Intrathoracic gastric perforation: a late complication of an unknown postpartum recurrent hiatal hernia

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Abstract

Diaphragmatic hernias occurring during pregnancy are an uncommon event. In very rare occasions, the clinical situation can suddenly worsen due to obstruction, torsion or infarction of the herniated viscera. Here, we describe a challenging case of a post-partum diaphragmatic hiatus hernia complicated by intrathoracic gastric perforation. A 23-year old woman was admitted to our hospital with a syndrome characterized by epigastralgia, dyspnoea and fever. She had previously undergone a laparoscopic antireflux surgery for hiatus hernia (6 years before) and a recent (4 months) unremarkable vaginal delivery. Due to the persistence of a pelvic pain after the delivery, she had been taking pain-killers as a self-administered medication. A CT scan showed a massive left pleural effusion and a complete herniation of the stomach into the left hemithorax. After placing a chest drainage and removing up to 3000 ml of brownish purulent fluid, a repeat CT scan (with water soluble contrast swallow) showed a leak at the level of the stomach. At surgery, we observed a complete intrathoracic herniation through a large diaphragmatic hiatal defect and a small well-defined gastric ulcer. A primary repair of both the stomach and the diaphragm was performed. We take the opportunity presented by this report to briefly discuss the patho-physiological mechanisms underlying this unusual complication.

Keywords: Gastric ulcer • Diaphragmatic hernia • Pleural empyema

Among diaphragmatic hernias, those occurring during pregnancy are an uncommon event. Although pregnancy usually occurs in the immediately antenatal period and seldom represent a life-threatening condition [1], here, we describe a very unusual and challenging case of a post-partum diaphragmatic hiatus hernia (HH), complicated by an intrathoracic gastric perforation. A 23-year old woman, with a history of a HH treated with laparoscopic antireflux surgery 6 years before and with a recent (4 months) vaginal delivery was admitted to the emergency department of our hospital with a subacute onset (3–5 days) of a clinical syndrome characterized by severe epigastralgia, moderate but worsening dyspnoea and fever. Due to the persistence of a pelvic pain after the delivery, she had been taking pain-killers as a self-administered medication. It is worth mentioning that, during the period of the pregnancy, no signs of recurrent HH were clinically suspected or instrumentally evidenced.

A remarkable (severe) hypoxia with SaO2% = 89% and paO2 = 51 mmHg was detected, and this was associated with a moderate increase in the white blood cells count (15.61 × 109/l with 81.4% neutrophils).

A chest X-ray showed a massive left pleural effusion confirmed by the chest CT scan that revealed, as well, a complete herniation of the stomach into the left hemi-thorax with an ample contralateral mediastinal shift (Fig. 1A and B). A 28-Fr chest drainage was immediately placed at the level of the fifth intercostal space, mid-axillary line and up to 3000 ml of brownish purulent fluid was removed. A repeat CT scan performed with a water soluble contrast showed a leak at the level of the stomach (Fig. 1C) and documented only a partial re-expansion of the pulmonary parenchyma (Fig. 1D). An emergency left thoracotomy was indicated and carried out: the stomach was entirely herniated in the left thoracic cavity through a large diaphragmatic hiatal defect and a small well-defined gastric ulcer. A primary repair with a double-layer closure of the gastric mucosa was performed; then, after having replaced the stomach in the abdominal cavity, a direct double-layer closure of the diaphragmatic hiatal defect was completed. The postoperative course was substantially uneventful.

A diaphragmatic hernia can remain unnoticed until the advanced stage of pregnancy when further herniation can be triggered by situations involving the increase to high intra-abdominal pressure levels. This, in turn, may be determined by an increase in the size of the uterus (relatively steep as of the second trimester) and, during delivery, by the Valsalva manoeuvres. Less frequently, early occurring hernias can happen due to repeated vomiting, a paraphysiological event in the first trimester [2].

More than 90% of maternal diaphragmatic hernias occurring during pregnancy are localized on the left side of the diaphragm [3]. Symptoms are usually vague (abdominal pain, nausea and/or...
vomiting, chest pain and dyspnoea) even if, on very rare occasions, the clinical situation can suddenly worsen due to obstruction, torsion or infarction of the herniated viscera [1–4]. So far and to the best of our knowledge, since 1928, 37 cases of this event have been published in the English literature [4].

In the case we report, the peculiarly late clinical onset (4 months after the partum), the relatively large diaphragmatic defect with no signs of visceral strangulation (single and well-defined ulceration in a context of normal gastric mucosa) and the history of long-term use of pain medications, configure a situation in which the typical pathophysiological mechanisms normally advocated in the aetiology of this condition, namely the vascular insufficiency due to visceral strangulation, appears to have a less causative role than a normal peptic ulceration due to non-steroidal anti-inflammatory drugs misuse, in an unusual clinical–anatomical condition.

Consent: Written informed consent was obtained from the patient for publication of this case report and the accompanying images.

REFERENCES

I read with interest the article entitled ‘Intrathoracic gastric perforation: a late complication of an unknown postpartum recurrent hiatal hernia’ by Lococo et al [1]. A 62-year-old man was referred to our centre with a case of left-sided traumatic chylothorax for the possibility of a thoracic duct ligation. A left-sided chest tube was draining several hundreds of millilitres of whitish milky fluid daily. Repeated chest X-rays were highly suggestive of a left traumatic diaphragmatic hernia. Chest CTs with both oral and intravenous contrast were done, which showed a defect on the left copula of the diaphragm with the whole stomach herniating into the chest and escape of oral contrast in the left pleural cavity. Exploratory thoracotomy was immediately decided upon. There was a 2-cm rupture in the lesser curvature of the stomach with the whole stomach in the chest with a thick pleural peel. The stomach defect was repaired in 2 layers of vicryl 1 followed by stapling. The stomach and omentum were reduced into the abdomen. The defect in the diaphragm was repaired with 2 layers of Prolene 1. The pleural peel was decorticated. The pericardial effusion was drained by a small pericardial window. The patient had a very smooth post-operative course.

To the best of our knowledge, very little is mentioned about traumatic ruptures of the herniated stomach in the chest.

Conflict of Interest: None declared

References