiter’s Syndrome in a Filipino patient.

Setting: Skin and Cancer Foundation – Dermatology OPD, Quirino Memorial Medical Center, Quenzon City.

Background: Reiter’s syndrome affects all population worldwide, without clear regional prevalence. In Finland, 8,806 cases were reported after a Chlamydia trachomatis infection. Frequency of Reiter’s has been increasing since 1998. German statistics reported an estimate of 10/10,000 versus 3.5/100,000 in the United States. There is no statistics available on the prevalence or incidence of Reiter’s syndrome in the Philippines. Reiter’s Syndrome (Reactive arthritis) presents with the classic triad of arthritis, conjunctivitis and urethritis. The term reactive arthritis implies a clinically characteristic reaction to a precipitating infection. Infection and immune response are thought to be the cause of Reiter’s syndrome. Reactive arthritis, which belongs to the group of spondyloarthopathies, has been known to be associated with HLA B27. There is no gold standard for the treatment of Reiter’s syndrome.

Summary: A 22 year old Filipino male presented with a 3 month history of bilateral symmetrical pain and swelling of both knees and ankles followed by the appearance of erythematous scaly plaques on knees, scalp and chest. Urethritis and conjunctivitis were also present. Patient presented with difficulty walking due to left knee pain. Physical examination revealed swollen upper and lower eyelids, with crusting on eyelash line, and bilateral conjunctivitis. No oral mucosal lesions were noted. His left knee was swollen and warm, with tenderness but no swelling and crepitation on right knee and both ankle joints. Skin examination showed histologic features consistent with the diagnosis of Reiter’s Syndrome, which is an immunologic reaction pattern usually precipitated by an infection. It presents with the triad of conjunctivitis, urethritis and arthritis. Therapy usually consists of a course of antibiotics and immunosuppressants. It has a variable course and usually lasts for 3 to 12 months but may progress to a chronic illness.

Keywords: Reiter’s syndrome, reactive arthritis, urethritis, seronegative spondyloarthopathies

INTRODUCTION

Reiter’s syndrome (reactive arthritis) presents with a classic triad of conjunctivitis, urethritis, and arthritis. It is an immunologic reaction pattern usually to a precipitating infection, which falls under the seronegative spondyloarthopathies (which also includes ankylosing spondylitis, psoriatic arthritis, the arthropathy of associated inflammatory bowel disease, juvenile-onset ankylosing spondylitis and juvenile chronic arthritis). This paper reports a classic case of Reiter’s syndrome, a rarely reported disease in Filipinos.

CASE REPORT

A 22-year-old Filipino male presented at Quirino Memorial Medical Center Dermatology outpatient department with a 3 month history of bilateral symmetrical pain and swelling of knees and ankles followed by the appearance of erythematous scaly plaques on the knees, scalp and chest. Patient also reported on and off low grade undocumented fever, weakness and dysuria. He was otherwise healthy. He denied previous blood transfusions or intravenous drug use, but admitted to having multiple sexual partners of both genders.

Patient presented with difficulty walking due to left knee pain. Physical examination revealed swollen upper and lower eyelids, with crusting on eyelash line, and bilateral conjunctivitis. No oral mucosal lesions were noted. His left knee was swollen and warm, with tenderness but no swelling and crepitation on right knee and both ankle joints. Skin examination revealed generalized involvement, including the genital area, with multiple well defined erythematous plaques and thick adherent scales. A skin biopsy specimen showed histologic features consistent with the diagnosis of Reiter’s Syndrome, which is an immunologic reaction pattern usually precipitated by an infection. Therapy usually consists of a course of antibiotics and immunosuppressants. It has a variable course and usually lasts for 3 to 12 months but may progress to a chronic illness.

A punch biopsy was taken from the chest. The patient was started on prednisone 20mg daily and tapered over a week, diclofenac 100mg once a day...
and betamethasone ointment twice daily. Patient improved after one week of therapy, however 2 weeks later, lesions recurred and worsened. Patient was then admitted and co-managed with Internal medicine.

Initial laboratory workup revealed anemia (hematocrit 28% and hemoglobin 98 g/dL) and leukocytosis (11,500/mm³). Urinalysis revealed pyuria (4-8 pus/hpf). HIV ELISA test, Hepatitis profile, Urethral swab culture, VDRL and TPHA tests were all negative.

Histopathologic examination of the skin punch biopsy taken from the chest (Figures 2 A and B) revealed parakeratotic scale, psoriasiform hyperplasia, a diminished granular layer and superficial perivascular dermal infiltrate with lymphocytes and few neutrophils. It was read as “psoriasiform dermatitis consistent with Reiter’s syndrome.”

He was started doxycycline 100mg BID which was later shifted to ceftriaxone 1gm/day given intravenously for a week. Diclofenac 100mg twice daily, loratadine 10mg daily, hydroxyzine 25mg once daily, methotrexate 2.5mg (given at three weekly doses) and betamethasone cream twice daily were continued. Patient was referred to ophthalmology, was diagnosed to have “conjunctivitis” and given prednisolone eye drops. On the 12th hospital day, he was discharged improved, however was lost to follow up.

**DISCUSSION**

The classic triad of arthritis, conjunctivitis and urethritis of Reiter’s syndrome (reactive arthritis) is clearly manifested by this patient, with knee and ankle joint pain and swelling, presence of bilateral conjunctivitis, and dysuria supported by pyuria indicative of urethritis.

The term reactive arthritis implies a clinically characteristic reaction to a precipitating infection. Infection and immune response are thought to be the cause of Reiter’s syndrome. Symptoms like arthritis are noted weeks after an infection in the gastrointestinal or urogenital tracts. The most commonly implicated organisms are Shigella, Campylobacter, Salmonella, Yersinia, Borrelia, Ureaplasma, Chlamydia and Neisseria.

Reactive arthritis, which belongs to the group of spondyloarthropathies, has been known to be associated with HLA B27. It is hypothesized that it functions as an antigen presenting molecule for an as yet unknown arthritogenicpeptide. Cytokines, T cell response and other immune modulators also play a role in the development of reactive arthritis.

Reiter’s syndrome affects all population worldwide, without clear regional prevalence. In Finland, 8,806 cases were reported after a Chlamydia trachomatis infection. Frequency of Reiter’s has been increasing since 1998. German statistics reported an estimate of 10/10,000 versus 3.5/100,000 in the United States. There is no statistics available on the prevalence or incidence of Reiter’s syndrome in the Philippines.

Any age group can be affected, however it typically affects young adults 20-40 years old and rare in children and older population. Four cases of juvenile Reiter’s syndrome was reported in Nigeria, with age ranging between 8-17 years old.

Symptoms of Reiter’s syndrome generally appear within 4 weeks after the infection but there is great variation in the severity and timing of clinical symptoms. A Nigerian patient manifested symptoms of arthritis 2 weeks after a gastrointestinal infection. A female patient manifested with arthritis 9 months after the onset of painful mucocutaneous lesions.

In a retrospective study of 432 cases of acute gastroenteritis, 27 patients developed arthritis 3 months after the infection, 9 patients after 4 months and 18 patients 5 years after infection.

Typical dermatologic manifestations include keratoderma blenorrhagicum, seen as thick scaling erythematos plaques on palms and soles; circinate balanitis, seen as brightly erythematous plaques with scales on genitalia, and nail dystrophy, which were all present in our patient. There are currently no established criteria for diagnosis of Reiter’s syndrome and no specific laboratory tests to clinch the diagnosis. The diagnosis of Reiter’s syndrome should therefore rest on the triad of symptoms, as well as typical clinical features seen on thorough history and physical examination.

There is no gold standard for the treatment of Reiter’s syndrome. There is a longstanding debate as to the efficacy of empiric antibiotic treatment, with some studies showing no role for antibiotics in the treatment of reactive arthritis. Immunosuppressants such as cyclosporine, etretinate and methotrexate have shown efficacy in the treatment of severe recurrent Reiter’s syndrome.

Our patient was managed conservatively with non-steroidal anti-inflammatory drugs (diclofenac), antibiotics (doxycycline and cefuroxime), methotrexate and topical steroids with slight relief of symptoms.
Reiter’s syndrome has a variable course, with usual duration between 3 to 12 months, and it may resolve spontaneously or progress to chronic illness. In conclusion, this is an interesting and unique case of classic Reiter’s syndrome in a Filipino patient.

Figure 1A. Generalized cutaneous involvement, multiple well defined erythematous annular plaques with thick adherent scales. Close up view reveals the thick adherent scales.

Figure 1B. Genital involvement reveals erythematous well defined scaly plaques. Penile involvement is described as circinate balanitis.

Figure 1C. Thick desquamation of the palms and soles.

Figure 1D. Nail changes includes onychotysis, nail dystrophy, subungual debris and periungual pustules.

Figure 2A. Histopathology reveals parakeratotic scale, psoriasiform hyperplasia, diminished granular layer and perivascular infiltrate with lymphocytes and few neutrophils.

Figure 2B. Hematoxylin and eosin stain, a at 200x original magnification; at 400 x original magnification.
REFERENCES

1. Andrew K: Reactive Arthritis, Fitzpatrick’s Dermatology in General Medicine, Seventh Edition/Klaus Wolff...[et al.]: 207.


10. Werner B, Kinim S; (Washington,D.C.: Washington Hospital Center)


