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Acute Massive Gastric Dialation—A Case Report

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Abstract

Acute gastric dilation leading to ischemia of the stomach is an underdiagnosed condition and can be potentially fatal. It can occur in various medical and surgical conditions such as post operative state, trauma, anorexia, spinal abnormalities, diabetes mellitus and electrolyte imbalance. Without proper and timely diagnosis and treatment, gastric perforation and hemorrhage can occur. In this report, we present a case of acute massive gastric dilation in a settling of type II diabetes mellitus and urosepsis. We will also present a review of literature and management options for such condition. Our report highlights the need for high index of suspicion and early intervention to avoid life threatening complications.

Keywords

Acute Gastric Dilatation, Infection, Urinary Tract Infection, Gastric Distension

1. Introduction

Gastric dilation occurs in various clinical situations. This could be either medical or surgical conditions. Patients develop abdominal distension with or without vomiting and pain. Diagnosis can be suspected clinically. However, plain X-ray of the abdomen is required in most cases. Although conservative treatment is usually successful in relieving the distension, treatment if delayed can lead to life threatening consequences. In this report we present a diabetic patient who developed acute massive gastric dilation during his stay in the hospital. We would also highlight the possible etiologies and discuss the clinical settings in which it can occur.

2. Case Report

An 85-year-old male patient was brought to us with five days history of fever and generalized body weakness.

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Fever was insidious in onset, moderate to high grade with chills. Maximal temperature recorded by patient was 39 degrees centigrade. Patient also gave history of decreased appetite and generalized body weakness. He was a known diabetic and was on oral Metformin 500 milligrams twice daily. On examination, patient was conscious. body temperature was 38.8 degrees centigrade, heart rate was 120 beats per minute, blood pressure was 90/60 mm of Hg, respiration was 20 cycles per minute and oxygen saturation was 97% on room air. Patient was dehydrated. Chest auscultation was clear. Abdomen was tender on superficial palpation with tenderness localized to epigastrium and mid abdomen. Patient was admitted into the intensive care unit. Laboratory investigations revealed neutrophilic leucocytosis with elevated serum urea and creatinine and normal potassium. Blood sugar was 220 mgs/dl. Urine Routine showed numerous pus cells/high power field. A diagnosis of urinatry tract infection with urosepsis was made. A urinary catheter was inserted and patient was started on broad spectrum antibiotics and intravenous fluids. Blood sugars were managed with insulin.

On the second day, patients developed abdominal distension and respiratory distress. Examination revealed a distended abdomen, hyper-resonant note on percussion and hyper active bowel sounds. There was also generalized tenderness on superficial palpation of the abdomen. A digital rectal examination revealed an empty rectum. An urgent abdominal X-ray was requested. Abdominal X-ray revealed a dilatation of the stomach occupying most of abdomen down to pelvis **(**Figure 1**)**.

The gastric shadow however, had maintained its normal anatomical shape to facilitate easy identification. An assessment of acute massive dialation of the stomach was made. Patient was placed on nil per orally and nasogastric tube was inserted with continuous drain. About 600 mL of gastric effluent was drained initially which contained ingested feeds.

After 24 - 36 hours of nasogastric tube insertion and drainage, patient's condition improved significantly. Abdominal distention decreased and bowel sounds were normal. A repeat abdominal X-ray revealed complete resolution of the gastric distension with normal gastric air bubble in the left hypochondrium (Figure 2).

He was continued on antibiotics for urosepsis and was discharged on day five in stable condition. Patient has remained stable on follow up. Blood sugars are well controlled. Patient had symptoms of upper abdominal bloating which has responded to prokinetic drugs.

3. Discussion

Acute gastric dialation is critical condition which can occur is diverse clinical situations. The causes of gastric

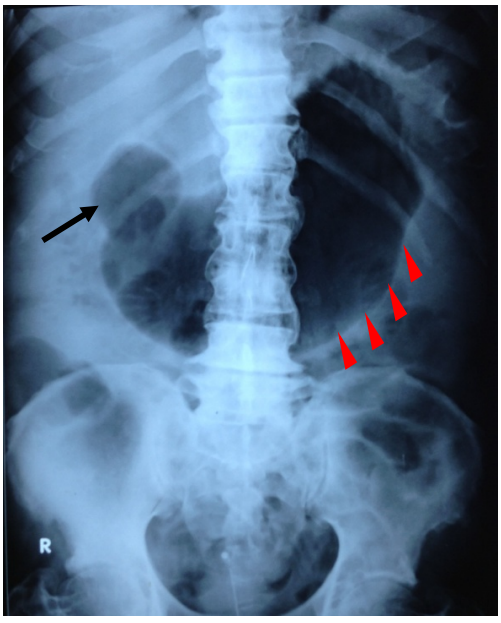

Figure 1. Plain abdominal X-ray showing massive dialation of the stomach with well delineated anatomical shape [pylorus (black arrow), greater curvature (red arrow heads)].

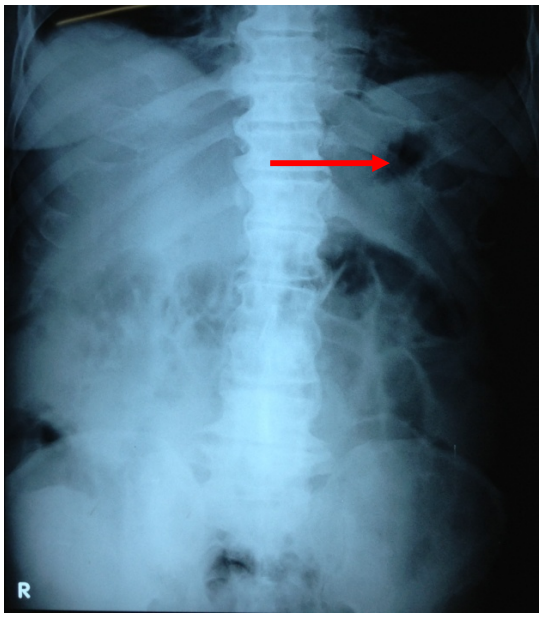

Figure 2. Plain abdominal X-ray showing resolution of gastric distension after 36 hours of nasogastric tube insertion and drainage (red arrow: normal gastric air-bubble in left hypochondrium).

dilatation are poorly understood. Gastric dilatation, can on rare occasions cause serious difficulties. If identified and treated early, recovery is complete. It can cause dyspnea either postoperatively or as a result of an underlying structural abnormality.

Gastric dilatation usually causes serious problems in the context of other structural abnormalities. If the dilatation occurs in the presence of a diaphragmatic or hiatal hernia, it can cause compression of the lungs and tracheal deviation [1]-[4]. Gastric rupture as a result of gastric dilatation has been reported to have caused death in patients with severe cerebral palsy. In these cases the dilatation might be caused by aerophagia as a result of autonomic neuropathy, malposition of the stomach, or severe kyphoscoliosis [5]. Gastric dilatation has been reported to cause sudden respiratory distress postoperatively as a result of abdominal compartment syndrome [6]. Postoperative ileus with subsequent gastric dilatation is not uncommon, although it usually resolves without serious sequelae; however, rarely it can cause sudden deterioration requiring prompt gastric decompression [7].

This case is unusual in that the patient was not suffering from a postoperative ileus and did not have an underlying structural abnormality, in the form of a hiatal or diaphragmatic hernia, or a skeletal abnormality, such as kyphoscoliosis. Also, there were no symptoms suggestive of intestinal obstruction. His serum electrolytes were normal. He did have features of urinary tract infection with urosepsis and diabetes mellitus with uncontrolled blood sugars. It is likely that combination of sepsis, dehydration and diabetes mellitus would have led to hypoperfusion of the stomach and led to gastric dialation. Autonomic neuropathy associated with longstanding diabetes mellitus is a likely contributory factor too. Nagai T. *et al.* [8] reported a similar case of Acute Gastric Dialation due to diabetic autonomic neuropathy in the stetting of bacterial pneumonia.

Acute gastric dialation (AGD) is radiographically defined as a gastric gas pattern larger than size of entire stomach. First described by S.E. Duplay in 1833, acute gastric dilatation has since been well documented in the literature. The common causes include postoperative state, trauma, eating disorders (e.g., anorexia nervosa, bulimia), medications, electrolyte abnormalities, spinal deformities, diabetes mellitus, debilitating chronic illnesses, diaphragmatic herniations, emotional stress and infections [9] [10]. Complications much known are perforation and sepsis. Mortality rate of up to 80 has been reported in cases of gastric ischemia and perforation [11]. Immediate identification and management are important to prevent complications such as gangrene, perforation, gastric emphysema (pnematosis) and emphysematous gastritis.

Early recognition of condition should prompt clinicians towards treatment consisting of nasogastric decompression along with fluid resuscitation. Diagnosis can be made on clinical grounds. However, a plain X-ray of

the abdomen is an easy and quick way of diagnosing the condition. Once a diagnosis is made, it is necessary to place the patient nil by mouth and a nasogastric tube should be inserted. A repeat radiographic evaluation is necessary after 24 hours to confirm adequate decompression the stomach. Our patient had complete resolution of the gastric dialation with nasogastric decompression within 36 hours. During this time however, care must be taken to monitor and correct any metabolic abnormalities such as hypokalemia and treat underlying cause (urosepsis—in our case) effectively.

4. Conclusion

Acute gastric dilation is a life threatening condition which occurs in various clinical situations. In the absence of trauma or surgery, it occurs in diabetic patients with evidence of sepsis. Conservative treatment in form of nasogastric decompression and intravenous fluids if instituted early is quite effective in relieving the gastric distension. In severe and prolonged cases, infarction and necrosis of gastric wall can set in and increase the morbidity and mortality.

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