MASSIVE GASTRIC DILATATION IN BULIMIC PATIENT

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Background: A 19-year-old patient presented to the emergency department with abdominal pain, inability to vomit and pain and paresthesia in her left leg. Her abdominal complaints began the evening before after drinking a large amount of milk and water. At that time she had bowel distention with inability to vomit and her symptoms intensified. The patient was a known bulimic with a tendency to ingest large amounts of food and vomit.

CT scan of the abdomen was performed on admission, and repeated after gastric decompression after insertion of a nasogastric tube which drained 8 litres of fluid. The following day, the CT scan was repeated.

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Work-up

Contrast-enhanced CT scan of the abdomen and pelvis (Fig. 1) includes transverse sections (A,B), a reformatted image (MPR) in the coronal plane (C) and a reformatted image (MPR) in the sagittal plane (D).

The images show a massive gastric dilatation: the stomach occupies most of the abdominal cavity and extends into the pelvis. Abdominal organs are displaced by the massively distended stomach, and the large abdominal vessels are compressed. There is an air-fluid level seen in the stomach on the axial and sagittal views with compression of the aorta (D). The right kidney shows signs of hypoperfusion due to compression of the renal artery (A).

Repeat contrast-enhanced CT scan after gastric decompression, transverse section at the level of the stomach (Fig. 2) shows improved perfusion of the abdominal organs and normalisation of the abdominal vasculature.

On a repeat CT scan performed the following day (Fig. 3) the presence of free abdominal fluid and marked thickening of the gastric wall, which show no mucosal contrast uptake are observed.

Radiological diagnosis

Massive gastric dilation was diagnosed with compression of the abdominal vasculature and hypoperfusion of the abdominal organs.

Repeat CT scan following gastric lavage suggested gastric necrosis, which was confirmed on endoscopy. Fortunately, no perforation was seen.

Discussion

Acute gastric dilatation is a rare and life-threatening condition. One of the causes of such a condition is eating disorders associated with binging. Severe complications, involving gastric infarction and compression of the aorta and large vessels with ischemia of the lower extremities are rare.

Mechanical compression of the cardio-esophageal and pyloroduodenal junctions can result in a closed loop which causes an increase in gastric pressure resulting in mucosal necrosis. Both the arterial and venous circulation has to be compromised before ischemia and necrosis occurs.

Plain abdominal films may be useful when a fluid level in a markedly distended stomach is present. The most useful diagnostic investigation however is an abdominal CT scan that can clearly demonstrate gastric distension. This case illustrates the important role of CT scan in establishing the diagnosis. Conditions such as gastric volvulus, duodenal obstruction, superior mesenteric artery syndrome, internal hernia and malrotation can be excluded by a CT scan. In addition a CT scan helps to exclude gastric gangrene or necrosis as well as perforation, conditions that will require immediate surgical exploration. Endoscopy is often necessary to rule out mechanical causes of obstruction and is also extremely important because it can show the general status of the gastric mucosa.

First line treatment for acute massive gastric dilatation consists of nasogastric decompression and fluid resuscitation. Even partial decompression may help because it can decrease the intragastric pressure and reduce the risk of necrosis and perforation. It also may allow vomiting as it may free the gastroesophageal junction obstruction If gastric necrosis or perforation is not recognized and the treatment is delayed, an 80% mortality rate is reported.

Bibliography