

Disseminated Tuberculosis Presenting as Gastric Outlet Obstruction

Eleonor G. Rodenas-Sabico and Germana Emerita V. Gregorio

Department of Pediatrics, College of Medicine and Philippine General Hospital, University of the Philippines Manila

ABSTRACT

A 12-year-old female had a three-year history of fever, non-bilious vomiting and abdominal pain. Upper gastrointestinal series showed a filling defect at the duodenum. Esophagogastroduodenoscopy exhibited circumferential mass extending from the duodenal bulb to the 2nd part of the duodenum which on histology disclosed chronic granulomatous inflammation. Chest X-ray suggested miliary tuberculosis; endotracheal tube aspirate was PCR positive for *Mycobacterium tuberculosis*. Patient was diagnosed as disseminated tuberculosis of the duodenum and lungs. Quadruple anti-tuberculosis medication was started but patient succumbed to nosocomial sepsis.

Key Words: duodenal tuberculosis, miliary tuberculosis, chronic granulomatous inflammation, disseminated tuberculosis, gastric outlet obstruction

INTRODUCTION

Tuberculosis (TB) is a life threatening disease that affects primarily the lungs but may involve any organ, including the abdomen. Infection may be spread through hematogenous or lymphatic route or directly through the mucosa from ingested bacilli or from the serosa contiguous to adjacent structures especially lymph nodes.¹

Abdominal TB, which comprises 1 to 3%² of all TB cases, may indicate infection of the solid organs, peritoneum, lymph nodes, stomach and the intestinal tract, from the duodenum to the anal area. Eighteen to 20% of abdominal TB involves the intestine, of which the most common site is the ileocecal area in 85% of cases.³ Tuberculosis of the stomach and the duodenum is rare, seen only in 0.4 to 2.5% of all TB cases.⁴

The features of gastro-duodenal TB are non-specific and require a high index of suspicion for diagnosis. Symptoms may include postprandial epigastric pain, vomiting, weakness, weight loss, fever and hematemesis. It can also present as recent onset of gastric outlet obstruction. Diagnosis is confirmed by the demonstration of either acid-fast bacilli and/or caseating granuloma in the biopsied material.³

We present a 12-year-old female who initially presented with abdominal pain and vomiting, and later was diagnosed to have disseminated tuberculosis with duodenal and lung involvement.

CASE REPORT

A 12-year-old female with Down Syndrome was admitted with a three-year history of undocumented high-grade fever, non-bilious and non-bloody vomiting, and on and off abdominal pain more prominent at the epigastric

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Corresponding author: Germana Emerita V. Gregorio, MD
Department of Pediatrics
Philippine General Hospital
University of the Philippines Manila
Taft Avenue, Manila 1000, Philippines
Email: gvgregorio@up.edu.ph

area and aggravated by food intake. The abdominal pain and vomiting persisted with no other associated symptoms until one month prior to her admission when she developed poor appetite and progressive weakness. She had bouts of loose yellow stools and one episode of melena. She was brought for consult to different hospitals given unrecalled medications but with no relief of symptoms. The patient was then brought to the emergency room due to weakness. She was in hypovolemic shock hence fluid boluses were administered and eventually started on inotropic support. She was intubated due to respiratory distress and hooked to mechanical ventilator.

The past medical and family histories were both non-contributory. She had no known exposure to pulmonary tuberculosis.

On admission, pertinent physical findings showed a severely malnourished patient in respiratory distress, tachypneic, tachycardic, with facial edema, pale palpebral conjunctivae and rhonchi on bilateral lung fields. The abdomen was distended and tender with ascites. Liver and spleen could not be palpated due to the presence of fluid. There was bipedal edema.

Initial laboratory examination showed leukocytosis ($12.9 \times 10^9/L$) with predominance of neutrophils, anemia ($54g/L$), reticulocytosis (0.023), hypoalbuminemia ($13g/L$) and elevated creatinine ($115\mu mol/L$). Serum electrolytes and transaminases were normal. Blood culture was negative. Chest X-ray showed findings of pulmonary cavitations on right upper lung fields; consolidation consistent with miliary tuberculosis; pleural effusion up to the right 6th intercostal space, right; and pleural effusion, left, mild. Chest ultrasound showed bilateral pleural effusion. Echocardiogram revealed pericardial effusion. The urine, pleural and pericardial fluids and endotracheal tube aspirate were all negative for acid-fast bacilli (AFB). These specimens were also sent for PCR studies for *Mycobacterium tuberculosis* and only the endotracheal tube aspirate was positive. On the basis of the clinical features of the patient and the results of the chest X-ray and microbiologic investigations, gastrointestinal tuberculosis was considered and abdominal imaging studies were done. CT scan of the abdomen showed massive ascites, calcified lymphadenopathy and long-segment thickening of the sigmoid and descending colon. No thickening nor filling defect was noted in the duodenum. Colonoscopy was contemplated once patient was more stable.

During her admission at the ward, there was note of bilious to coffee ground nasogastric tube output. An upper gastrointestinal series (UGIS) (Figure 1) demonstrated delayed gastric emptying and filling defect on the first part of the duodenum. A bed side esophagogastroduodenoscopy was done and showed a snake-skin-like mucosa in the antral area, erythematous and edematous pylorus, an 8x5 mm ulcer at the duodenal bulb and presence of a smooth and circumferential mass from the duodenal bulb extending to the 2nd part of the duodenum with nodularities (Figure 2).

Histology of the duodenal tissue was interpreted as chronic granulomatous inflammation (Figure 3) compatible with tuberculous etiology but the AFB smear was negative. There was no specimen sent for TB PCR.

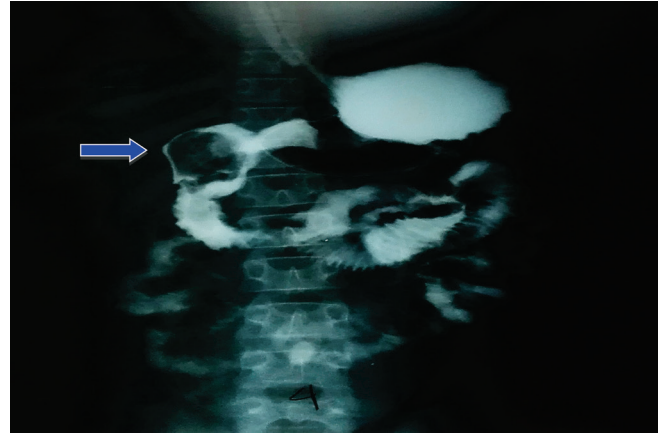


Figure 1. Filling defect at the first part of duodenum on upper gastrointestinal series (arrow).



Figure 2. Mass at the duodenal area on esophagogastroduodenoscopy (arrow).

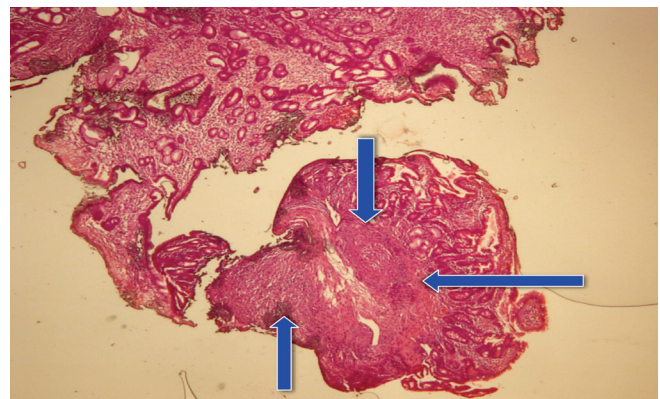


Figure 3. Granuloma on esophagogastroduodenoscopy of the second part of the duodenum (arrow).

The patient was diagnosed with disseminated tuberculosis with involvement of the duodenum and the lungs on the basis of the military findings on chest X-ray, the microbiologic demonstration of *Mycobacterium tuberculosis* on endotracheal tube aspirate and the chronic granulomatous inflammation of the duodenal tissue. She was started on quadruple anti-tuberculosis medication but after only months of treatment, succumbed to nosocomial sepsis. Human immunodeficiency virus testing was not determined.

DISCUSSION

We presented a 12-year-old female with Down's syndrome who had a three-year history of undocumented fever, epigastric pain, non-bilious vomiting that was aggravated by food intake and weight loss. The presence of these symptoms in the history is indicative of an organic cause for the chronic abdominal pain. Since the vomitus is non-bilious in character, gastric outlet obstruction was considered and further work up led to the identification of a duodenal mass secondary to tuberculosis.

There are only seven cases of duodenal tuberculosis in the pediatric age group reported in published literature and all presented as a gastric outlet obstruction. All cases were from India and all presented as vomiting with a mean duration of approximately three months. Our patient had disseminated tuberculosis with duodenal involvement. Although there was a protracted history of abdominal pain and vomiting, she was brought to different hospitals for consult and given unrecalled medications but there was no relief of symptoms. In adults, vomiting and abdominal pain are also the most common presenting symptoms of duodenal TB seen in 60-70% of cases. Around 25% of these patients may present as upper gastrointestinal bleeding, either from duodenal ulcer, fundal varices from obstruction of perihilar lymph nodes or presence of superior mesenteric artery-duodenal fistula.

The National Tuberculosis Control Program Manual of Procedures classified TB disease as bacteriologically-confirmed if with a smear/culture/rapid diagnostic test from a biological specimen in an extra-pulmonary site positive for AFB or MTB complex; and clinically-diagnosed if with histological and/or clinical or radiologic evidence consistent with active extrapulmonary TB and there is a decision by a physician to treat the patient with anti-TB drugs. These may consist of at least one of the following: (1) histologic evidence of caseation granuloma and/or Langhans multinucleated giant cells in the intestinal tissue and/or evidence of inflammation in any area of the intestine; (2) radiologic findings either on abdominal contrast studies, X-ray, ultrasound or CT scan demonstrating thickening, dilatation and/or palisading of bowel loops involving any part of the intestine; and (3) clinical improvement following anti-TB treatment including resolution of fever, decrease in abdominal pain or improvement in appetite.⁵

Our patient had clinically diagnosed duodenal tuberculosis based on associated clinical features and imaging studies and confirmed by demonstration of caseation granuloma on duodenal biopsy. Chest X-ray findings maybe suggestive in 20% of cases and was seen in our patient who had miliary tuberculosis. On CT scan, the presence of duodenal thickening or perigastric or periduodenal lymph nodes may give a clue to the diagnosis. An upper gastrointestinal series will reveal any gastric outlet obstruction either from an extrinsic compression from lymphadenopathy^{1,6} or an intrinsic mucosal ulceration or hypertrophy³, demonstrated as a filling defect in the first part of the duodenum in our patient. Endoscopy is performed in order to visualize any mucosal involvement and if possible, to obtain duodenal tissue for histologic diagnosis. Multiple mucosal biopsy and if possible endoscopic mucosal resection has been suggested due to the submucosal nature of tuberculous granuloma.⁷ In our case, endoscopy confirmed the presence of a duodenal mass and the biopsy was interpreted as chronic granulomatous inflammation although the AFB smear of the tissue was negative. In patients in whom it is not safe endoscopically to do a duodenal biopsy due to mucosal inflammation or presence of adhesions, tissue diagnosis is obtained operatively. In adults, only two of 20 cases had positive endoscopic biopsy⁸. In the reported cases in children, the diagnosis of primary gastroduodenal TB is difficult as there are no specific signs and symptoms and no characteristic endoscopic findings and is often suspected intra-operatively.

The management of duodenal TB is primarily medical and surgery is reserved for patients in whom there is complete obstruction, fistula formation or intractable ulcers. Standard quadruple anti-TB medications consisting of six months of isoniazid and rifampicin and two months of pyrazinamide and ethambutol has been effective in improving symptoms and resolution of lymph nodes in 100% of cases.⁹ Unfortunately for our patient, she succumbed to nosocomial sepsis even before treatment could be completed.

In summary, we presented a case of duodenal tuberculosis presenting as gastric outlet obstruction. Despite the rarity of the site of infection, tuberculosis should be part of the differential diagnosis especially if the signs and symptoms are supported by other radiologic, microbiologic and histologic findings.

Statement of Authorship

All authors participated in data collection and analysis, and approved the final version submitted.

Author Disclosure

All authors declared no conflicts of interest.

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