Gastrointestinal Histoplasmosis Mimicking Abdominal Tuberculosis

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Abstract

We report an elderly male who presented with history of chronic diarrhoea. The patient underwent colonoscopy and CT scan of the abdomen which strongly suggested tuberculosis; however histopathology showed presence of budding forms of Histoplasma capsulatum. The patient was started on oral itraconazole on which he improved remarkably. ©

INTRODUCTION

Gastrointestinal histoplasmosis (GIH) is an uncommon condition with protean manifestations. Gastrointestinal involvement occurs due to haematogenous seeding of the gastrointestinal tract or as a result of mediastinal involvement. It affects both immunocompetent and immunocompromised patients. It needs to be considered in the differential diagnosis of malignancy, inflammatory bowel disease and intestinal tuberculosis. GIH has excellent long term survival with aggressive therapy.

CASE REPORT

60 year old male, an ex smoker, was admitted in February 2008 with complaints of loose motions since 6 months. The patient used to be a farmer in Uttar Pradesh and was residing in Mumbai since 5 years. He complained of watery stools 7 – 8/day, without blood or mucus, 1 – 2 hours after meals. There was no history of bleeding per rectum or abdominal pain or lump. Occasionally he complained of passage of undigested food, and aggravation of symptoms on eating spicy food. He had developed pedal edema since 2 months. There was history of stomatitis and glossitis. He complained of low grade fever and generalized weakness.

In October 2007, the patient was seen in plastic surgery department at our hospital for a skin lesion (rodent ulcer) over the face which was surgically excised completely. Histopathologic diagnosis was basal cell carcinoma.

On examination, he was cachexic, afebrile with pulse rate of 78/min and BP of 120/80 mmHg in right upper limb. He had Gr.II clubbing and bilateral pitting edema upto the knees. He had stomatitis and glossitis. Per abdomen there was mild hepatomegaly, no lump was palpable. Rest of the examination was normal.

The patient was investigated with the differential diagnoses of inflammatory bowel disease, intestinal tuberculosis, malignancy or immunocompromised state. Investigations showed hemoglobin 10.1g/dl, total white cell count 6700/mm³, differential count-86 polymorphs, 14 lymphocytes, platelet count 3.34 lacs/mm³, MCV 81.8 fl, MCH 25.7 pg and ESR of 20 mm/hr. Blood sugar fasting was 96mg/dl , post lunch 108 mg/dl. Blood urea nitrogen was 10mg% and creatinine 0.7mg%, T.bilirubin 0.3mg/dl, SGOT / PT were 25/16 IU/ml (normal value 5-40 IU/ml), serum Na/K were 133 and 4.8 mEq/L respectively, alkaline phosphatase was 581 IU/L (normal up to 280 IU ) and LDH was 430 U/L. HIV, HBV and HCV serology was negative. CD4 count was 550/mm³. Chest X ray, ECG and 2D Echo were also normal. Stool routine showed plenty of pus cells, without occult blood. Stool culture including fungal culture was negative. Urine protein was negative. USG abdomen showed abdominal lymphadenopathy (preaortic paraaortic, precaval, and mesenteric lymph nodes) and minimal fluid. The patient underwent colonoscopy which revealed edematous colon with nodular lesions. Ascending colon showed circumferential thickening, caecum was nodular and edematous. Biopsy was taken from these lesions. The impression was multifocal tuberculosis, to rule out malignancy. Carcino embryonic antigen (CEA) was 1.45 ng /ml (normal 0.37 - 3.3ng/ml).

CT abdomen showed circumferential asymmetrical mural thickening of ileoacaecal junction, caecum, ascending colon, splenic flexure, descending colon with multiple sub centimeter sized peripancreatic, gastric and para aortic lymph nodes. Liver, spleen, kidneys, adrenals, pancreas, gall bladder, urinary bladder and prostate were normal.

Histopathology revealed focal cryptitis with multiple budding yeast forms resembling Histoplasma capsulatum
which stained positively on Periodic acid Schiff (PAS) stain involving large intestine and ileum. Abdominal lymph node fine needle aspiration cytology also showed histoplasma on Papanicoleau stain (Fig. 2).

CT chest showed small peribronchovascular nodules with tree in bud appearance, bilateral pleural effusion and mediastinal lymphadenopathy. The slides of skin ulcer were reviewed for presence of Histoplasma. It was negative.

The patient was started on oral itraconazole in the dose of 200 mg twice a day. There was a dramatic response with a quick change in consistency of stools followed by decrease in frequency within 1 week. The patient was discharged and was asked to continue itraconazole for 6 months. He was asymptomatic at last follow up in June 2008. USG abdomen and laboratory parameters repeated at the time were normal. The patient has returned to his native place and reported to be very well when telephonically contacted in October 2008.

DISCUSSION

Inhaled Histoplasma microconidia are transformed into yeast phase in the lungs. The yeasts are translocated to the local draining lymph nodes with the help of phagosomes from where they disseminate hematogenously. Gastrointestinal involvement occurs in 50–70% of cases of histoplasmosis due to hematogeneous seeding of the gastrointestinal tract.1 Gastrointestinal histoplasmosis (GIH) can affect both immunocompetent and immunocompromised individuals especially patients with acquired immune deficiency syndrome (AIDS).2 Of the several case series of GIH, largest identified 77 cases, three quarters of whom were immunocompetent.1 Besides AIDS, other causes of dissemination are extremes of age, immunosuppression, idiopathic CD4 lymphocytopenia, Job’s syndrome and recently with biological agents like infliximab and etanercept.1 In our patient age was the only obvious risk factor.

Patients mostly present in the 5th decade of life. Males are more commonly affected. GIH produces symptoms in only 3-12% patients.4 Abdominal pain, diarrhea and weight loss are present in 30–50% patients with GI involvement.5 Though usual manifestations of histoplasmosis are in the form of respiratory illness, with GI involvement pulmonary symptoms are uncommon and GI symptoms predominate. Intraabdominal lymphadenopathy can be seen on CT scan in 2/3rd of patients.

Terminal ileum and caecum are frequently involved. Depending on the layer of bowel wall involved, patients present with bleeding, obstruction, perforation or peritonitis.5

Pathologic spectrum of GI histoplasmosis includes ulcers, nodules, hemorrhage, obstructive masses and occasionally normal mucosa. Microscopic gastrointestinal findings are diffuse lymphohistiocytic infiltration, ulcerations, lymphohistiocytic nodules, minimal inflammatory reactions. Well formed granulomas are rare.6

It is possible that GIH is underdiagnosed. In our patient with involvement of terminal ileum and caecum, presence of strictures, nodules and intraabdominal lymphadenopathy tuberculosis was strongly considered till the biopsy report was available.

We report this case for its rarity and to make physicians aware of the possibility of histoplasmosis in the differential diagnosis of inflammatory bowel disease, malignancy and other intestinal disorders like TB. Co- existence of GIH with GI tuberculosis has also been reported in an HIV patient.7 Importantly GIH has excellent long term survival with aggressive therapy with antifungal drugs. Untreated histoplasmosis can be fatal especially in immunocompromised patients.

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