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Primary duodenal tuberculosis presenting as gastric-outlet obstruction: Its diagnosis

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ABSTRACT

Introduction: Gastrointestinal tuberculosis often involves the ileocecal region. Duodenal and gastric tuberculosis found in only 1% of patients suffering from pulmonary tuberculosis with associated HIV infection in non-endemic areas. Duodenal obstruction due to tuberculosis is very rare and needs high index of suspicions for diagnosis. Mostly this entity is suspected on intraoperative findings. In this manuscript we emphasized on ways and means for establishing histopathological diagnosis before starting anti-tubercular treatment in such cases. **Method and Material:** All patients of suspected gastroduodenal tuberculosis presented with feature of gastric-outlet obstruction managed during Jan 2009 to June 2011 were included in the study. After proper evaluation (routine hematological and biochemical examination, microbiological examination, serological and endoscopic evaluation) exploratory laparotomy was done and if there is no mesenteric lymphadenopathy or it is not safe to take biopsy form the diseased duodenum, multiple FNAC were taken from the diseased portion for histopathological and microbiological diagnosis. **Result:** A total of five patients were treated during this period. The most common presentation was vomiting followed by failure to thrive and weight loss; two patients had abdominal pain. Biopsy of mesenteric lymph node was possible in two cases. FNAC from diseases portion was taken in all cases. FNAC showed granulomas in four cases. Cases where even FNAC finding was non-conclusive on HPE/Microbiology was not subjected to antitubercular drug. **Conclusion:** Multiple intra-operative FNAC may be taken from the diseased portion of the duodenum to establish the histopathological diagnosis if diagnosis is not established by any other mean.

Key words: Duodenum, fine needle cytology, tuberculosis

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INTRODUCTION

Gastrointestinal (GI) tuberculosis (TB) often involves the ileocecal region. The stomach and duodenum are rare[1] sites for TB and usually occurs as a result of secondary spread from the pulmonary disease. Duodenal and gastric TB found in only 1% of patients suffering from pulmonary TB[2,3] with associated HIV infection[4] in non-endemic areas. TB may affect any portion of the GI tract, in 85% of cases ileocecal region is affected whereas gastroduodenal region is involve only 0.3-2.3% of TB cases of abdominal TB[2] According to autopsy series the incidence of gastroduodenal TB is only 0.5%.[3] When gastroduodenal TB is seen without any other lesion of TB in body is known as primary gastroduodenal TB, which is very rare and only few cases had been reported in the literature.[1,5-7] Duodenal obstruction due to TB is very rare and needs high index of suspicions for diagnosis. and usually it is suspected on intraoperative findings[8,9] in this manuscript we emphasised on ways and means for establishing HPD before starting anti-tubercular treatment in such cases.

MATERIALS AND METHODS

All patients of suspected gastroduodenal TB presented with feature of gastricoutlet obstruction managed during Jan 2009 to June 2011 were included in the study. A total of 5 cases of duodenal TB were treated at our centre during this period. Patients with associated pulmonary TB were excluded. All patients were evaluated with haematological investigation, biochemical investigation, and upper GI contrast studies followed by upper GI endoscopy. After proper evaluation exploratory laparotomy if there is no lymphadenopathy or it is not safe to take biopsy form the diseased duodenum, multiple fine needle cytology (FNAC) was taken from the diseased portion before gastrojejunostomy.

RESULTS

A total of five patients were treated during this period in our department. Mean age was 5.2 years. Mean duration of symptoms was 85 days. The most common presentation was vomiting followed by failure to thrive and weight loss; only two patients had abdominal pain. In all cases, haematological and biochemical investigation [Table 1] showed mild decrease in haemoglobin level, ESR was border line (ranging between 12 and 26) and serum albumin level was low (ranging between 2.4 and 3.2 g/dl). Upper GI contrast study showed dilated stomach, dilated first or up to second part of the duodenum and minimal flow of the contrast in rest of the intestine (Figure 1 and Figure 2). In all patients on upper GI scope cannot be negotiated into the diseased portion of the duodenum and biopsies as well washing were negative for acid fast bacilli and granulomas respectively. On exploration, all cases had dilated stomach, dilated duodenum up to second part; duodenum was severely inflamed and densely adherent to the underlying structure, there were no periduodenal or mesenteric lymph nodes except in one patient. FNAC taken from the diseased duodenum showed granulomas in four out of five cases. In cases where FNAC finding was negative for granuloma and QuantiFERON-TB Gold test (QFT-G) Gamma assay was negative, were not subjected to antitubercular drug.

In all cases, gastrojejunostomy was done and after their surgical treatments, all patients except the one where FNAC and TBFN-Gamma was negative were given a standard four-drug regime of antitubercular treatment (isoniazid, rifampicin, pyrazinamide and ethambutol) for 4 months followed by an additional 8 months on two drugs (isoniazid and rifampicin).

DISCUSSION

TB of GI tract most often affects the ileocaecal region. Gastro-duodenal involvement accounts for 2.5% of TB enteritis.[8] Rarity of involvement of duodenum may be due to gastric acidity, rapid transit of ingested organisms, and the scarcity of lymph follicles in the gastric wall, and an intact gastric mucosa.[5] Involvement of duodenum may be either extrinsic or intrinsic or both.[8-10] In the extrinsic type there can either be primary duodenal involvement or compression due to enlarged peri duodenal lymph node whereas intrinsic type may present with ulcerative, hypertrophic or

Table 1: Hematological, biochemical and serological investigations

Serial no. of Pt.	Hb. g/dl	TLC × 1000/ul	ESRmm in 1st h	Total bilirubin mg/dl	SGPT/SGOT U/L	HIV	GGT U/L	Serum albumin g/dl	Mantoux test	TBFN-gamma assay
@1+^	9.1	7500	12	0.48	12/19	−ve	17	2.3	Non-reactive	−ve
@2!	8.7	12600	18	0.78	14/19	−ve	27	3.1	Non-reactive	+ve
3+^	10.6	8500	22	1.1	56/47	−ve	12	3.0	Non-reactive	+ve
4+	8.5	11000	26	0.9	28/34	−ve	18	2.4	Reactive	−ve
5+*%	10.2	9600	22	0.7	21/37	−ve	22	2.9	Non-reactive	−ve

PT: Patient, Hb: Hemoglobin, TLC: Total Leucocyte count, ESR: Erythrocyte sedimentation rate, SGOT: Serum glutamic oxaloacetic transaminase, SGPT: Serum glutamic pyruvic transaminase, HIV: Human immunodeficiency virus, GGT: Gamma-glutamyltransferase, TBFN gamma assay: QuantiFERON® – TB Gold In-Tube test (Interferon-Gamma Release Assays – Blood Tests for TB Infection), Indication: @- patients operated at other center, + - Patients in which endoscopic washing and biopsy was done, ^ - Patients in which mesenteric lymph nodes were present

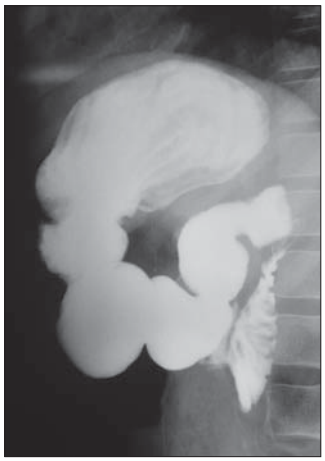


Figure 1: Dilated stomach with dilated 2nd part of duodenum case-1

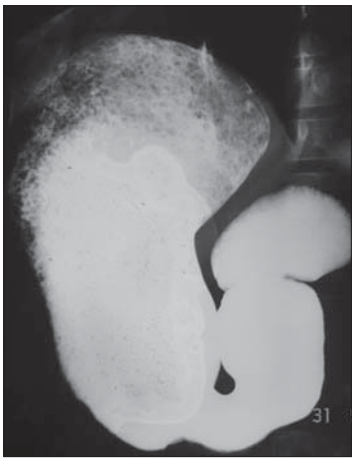


Figure 2: Dilated stomach with dilated 2nd part of duodenum

ulcerohypertrophic lesions.

Proximal duodenum is the commonest site involvement in cases of duodenal TB.[3,9,11] In most of these cases on exploration; stomach is grossly dilated, pylorus appeared normal, duodenum grossly dilated, hypertrophied and inflamed with or without peri-duodenal or mesenteric lymph nodes. If periduodenal or mesenteric lymph nodes are present they can be used for HPD but in cases where there is no mesenteric or periduodenal lymph nodes, and duodenum is highly inflamed and adherent it is not safe to take biopsy for HPD.

The diagnosis of primary gastoduodenal TB is difficult and is often suspected intra-operatively[3,9] Symptoms of gastro-duodenal TB are postprandial epigastric pain, vomiting, weakness, weight loss, fever, hematemesis.[12,13] Gastoduodenal TB can also present as recent onset of gastric outlet obstruction (GOO) and weight loss[13] with no history of fever or hematemesis and malena making the diagnosis of gastro duodenal TB difficult. On review of literature vomiting was the most common presentation followed by GI bleeding.,[3,9] In our series vomiting and weight loss were the constant finding and none of our patients presented with GI bleed. Differential diagnosis in pediatric age group are malrotation or lymphoma which can be excluded on the basis of associated feature and investigation. Diagnosis of duodenal TB is made by demonstration of either acid-fast bacilli and/or caseating granuloma in the biopsied material.[3,14] At time it is difficult to get any histopathological evidence of TB even on exploration as there are no mesenteric or periduodenal lymph node which can be biopsied and duodenum is grossly inflamed, densely adherent to the adjacent structure making it difficult to get any biopsy material safely for HPD, in these cases we did multiple FNAC for confirming the diagnosis. Though it is possible to take such FNAC under ultrasonographic guidance but since all patients in our series had significant weight loss because of GOO we decided to explore them and by pass the obstruction and establish HDP before starting antitubercular treatment, which is a recommended management[3,9] and secondly we are worried will antitubercular treatment will be effective in presence of significant GOO or not.

Management of duodenal TB is primarily medical and if the diagnosis is made prior to surgery, most lesions improve with appropriate treatment[15] but in majority of the cases diagnosis is made per-operatively and treatment for these cases is gastrojejunostomy along with proper course of anti tubercular therapy Anti tubercular therapy (ATT).[3,4,9,10] Endoscopic biopsy for HPD and feeding jejunostomy along with ATT had been described but effectiveness of delivering drugs via jenjunostomy is doubtful[2]. 0Another group of authors had advised endoscopic biopsy followed by endoscopic dilatation of the diseased portion along with ATT,[16] but in our cases it was difficult to negotiate endoscope in the diseased part of the duodenum hence washing or biopsy from the duodenum was not conclusive. (to establish the diagnosis). In such cases when diagnosis is not established by any other method including radiological investigation and endoscopic biopsy and per-operatively there is no mesenteric or paraduodenal lymphadenitis and duodenum is grossly adherent and inflamed, we did multiple FNAC from the lesion for histopathology along with Roux–en–Y retro colic gastro-jejunostomy. Patients were given a standard four-drug regime of antitubercular treatment (isoniazid, rifampicin, pyrazinamide and ethambutol) for 4 months followed by an additional 8 months on two drugs (isoniazid and rifampicin). In one patient HPD was established by mesenteric lymph node whereas in one case we are not able to establish diagnosis of tuberculosis was not managed with ATT. After completion of ATT patient were evaluated for patency of diseased duodenum which showed free passage.

CONCLUSION

Duodenal TB though rare should be considered in the differential diagnosis of GOO of recent origin in endemic countries like India. Secondly multiple intra-operative FNAC may be taken from the diseased portion of the duodenum to establish the HPD if diagnosis is not established by any other mean.

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