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Respiratory Surgery Online 'Mini Series'

Session 1: Upper Airway Surgical Conditions

Jonathan Bray

MVSc MACVSc CertSAS MSc (ClinOnc) DiplECVS
RCVS and European Specialist in Small Animal Surgery

Lower Airway Surgical Conditions 1

The Chest Trauma Patient

Incidence

Injury to the structures of the chest should be suspected following any traumatic injury, particularly those involving a fall, blunt injury or an automobile/vehicular event. Thoracic injuries may be a 'silent' component of many emergency presentations, and it can be easy to be distracted by the more significant fractured bones, open wounds etc. It is known from human Emergency Rooms that there are three intervals where death may occur as a result of injury.

- the first hour (termed the 'survival hour'), when most deaths occur as a result of severe disruption to the cardiovascular or respiratory systems.

- the 2nd and 4th hour following injury (the 'Golden Period'). During this period, the astute clinician and nursing team have the greatest ability to influence a patient's outcome following an injury – by either promptly recognising and successfully managing underlying cardiorespiratory compromise, or by neglecting appropriate disease signs leading to the patient succumbing to its injuries with cardiorespiratory arrest.

- Death can still occur several days after an initial injury due to complications or irreversibility of the trauma.

Causes of Injury

Injuries to the chest can occur following a variety of events, including road traffic accidents, falling from a height, kicks, dog fights etc. However, the nature of the trauma may increase our suspicions for the potential for thoracic injury. In a large review of 300 dogs presenting with musculoskeletal injuries, 31% of animals involved in a road traffic accident were found to have concurrent thoracic injuries. This compared to only 2.4% when the musculoskeletal injury occurred through other means (e.g. falling from a height, fracture while running etc.). A similar high rate (52%) of thoracic injury was detected in cats following RTA, but cats generally had a higher rate of injury regardless of the inciting cause (38.7%), compared to the dog.

Penetrating injuries to the chest from dog fights, ballistics or other sources should always be surgically explored (once the patient has been adequately stabilised), even if the penetrating wound appears small and inconsequential. A high rate of 'hidden' underlying thoracic injury is reported following such events.

Pulmonary contusions (bruising) represent the most common type of pathology seen following thoracic injury – seen in 55% or more of all cases. Haemorrhage and swelling/oedema of the small airways leads to disruption in air exchange (ventilation:perfusion mismatch); the clinical impact will depend on the amount of lung tissue affected by the injury. Haemoptysis, or the presence of bloody froth in the oropharynx is an indicator of severe pulmonary contusion.

Recognition of Injury

Injury to the respiratory system usually causes a change in breathing efficiency – this is noted initially by an increase in effort. As the severity of injury increases, the animal may begin to show signs of distress due to oxygen depletion; the animal may be unable to breathe effectively if laying or sitting down (orthopnoea), or each respiratory effort is extremely laboured. Noises (stertor/stridor) may become apparent with the increased effort, particularly if there is obstruction to the airway (either as a result of injury, or due to the dog's normal conformation); the source of these noises may give some clue to the underlying pathology.

Physical Assessment

Clinical signs of respiratory injury are not always obvious; in a recent study, 75% of patients had evidence of thoracic trauma on radiography, blood gas evaluation or ECG that had gone undetected on clinical evaluation. This may have significant consequences for an individual – failure to recognise thoracic injury before proceeding with a prolonged general anaesthetic, or leaving the animal unmonitored overnight, for example, may result in death due to the effects of progressive hypoventilation, hypoxia (low oxygen) and hypercapnia (high carbon dioxide).

Careful assessment of respiratory movement often provides the first clues about underlying thoracic problems. Watching the animal when it believes it is being unobserved (and is therefore "relaxed") may reveal signs of altered respiratory effort. The respiration pattern may be shallow and rapid with 'space filling' conditions like effusions, diaphragmatic rupture, pneumothorax; with contusions and pulmonary injury, the breathing is likely to be more laboured, with a greater abdominal component evident. Other signs that may be noted include exercise intolerance, tachypnoea, tachycardia, cyanosis and open-mouth breathing. The latter is a very concerning sign in the cat – IT MEANS HANDLE WITH CARE!! Other signs may be more referable to the causative disease, and include fever, depression, vomiting/diarrhoea, anorexia, ascites, peripheral oedema, orthopaedic trauma, cardiac murmur or arrhythmia.

Thoracic auscultation with a stethoscope allows air sounds to be assessed over the whole area of the chest. Dullness, and an absence of breath sounds suggests the presence of effusion, or interfacing tissue (diaphragmatic hernia); these signs may be gravity dependant. Harsh sounds, crackles or wheezes may suggest the presence of fluid in the smaller airways, and indicate pulmonary contusion. Increased resonance of the chest may be detected in animals with pneumothorax.

Monitoring Techniques

Physical evaluation alone is insensitive for the detection of many thoracic injuries. In order to improve detection of subtle and/or progressive injuries, a clinician may rely on various objective monitoring aids: blood gas machines, pulse oximetry, thoracocentesis and electrocardiography (ECG).

Pulse oximetry: Pulse oximetry is a simple, and readily available technology. It can provide instantaneous and continuous recording of oxygen status, and provide early detection of hypoxia. The probes are usually well tolerated by the critical patients, and should form the first-line monitoring technique for all trauma patients. However, it is important to recognise the limitations of pulse oximetry. The value displayed on the screen is simply a percentage oxygen saturation of haemoglobin (should be >92% on room air); thus if the patient is anaemic, has low haemoglobin concentrations, or has poor peripheral circulation (due to shock, hypovolaemia etc) then the actual oxygen delivery to tissues can still be sub-optimal, even though the SpO2 is recording a high value.

Blood Gas measurement: Blood gas measurement provides the most accurate and comprehensive evaluation of respiratory function – i.e. ventilation efficiency and perfusion, a measure of gaseous exchange at alveolar level. It requires specialised equipment, and the collection of arterial blood samples (though central venous samples can also be used). Arterial blood sampling is normally performed from the dorsal pedal artery, which courses on the medial aspect of the hock; this may be catheterised if repeat sampling is required.

An animal's ability to ventilate effectively is determined by the PaCO2 value – if it is low, the patient is probably hyperventilating. With shallow respiratory movements due to pain, or altered pulmonary function, the CO2 may be high. Because CO2 is an important 'buffering' agent for balancing the build-up of metabolic acids in the blood stream (lactic acid etc), the pH value of the blood gas can also provide some important information on the patient's systemic status.

Estimation of the effectiveness of alveolar perfusion/gaseous exchange is measured by the PaO2 value. In patients breathing room air, a value less than 80mmHg is concerning and indicates a need for oxygen supplementation.

Thoracocentesis: If altered respiratory efficiency is suspected (based on disturbed respiratory efforts, or auscultative findings), thoracocentesis can provide a rapid and effective technique to rule out the presence of a pleural effusion or pneumothorax. If performed properly, thoracocentesis can allow these concerns to be swiftly eliminated from the diagnostic list; if confirmed, therapeutic relief is achieved in the same step.

Imaging Assessment

Radiography: Radiography can be a relatively unhelpful media in the investigation of thoracic trauma. Accurate positioning can be very difficult in the dyspnoeic patient, and poorly positioned radiographs are of little diagnostic value. Excessive and inappropriate handling can be very stressful, and extreme care is required with dyspnoeic patients. Oxygen supplementation should be considered at all times – a flow-by technique is the least stressful.

Initial imaging should be performed with the patient in sternal recumbency only. This view is the most sensitive for pleural effusion, and thus will provide information to proceed with further support or treatment. It will also allow assessment of the severity of any effusion, and may provide some guidance as to whether imaging in lateral recumbency will be tolerated by the patient. Rib fractures can be difficult to detect, but if seen will provide some indicator of the potential for underlying bruising to the lung tissue.

Radiographic evidence of pulmonary contusions is often not immediately apparent after an injury – it may take 6-12 hrs for these changes (increased interstitial density; air bronchograms etc) to become evident.

Ultrasonography: Ultrasonography can be invaluable for the identification of neoplastic lesions, diaphragmatic hernia, or for collection of pleural fluid when only small quantities exist. Ultrasonic examination is a minimally stressful technique, and can usually be performed with the patient in its most comfortable position (standing, or sternal recumbency). The presence of a fluid effusion in the pleural cavity provides an excellent acoustic media, and good images can usually be obtained.

Clinical Interventions

Provision of Oxygen Supplementation

In animals with only moderate dyspnoea, increasing the inspired fraction of oxygen above that of room air (21%) may be all that is required to relieve respiratory distress. The technique chosen should be simple and effective and not cause undue stress to the patient. Many patients often resent oxygen masks and continued struggling to maintain the mask in place serves only to heighten anxiety and worsens the dyspnoea. Several techniques are described for provision of long-term oxygen supplementation, including intranasal oxygenation, oxygen tents, and oxygen cages.

INTRANASAL OXYGEN: If the patient will tolerate placement of the catheter, nasal cannulation is an excellent method for providing long-term oxygen therapy. An intranasal catheter will increase the inspired fraction of oxygen to about 40% at flow rates of 100 ml/kg/minute, and is considerably less wasteful of oxygen than other methods. Gastric distention can occur with excessively high flow rates so a regulated oxygen source must be used (e.g. anaesthetic machine). Humidification of the oxygen is imperative to prevent dehydration of the airways.

Nasal cannulation can be performed in the conscious patient, with any standard polyurethane nasogastric feeding tube. The tube is inserted into either nasal cavity in a similar fashion to nasogastric intubation. In this instance, however, the tip of the tube terminates in the mid-nasal cavity (at the level of the carnassial tooth). Prior instillation of local anaesthetic (Opthaine®) into the nostril, and lubricating the tube with xylocaine gel, will improve patient compliance. Once the tube is inserted, it is secured to the perinasal skin with suture or superglue. An Elizabethan collar may be necessary to prevent interference with the tube by the patient.

OXYGEN TENT: A personal oxygen tent can be created from an Elizabethan collar and cling film. Oxygen is supplied via a tube which enters the Elizabethan collar at the back. High flow rates (> 10 L/min) are required to prevent carbon dioxide building up in the tent, but humidification is not required.

INCUBATOR/OXYGEN CAGE: Oxygen cages are considerable more wasteful of oxygen, and restrict direct access to the patient for examination. If the door is opened, the benefit of oxygen supplementation is lost. Precise control of inspired oxygen fraction is therefore difficult to achieve.

Thoracocentesis

Thoracocentesis is easily performed, with care. Equipment required includes a small butterfly catheter (or an 'over-the-needle' intravenous catheter (20g) attached to extension tubing), a three-way stopcock, and syringe. The site of thoracocentesis will be dictated by physical examination or imaging studies, particularly if a unilateral effusion is present. In general, the mediastinum is an imperforate membrane, and it is usually possible to drain both sides of the chest from a single puncture site.

Chest Drain placement and management

Thoracostomy tubes are available from several commercial suppliers. They consist of a tube made of PVC or silicone, together with a metal stylet to facilitate placement. Tubes come in a range of sizes. The correct size for an individual patient may vary on the type of effusion present, but a usual rule of thumb is to place a tube approximately equal to the diameter of the main stem bronchus. A wide bore may be required for thicker inflammatory secretions. Other equipment that will be required includes a scalpel blade, three-way tap, appropriate adaptors from the tube to the three way tap, syringe, tube clamp and suture material. Some method of providing adequate security of the various connections to the chest drain is also essential to prevent dislodgement.

Complications of chest drains

When appropriate care is taken with the placement of chest drains, it is unusual for significant complications to occur. Potentially, a chest drain trocar can bruise/perforate the underlying lung tissue, causing pneumothorax and/or haemorrhage. Because there is usually air of fluid present within the pleural cavity, this can act as a reasonable buffer to prevent such injury from occurring.

Following placement of the chest drain, the most common complications include pain, stoma infection/irritation, subcutaneous emphysema, and poor 'seal' of the stoma due to an inadequate subcutaneous tunnel (particularly in lean animals, or due to poor placement). Catastrophic pneumothorax may occur if the patient interferes with the tube, due to dislodgement of sealing bungs, or by biting through the tube.

Chest drains can be very uncomfortable for some patients (especially cats), either due to direct pleural irritation, impingement on intercostal nerves, or other reasons. Appropriate use of opiate analgesia may be necessary. Regional anaesthesia may be of value in other patients.

Prevention of stoma infection requires careful attention to asepsis during placement and handling of the drain. The use of protective dressings to prevent contact contamination of the area during hospitalisation should also be considered. Latex or polyurethrane drains are likely to induce a minor cellular reaction if a drain is left in place for over 5 days due to chemical irritation of the tissues. This is usually self-limiting, but may be the cause of some discomfort for the patient.

Subcutaneous emphysema can develop if there is an insufficient seal about the surface of the drain, either as a result of air leaking directly from the pleural space (with a pneumothorax) or by direct extension from the skin surface. This is unusual, but may occur when a drain has been left in situ for a prolonged period (with progressive necrosis of intercostal muscles about the pleural wall stoma), or if an inadequate subcutaneous tunnel was performed at the time of tube placement.

The importance of creating a good subcutaneous tunnel at the time of chest tube placement cannot be understated. In very lean animals, it can be difficult to achieve a good seal, however. Without

adequate tunnelling, there is a potential for atmospheric air to track along the chest drain during respiratory movements. In addition, if there is a poor ‘seal about the skin/tube interface as a result of poor tunnelling, during aspiration of the chest drain large volumes of air may be obtained giving the impression of pneumothorax, when in fact this air is simply being drawn from the atmosphere. This complication can sometimes be difficult to differentiate – although if >1000ml of air has been removed from a dog, with no signs of dyspnoea or change in respiration pre- and post- drainage, then it is likely that the air being withdrawn was not within the pleural space.

Pneumothorax

Pneumothorax occurs when there is an accumulation of air within the pleural cavity. A pneumothorax may be classified as open or closed, depending on whether there is patent communication between the pleural cavity and the external environment. A tension pneumothorax occurs when a damaged section of tissue acts as a one-way valve, effectively allowing air into the pleural cavity during inspiration, but preventing its escape. A tension pneumothorax will lead to rapid respiratory failure as the lungs are quickly prevented from expanding effectively due to the high intrapleural pressures. A pneumothorax may also occur as a result of a traumatic incident (blunt injury, penetrating injury), or can occur spontaneously.

Traumatic Pneumothorax

Traumatic causes represent the most common cause of pneumothorax in the dog and cat. Blunt trauma (e.g. kicks, falls, road traffic accidents etc) can cause rupture of the lung tissue or bronchial tree due to pressure overload or shearing forces [a closed pneumothorax]. Less commonly, direct puncture of the lung may occur due to penetrating injuries such as stake wounds, rib fractures, or ballistic injuries. Thoracic injury is common in both the dog and cat following road traffic incidents, with about a third of all cases have some degree of thoracic involvement. In two separate studies, pneumothorax occurred in between 36 and 50% of these cases.

Spontaneous Pneumothorax

Spontaneous pneumothorax occurs in purportedly healthy dogs, without any evidence of antecedent trauma. The pneumothorax may occur as a primary event (i.e. without any evidence of underlying pulmonary disease) or may be secondary to disease of the pulmonary parenchyma (e.g. pneumonia, neoplasia, parasitic disease. Perhaps the most common cause of spontaneous pneumothorax in the dog is due to rupture of sub-pleural blebs, cysts or bullae, (also termed bullous emphysema). The reason for development of sub-pleural blebs in lung parenchyma is unknown, but they occur with high frequency in tall male humans. Cigarette smoking is an important risk factor. Spontaneous pneumothorax appears to occur more commonly in the large breed, deep-chested dog suggesting a similar predisposition for sub-pleural bleb formation.

Diagnosis

Clinical signs of pneumothorax depend on the amount of air present within the pleural cavity, and the presence of underlying pulmonary disease. For example, in the road traffic injury patient, concomitant pulmonary contusions may have a significant impact on an animal's ability to tolerate a smaller amount of pneumothorax than an animal without this additional complication. Other physical injuries (e.g. fractures) may cause pain, leading to an increased respiratory rate and exacerbation of dyspnoea.

Thoracic auscultation usually reveals dull, muffled lung and heart sounds, especially dorsally. The chest will be hyper-resonant to percussion. Respirations will be shallow and rapid. With tension pneumothorax, respiration becomes ineffective, the chest is barrel-shaped and in maximal extension. The animal will be very distressed, cyanotic, and close to death without immediate relief of the pneumothorax.

Radiography

Bilateral pneumothorax is usually present, as air diffuses easily across the mediastinum. The sternal projection is again the most sensitive for detection of even a minor pneumothorax. The vascular pattern of the lung should extend to the lateral recesses of the diaphragm. Careful inspection of the radiograph, using a hot-light if necessary, may be required in some cases. When larger amounts of air are present, the lung lobes will be collapsed and atelectic. In a lateral projection, the heart will appear elevated from the sternum.

Radiographs should be carefully inspected for evidence of associated pulmonary disease (e.g. rib fracture, pulmonary contusion, neoplasia). Pulmonary blebs are only rarely visible.

Management

If the animal has an open pneumothorax, it is important to immediately cover the open thoracic wound with a sterile pad, and thus convert the chest to a closed system. Needle thoracocentesis can then be performed to evacuate air from the chest. Oxygen supplementation may be required, particularly if there is underlying pulmonary injury. The patient should be monitored carefully, to assess for recurrence of the pneumothorax. Needle thoracocentesis may need to be repeated if there is continual accumulation of air.

Placement of a chest drain should be considered if more than two thoracocenteses are required within a 24-hour period, or if air rapidly reaccumulates within the chest. Continuous drainage may be indicated for animals with large traumatic defects. This may enable quicker resolution of the injury as the chest is kept inflated, and an effective fibrin seal can form without being regularly displaced. Heimlich valves may be considered as an alternative to continuous drainage in some animals. However, they should only be used in medium to large breed dogs, and smaller animals are unable to generate sufficient expiratory pressure. The animal must also be monitored constantly, as the presence of fluid within the valve chamber will completely negate their function.

Surgical intervention is rarely required in animals with traumatic pneumothorax, and spontaneous resolution is the norm. Intermittent drainage may be required for 3-5 days, while the pulmonary injury heals.

Conversely, spontaneous pneumothorax rarely resolves without surgery. In two large studies, the pneumothorax in over 80% of animals did not respond to a combination of needle thoracocentesis or thoracic drainage, and surgical intervention was required. Surgical management was found to provide a lower recurrence rate than conservative care, the animals had a short hospital stay. However, surgical management is not straightforward. A large proportion of animals (75%) will have bilateral pulmonary disease, and this may necessitate the need for multiple complete or partial lobe resections. Exploration of the chest via a median sternotomy is recommended due to this high rate of bilateral involvement. The use of stapling equipment can greatly facilitate lung resection, which can otherwise be difficult from this approach.

A mortality rate of up to 30% is described for animals with spontaneous pneumothorax following medical or surgical therapy. Recurrence in the surviving animals may occur immediately many months after apparently successful management. Some authors also advocate the use of pleural abrasion intra-operatively in order to cause pleurodesis. They suggest that this may prevent recurrence of pneumothorax in animals with generalised pulmonary involvement.

Inflammatory Exudate ('Pyothorax')

Pyothorax is an accumulation of purulent exudate in the pleural cavity. The majority of pyothorax conditions will have a septic aetiology, with bacterial/fungal inoculation of the pleural cavity occurring as a result of penetrating wounds, migrating foreign bodies, haematogenous spread, or by direct extension of local disease.

Pyothorax can occur in any animal, at any time. There is no breed, sex or age predilection. However, younger male cats may be more prone than other cats (due to fighting and bite wounds to the chest).

Clinical Signs

Affected animals are presented due to signs referable to the pleural effusion. Septic signs may also be seen in these animals, including pyrexia, anorexia, weight loss and severe malaise. There may be a delay of several weeks between the injury which established the infection, and the onset of obvious clinical signs, so historical data may be unhelpful.

Gross inspection of thoracic fluid, combined with routine fluid analysis is useful diagnostic for pyothorax. The fluid is purulent in appearance (red/creamy/amber in colour, with marked turbidity due to high cell numbers). Heavy 'granules' may be evident within the fluid with *Nocardia* or *Actinomyces* infection. The fluid will have a high protein content, and cytological examination will reveal large numbers of neutrophils. The neutrophils may be degenerate, or non-degenerate, depending on the causative agent.

Fluid samples should be submitted for specialist laboratory analysis, including anaerobic and aerobic culture. However, it is not uncommon for a negative culture to be obtained, even when obvious features of infection are present clinically.

Treatment

Successful treatment of pyothorax requires prompt and aggressive management. Systemic stabilisation of the septic patient is essential, using intravenous fluids, antibiotics and nutritional support. This is combined with aggressive drainage of what is essentially a large 'abscess' within the pleural cavity. Effective management can only be obtained with a chest drain.

Following initial placement of the chest drain, the pleural cavity is copiously lavaged performed with warmed, sterile saline. No more than 10 ml/kg of fluid should be instilled into the thoracic cavity at any time. Irrigation is continued until the drainage fluid become clear. Ideally, the initial irrigation of the pleural cavity is performed under general anaesthesia, as this will allow higher volumes of lavage to be circulated through the chest without distress to the patient. As outlined above, initial stabilisation of the patient may be required with needle thoracocentesis and oxygen supplementation before anaesthesia is contemplated.

Following this initial lavage, subsequent irrigations of the chest cavity can be performed in the conscious patient. Irrigation is performed 2-4 times daily, depending on the severity of the condition. The addition of an antibiotic to the lavage fluid offers no advantage over systemic treatment. The choice of a systemic antibiotic will ideally be based on culture results, but in the event of a negative culture, the choice is often empirical. In most instances, a broad spectrum antibiotic with anaerobic cover, is effective. Treatment must be continued for at least 4-6 weeks to prevent relapse of infection.

With a pyothorax, there is a possibility that pockets of fluid/infection may become walled off from the rest of the chest due to thickening of mediastinal folds. A chest drain may also become entrapped within such a pocket. Two or more chest drains may need to be placed initially, and repositioning of the patient during drainage may also be required to facilitate complete evacuation of all lavage fluid. A common finding during treatment is to instil a large volume of saline, yet only obtain small amount back! In this instance, radiographs are useful to determine the position of the tip of the chest drain. It is not unusual for a drain to end up in the dorsal thoracic cavity as a result of respiratory movements. In this position, it is not surprising that it can be relatively ineffective in draining fluid from the standing

animal. Heparin added to the lavage fluid (1500 units/100ml of lavage) is also beneficial in reducing fibrinous pockets.

Surgical intervention is indicated if there is no improvement in the animal's condition within 3-4 days of treatment. Exploration is best performed via median sternotomy, as this allows both sides of the chest to be explored with ease. However, surgical manipulation of intrathoracic organs is more difficult via this approach, however. Surgical exploration allows necrotic tissue, foreign material, or localised abscesses to be removed. A thorough irrigation of the thoracic cavity should be performed prior to closure.

Prognosis

A reasonable prognosis is expected with prompt and aggressive management. Recent papers suggest that complete resolution of infection occurs in over 75% of patients that are managed with a combination of pleural drainage and antibiotics.

Fibrosing pleuritis may limit lung expansion and be a cause of peristent dyspnoea or exercise intolerance. Decortication of the lungs may be required in this instance.

Chylothorax

Idiopathic chylothorax is a frustrating disease, with consistently poor results being traditionally reported for a variety of treatment strategies. Thoracic duct ligation is the most widely accepted surgical method for treating animals with chylothorax. A recent paper has provided some encouragement for the successful management of chylothorax; the results of this paper are outlined in this short summary of chylothorax.

Introduction

The presence of chylothorax in an animal is usually suspected following removal of creamy-white or strawberry-milkshake appearing liquid on thoracocentesis. Chyle is lymphatic fluid that arises from the intestinal circulation. The chyle is returned to the systemic circulation via the thoracic duct, which travels in the dorsal thoracic cavity and drains into the major veins about the heart.

Traumatic 'laceration' or rupture of thoracic duct is not considered a significant cause of clinical chylothorax. If the duct is accidentally ruptured, healing has been shown to occur rapidly, and any effusion that may develop usually resolves without treatment.

Clinical disease is considered to be due to a lymphangiectasia of the thoracic duct, with diffuse leakage of chyle from along the length of the thoracic duct. The lymphangiectasia is due to increased flow, or altered pressures within the thoracic duct. Because the thoracic duct is a low pressure system, any disease or process that increases systemic venous pressures may impede chyle flow and lead to the development of a dilated and distended thoracic duct. Chylothorax is thought to arise in association with conditions that increase right-sided venous pressure or from obstructions to the flow of thoracic duct lymph into the venous circulation. Possible causes of chylothorax therefore include mediastinal lesions, heart disease (cardiomyopathy is a common cause of chylothorax is cats), cranial vena cava occlusion, increased hepatic lymphatic flow, and more generalised lymphatic disease. However, in the majority of dogs, no underlying disease is identified and the chylothorax is considered to be idiopathic in nature.

Diagnosis

Chylothorax may occur in both cats and dogs. Any breed, sex or age of animal may be affected. The Afghan Hound and Oriental breeds have a higher prevalence.

Coughing (which may precede the development of chylothorax by several months) has been recognised as a reasonably consistent feature of disease in some animals. The cough may be due to the irritation of the effusion, or may be part of the underlying disease process.

Once the effusion develops to a significant degree, classical signs of pleural effusion will be recognised. On thoracocentesis, the fluid classically has a 'strawberry' milkshake appearance, but may be creamy white in colour in some cases. The colour difference depends on the dietary fat content, and the presence of haemorrhage. Samples of fluid should placed in both EDTA and serum tubes for cytological and biochemical evaluation. A peripheral blood sample should also be submitted with the sample of pleural fluid.

The laboratory characteristics of chyle includes:

- Trigylceride content > serum
- Cholesterol content $\leq$ serum
- Cholesterol: Triglyceride ratio of fluid < 1.0
- Numerous small lymphocytes and non-degenerate neutrophils may be present

Following confirmation of a diagnosis of chylothorax, clinical investigation should turn to identifying a possible underlying cause of the effusion. Ultrasonographic examination of the pleural cavity, mediastinum and heart is essential before embarking upon management of the chylothorax. Other investigations (blood pressure evaluation, venography) may be selected in individual cases.

Management

Idiopathic chylothorax is a frustrating disease, with consistently poor results reported for successful management by a variety of treatment strategies. Although aspects of medical management will be consistently employed in many dogs, thoracic duct ligation is the most widely accepted surgical method for treating animals with chylothorax.

Medical Management

If an underlying disease is identified, treatment should be directed at reversal of the effects of this disease on the systemic circulation. Needle thoracocentesis of the chylous effusion will be required to relieve the dyspnoea.

Dietary management with a low-fat formulation has been recommended in an effort to reduce the amount of chyle being formed. There is no evidence that this strategy actually reduces the chyle volume, and most likely merely reduces the fat content of the fluid. This may render the fluid less irritable to the pleura and thus reduce a secondary inflammatory effusion. When regular intermittent needle thoracocentesis is being performed, the author's preference is to feed the animal a nutrient-rich, high calorie diet. This is because the animal is prone to loss of significant condition during the treatment as large amounts of fat and body proteins are being removed with the fluid.

Benzopyrone drugs (Rutin) have been used for the treatment of lymphoedema, and have been used with variable success for chylothorax. Medical grade Rutin has now been withdrawn from the market, but most natural health food shops will have an equivalent product. The potency of these 'natural' products may be questionable, however. The dose of rutin is 50 mg/kg, TID.

Surgical management

Surgical management of the chylothorax is indicated if there is no resolution of the effusion despite appropriate medical intervention. Thoracic duct ligation has become the procedure of choice. However, previous experience with thoracic duct ligation has been associated with only a limited success (50-60% in dogs, <40% in cats) In addition, in some animals, the chylous effusion changes to a serosanguineous effusion following surgery, which is equally intractable to treatment. Limited non-surgical treatment options are available and euthanasia is frequently performed in animals that do not respond to surgical therapy or medical palliation of clinical signs.

In animals with chylothorax, it is frequently observed that the pericardium and pleural tissues can be considerably thickened, probably as a result of chronic irritation by the chyle. A recent study (Fossum et al, 2004) has tested the hypothesis that this thickened pericardium would lead to continued elevations in systemic venous pressures and thus impede the drainage of chyle into existing lymphaticovenous communications following conventional thoracic duct ligation. The authors also hypothesized that serosanguineous effusions that occurred after TD ligation could effectively be treated or prevented by pericardectomy in affected animals. Treatment included a combination of thoracic duct ligation and sub-total pericardectomy. Thoracic duct ligation was performed through a caudal intercostal thoracotomy (8^{th}, 9^{th} or 10^{th} intercostal space). Subtotal pericardectomy was then performed through a separate intercostal incision (4^{th} intercostal space) or by reaching cranially in the thorax from the original incision. Ten dogs and ten cats were included in this study.

Excellent success rates were achieved in this study with effusion resolving in 90% of patients (100% in dogs, 80% in cats). This represents a considerable improvement on current treatment methods and thus provides some encouragement for the future management of this disease. Pericardectomy alone was not consistently successful with one dog requiring subsequent thoracic duct ligation to resolve a continued chylous effusion. The duration of 'reported' clinical signs was found to be a poor predictor of surgical success, since many owners may not be aware of low grade clinical signs until the disease is

quite advanced. Based on the results of this study the authors recommend the concurrent use of thoracic duct ligation and subtotal pericardectomy in any animal with idiopathic chylothorax, or when a serosanguineous effusion has occurred after thoracic duct ligation.

Thoracic duct ligation is a technically demanding procedure. Identification of the duct may be difficult in some animals, and a variable configuration of the duct is recognised, with some animals having multiple separate ducts. These issues can lead to uncertainty as to whether complete thoracic duct ligation was successfully accomplished. The use of intra-operative mesenteric lymphangiography is recommended to improve recognition of the duct and its various anatomical configurations, and to document successful closure of the duct. In the recent study discussed previously, a successful outcome was attributed to the role of an experienced surgeon. In this study, previous surgery (attempted thoracic duct ligation) had been performed in 9 animals by previous surgeons. Resolution occurred in 100% of these animals, suggesting that clinical experience may have a positive influence on treatment outcome.

Alternative remedies

When surgical management has proven unsuccessful, alternative interventions may be considered. These include pleuroperitoneal shunting, omental drainage, diaphragmatic mesh, and pleurodesis.

Pleuroperitoneal shunting requires the owners to operate a pump that has been surgically implanted into the dog. Although effective in some cases, significant short and long term complications can occur.

Sporadic cases reports are available for the other alternative therapies, and may not be suitable in every case. Again, high rates of failure are recognised as a potential complication.

Conclusion

Based on current experiences, thoracic duct ligation in combination with pericardectomy should be the recommended management strategy for animals with idiopathic chylothorax. It is important to state however, that until the aetiology of chylothorax is better understood, the success of palliative interventions is likely to be limited.

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Diaphragmatic Hernia

Introduction

Diaphragmatic hernia is a relatively common condition that may occur following a traumatic incident such a road traffic accident. Although most animals present almost immediately with signs referable to the traumatic incident, a proportion of animals (about 20%) will present many weeks to months after injury, despite having been apparently normal during the intervening period. Congenital forms of the disease also occur, but many of these animals will die immediately after birth due to severe respiratory compromise. However, congenital pericardioperitoneal diaphragmatic hernia causes less respiratory embarrassment and animals with this condition may not present until they are middle-aged or older, and indeed, may even remain asymptomatic for life.

Because road traffic accidents constitute the most common cause of traumatic diaphragmatic hernia, the signalment of animals that typically present with the condition reflects a group with a tendency to roam and a relative näivety of traffic i.e. young, adult male dogs. However, a wide variety of breeds and ages can be affected, and diaphragmatic hernia should be considered in all animals that present with acute respiratory distress.

Presentation and Clinical Signs

Animals with traumatic diaphragmatic hernia are usually presented after a road traffic accident. Cardiovascular compromise, due to shock, blood loss and pain, is the most significant concern and requires immediate attention. Assessment of mucous membrane colour, pulse rate, rhythm and pressure, and respiratory status should occur immediately. The violence of the trauma suggests that other soft tissue injuries may be present, including liver lobe laceration, bladder rupture, myocardial or pulmonary contusions. Diaphragmatic hernia is relatively uncommon (about 2% incidence) in combination with musculoskeletal fracture. Because most animals will tolerate disruption of the diaphragm without significant distress, the occurrence of progressive dyspnoea or cyanosis suggests the presence of significant concurrent injuries that are compromising the animal's ability to compensate adequately. Concurrent injuries that require attention include myocardial or pulmonary contusions, fractured ribs, flail chest and pain. Provision of intravenous fluids, colloids or hypertonic saline to stabilise the cardiovascular parameters, in combination with oxygen therapy and pain relief should be the first priority.

Animals with more chronic traumatic diaphragmatic hernia, or congenital pericardiodiaphragmatic hernia, may present with a wider variety of clinical signs, which may reflect a degree of respiratory compromise (dyspnoea, exercise intolerance), or may be attributable to the herniated organ (e.g. chronic vomiting, hepatic disease, ascites). The clinical status can deteriorate quite acutely in some of these animals, and will require an astute clinician to identify the cause (e.g. a liver lobe that becomes acutely entrapped in an old diaphragmatic rent, leading to significant pleural effusion and respiratory compromise). Aspects of the physical examination that may raise an index of suspicion for a diaphragmatic hernia include distortion of the apex beat, boborygmus audible on thoracic auscultation, and a general 'emptiness' of the abdominal cavity of intestinal content on palpation.

Diagnosis

Definitive diagnosis of traumatic pleuroperitoneal diaphragmatic hernia requires radiography or ultrasonography. Reliable interpretation of radiographs is often compromised if significant pleural effusion is present, thus obscuring soft tissue detail. In this instance, thoracocentesis may be necessary. Alternatively, ultrasound examination provides excellent visualisation of the diaphragmatic line and the pleural cavity, and is particularly helpful for animals with very subtle herniations (e.g. partial hepatic herniation only). The presence of fluid in the thorax will considerably improve the acoustic resolution of the ultrasound images. Radiographic signs of diaphragmatic hernia include loss of the diaphragmatic line, loss of the cardiac silhouette, presence of gas-filled viscus within the thoracic cavity, and cranial displacement of the gastric fundus or splenic shadow.

Abnormalities in laboratory tests with diaphragmatic hernia usually reflect compromise of the displaced organs, and may include increases in liver, biliary or pancreatic enzyme activity. The fluid obtained by thoracocentesis is usually serosanguineous, and analysis is consistent with a modified transudate.

Stabilisation and repair

Pre-operative stabilisation: Immediate surgical repair of the acutely ruptured diaphragm is usually contraindicated, unless there is life-threatening haemorrhage from the liver or other vessels, or the stomach is herniated and distended in the thoracic cavity. Mortality rates for surgery performed within the first 24 hours are significantly higher than if the animal is adequately stabilised first. Correction of cardiovascular shock with adequate intravenous fluid resuscitation is therefore the main priority. If the animal is dyspnoeic, immediate oxygen supplementation via a face mask, oxygen cage or nasal tube should be provided and attention given to the cause of the dyspnoea. Sitting the animal in sternal recumbency may improve pulmonary dynamics. Significant pleural effusion should be drained by thoracocentesis. Pain from abdominal or thoracic bruising, rib fractures or other orthopaedic injuries should be managed with effective opiate analgesia and splintage. Pulmonary contusions will require careful titration of intravenous fluids (to prevent worsening of pulmonary oedema), oxygen supplementation, corticosteroids, antibiotics and analgesia. Severe contusions may require ventilatory support if significant ventilation:perfusion deficits are present. Arrhythmias due to myocardial contusion and ischaemia are a significant concern, and can be associated with a higher mortality. They are frequently refractory to most antirhythmogenic drugs; ideal treatment consists of oxygen supplementation, analgesia and fluid support.

Anaesthesia considerations: A variety of anaesthetic agents may be used to induce anaesthesia in these patients. However, doses used in the trauma patient are likely to be significantly reduced. A smooth intravenous induction and rapid endotracheal intubation should be the objective of any protocol employed; mask induction should be avoided in animals with respiratory compromise. Providing oxygen supplementation prior to induction will improve myocardial oxygenation and minimise compromise during induction and intubation. Maintenance of anaesthesia is performed with an inhalation agent such as halothane (or isoflurane if myocardial arrhythmias are present). Nitrous oxide should not be used.

Surgical access for repair of the diaphragmatic hernia is best achieved via a ventral midline cranial laparotomy. Extension of the incision into a median sternotomy may be useful in some cases where extensive herniation, organ entrapment or adhesions exist. Careful monitoring of the animal is essential when the patient is placed into ventral recumbency, as significant respiratory compromise may be seen. Intermittent positive pressure ventilation should be performed if respiratory difficulties are seen.

As soon as the abdominal cavity is entered, anaesthetic management for an ‘open chest’ should be started. Controlled ventilation via intermittent positive pressure ventilation is obligatory, and will require a dedicated anaesthetic assistant. Good observation and communication between the surgeon and anaesthetist will prevent excessive over-inflation of the lungs (which may lead to expansion pulmonary oedema in chronic cases), and will allow the surgeon to perform manipulations (e.g. dissection of adhesions, suturing) within the thoracic cavity between inspiration.

Once the diaphragm is closed, ‘closed chest’ respiratory dynamics may resume and the animal may start to ventilate for itself, with assistance (by IPPV) if required. Depending on the chronicity of the case, it is usually not necessary to evacuate all of the air from the chest immediately after surgery. Indeed, it may be unsafe to do so, with re-expansion pulmonary oedema being a significant concern. Placement of a temporary chest drain will allow intermittent thoracocentesis in the post-operative period. Criteria for ‘how much air to remove’ are vague; I typically rely on pulse oximetry and ventilatory effort to gauge progress. Careful monitoring of mucous membrane colour, respiratory effort and rate in the post-operative period is essential. Oxygen supplementation via a nasal oxygen catheter should be considered in all cases. Provision of effective pain relief is also important.

Surgical management

Surgical management of most diaphragmatic hernias is relatively straightforward. However, complications can occur if inappropriate care is taken when repositioning organs, or when reconstructing the defect in the diaphragm.

A midline cranial laparotomy is the preferred technique for the repair of most diaphragmatic hernia. This approach can be extended into the sternum if necessary to maximise the exposure. Every care should be taken during replacement of herniated organs into the abdominal cavity. Careful assessment for injury or tissue ischaemia that may have occurred as a result of the traumatic incident or though vascular compromise as a result of incarceration should be made. Damaged and displaced parenchymal organs such as the liver and spleen are often considerably more friable than normal, and have a tendency to fragment and bleed during handling. Adhesions between abdominal and thoracic organs may be a significant concern, particularly in hernias that are more than a few days old. Early fibrous adhesions (3-8 days) can usually be broken down easily with gentle manipulation. More chronic adhesions require greater care, as damage to pleural surfaces may occur, resulting in pneumothorax or haemorrhage. Careful sharp dissection of the adhesions should be performed wherever possible. If the adhesion is too large, then a partial lobectomy of lung from the adherent organ should be considered.

Assessment of the diaphragmatic rent can usually be performed once all of the organs have been replaced in the abdominal cavity. The diaphragm may rupture circumferentially or radially, or may be a combination of the two. The left side is injured more frequently than the right side. Suturing should begin in the dorsal recesses of the wound, and advance ventrally. The diaphragm can be repaired with a simple continuous or interrupted pattern using a non-absorbable monofilament material, or an absorbable material with a prolonged duration of tensile strength retention (e.g. polydiaxinone). At all times, ensure suture bites are adequate to minimise the potential for dehiscence. Resection of scarred margins should be avoided as it increases the size of the defect, and risks dehiscence of the repair through excessive tension. If the diaphragm has torn circumferentially from the ribs, incorporate a rib in the repair for additional strength.

The use of synthetic or autogenous materials (e.g. omental flap) may be considered if the defect is too large to close primarily, or there is significant tension on the repair. This may occur particularly in chronic traumatic cases due to induration of the wound edges, or with large congenital pericardioperitoneal diaphragmatic hernias. The use of a vascularised muscular pedicle graft from the transverse rectus abdominal muscle has also been reported.

Closure of the abdominal wound may also be a problem in animals where the herniation has been long-standing. Contraction and atrophy of the abdominal muscles may lead to some difficulty in closing the linea alba when all organs have been returned to their rightful place. Abdominal closure needs to be performed with extreme care to ensure that the repair is sufficiently strong to tolerate the transiently increased tension on the wound, and to prevent inappropriate entrapment of viscera in the suture line.

Prognosis

Successful management of the animal with a traumatic diaphragmatic hernia requires application of a variety of resuscitative, diagnostic and peri-operative skills. The prognosis for recovery is good, but reported mortality rates range from 12-48%. Mortality rates are higher for animals operated within the first 24 hours or over a year after injury. With appropriate care, however, good success should be expected in more than 75% of animals. Possible complications include: pneumothorax, haemothorax and continued pleural effusion due to injury or damage to intracavitary organs (lungs/liver/spleen) that was not recognised at the time of surgery; infection; re-expansion pulmonary oedema; and reherniation due to dehiscence of the diaphragm wound. Reherniation has been reported up to 5 months after the original repair.