Anaesthetic management of acute gastric volvulus in an adult

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We describe an adult patient with gastric volvulus caused by a congenital diaphragmatic hernia. Anaesthetic management was complicated by cardiovascular instability, respiratory distress and unexpectedly difficult intubation.

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A previously healthy 49-yr-old 75 kg female was admitted to hospital with a history of pain in the left upper abdominal quadrant and left flank for 2 days. She had symptoms of bowel obstruction (nausea and vomiting, abdominal distension, and absolute constipation), pain upon deep inspiration and dyspnoea upon lying flat.

Her heart rate (HR) was 140 beats min⁻¹, mean arterial pressure (MAP) was 115 mm Hg. Oxygen saturation was 94% with \( F_{O2} \) 0.4. There was decreased air entry at the left base of the chest, but no other abnormality on auscultation. The abdomen was distended, generally tender and bowel sounds were absent.

An electrocardiogram (ECG) showed sinus tachycardia with rate-related ST segment depression. A portable semi-erect chest x-ray showed a gas-filled bowel loop occupying approximately 50% of the left thoracic cavity; with compression of the left lung and deviation of mediastinal structures to the right; and gas beneath the right hemidiaphragm (Fig. 1).

Respiratory distress increased and cardiovascular state rapidly worsened: HR was 160 beats min⁻¹, MAP was 60 mm Hg, jugular venous pressure was increased and \( S_{P2} \) was 92%. Oxygen 100% was given by a face mask and reservoir bag and she was immediately transferred to theatre receiving i.v. colloid (hydroxyethyl starch 6%, 1000 ml).

Anaesthesia was induced in theatre after preoxygenation. Cricoid pressure was applied, and a modified rapid sequence induction was performed using fentanyl 100 \( \mu \)g, etomidate 14 mg and succinylcholine 100 mg.

The intention was to insert a 37-Fr double-lumen bronchial tube and start ventilation of the right lung. Unfortunately, the patient had a small mouth (<2.5 cm inter-incisor gap) and a prominent maxilla. The larynx could be seen (Cormack–Lehane grade II), but passage of a double-lumen tube was not possible. A gum elastic bougie was passed through the glottis, but the double-lumen tube could not be passed over it. Oxygen saturation decreased to <80%.

A standard 8.0 cuffed orotracheal tube was therefore passed over the bougie, and the right mainstem bronchus was intentionally intubated by inserting the tube to a distance of 24.5 cm at the upper incisors. The cuff was inflated, one-lung ventilation was confirmed by auscultation and cricoid pressure was removed. Adequate gas exchange (\( E_{CO2} \), 44 mm Hg, \( S_{P2} \) 98%) was achieved using volume controlled ventilation (tidal volume 400 ml, frequency 12 bpm, \( F_{O2} \) 0.97, PEEP 5 cm H₂O). Anaesthesia was maintained using atracurium 30 mg and sevoflurane 1.4–3.0% in 100% oxygen, and supplemented with i.v. morphine.

A midline laparotomy showed a large congenital defect of the left hemidiaphragm. The left hemithorax contained the
stomach, spleen, left lobe of the liver and omentum. There was torsion of the stomach; the greater curvature of which was gangrenous and perforated in two places, with extensive contamination of pleural and peritoneal cavities. The left hemithorax was decompressed by returning the viscera to the abdominal cavity, the tracheal tube was withdrawn to 21 cm at the upper incisors, and two-lung ventilation was commenced. Gastropexy and lavage of the peritoneal and pleural cavities was performed. The diaphragmatic defect was not repaired because of the contamination and possible infection after operation. Chest drains on both sides and an intra-abdominal drain were inserted.

After the procedure, which lasted 90 min, the right internal jugular vein was cannulated for monitoring of central venous pressure. A thoracic epidural catheter was inserted, and the patient was transferred to the intensive care unit for stabilization after the operation. She recovered completely, and left hospital 2 weeks later.

Discussion

The incidence of congenital diaphragmatic hernia (CDH) is 1:2000–1:5000 live births with equal gender preference.\textsuperscript{1–4} Autopsies performed on Atacama mummies from the third century AD show that it is not a new phenomenon.\textsuperscript{5} Up to 20% of cases are associated with other congenital anomalies (e.g. mental retardation, Down’s syndrome, heart defects, cleft lip).\textsuperscript{2–4}

The defect arises during embryological development because of incomplete closure of the diaphragm or early migration of the midgut from the umbilical coelom into the abdominal cavity before the diaphragm is fully formed (around 10 weeks); thus allowing the midgut to herniate into the chest cavity. Related abnormalities include complete agenesis of one or both hemidiaphragms, and diaphragmatic eventration, where the diaphragm mainly consists of pleuroperitoneal membrane with little or no muscular component, allowing it to be pushed upwards by the abdominal contents.\textsuperscript{1–4}

Herniation may occur at three sites: (i) posterolaterally through the foramen of Bochdalek (78–90% cases); (ii) retrosternally via the foramen of Morgagni (1.5–6% cases); (iii) via the oesophageal hiatus (14–24% cases). Left-sided herniation is more common, as the right hemidiaphragm develops slightly earlier than the left, and is somewhat protected by the liver.\textsuperscript{13–4} The lung on the affected side is often hypoplastic, resulting in right-to-left shunting of blood.

A total of 70–95% of cases are diagnosed in the neonatal period based on a triad of: (i) respiratory distress, often severe with cyanosis and decreased breath sounds on the affected side; (ii) scaphoid abdomen; (iii) contralateral mediastinal shift. Auscultation of bowel sounds over the involved hemithorax is an unreliable sign. Emergency operative correction is required in these cases.\textsuperscript{16}

If CDH does not present acutely in the neonatal period, diagnosis of the defect is more difficult as the signs and symptoms become chronic, vague and inconsistent. Symptoms can be either cardiorespiratory or gastrointestinal, with the latter becoming more prominent as the age of onset of symptoms increases. Chronic cardiorespiratory symptoms include coughing, dyspnoea or recurrent chest infections. Chronic gastrointestinal symptoms include epigastric or subcostal discomfort, nausea and vomiting, fullness, bloating, cramping or diarrhoea.\textsuperscript{2–4,7}

Plain chest radiographs may show bowel loops in the thoracic cavity; however the hernia may reduce spontaneously; or may mimic a mediastinal mass, pneumatocele or pulmonary consolidation, making diagnosis difficult. Upper gastrointestinal contrast studies may aid diagnosis.\textsuperscript{7}

Acute herniation of viscera through a congenital diaphragmatic defect in the adult is usually precipitated by increased intra-abdominal pressure because of pregnancy, postural changes or trauma.\textsuperscript{8–9} Once in the thoracic cavity, the viscera usually remain there because of the abdomino-thoracic pressure gradient. Small defects tend to present with strangulation of the viscera leading to necrosis, perforation and peritonitis; whereas larger defects present with respiratory distress and compromise of the circulation. Often, as in this case, a combination of both is seen. It is interesting to note that this patient had a normal pregnancy and delivery by Caesarean section 14 yr previously without incident. There was no history of trauma or oesophageal reflux.

The mass effect of the intra-thoracic viscera causes cardiovascular impairment by direct compression of the heart and mediastinal shift, which can kink the vena cavae and pulmonary veins, impair venous return to the heart, and cause cardiac output to decrease. A plain chest radiograph
will confirm the presence of bowel loops in the thoracic cavity. Once the diagnosis has been established, urgent surgical correction is required. Anaesthetic management of CDH presenting acutely in the adult is poorly described, but the principles resemble management of late-diagnosed traumatic diaphragmatic herniae.9

Large-bore i.v. access should be gained and fluid resuscitation commenced. Invasive arterial and central venous pressure monitoring should be considered, but if the patient is in extremis, these should not delay commencement of surgery. The patient is at high risk of aspiration because of gastrointestinal obstruction, and therefore antacid premedication should be given, and a nasogastric tube should be inserted and aspirated if possible before rapid sequence induction with cricoid pressure. If difficult intubation is anticipated, awake fibreoptic intubation or tracheostomy under local anaesthesia should be considered. If the patient is haemodynamically unstable, induction should be in theatre with the surgeon ready to operate immediately. Agents less likely to decrease MAP (etomidate and fentanyl) should be used. Expansion of the viscera is likely to worsen the mass effect and impair circulation and respiration. Face-mask ventilation, with potential gastric insufflation; and nitrous oxide anaesthesia should therefore be avoided.

In theory, positive-pressure ventilation might preferentially ventilate the normal lung rather than the collapsed lung. However, any re-expansion of the collapsed lung may exacerbate the mass effect, with rapid and disastrous worsening of the circulation. The collapsed lung should therefore be isolated and ventilation of the normal lung started with small tidal volumes and pressures, using a double-lumen tube, until the affected hemithorax has been decompressed. If this is not possible, a single-lumen tracheal tube with a bronchial blocker should be considered. The use of a single-lumen tube in the manner described in our case was an expeditious solution to an unexpected difficult situation, and is not recommended.

In conclusion, CDH in an adult is a diagnostic and anaesthetic challenge. The diagnosis should be considered in any patient presenting with an acute abdomen and respiratory distress.

References
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