INTRODUCTION

Historically speaking, tuberculosis has been present in the human population since antiquity. Definite signs of tuberculosis have been found in the Egyptian mummies. It is a leading killer causing three million deaths globally, and 62,000 deaths annually in Pakistan.\(^1\),\(^2\) Gastrointestinal tuberculosis is the fourth commonest site in extra-pulmonary tuberculosis, after lymph nodes, bones and kidneys. Hypertrophic pyloroduodenal tuberculosis presenting as gastric outlet obstruction is one of the rarest manifestations of gastrointestinal tuberculosis.\(^3\)

The aim of reporting this case is to create awareness among the healthcare professionals of this unique presentation of gastrointestinal tuberculosis.

CASE REPORT

A 19 years old girl belonging to low-socioeconomic class, presented with history of epigastric pain and discomfort for one year. Pain was mild to moderate in intensity and associated with anorexia, nausea, vomiting, fatigability, loose motions, and low-grade fever (PUO). Vomiting was effortless and projectile, occurring two hours postprandially. Vomitus was non-bilious and contained partially-digested food particles eaten a day or two before. She had lost 10 kg body weight over this period. A wide range of anti-ulcer medications had failed to relieve the symptoms. There was no history of cough, expectoration, hemoptysis, or any other significant past medical or surgical illness.

General physical examination revealed features of anemia, koilonychia, dehydration and malnutrition. Systemic examination was unremarkable. Abdominal examination revealed a large non-tender tympanic mass in the epigastrium. Succession splash was demonstrable on shaking the patient. Digital Rectal Examination (DRE) showed no pathology.

On passing nasogastric tube, about four litres of dirty coloured fluid was aspirated out and the mass got reduced in size, and became soft. Over the next 24 hours, another 3 liters of fluid was aspirated from the stomach, and the abdomen became scaphoid.

Laboratory investigations revealed iron-deficiency anemia with Hb of 8.5 g/dl. ESR was 26 mm after first hour and Mantoux’s test was positive within duration of 15 mm. Serum electrolytes showed sodium 130 mmol/L, potassium 3.0 mmol/L, chlorides 85 mmol/L and HCO\(_3\) 35 mmol/L. Arterial Blood Gases (ABG) analysis showed hypochloremic metabolic alkalosis.

Chest X-ray showed no cardiopulmonary pathology. Abdominal ultrasonography depicted thickening and deformity of pyloroduodenal area with proximal gastric dilatation. Contrast-enhanced CT scan of abdomen demonstrated marked gastric dilatation, pyloroduodenal thickening and narrowing, massive mesenteric lymphadenopathy and minimal ascites. (Figure 1) Barium meal X-ray displayed gross gastric dilatation and pyloroduodenal narrowing (Figure 2). Upper GI endoscopy confirmed gastric dilatation with tight pyloric opening, which was difficult to negotiate with tip of the endoscope. Multiple endoscopic biopsies taken from antral mucosa revealed nonspecific gastritis.

After thorough gastric lavage with normal saline, and correction of fluid and electrolyte imbalance, the patient was explored through upper midline incision. The pyloroduodenal area was found to be markedly congested, erythematous, thickened and deformed, with proximal gastric dilatation. It gave the impression of

ABSTRACT

Hypertrophic pyloroduodenal tuberculosis is a rare cause of Gastric Outlet Obstruction (GOO) often forgotten in the differential diagnosis of gastric outlet obstruction. Since laboratory and radiological investigations often prove inconclusive in reaching the diagnosis of hypertrophic pyloroduodenal tuberculosis, surgery has a key role in the management of hypertrophic pyloroduodenal tuberculosis. Postoperative anti-tuberculosis chemotherapy (ATT) becomes imperative for complete resolution of hypertrophic pyloroduodenal tuberculosis. This case report describes the condition and management in a young girl.

Key words: Hypertrophic pyloroduodenal tuberculosis. Gastric outlet obstruction. Diagnosis.
irresectable pyloroduodenal growth (linitis plastica). There were five ileal strictures at 5, 10, 25, 35 and 40 cm from the ileocaecal junction. Caecum was also boggy and indurated. Massive intra-abdominal lymphadenopathy involving mesenteric, mesocolic, perigastric, celiac, periportal, peripancreatic and paraaortic lymph nodes was found. Minimal amount of serous ascites (about 150 ml) was found in the Pouch of Douglas.

Side-to-side gastrojejunostomy and right hemicolectomy, with primary ileotransverse anastomosis was performed. Multiple juxtapyloric and mesenteric lymph nodes were removed for biopsy. Histopathological examination of caecum, terminal ileum, appendix and lymph nodes showed typical caseating granulomas with epithelioid cells, Langhan’s giant cells and lymphocytes. No evidence of malignancy was found in the resected specimens. Postoperatively, the patient recovered uneventfully and was put on Anti-tuberculous Treatment (ATT). She became symptom-free within a couple of weeks, and gained 10 kg body weight within 4 months. Six months later, she was re-evaluated with endoscopy and barium, which demonstrated a well-functioning gastrojejunal stoma and patent pyloric opening.

DISCUSSION
Gastrointestinal tuberculosis is a universal health problem in the underdeveloped countries, especially in South-East Asia and Africa. With the advent of AIDS epidemic, incidence of gastrointestinal tuberculosis has escalated even in the developed countries. The causative agent enters gastrointestinal tract either by ingestion of contaminated milk (Mycobacterium tuberculosis) or by swallowing of infective sputum (Mycobacterium tuberculosis). Although any part of gastrointestinal tract from mouth to anal verge may be involved by gastrointestinal tuberculosis, the ileocaecal region is the most frequently involved segment of the gut (85%) due to the abundance of lymphoid follicles (Peyer’s patches). Involvement of pyloroduodenal area by tuberculous is exceptionally rare and accounts for only 2.5% of tuberculous enteritis. This is due to bactericidal effects of gastric acidity, rapid transit due to high gastric motility and paucity of lymphoid follicles in the gastric wall.

Morphologically, there are three main variants of pyloroduodenal tuberculosis; ulcerative (60%), hypertrophic (10%) and ulcerohypertrophic (30%). It is the hypertrophic variant of pyloroduodenal tuberculosis which primarily causes gastric outlet obstruction by inducing exuberant fibroblastic and inflammatory reaction in the wall of the pyloroduodenal area. However, extrinsic compression caused by the matted juxtapyloric tuberculous lymph nodes, although less important, is another mechanism for production of gastric outlet obstruction. Both pyloroduodenal fibrotic thickening and extrinsic compression by matted juxtapyloric lymph nodes were present in this patient.

Clinical features of hypertrophic pyloroduodenal tuberculosis are nonpathognomonic. Mostly such patients present with epigastric pain, discomfort, anorexia, nausea, vomiting, anemia, weight loss, fatigue, loose motions, and pyrexia of unknown origin. All these features were present in this patient. Due to the non-specific clinical features, it is difficult to differentiate hypertrophic pyloroduodenal tuberculosis from other common causes of gastric outlet obstruction like chronic Peptic Ulcer Disease (PUD) and gastric malignancy (linitis plastica) merely on clinical grounds.

Pre-operative diagnosis of pyloroduodenal tuberculosis is rarely established with the help of laboratory and radiological investigations. ESR and Mantoux’s test are usually inconclusive. Chest X-rays may show concomitant pulmonary tuberculosis only in 25% of the cases. Abdominal ultrasonography, CT scan and barium...
X-rays, apart from showing gastric outlet obstruction and pyloroduodenal thickening and deformity, cannot clinch the exact underlying pathology. Upper GI endoscopy is helpful in excluding chronic peptic ulcer disease and gastric malignancy but fails to disclose the basic pathological process responsible for gastric outlet obstruction. Most of the endoscopic antral mucosal biopsies turn out to be non-specific like that of the presently reported patient. Nowadays, laparoscopy has emerged as an important modality for diagnosis of hypertrophic pyloroduodenal tuberculosis with promising results.

Management of hypertrophic pyloroduodenal tuberculosis is primarily surgical. Surgery has an important role to play in the management of gastric outlet obstruction caused by hypertrophic pyloroduodenal tuberculosis both diagnostically as well as therapeutically. Diagnostically, surgery not only provides the opportunity for naked-eye examination of the whole abdomen but also the tissue(s) for histopathological diagnosis. Therapeutically, side-to-side gastrojejunostomy is performed to bypass the pyloroduodenal obstruction. Occasionally, surgical intervention is also needed to deal with other complications of pyloroduodenal tuberculosis like haematemesis and perforation or fistulization.

Surgery is effective in relieving the obstructive symptoms of hypertrophic pyloroduodenal tuberculosis. Postoperative antituberculous therapy (ATT) is imperative for complete resolution of hypertrophic pyloroduodenal tuberculosis. Patients suffering from hypertrophic pyloroduodenal tuberculosis show extremely good response to postoperative antituberculous therapy (ATT) and become symptoms-free within a couple of weeks as observed in this patient.

**REFERENCES**