Letter to the Editor

Reactive Arthritis Caused by Blastocystis hominis

To the Editor,

Reactive arthritis is defined as an acute arthritis caused by enteric or genitourinary infection. This infection usually precedes arthritis by a month. The most frequent reactive arthritis due to Salmonella, Shigella, Chlamydia, Versinia and Campylobacter. However, there are cases described in the literature of reactive arthritis caused by other microorganisms, such as protozoa.1

Currently there are no validated criteria for the classification or for the diagnosis of reactive arthritis. According to the working definitions of the Third International Workshop on Reactive Arthritis, there are two clinical features that are considered very important2:

1. Acute inflammatory arthritis, inflammatory back pain or enthesitis.
2. Evidence of infection preceding the disease between 4 and 8 weeks.

Here we report a case of reactive arthritis due to Blastocystis hominis in immunocompetent patients.

A 45-year-old Spanish woman, with no toxic habits, who had made multiple trips to places like India, Nepal, Kenya, Tanzania and Burma was seen. Her last trip to Egypt was made a year ago. Among her medical history featured a recurring tapeworm infection and isolation of Blastocystis hominis (B. hominis) in a control stool. She consulted for malaise, and joint and back pain accompanied by mild fever of 2 days duration. The latency period from the isolation of B. hominis to the onset of symptoms was 10 days. Physical examination showed fever (38°C), monoarthritis of the left knee, heel pain and pain with redness of the eye. The rest of the examination was normal, including oral and genital mucosa. The chest radiograph showed no abnormalities. Chest and hand X-rays were normal. Knee arthrocentesis was performed, resulting in inflammatory synovial fluid: 21 250/mm³ leukocytes (94% polymorphonuclear) and glucose: 143.4 mg/dl. The examination of joint fluid under a polarized optical microscope showed no birefringent crystals. Joint fluid and blood cultures, urine cultures, stool cultures were negative for bacteria and parasites. ESR was 67 mm at 1 h, she had 15.8 x 10⁹ leukocytes (88% neutrophils), hemoglobin 12.2 g/dl, hematocrit 35%, CRP: 56 mg/l and fibrinogen 601 mg/dl. Liver and kidney functions were normal. Serologic testing for hepatitis B and C, HIV, Salmonella, Brucella, and Treponema pallidum were negative. Antinuclear antibodies and rheumatoid factor were negative. HLA-B27 was positive. Ophthalmologic examination revealed bilateral anterior uveitis, predominantly on the right eye. Reactive arthritis due to B. hominis was diagnosed, and treatment with metronidazole 250 mg every 8 h was continued, which she had begun beforehand. In addition, indomethacin was started 125 mg every 24 h, with resolution of arthritis. The patient was intolerant to metronidazole and tinidazole and was changed to 1000 mg every 12 h. Upon discharge, the patient had resolution of arthritis and improvement of uveitis. He continued with indomethacin and tinidazole. Subsequently, the patient came for a control visit, with improvement of arthritis, which allowed her to gradually reduce the anti-inflammatory dose. Further episodes of uveitis was controlled with topical treatment. Stool cultures were repeated at three weeks until deworming was positive. Subsequently, the patient underwent another round of treatment, repeated cultures after a month, which were negative.

Protozoal reactive arthritis is rare. The microorganisms most frequently implicated are Giardia lamblia, Entamoeba histolytica, Trichomonas vaginalis, Toxoplasma gondii, and rarely, B. hominis. This is a protozoa that is transmitted between animals and humans by ingestion of cysts in water or food contaminated by a carrier. Its pathogenic role is not universally accepted, but has been associated with episodes of diarrhea and extraintestinal manifestations, and its relationship with reactive arthritis is less common. Different morphological and ameboid vacuolar types have been described, which could be related to its pathogenic capacity.

The mechanisms by which different parasites can cause joint disease are multiple. For example, local invasion from neighboring bones or muscles, via the blood or lymphatic with the presence of adult individuals, larvae or eggs in the joint cavity. They could also trigger a reactive inflammatory reaction to the presence of the parasite in the surrounding tissue, without an actual joint invasion. In our case, we assume that the latter was the most likely mechanism of action of B. hominis.

On the other hand, some authors use the term “parasitic rheumatism” in the case of inflammatory conditions without the presence of the parasite in the joint or in its vicinity, probably triggered by an immune mechanism.5

In a 1990–2010 PUBMED literature review (keywords: Blastocystis hominis and arthritis) we found 4 cases of arthritis associated with B. hominis, treated in 2 cases as infectious arthritis.6,7 The other 2 cases were reactive arthritis associated with B. hominis.8,9 This case would be the fifth related to this parasite and the third description of reactive arthritis due to this organism.

As in other cases, this was a middle-aged, immunocompetent woman, who had traveled to risk countries. However, unlike the cases described above, this case provided had a positive HLA-B-27.

References


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