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Reactive Arthritis Due to Influenza Virus in a Male Patient: A Case Report

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1. Abstract

Myalgias and arthralgias are a common symptom of influenza viruses' infection. Reactive arthritis due to influenza vaccination has been described. Here we report the case of a 40-year-old Greek patient with free history who suffered from reactive arthritis due to influenza A infection. He proved negative to other viral or bacterial infections or autoimmune diseases related to arthritis. The affected joints were typical of post-infectious arthritis. The patient responded well to corticosteroid administration. All the above led to the suggestion that our patient's arthritis represents a rare case of reactive arthritis after influenza A virus infection. To our knowledge this is the first case of reactive arthritis reported due to influenza virus A in adults.

2. Introduction

Myalgias and arthralgias are a common symptom of influenza viruses' infection. [1] Reactive arthritis due to influenza vaccination has been described. [2-4]

Here we report the case of a 40-year-old Greek patient with free history who suffered from reactive arthritis due to influenza A infection. He proved negative to other viral or bacterial infections, such as hepatitis virus B and C, Human Immunodeficiency Virus (HIV), Cytomegalovirus (CMV), Epstein-Barr Virus (EBV), adenovirus, parvovirus, mumps, and rubella. Toxoplasma, syphilis, Chlamydia trachomatis, Streptococcus pyogenes and Brucella infection were also excluded. The affected joints were typical of post-infectious arthritis. To our knowledge, this is the first reactive arthritis reported due to influenza virus A in adults.

3. Case Presentation

A 40-year-old male patient, with free history visited his family doctor due to low-grade fever, myalgias, fatigue and sore throat that commenced 48 hours before. Physical examination revealed that the patient was in good general condition with signs of pharyngitis and without shortness of breath. Temperature of 380C, blood pressure of 110/60 mmHg, pulse rate of 75 beats/min, and oxygen saturation of 99 % (FiO2: 21 %). Chest auscultation revealed normal lung sounds. Kernig's, Brudzinski, Giordano, and Murphy signs were negative. No swollen lymph nodes splenomegaly, or hepatomegaly were detected. Due to the Covid-19 pandemic, a Point-of-Care (POC) test and RT-PCR were performed and proved negative. The patent was subscribed antipyretic drugs and clarithromycin 500 mg twice daily. Three days later he returned for examination due to persistence of fever. A second Point-of-Care (POC) test and RT-PCR were performed for COVID-19 and proved negative. Chest X-ray was without pathological findings. Electrocardiogram revealed negative T-waves in leads V1-6. Troponin levels and D-dimers were within normal range. Heart, thyroid, and abdominal ultrasounds were normal. A rapid influenza A test was performed, and the patient proved positive for influenza A virus.

One day later he was afebrile and in good general condition. On the seventh day from initial symptoms onset, he felt intense acute pain in lower left abdomen and fever (38oC). He attended the local hospital on his therapist instructions, where abdominal Computed Tomography revealed diverticulitis of the sigmoid. He was

administered antibiotics -ciprofloxacin and metronidazole- and the fever and pain subsided. After having taken the antibiotic therapy for 6 days, he developed migratory arthralgias of lower and upper extremities and fever (38oC). To this point physical examination revealed no tenderness, swelling, or restricted motion of the affected joints. C-reactive protein, erythrocyte sedimentation rate, antinuclear antibodies, rheumatoid factor, and antistreptolysin titer were within normal range or negative. The patient was negative for hepatitis virus B and C, HIV, CMV, EBV, adenovirus, parvovirus, mumps, and rubella. Toxoplasma, syphilis, Chlamydia trachomatis and Brucella infection were also excluded by serologic tests. Rheumatoid factor and antinuclear antibody tests were negative. Serologic tests for Borrelia burgdorferi were not performed as there is low prevalence in Western Greece, the patient had not travelled abroad recently and there was no such evidence from the patients' present history. Due to lack of diarrhea and other symptoms from the gastrointestinal tract, a stool culture was not performed. Human leukocyte antigen (HLA) typing was not performed.

To the light of these new symptoms and laboratory results, either side effects to ciprofloxacin or reactive arthritis were suspected. Ciprofloxacin was stopped and ibuprofen 600mg twice daily was administered. After commencing ibuprofen, arthritis subsided for four days. On the fifth day of ibuprofen administration, he complained to suffer from acute joint pain, stiffness, warmth, swelling and redness of both knees. The patient was admitted to the University Hospital of the region where he lives, where the diagnosis of reactive arthritis was confirmed. He was prescribed methylprednisolone 24 mg daily with immediate clinical improvement. On follow-up he was in good general condition and all symptoms had subsided.

4. Discussion

Acute arthritis related to viral infections is usually self-limiting and requires no specific therapy. [5] Approximately 1% of acute arthritis is of viral etiology. [1] The most common viruses related to joint involvement are hepatitis B and C viruses and HIV. [5] Other viruses related to arthritis are parvovirus B19, rubella virus, Epstein-Barr, varicella zoster and mumps. [2-4, 6]

Post- infectious arthritis or reactive arthritis formerly known as Reiter's syndrome, is defined as a sterile inflammatory arthritis occurring secondary immediately or months after initial infection. The most common joints affected are the knees and ankles. [2-4, 6] It is related to infection by certain bacteria, such as Chlamydia trachomatis, Campylobacter, Salmonella, Shigella and Yersinia. [7] Our patient was negative for Chlamydia trachomatis, whereas had no history for gastrointestinal infection.

Human Leukocyte Antigen (HLA)-B27 is related to the manifestation of arthritis. [4] The most common age group affected are men aged between 20 and 50 years. [6] Our patient was not tested for HLA-B27.

Differential diagnosis also includes rheumatoid arthritis or systemic lupus erythematosus. [8-9] Our patient had negative rheumatoid factor and antinuclear antibody tests; therefore, these entities were excluded.

Many viruses and bacteria are also related to arthritis. Our patient was negative for hepatitis virus B and C, HIV, CMV, EBV, adenovirus, parvovirus, mumps, rubella, Toxoplasma, syphilis, Chlamydia trachomatis, Streptococcus pyogenes and Brucella infection.

Clinical manifestations of reactive arthritis include pain and swelling of joints, mostly of the lower extremities, mainly the knees and the ankles. In some patients there is also ocular involvement (conjunctivitis), tendonitis, enthesitis, and urithtitis. [7] Our patient suffered initially from migrating arthralgia of the upper and lower extremities which evolved to joint stiffness and signs of inflammation of both knees.

Viral arthritis is treated symptomatically. Usually Nonsteroidal Anti-Inflammatory Drugs (NSAIDs) are the treatment of choice. Corticosteroids are used to treat severe symptoms or if there are contraindications to NSAIDs usage. [5] Our patient was initially treated with ibuprofen and the symptoms retreated after the use of corticosteroids.

To our knowledge, in adult patients, reactive arthritis has only been related to influenza immunization. [4] T cells, macrophages, and dendritic cells that are present in low numbers in a normal synovium could be implicated in synovitis in a previously healthy joint. The mechanism is molecular mimicry between synovial and viral antigenic epitopes. It seems that T cells attack the respective antigens in the synovial membranes of adult patients with rheumatoid arthritis after immunization with influenza vaccines. The cascade of cytokine synthesis that follows leads to autoimmune-mediated tissue injury. Probably, host and environmental factors are also involved in pathogenesis. [6] The mechanism of reactive arthritis development after influenza infection needs further elucidation. Our patient had negative rheumatoid factor and was not previously vaccinated for influenza.

Although we present a single case, we concluded that our patient's arthritis was triggered after infection with influenza A virus. This was supported by the following data: Our patient proved to suffer from influenza A infection two weeks prior to onset of rheumatologic manifestations. He proved negative to other viral or bacterial infections related to arthritis development. The affected joints were typical of post-infectious arthritis. The patient responded well to corticosteroid administration. All the above led the authors to suggest that our patient's arthritis represents a rare case of reactive arthritis after influenza A virus infection. To our knowledge this is the first reactive arthritis reported due to influenza virus A in adults.

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