



Acute Gastric Dilatation Because of Binge Eating in a Mentally Retarded Child

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ABSTRACT

Acute gastric dilatation (AGD) is a rare entity and has multiple clinical presentation. It is important for a clinician to be aware of this condition as it helps in early diagnosis and treatment. We present this case of a young girl who had presented with sudden onset abdominal distention and vomiting. Diagnosis of acute gastric dilatation was established by clinical examination and X-ray abdomen. Gastric decompression was done by nasogastric tube for 3 – 4 days. It allowed the normal gastric motility to return gradually. Gastric motility agent also helped in early recovery. It is important to diagnose acute gastric dilatation early and treatment with gastric decompression and gastric motility helps in quick recovery.

Introduction

Diagnosis of acute gastric dilatation is challenging. It can sometimes cause severe complications such as gastric perforation, pressure necrosis of gastric wall and compression of abdominal aorta with its occlusion. An accurate history and physical examination is very important in the diagnosis of AGD because delay can lead to ischemic necrosis and rupture. Therefore prompt diagnosis and treatment is of vital importance. This case is one such rare presentation of acute gastric dilatation.

Clinical Findings

A 15 yrs old girl known case of meningoencephalitis in childhood with mental retardation since then presented with constipation, vomiting, generalised pain over abdomen and distention of abdomen since 2 – 3 days.

She was only taking Juices and liquids orally since many years. However since last 8 – 10 days she had started eating solid food in large quantity. She started having symptoms central abdominal pain and distention of abdomen since last 3 – 4 days. She also had constipation since 3 – 4 days. She did not have any history of fever.

When she was received in casualty she was in hypotension and acute confusional state. Her abdominal examination showed gross abdominal distention. Bowel sounds were feeble. Tenderness was present all over abdomen but no rigidity was present. Per rectal examination showed no content in rectum.

She was resuscitated with IV fluids but she still remained in hypotension, therefore inotropic support was started for correcting hypotension. Nasogastric tube was inserted – it showed dark colour aspirate of 2500 ml. She was shifted to

intensive care unit and was kept under observation.

Her investigation showed Hb 11.2 gm / dl, WBC 9,000/cmm, SGOT 34U/L, SGPT – 25U/L, Total bilirubin - 0.3 mg/dl and direct bilirubin 0.2 mg /dl and Alkaline phosphatase–59 IU / L, Total protein 4.5 gm/dl, Albumin 2.5 mg /dl, Her Serum Creatinine was 1.7mg/dl on admission, which was normalised after resuscitation with IV fluids to 0.7mg /dl. Her Serum electrolyte were Sodium–136meq/L, Potassium -4.8 meq/L and Chloride of– 97 meq/L. Her X -ray abdomen showed ground glass appearance [Figure 1], suggestive of large fluid in abdomen. After insertion of nasogastric tube it was seen that it was reaching right up to the lower abdomen [Figure 2].

Her CT scan of abdomen and pelvis [Figure 3] was done after serum creatinine was normalised and she became hemodynamically stable. It revealed finding of massive dilatation of stomach with oral contrast passing into small and large bowel which were essentially collapsed thus confirming diagnosis of acute gastric dilatation of stomach.

Over a period of next 3 days, her abdominal distention was settled and pain in abdomen reduced to a significant extent. Her nasogastric tube aspirate progressively decreased and it was removed. Her 2D-echo was done to rule out any cardiac pathology and it was found to be normal. She was investigated for malarial parasite in peripheral smear and was found to be absent, leptospirosis and Dengue IgM antibody was found to be negative. Her Antinuclear antibody and ds DNA antibody was found to be negative. She was given IV antibiotic piperacillin and tazobactam along with GI motility agent Inj. Perinorm, Inj. Emeset and T. Ganaton in a thrice a day dose.

She started having diet on post admission day 5 and was discharged in a stable condition.

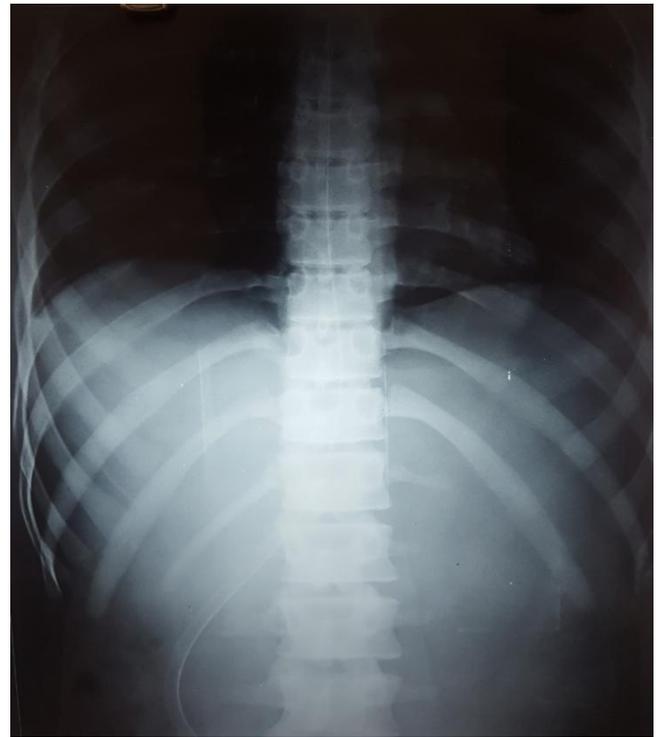


Figure 1: X ray abdomen showing ground glass appearance in entire abdomen caused because of massive dilatation of stomach.

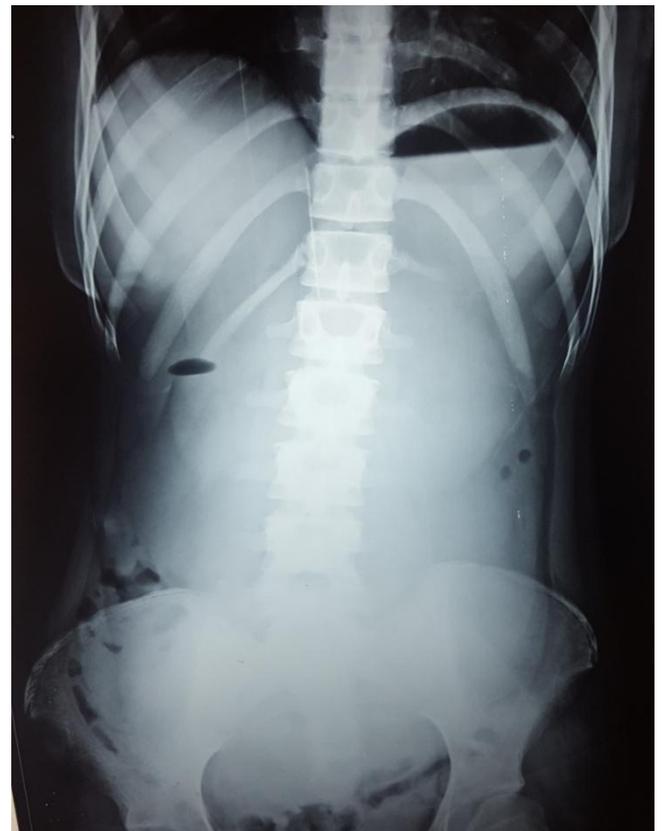


Figure 2: X ray abdomen showing nasogastric tube reaching right up to pelvis because of gross dilatation of stomach.



Figure 3: CT Scan of abdomen and pelvis showing, Massively distended stomach with ascites.

Discussion

Binge eating as a cause of acute gastric dilatation is a rare entity. The exact cause of acute gastric dilatation because of binge eating is still unknown. However there are many theories proposed for cause of acute gastric dilatation.

Acute gastric dilatation is seen in superior mesenteric artery syndrome where duodenum gets compressed between superior mesenteric artery and spine, thus leading to gastric outlet obstruction. Ho-Jun Lee et al, described a case of superior mesenteric artery syndrome causing acute massive dilatation of stomach.^[1]

Anaesthesia and debilitation can causing relaxation of the upper oesophageal sphincter with aerophagia causing acute gastric dilatation and regional diseases such as pancreatitis, peptic ulcer, gall bladder disease, appendicitis etc. have also been proposed as cause of acute gastric dilatation.^[2]

In this case the patient had been on liquid diet for long duration of time and she had started eating recently large quantity of solid food recently. Probable cause of acute gastric dilatation is, long duration of liquid diet resulted in decreased tone

of stomach muscle and sudden binge eating resulted in acute gastric dilatation.

Once the diagnosis of AGD is made, urgent nasogastric decompression and appropriate fluid resuscitations is required. It is always better to use large calibre nasogastric tube for drainage of gastric content in order to achieve complete emptying of stomach. Decompression may help because it can decrease the intragastric pressure and reduce the risk of necrosis and perforation. If conservative management fails or gastric necrosis with or without perforation is suspected, urgent surgical intervention is required.^[3] Adequate resection of the gangrenous portion is essential and gastrointestinal continuity is established depending upon resection. Total gastrectomy with oesophagojejunostomy, partial gastrectomy as well as wedge resection for acute gastric dilatation have been described.^[4]

Mishima T. et al, reported gastric necrosis and rupture in anterior part of stomach in a 12-year-old boy without any underlying disorders.^[5] Sung-Ui J et al, reported a case with rupture in the fundus in a 23-year-old-lady.⁶ Turan et al and Trindade EN et al, described a case of gastric necrosis caused by acute gastric dilatation in 2003 and 2008 respectively.^[7, 8] Manish Dewangan et al, discussed binge eating leading to acute gastric dilatation and ischemic Necrosis.^[9] Breslow M et al reported a case of Spontaneous rupture of the stomach as a complication of bulimia.^[10]

Severe ischemia with extensive mucosal necrosis in AGD is not always mandatory indication for surgery. Prompt adequate conservative therapy may avoid an un-necessary laparotomy with its added complications. In the present case, we performed early decompression for the distended stomach and fortunately, the patient recovered well using conservative management, and laparotomy was not required.

Conclusion

The clinician's awareness is of utmost important in diagnosis of acute gastric dilatation. High index of suspicion is required in any patient

presenting with history of binge eating, with prior history of fasting as this condition can be rapidly progressive and fatal. Early diagnosis and prompt management is of critical importance for better outcome.

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