Diaphragmatic hernia repair and after care

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Rupture of the diaphragm is most commonly seen with blunt thoracic trauma, with 77-85% of all cases of diaphragmatic rupture being traumatic, congenital pleuroperitoneal diaphragmatic ruptures are very occasionally seen. Affected animals are presented with different clinical signs starting from no respiratory distress to catastrophic life endangering dyspnoea depending on the amount of herniated abdominal organ material.

The abrupt increase in intra-abdominal pressure accompanying forceful blows to the abdominal wall causes the lungs to rapidly deflate (if the glottis is open), producing a large pleuropertitoneal pressure gradient. Alternately, the pressure gradient that occurs between the thorax and the abdomen may cause the diaphragm to tear. The tears occur at the weakest points of the diaphragm, generally the muscular portions. Location and size of the tear or tears depend on the position of the animal at the time of impact and the location of the viscera. Traumatic diaphragmatic hernias are often associated with significant respiratory distress; however, chronic diaphragmatic hernias in asymptomatic animals are not uncommon.

Animals with recent traumatic diaphragmatic hernias frequently are in shock when they present for treatment; therefore, clinical signs may include pale or cyanotic mucous membranes, tachypnoea, tachycardia, and/or oliguria. Cardiac arrhythmias are common and associated with significant morbidity. Other clinical signs depend on which organs have herniated and may be attributed to the gastrointestinal, respiratory, or cardiovascular system. The liver is the most commonly herniated organ, a condition that often is associated with hydrothorax caused by entrapment and venous occlusion.

What is the cause of respiratory impairment following diaphragmatic hernia?

- Loss of functional residual capacity (mass effect from herniated organs and or pleural effusion / pneumothorax)
- Pulmonary contusions
- Atelectasis of the lung lobes
- Rib fractures
- Flail chest
- The effects of shock
- The effects of pain

Myocardial contusion often present and may decrease cardiac output. When myocardial injury is concomitant with impaired ventilation, tissue hypoxia can result. Pain resulting from chest and abdominal contusion and accompanying injuries causes voluntary restriction of thoracic excursion and can therefore further compromise ventilatory capability.

Diagnosis

Definitive diagnosis of pleuroperitoneal diaphragmatic hernia usually is made by radiography or ultrasonography. If significant pleural effusion is present, thoracocentesis may be necessary before diagnostic diagnostic radiographs are performed. Radiographic signs of diaphragmatic hernia may include loss of the diaphragmatic line, loss of the cardiac silhouette, dorsal or lateral displacement of lung fields, presence of gas or a barium-filled
stomach or intestines in the thoracic cavity, pleural effusion, and/or failure to observe the stomach or liver in the abdomen. It may be difficult to diagnose diaphragmatic hernias radiographically if only a small portion of the liver is herniated. Ultrasound examination of the diaphragmatic silhouette may help when herniation is not obvious radiographically (i.e., hepatic herniation, pleural effusion). Ultrasonography may be particularly difficult if severe pulmonary contusions are present which make the lung appear ultrasonographically similar to liver, if only omentum is herniated, or if adhesions between the liver and lung are present. Also, care should be taken not to mistake a normal mirror-image artefact (usually seen as apparent liver parenchyma on the thoracic side of the diaphragmatic line) for herniated liver.

Positive contrast coeliography occasionally may be helpful. Pre-warmed water-soluble iodinated contrast agent is injected into the peritoneal cavity at a dosage of 1.1 ml/kg (the dose is doubled if ascites is present), the patient is gently rolled from side to side or the pelvis is elevated, and films are taken immediately after the injection and manipulation. Criteria used in evaluating these images should include the presence of contrast medium in the pleural cavity, absence of a normal liver lobe outline in the abdomen, and incomplete visualization of the abdominal surface of the diaphragm. Positive-contrast celiograms should be interpreted cautiously, because omental and fibrous adhesions may seal the defect, resulting in false negative studies.

**Pre-operative considerations**

**Oxygen supplementation**

If the patient is dyspnoeic, oxygen should be provided by face mask, nasal insufflation, or an oxygen cage/incubator as all cases of diaphragmatic rupture are likely to have a VQ mismatch, a minimum FiO2 of 50% should ideally be administered. Oxygen supplementation must not induce undue stress that can result in a deterioration of the animal’s condition. Positioning the animal in sternal recumbency with the forelimbs elevated may help ventilation. If moderate or severe pleural effusion is present, thoracocentesis should be performed.

Cyanosis is a late sign of the need for oxygen, and any signs suggestive of hypoxia should be treated promptly to prevent this happening i.e., nasal flaring, dyspnoea, reduced mentation and signs of oxygen hunger such as abducted elbows, extended head and neck, and open-mouthed breathing. Patients that fail to respond to oxygen supplementation may have severe ventilation perfusion mismatching as a consequence of atelectasis or pulmonary contusions.

**Fluid therapy**

Adequate volume replacement is essential. However, vascular support must be delivered with the knowledge that these patients often have concurrent pathology such as atelectasis and pulmonary contusions that can be exacerbated by over aggressive fluid administration.

**Antibiosis**

Prophylactic antibiotics should be given before induction of anaesthesia in animals with devitalized tissue, e.g. due to hepatic herniation or significant lung atelectasis. Massive release of toxins into the circulation may occur with hepatic strangulation or vascular compromise.

**Timing of surgery**

Surgery is best performed after a period of patient stabilisation. Approximately 15% of patients will die prior to surgery. If surgery is performed within the first 24 hours of presentation, mortality rates are highest (33%) reflecting acute cardiorespiratory
deterioration in these unstable, shocked and compromised patients. In general, patients are best managed by a period of stabilisation (24-72 hrs) to improve their respiratory function and tissue oxygenation, to correct fluid deficits and to diagnose and manage other potentially life-threatening complications such as cardiac arrhythmias. However, surgery should be performed as soon as the patient is stable and should not be delayed if a patient is deteriorating despite supportive care. However, some cases cannot be left for a period of stabilisation and will require immediate surgical intervention because of the risk of acute decompensation. These include:

- Diaphragmatic hernia with intrathoracic gastric dilatation or GDV: if the stomach or proximal small intestine are herniated, the risk of pyloric outflow and oesophageal cardial obstruction are high and cases may present with a tension gastrothorax as a result of GD or worse, GDV within the thoracic cavity. As normal a stomach tube may be passed in an attempt to relieve the pressure within the stomach.
- Rupture of the gastrointestinal tract;
- Rupture of the biliary tract;
- On-going life threatening intrabdominal or intrathoracic haemorrhage;
- Tension pneumothorax secondary to lung damage.

Pre-anaesthetic considerations

Supplementing oxygen before induction improves myocardial oxygenation. Because of the animal's already compromised ventilation, drugs with minimal respiratory depressant effects should be used. Injectable anaesthetics allowing rapid intubation are preferred. Inhalation anaesthetics should be used for maintenance of anaesthesia.

Intra-operative considerations

Intermittent positive pressure ventilation should be performed, and high inspiratory pressures should be avoided to help prevent re-expansion pulmonary oedema. The lungs should be allowed to expand slowly after surgery.

Goals of surgery

- Identify the position of the hernia;
- Reduce hernia contents;
- Assess abdominal viscera for viability;
- Assess thoracic viscera for injury;
- Repair diaphragmatic defect: tension free repair of viable tissue;
- Remove air and fluid from the thorax.

Re-establish negative intrathoracic pressure

There are a number of methods that can be used to re-establish negative intrathoracic pressure:

- Thoracostomy tube placement
- Trans-diaphragmatic thoracocentesis
- Transthoracic needle thoracocentesis.

Lung overinflation
The traditional technique of overinflating the lungs prior to final suture placement in order to re-inflate atelectatic areas of lung and to evacuate air from the thorax is contraindicated and most probably contributed to the high mortality rates in the early reports of diaphragmatic hernia management. This practice can lead to pulmonary re-expansion injury leading to acute alveolar flooding. In this syndrome, increased permeability of the alveolar membrane leads to rapid pooling of fluid in the alveolar space and respiratory collapse. This is seen within a few hours of re-expansion and is usually progressive and fatal. The aetiology is uncertain and could relate to membrane injury secondary to endotoxaemia, hypoxia or reperfusion injury but what is clear is that its development is directly linked to rapid re-inflation and over-inflation of lung. It is far safer to slowly re-establish negative pressure in the thorax and to allow atelectatic areas of lung to re-inflate over time.

**Recovery and complications**

Patients should be monitored postoperatively for hypoventilation, and oxygen should be provided if necessary. Most cases that survive surgery but die do so in the immediate postoperative period as the result of acute respiratory collapse. This may be secondary to re-expansion pulmonary injuries or pneumothorax due to previously undiagnosed lung injuries that become apparent as the lungs re-expand or due to thoracostomy tube complications. Patients need to be carefully monitored postoperatively to ensure that their respiratory status is not deteriorating and should continue to receive oxygen supplementation well into the recovery period. If there is any deterioration, diagnostic thoracocentesis and radiography early in the course of the problem to identify the cause is the safest option. Animals with empty abdomen syndrome may show signs of respiratory distress as a result of raised intra-abdominal pressure. Ventricular arrhythmias are also common. Less frequently, complications associated with the organs that have herniated are encountered. Gastrointestinal tract perforation and haemorrhage from splenectomy and partial hepatectomy are potential complications. Post-operatively as the pancreas may have been traumatised either at the time of the original injury or subsequently during reduction of the hernial contents.