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Anti-tubercular Agent for Intestinal Tuberculosis Induced Hepatotoxicity - A Case Report

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INTRODUCTION

uberculosis typically affects the lungs, but may involve other sites which referred as extra pulmonary TB. Intestinal tuberculosis is still rare disease in India and its related case reports are seems to be rare. About 20-25 case

ABSTRACT

Tuberculosis (TB) is an endemic airborne disease. TB is the 13th leading cause of death and second leading infectious killer after covid 19. In worldwide, 10.6 million people suffering from TB. Abdominal TB cases make up above 3% of all extra pulmonary TB as per the Index TB Guidelines. Abdominal tuberculosis is the type of extra pulmonary tuberculosis that is TB infection of any organ in the abdominal cavity. Intestinal tuberculosis is a rare clinical manifestation of extra pulmonary TB. Intestinal tuberculosis is a rare clinical manifestation of extra pulmonary TB. A 63 year old female patient was admitted with complaints of fatigue for one week. She has fever for one month on and off with significant loss of weight and had recent history of deep vein thrombosis (DVT). Now she was newly detected with type 2 diabetes mellitus too. CECT scan of abdomen and pelvis was performed with finding of wall thickening of cecum and terminal ileum, mild splenomegaly, multiple adjacent enhancing mesenteric nodes, multiple enlarged enhancing conglomerated para aortic, retroperitoneal and common iliac nodes, multiple short discontinuous segments of bowel wall thickening with luminal narrowing involving ascending, transverse and descending colon. A detailed examination of colonoscopy concluded intestinal tuberculosis. Although Expert MTB/RIF ultra-extrapulomonary showed MTB not detected as well as the rifampicin resistance not detected. MTB treatment was initiated. After the drug had taken which resulted in hepatitis that is, ATT induced hepatitis. Therefore the particular drug had stopped and alternative was started. This case points outs the diagnostic challenges of intestinal tuberculosis and commonly caused anti tubercular agent induced hepatotoxicity. In this case it takes time to find out the disease condition and after starting the TB drugs which leads to hepatotoxicity. This further leads to the elongation of the treatment duration.

reports and 2 case series were presented in worldwide. Intestinal tuberculosis is a diagnostic challenge, especially when absence of active pulmonary infection. It may mimic various other disease (commonly crohn's disease). The ileocaecal region is the most common area involvement in the intestinal tract. Clinical manifestations of intestinal TB include fatigue, abdominal pain,







Fig. 1

Fig. 2

Fig. 3

Table 1 : Colonoscopy revealed large multiple ulcers with patulous ileocecal valve

weight loss, anemia and fever with night sweats. The mechanism by which the tubercle bacilli may enter the intestinal tract through the ingestion of infected milk/sputum or hematogenous spread from the primary lungs or direct spread from adjacent organs or through lymph channels from infected nodes. TB peritonitis, Hemorrhage and intestinal obstruction are the recognized complications of intestinal tuberculosis. This case contributes the prevalence of intestinal tuberculosis in southern region of India and increased occurrence of drug induced hepatotoxicity.

CASE REPORT

A 63 year old female patient was admitted with complaints of fatigue for one week. She has fever for one month on and off with significant loss of weight and had recent history of deep vein thrombosis (DVT). Now she was newly detected with type 2 diabetes mellitus too. General examination revealed conscious, febrile (101°F) and soft abdomen. Her laboratory investigation showed a variation in hemoglobin (10.8 mg/dl), sodium (127 and 129 mmol/L) and potassium (2.4 mmol/L). On peripheral smear test, smear showed dimorphic anemia (Normocytic, normochromic to microcytic hyperchromic). CRP and ESR levels were elevated.

Dengue antibody IgM, typhoid M, Scrub typhus IgM, rapid malarial test, Widal test, Flu real time PCR panel were all

negative. Expert MTB/RIF ultra-extrapulomonary showed MTB not detected as well as the rifampicin resistance not detected and urine analysis pus cells were slightly elevated there was no significant bacteriuria. Here USG Abdomen and pelvis showed Grade I fatty liver. On examination of cardiovascular system there was abnormality detected (sinus tachycardia and right ventricular hypertrophy).

CT chest revealed bilateral diffused distribution of centrilobular nodules with tree-in-bug pattern and focal patchy central and peripheral areas of found glass densities in lower lobe and CECT scan of abdomen and pelvis was performed with finding of wall thickening of cecum and terminal ileum with thickened ileocecal valve and pericolonic fat stranding, mild splenomegaly, multiple adjacent enhancing mesenteric nodes, multiple enlarged enhancing conglomerated para aortic, retroperitoneal and common iliac nodes, multiple short discontinuous segments of bowel wall thickening with luminal narrowing involving ascending, transverse and descending colon. A detailed examination of colonoscopy concluded intestinal tuberculosis. The pathobiologic assessment confirmed granulomatous colitis on caecum and active ileitis with ulceration.

Initially patient was started with antibiotics like Inj.

Table 1

TESTS	D1(after the drug given)	D4	D7	D11	D13
T. Bilirubin (mg/dl)	1.14	2.16	2.24	3.32	1.68
D. Bilirubin (mg/dl)	0.55	0.52	1.65	2.52	0.98
AST (U/L)	89	97	229	231	106
ALT (U/L)	62	79	144	111	72
ALP (U/L)	526	405	314	435	380
T. Protein (g/dl)	5.4	5.2	4.8	5.6	4.9
Albumin (g/dl)	2	1.9	1.8	1.6	1.7
Globulin (g/dl)	3.6	3.6	3.4	3.2	3.7

CEFOPERAZONE AND SULBACTAM 1.5mg, Inj. MEROPENEM 1gm, POTKOLR syrup for declined potassium levels and after verifying the impression of CECT and colonoscopy, anti-tuberculous agents (Tab. AKT-4) and PYRIDOXINE was started. There was marked elevations in LFT parameters after the drug had taken which resulted in hepatitis that is, ATT induced hepatitis. Therefore the particular drug had stopped and alternative (Inj. STREPTOMYCIN, Tab. ETHAMBUTOL & Tab. LEVOFLOXACIN) was started. She had hospitalized for one and half months and finally the patient got symptomatically improved and discharged.

DISCUSSION

Intestinal Tuberculosis can be misdiagnosed as many other inflammatory condition. MTB culture is considered as the gold standard diagnostic test but this case revealed a negative result due to its long incubation period of bacteria. Intestinal TB can be of three forms; hypertrophic or ulcero hypertrophic and fibrous stricturing. For this case, it was confirmed ulcerative form from CECT scan report. The points which favor the diagnosis of intestinal tuberculosis are loss of weight, anemia, elevated ESR, CRP and hepatic function tests, CECT showing wall thickening of cecum and terminal ileum, mild splenomegaly, enhancing mesenteric nodes and bowel wall thickening with luminal narrowing in colon, colonoscopy and biopsy revealing intestinal tuberculosis. After starting the antituberculous agents patient had experienced ATT-induced hepatitis and this made a way to add alternatives. She had hospitalized for one and half months and was discharged. Certain studies declared that there is an option to prevent hepatotoxicity by introducing herbal formulation with anti-tubercular agents such as Curcuma longa (Turmeric). It showed an improved disease outcome without any sideffects or toxicity.

CONCLUSION

Intestinal tuberculosis is an uncommon clinical manifestation of extra pulmonary tuberculosis. Here the patient presented with symptoms of fatigue for one week, fever for one month on and off. The colonoscopy and histopathological findings could help to confirm intestinal tuberculosis. After starting the anti-tuberculous agents the patient had experienced ATT- induced hepatitis. So the particular drug had stopped and alternative was started.

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CONFLICT OF INTEREST

The authors declared that they do not have anything to disclose regarding the conflict of interest.

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