Thrombosis, an Early Presentation and Extensive Spondylosis, a Late Complication of Inflammatory Bowel Disease

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Inflammatory bowel disease(IBD) comprises two major disease entities, ulcerative colitis and Crohn's disease, that may be complicated by a variety of extra-intestinal manifestations. We reported a 55 years old male patient presented by leg thrombosis years before developing florid intestinal manifestations of IBD. After a detailed history taking and investigations, the patient proved to have ulcerative colitis with extensive spondylosis of the spine and had thrombocytosis. The condition of the patient improved on medical therapy for ulcerative colitis plus anti-platelet drug.

INTRODUCTION

Inflammatory bowel disease (IBD), comprise two major disease entities: ulcerative colitis (UC) and Crohn's disease (CD). IBD is associated with a variety of extraintestinal disorders, including thromboembolism (TE), arthritis, ankylosing spondylitis,uveitis, pyoderma gangrenosum, and erythema nodosum[1].

CASE PRESENTATION

A 55 years old male patient presented to the outpatient clinic with dizziness, fatigue, weakness, mild attacks of haematochezia. On detailed history taking the patient gives troublesome since 1996, when he developed severe pain in the left lower limb below the knee and diagnosed as thrombosis, 6 months later the patient developed numbness and tingling in both lower limbs and the patient improved after hospital admission , 2 months later he experienced the same complain, he consulted a neurosurgeon , he was examined by MRI and diagnosed as having L2-3 disc prolapse and operated upon, when the patient visited our outpatient clinic due to the previous non-specific symptoms he was investigated by CBC that showed HB: 6 gm/dl (microcytic hypochromic), WBCs :18x10³/dl (mainly neutrophils), platelets :1066x10³/dl, reticulocytic count (R.C): 3%, serum iron : 53.7 ug/dl, serum ferritin: 11.4 ng /ml, bone marrow aspiration was hypercellular with increased megakariocytes series in immature and dwarf forms, while myeloid and erytheroid series were normal, kidney functions were normal, liver functions apart from low albumin level (2.8 gm/dl) were normal, ESR>100, stool analysis showed pus and mucus, urine analysis was normal, US examination revealed mildly enlarged homogenous liver, CT scan detected enlarged para-tracheal lymph nodes. The patient was discharged to be followed and prescribed aspirin 150 mg daily as antiplatelet aggregation drug. On revision the patient was still complaining of small recurrent attacks of haematochezia and still had high platelet count, decision was taken to examine the patient

by colonoscopy, findings revealed interno-external piles, severe inflammation and ulceration seen all over the rectum, descending and transverse colon, multiple biopsies were taken for histopathological examination that revealed microscopic picture of ulcerative colitis (focal surface sloughing, the glands showed goblet cell depletion and reduced mucous secreting activity with early crept abscesses formation with laminal edema and inflammation). The condition of the patient was controlled by blood transfusion, antibiotics, salazopyrine 500 mg 2 tablets/ 8 hours, prednisone 60 mg/day that later tapered gradually over 2 weeks. One month later the patient suddenly stopped salazopyrine and experienced bleeding per rectum and abdominal pain, upon giving the original dose salazopyrine the condition controlled. Few months later the patient admitted due to exacerbation in the form of severe abdominal pain and distension, dizziness, tingling and numbness of both lower limbs, the condition was controlled by antibiotics, steroids and salazopyrine, CBC showed mild polymorphonuclear leucocytosis ($11.5x10^3$), mild microcytic hypochromic anemia (10.1g/dl), thrombocytosis ($829x10^3$), kidney functions were normal, serum albumin level was low (1.9 gm/dl), to evaluate the numbness of both lower limbs, lumbar X-ray was done and followed by X-ray examination of the cervical spine both showed sondyltic lesions, osteophytes and calcified disc spaces but no soft tissue pathology was noticed.

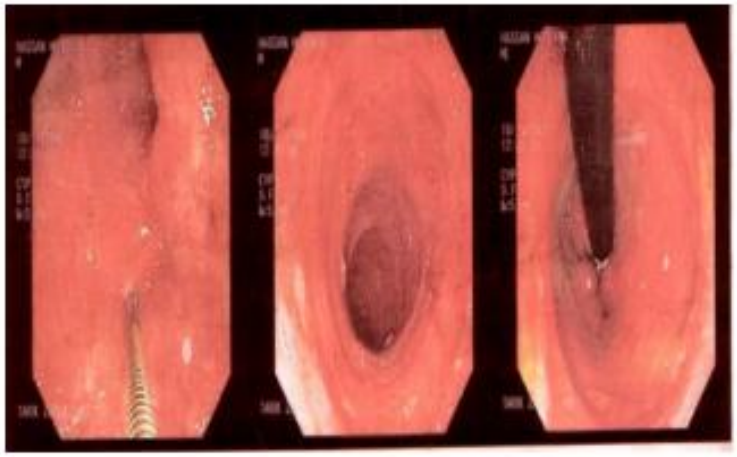

Fig 1: Colonoscopic findings of ulcerative colitis.

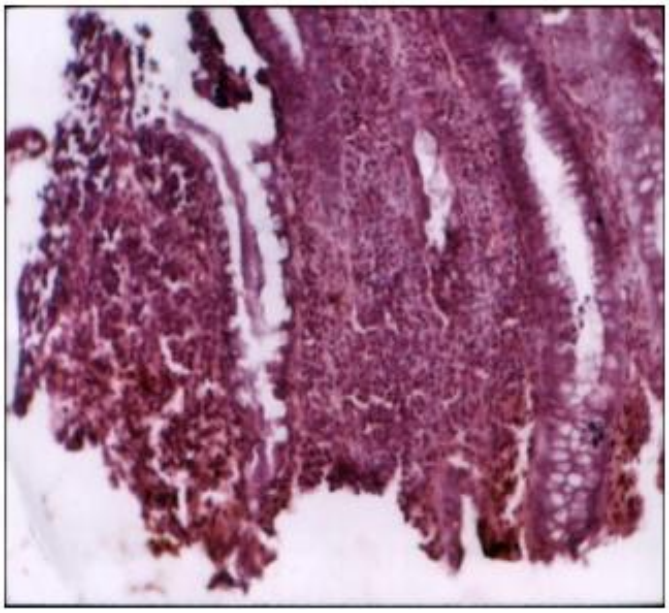

Fig 2: Histopathological findings of UC

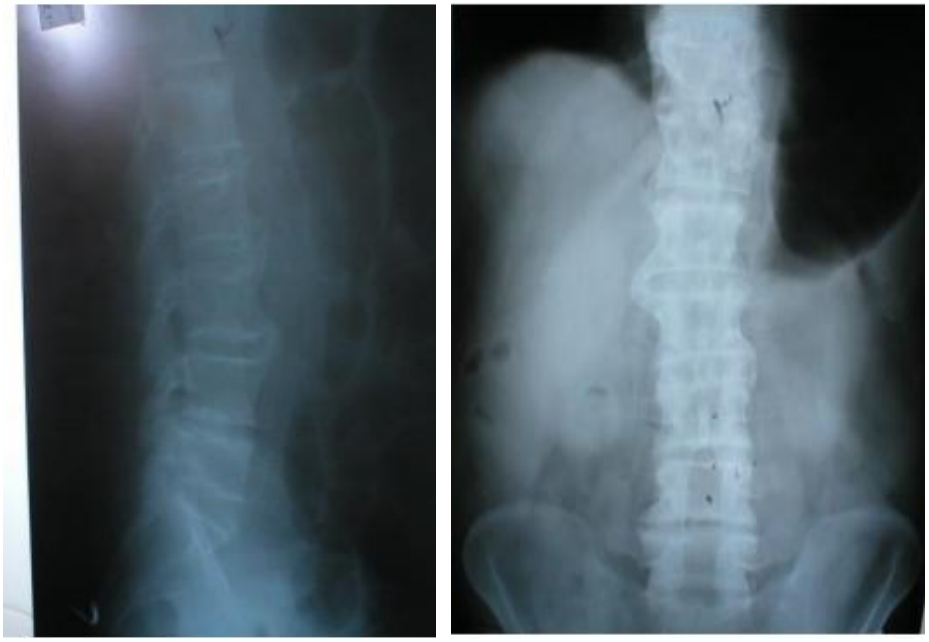

Fig 3: X-ray lumbar spine shows extensive spondylosis.

DISCUSSION

Prevalence of TE in IBD, varies between 1.2% and 6.7% in clinical studies and rising to 39% in postmortem studies. IBD found to be a 3.6 fold higher in the risk of TE compared with controls matched for age and sex. This represents a relevant extra-intestinal complication of IBD, including life threatening pulmonary embolism[2]. The mechanism of enhanced pro-coagulant activity is not understood. Thrombosis in inflammatory bowel disease is important because it occurs in a young population, often in unusual sites, and has a high mortality. The development of thrombosis is related to active inflammatory disease in most patients with Crohn's disease but apparently not in those with ulcerative colitis[3]. In IBD, the platelet count correlates with disease activity, high counts are more likely associated with severe UC than with mild disease[4], this coincide with our case. In our patient thrombocytosis and thrombotic diathesis preceded florid colonic manifestations by a long period of time, this should raise the suspicion of IBD in causes of acquired thrombophillia even in absence of colonic manifestations. Spontaneous platelet aggregation is common with IBD but there is no correlation with disease activity. Patients with IBD have abnormal platelet activity, which may contribute to the inflammatory process[5]. Inflammatory arthropathies are the most common extraintestinal manifestations in IBD patients with a prevalence ranging between 7% and 25% [6]. Articular and musculoskeletal manifesttations are included in the spondyloarthropathies (SpAs) that are a group of seronegative autoimmune related disorders with common characteristics including: ankylosing spondylitis, reactive arthritis, psoriatic arthritis, inflammatory bowel disease, some forms of juvenile arthritis and acute anterior uveitis[7]. Articular involvement (peripheral or axial) can precede, be synchronous or begin afterward the diagnosis of IBD, it is characteristically pauciarticular, asymmetrical, transitory, migrating, prevalently non deforming. This occurred in our patient, where he consulted a neurosurgeon and operated upon for disc prolapse with unsatisfactory improvement. The axial involvement can vary from asymptomatic sacroiliitis to inflammatory lower back pain to ankylosing spondylitis (that occurs in 3% of IBD patients)[6]. Ankylosing spondylitis (AS) affects the vertebral column by progressive ankylosis of the vertebral facet joints and the sacroiliac joint. The prevalence of AS in IBD (1%-6%) is higher than in the general population (0.25%-1%)[8]. Bacteria and gut inflammation seem to play an important role in the pathogenesis of AS. Interestingly, ileocolonoscopy in patients with idiopathic spondylarthropathies reveals ileal inflammation in more than two thirds of patients[9]. The

clinical course of AS in IBD is similar to idiopathic AS, and disease progression leads to increasing immobility of the spine resulting in ankylosis (bamboo spine). Secondary to reduced chest expansion, poor lung expansion with fibrosis and dilatation of the aortic root can occur. AS is associated with peripheral arthritis in about 30% of patients and with uveitis in 25% of patients[10]. Although in our patient mobility disorders were evident, no pulmonary or aortic damage were seen.

In conclusion: Thrombocytosis and thrombosis may precede colonic manifestations and add to the risk of TE in IBD, while extensive spondylosis may complicate IBD and results in marked disability.

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