Case Report

Arthritis Predating Inflammatory Bowel Disease

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Abstract
Polyarthritis can occur in many Gastrointestinal diseases and 6 to 46 percent of patients with Inflammatory Bowel Disease, but usually along with the active disease or later. Only in one to two percent of people arthritis may predates IBD. As there will be no symptoms of Gut inflammations it is difficult to hook them together. Here we introduce a case who presented with peripheral arthritis and stiff back. Progress of the disease to the point of presenting as IBD detailed. Several types of arthritis that are associated with IBD is discussed. Management of arthritis associated with IBD briefed.

Keywords: Peripheral arthritis; predates; inflammatory Bowel disease; NSAID; Sulphasalazine

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Introduction

Arthritis is a frequent extraintestinal manifestation of Gastrointestinal diseases of varied etiology. Behcet's disease, Reactive arthritis, Whipple's disease, celiac disease, Parasitic rheumatism, Pseudomembranous colitis, Intestinal bypass arthritis and Inflammatory bowel disease are known to present with arthritis. Arthritis may occur in 6 to 46 percent of patients with IBD [1]. Arthritis is more common in Crohn's disease than Ulcerative colitis [2]. In both the conditions it is more common with large bowel involvement and in the presence of abscess, perianal disease, uveitis, stomatitis, erythema nodosum, massive haemorrhage and pyoderma gangrenosum. It affects males and females equally and children are at the same risk. Subclinical Gut involvement is documented endoscopically up to two thirds of patients with the spondyloarthropathy. Three types of arthropathies have been described, spondylitis, peripheral arthritis type 1 & 2. Spondylitis may occur up to 26% of patients of IBD, usually presents with stiff back often relieved by exercise, do not parallel with Gut inflammation and may occur in association with type I peripheral arthropathy. Type I peripheral arthropathy usually is acute, pauciarticular often parallel with Gut inflammation. In up to 90% of patients it resolves within 3 months and does not deform a joint. Joint symptoms may predate IBD in this type. Type II arthropathy is chronic, polyarticular, MCP is frequently involved, exacerbations and remissions may occur for years and commonly do not predate the IBD [3]. HLA-B27, DRB1*0103 is frequently found in the patients with sacroiliitis and type I peripheral arthritis, in contrast HLA-B44 is frequently linked with type II peripheral arthritis. No pathognomonic test exists for these arthritis and inflammatory markers do not differ from those of IBD. The Radiographs of the spine may resemble ankylosing spondylitis, and of peripheral joints is without erosions and destructions. Efficient treatment of IBD may alleviate the symptoms of peripheral arthritis but do not slow down the radiological progression of spondylitis. NSAIDs are effective but should be used withprecaution as it may flare up the IBD. Sulfasalazine, methotrexate, local or systemic steroids, are the drugs that of help for IBD and arthritis. TNF Alpha antagonists have proven to be of use in spondyloarthropathy, but mesalamine has no effect on the joint inflammation.

Case presentation

23 years aged male patient was referred to us by an orthopedic surgeon for polyarthralgia of two months duration. He had pain of both shoulders, knees, ankles and back. He also experienced mild pain of the hands and feet. He suffered a fever for the last five days. His the most troubled problem was the stiffness of back and he had to toss in the bed to get up. Clinical examination revealed cachectic, agitated patient with angular stomatitis. He was unable to bend or reach his knees on the examination couch. But there was no sign of inflammation in any articulation. He was febrile temperature was 38°C, Pulse was 100 per minute, BP was 120/80 mmHg. Systemic examination did not unveil a significant abnormality. HB: 9.4, TWBC: 13,300, P 72 L20 E 8, PLAT: 7,63,000, ESR :102/ 1hr.FBS: 77, S.Cr: 0.8, LFT: normal, Widal: neg, HIV: neg, Hbsag: neg, Mantoux: neg, ANF: neg, R.Factor : neg, CRP (+): 2.4 mg, Urine exam: alb (-), micro 6-8 pus cells, RBC( -), Alk phosphatase: normal. No abnormalities detected on radiological
examination of bones and joints. In the view of above clinical findings and investigation reports we suspected seronegative spondyloarthropathy. We consulted a Rheumatologist who also had opined the same. He was started on Prednisolone 60 mg a day and NSAID orally. He responded well to the treatment and got complete relief from joint and back pain. He could walk about normally. He was discharged home and didn’t turn up for follow up as he considered he was cured of the disease. He was brought to our hospital two months later with complaints of repeated vomiting, pain abdomen and loose motions of two weeks duration. Clinical examination found a dehydrated patient with multiple oral ulcers, mild distended abdomen tender in left iliac fossa. Ultrasound examination of the abdomen was asked for, which revealed fluid and air filled bowel loops in the peritoneal cavity with thickened bowel loops in the left lumbar region. Severe esophagitis, gastritis and inflammatory narrowing of pyloric opening was found on upper GI endoscopy. Sigmoidoscopy unveiled pancolitis suspicious of IBD (Fig1). Multiple biopsies were examined histopathologically, confirmed the diagnosis of ulcerative colitis (Fig 2). He was started on Prednisone 60 mg and Sulphasalazine 1.5 grams a day, responded well to the treatment and got relief from GIT symptoms within three months. Arthritis did not recur for the last one year.

Fig. 1 Sigmoidoscopy: Inflamed, ulcerated colonic mucosa suggestive of Inflammatory Bowel Disease
Discussion

Our patient presented with polyarthralgia and very significant stiffness of back restricting his mobility. He has no symptoms of the gastrointestinal tract. Serological tests for arthritis were negative. His hemoglobin was low, platelets were increased in numbers and ESR was grossly elevated. We suspected seronegative arthritis in the view of his symptoms. We did not have the hint of gastrointestinal involvement. Arthritis is a common extraintestinal manifestation of IBD. This arthritis also can be grouped under seronegative arthritis. In 2 percent of patients, arthritis may predate IBD, posing diagnostic difficulties [4]. Our patient presented with significant spinal symptoms and peripheral arthralgia signifying the presence of type I peripheral arthritis along with spondyloarthropathy, a combination known to occur. Their association with HLA-B27 was well documented. Peripheral arthritis type I was acute and self-limiting as in our patient. He went home stopped medications as he felt completely cured notifies transient nature of illness. Unexplained anaemia, aphthous stomatitis, persistently elevated Inflammatory markers even after resolution of arthritis may give a hint to underlying Inflammatory bowel disease. NSAIDs are the drugs of choice in these arthritis to relieve the pain and inflammation of the joints, COX-2 inhibitors are preferred choice as they are known not to induce flare in IBD [5]. Corticosteroid injection into the inflamed joint can be attempted if involvement is monoarticular, systemic steroids if patient and do not tolerate other drugs. Sulfasalazine, the first drug used in ulcerative colitis is useful in relieving the symptoms of mild to moderate peripheral arthritis. Methotrexate, a preferred DMARD for Rheumatoid arthritis was also tried in IBD related peripheral arthritis successfully, in spite of lack of control studies. NSAIDs, specially naproxen and COX-2 inhibitor are chosen for pain relief in spondyloarthropathy along with physical measures. DMARDs like Sulfasalazine, Methotrexate, Azathioprine have a very marginal effect on IBD associated spondyloarthropathy. Introduction of TNF Alpha antagonist has revolutionised the management IBD and its extraintestinal manifestations. This is the drug of choice in spondyloarthropathy associated with IBD, as it may modify the course of disease [6].
Conclusions

Polyarthritis is a frequent extra intestinal manifestation of Inflammatory Bowel disease. But on rare occasion may predate IBD gives a diagnostic dilemma. All the inflammatory markers that are elevated in IBD are also so in arthritis denying a clue. Persisting anaemia and inflammatory markers, weight loss and aphthous stomatitis in the quiescence of arthritis, may raise suspicion of underlying gut inflammation. Treatment of IBD usually suffices management of arthritis, but the choice of NSAIDs must be selective.

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References
