Boerhaave syndrome in pregnancy - A case report

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Case report:

History: We present the case of a 18 year old female, who was admitted in December 2020 hyperemesis gravidarum in the first trimester of pregnanc y. She was complaining of severe central chest pain worse on inspiration and ntractable vomiting which progressed to co ffee ground coloured vomit. Mallory-Weiss syndrome was suspected but in view of the severity of her pain, CT angiography was arranged.

Investigations:

CTPA: Evidence of pnuemomediastinum with free gas around the level of the carina and tracking up into the base of the neck. Clear lungs, no evidence of pulmonary emboli. Pneumomediast inum, given the history of hyperemesis is likely to represent a ruptured oesophagus (Boerhaave syndrome).

Surgical opinion was sought and, in light of her stable observations, conservative management with parenteral fluids, nil by mouth, antibioti cs, antiemetics and analgesics.

Two days later patient discharged herself against medical advice.



She was re - admitted with same complaint six weeks later with increasing chest pain and hyperemesis symptoms after period of settling at home. Assessed by surgeons and started on IV fluids, antitbiotics, antiemetics, analgesics and anxiolytics.

CT thorax: The extent of pneumomediastinu m has significantly reduced with only small foci of free air at the root of the right neck and sur rounding the mid of the



oesophagus. No evidence of frank oesophageal perforation.

She was discharged in good condition after 5 days of conservative management.

Background:

In 1724, Hermann Boerhaave, a Dutch physician and professor of clinical medicine, first described spontaneous, as opposed to traumatic, rupture of the oesophagus, which typically occurs after forceful emesis. Boerhaave syndrome, a transmural perforation of the oesophagus, should be distinguished from Mallory-Weiss syndrome, a nontransmural oesophageal tear that is also associated with vomiting.

Diagnosis of Boerhaave syndrome can be difficult, because often no classic symptoms are present and delays in presentation for medical care are common. Although Boerhaave syndrome classically presents as the Mackler triad of chest pain, vomiting, and subcutaneous emphysema due to oesophageal rupture but these symptoms are not always present.

Discussion:

Although vomiting is thought to be the most common cause, other causes include weightlifting, defecation, epileptic seizures, abdominal trauma, compressed air injury, and childbirth, all of which can increase the press ure in the oesophagus and cause a barogenic oesophage al rupture. Most cases occur in patients with a normal underlying oesophagus, although the presence of oesophagitis or ulcers has also been found in a subset of individuals. Two common risk factors include alcoholism and excessive indulgence in food.

Today, Boerhaave's syndrome accounts for 15% of all cases of oesophageal rupture. Although the actual incidence of oesophageal perforation worldwide is unclear, an incidence of 3.1 per 1,000,000 per year is estimated.

The actual clinical presentation will depend on the level of the perforation, the degree of leakage, and the time since the onset of the injury. Typically, the patient will present with pain at the site of perforation, usually in the neck, chest, epigastric region, or upper abdomen. Cervical perforations can present with neck pain, dysphagia, or dysphonia; intra-thoracic perforations with chest pain; and intra-abdominal perforations with epigastric pain radiating to the shoulder or back.

Differential diagnosis:

Aortic dissection

- Acute pancreatitis
- Myocardial infarction Pneumothorax
- Mallory Weiss: A tear of the mucous membrane in the gastroesophageal junction results in severe bleeding from the gastrointestinal tract.

Treatment:

Treatment is typically tailored to the patient's presentation, the type, and extent of the rupture, the time to diagnosis, and the viability of the oesophageal wall.

Early perforations, those diagnosed within 12-24 hours, have the best outcomes. Three common treatment options include conservative, endoscopic, or surgical.

The mainstay of treatment includes volume replacement, broad-spectrum antibiotic coverage, and surgical evaluation.

Surgical intervention includes primary oesophageal repair through open thoracotomy vs. VATS (video assisted thoracoscopy surgery) with fundic reinforcement, which is the gold standard within the first twenty-four hours.

Endoscopic placement of stents has been used to prevent fistula formations or seal oesophageal leaks in patients with delayed diagnoses and those with the early diagnosis without widespread contamination.

Conservative measurements are usually rese rved for small or contained ruptures.

Conclusion:

While this scenario is very unusual in pregnancy, all obstetricians need to be aware of the potential diagnosis of this syndrome when patient presents with chest pain and hyperemesis as one of the possible differential diagnoses that requires early recognition.

References:

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