Reactive Arthritis Associated with an Amebic Liver Abscess

Pages with reference to book, From 105 To 106

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Introduction

The term "reactive arthritis" was first introducedby Aho et al. in 1973[1] for conditions following infection in other organs in which no microbial antigens could be isolated from the joint. Rheumatic fever[2], Reiter's syndrome[3] and postenteric reactive arthritis[4] constitute the major clinical forms of the disease. A number of unrelated organisms have been implicated in association with the disease. These include bacteria[5-7] viruses[8,9], protozoa[10-12], fungi[13,14] and helminths 5. The protozoa constitute a rare cause of reactive arthritis. Those reported in association with the disease to produce arthritis and/or arthmlgia include Giardia lamblia, trypanosomes, plasmodia, trichomonas, Babesia microti, Blastocystis hominis and cry ptosporidium[10-14]. We believe that amoebiasis has never been reported in association with reactive arthritis. Here we report a case of reactive arthritis following an amebic liver abscess.

Case Report

A 38 years old male presented with fever, pain in right upper abdomen for 12 days and pain and swelling of both ankles and right knee for4 days. The fever was high grade and continuous and associated with chills and nausea. It started abruptly with severe upper abdominal pain mainly on the right side. Eight days after the onset of illness the patient noticed swelling and pain of both ankle joints. On 9th day similar symptoms appeared in the right kneejoint as well. There were no symptoms related to throat, bowel or urinary tract. Examination revealed a young man of average height andbuilt who was well oriented. His pulse was 100 perminute, temperature 101°F, blood pressure 110/70 mm of Hg and respiratory rate 24 per minute. He was anemic. There was tenderness in right hypochondrium and liver was enlarged 5cmbeneath costal margin with a span of 15 cm. Examination of neurological, respiratory and cardiovascular system was unyielding. Both ankles, elbows and right knee joints were swollen, warm and tender with limitation ofjoint movements. Investigations showed haemoglobin 9. lgm/dl, total leukocyte count 11,000/mm3 with neutrophils 75%, lymphocytes 19%, monocytes 1% and eosinophils 5%. ESR was 130mm for 1st hour, Westergren. Liver function tests revealed total bilirubinof 1.9 mg/dl, ALT44 UIL (upto 44), AST 52 U/L (upto 33), gamma- GT 120 U/L (11-50) and alkaline phosphalase 383 U/L (39-117). Fasting blood sugar, urea, creatinine, electrolytes, urine examination, prothrombin time, semmpmteins, ECG and chest radiographwere within normal limits. Stool examination showed few white blood cells, scanty mucus and cysts ofEntameba histolytica; no trophozoites were seen. Ultrasound of the liver revealed an area of complex echogemcity, measuring 75mnix52mm which upon needle aspiration yielded 320 ml of brown sauce pus. Microscopy of the pus revealed few leukocytes and no tmphozoites. Cultures of blood, stool, urine, liver aspirate and throat swab also yielded no growth. Echocardiography of the heart was normal. Indirect hemagglutination (IHA) titre for amebiasis was 1:1024(1:32). Serum uric acid was 4.5 mg/dl, ASO titre 200 Todd U/L (50-250), rheumatoid factor (RF) positive with a titre of 1:16 (>1:80 is significant), ANA negative and anti-dsDNA within normal range. Radiographs of ankle, knee and elbow joints revealed no abnormality. Examination of right knee joint aspirate revealed thick light yellow fluid having 4000 WBC/mm3 with 70% PMN's and a glucose of 70 mg/dl; fluid culture was negative. Patient was given meteronidazole 800 mg thrice daily for 14 days and NSAJDs. The joint symptoms disappeared within

three days and liver abscess diminished rapidly in size; no ultrasonographic evidence was present at 6 months. Patient was perfectly healthy on follow-up at 18 months.

Discussion

Amebiasis is endemic in Pakistan. Commonly it presents as intermittent diarrhea or dysentery; extraintestinal manifestations are not uncommon. Liver abscess may develop abruptly with acute fever, right upper abdominal pain and polymorphonuclear leukocytosis (PMN); right dome of diaphragm may be elevated on chest radiograph. Ultrasound scanning of liver is safe and reliable method of confirming the cystic nature of the lesion. Serologic tests are positive in over 90%of cases[16]. IRA titre of >1:256 is sensitive and specific[17]. Reactive athritis refers to acute non-purulent arthritis complicating an infection elsewhere in the body. It usually follows intestinal or urogenital infections. Pathogens commonly triggering the disease include Shigella, Salmonella. Yersinia and Campylobacter in the bowel and Chiamydia in the urogenital tract[18]. The disease is most common in individuals between 18 to 40 years of age and affects both sexes almost equally[4]. Typically it starts as an abrupt onset febrile oligoarthritis principally affecting knees and ankles[2]; wrist, fingers and toes come next in frequency[5,19,20]. Usually some evidence of an antecedent infection 1-4 weeks before the onset of symptoms is present. Urogenital, mucocutaneous and ocular involvement may occur. Reactive arthritis in not known to occurwith amebiasis. In our case no previous or subsequent (upon 18 months follow-up) histoiy of joint involvement, absence of typical features of rheumatoid arthritis, 8 days interval between the symptoms of liver abscess and arthritis, low titres of RF, dramatic response to therapy and high titres of IHA for amebiasis made us to make this diagnosis. The RF was positive in our case, though in low titres. Many parasitic diseases (e.g., malaria, shistosomiasis, trypanosomiasis, filariasis) are known to be associated with positive RF in a small proportion of cases[17,21].

The pathogenesis of reactive arthritis is not known, although both humeral and cellular immune mechanisms participate. Occurrence of the disease with such diverse types of organisms, strong association with HLA B27, isolation of bacterial antigens from synovial fluid, immune complex formation and autoantibody response (molecular mimiciy) are the facts known yet. Few points regarding amebiasis deserve mention. Specific humeral antibodies are found in carriers of pathogenic zymodenes of E. histolytica, cell mediated immunity, though incomplete, develops particularly after invasive liver disease. There are reasons to believe that amebic hepatitis which occasionally occurs in association with amebic colitis perhaps does not occur secondaiy to spread of trophozoites from the intestine, but is rather a non-specific accompaniment of amebic colitis[16]. Finally, immune complex glomerulonephritis has been reported with amebic liver abscess[22]. These findings merely suggest that invasive amebiasis has a potential to behave like a systemic disease, but the relevance of these findings to reactive arthritis is not known.

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