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CLOTS KILL: PULMONARY THROMBOEMBOLISM

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Pulmonary Thromboembolism (PTE) is a complex, challenging disease in small animal patients. Challenges exist not only in diagnosis of the disease, but also in differentiating PTE from other causes of diseases, which exhibit the same clinical signs. Excellent nursing management of these patients is especially important to maximize the chances of a successful outcome in a disease which has exceptionally high morbidity and mortality rates.

An embolus is defined as an obstruction of a vessel or vascular bed by an object such as a blood clot, fat, air, hair, bacteria, parasites, tissues, or foreign body. A thrombus is a blood clot. A PTE occurs in the pulmonary vessels. Thrombi may form within the right heart or somewhere else in the venous system, travel through the heart, and on to the pulmonary system once exiting the heart. Thrombi known as primary thrombi form in the pulmonary system itself; however, in veterinary medicine, both forms are referred to as PTE.

In our veterinary patients, PTE is often associated with other common underlying diseases and conditions. This fact often makes diagnosis of PTE difficult because common signs often include tachypnea and dyspnea, which are also prevalent in other cardiopulmonary diseases. Other signs may include cough, syncope, lethargy and altered mental state. When dyspnea is present, crackles or harsh lung sounds may be present on auscultation. Often, patients are also tachycardic. PTE should be suspected in patients who present acutely dyspneic who have had no previous diagnosis of cardiopulmonary disease especially if the animal has a disease considered to be a risk factor for PTE.

Predisposing diseases associated with PTE include, but are not limited to; heart worm disease, immune-mediated hemolytic anemia (IMHA), hyperadrenocorticism (Cushing's disease), pancreatitis, diabetes mellitus, protein-losing nephropathy, nephrotic syndrome, sepsis, systemic inflammatory response syndrome (SIRS), cardiomyopathy, disseminated intravascular coagulation (DIC), polycythemia, neoplasia, and trauma. Any disease that contributes to hypercoagulability, stasis of blood, or endothelial damage (Virchow's Triad) is a risk factor for PTE.

Once an area of the lung is obstructed, a complex series of events occurs resulting in hypoxemia of the animal. Ventilation-perfusion (V/Q) mismatch occurs when shunting of blood occurs due to the obstruction. Other changes in the lungs that occur with PTE include bronchoconstriction, atelectasis, pulmonary edema, and pleural effusion. Any area of the lung may be affected by a PTE; however, the caudal lungs lobes are more frequently involved since these lung lobes receive most of the right ventricular output.

This population of patients almost always requires IV catheterization; however, IV catheters should be considered an additional risk factor since catheterization damages vascular endothelium. The infusion of non-isotonic or fluids containing dextrose causes additional damage to vascular endothelium and may lead to PTE. It is important to consider catheter type and length in any patient at risk for PTE and even though central lines are often indicated in critical patients, these can become a cause for thrombi formation. Central lines are often contraindicated for patients with hypercoagulability. For peripheral catheterization, attempts to minimize tissue damage should be made. Catheter sites should be frequently examined for redness, swelling or the presence of phlebitis.

Thoracic radiographs are indicated with patients suspected of PTE; however, dyspneic patients should be stabilized prior to radiographs. Oxygen administration is indicated prior to and during radiographs. Sedation is often indicated in low doses to reduce anxiety and stress in these patients. Lateral and V/D views should only be attempted if the animal tolerates this positioning and does not struggle as the added stress of this positioning can cause the animal to decompensate. Despite excellent technique, radiographs in PTE patients are often normal or inconclusive. Radiographic changes that may indicate PTE include, right ventricular enlargement, decrease peripheral vessels in the affected area, and poorly defined alveolar or interstitial patterns. Pleural effusion may also be present. The lack of radiographic changes may not correlated with the severity of the patient's degree of dyspnea.

Obtaining an arterial blood gas is indicated in patients with suspected PTE. Technicians should be proficient in obtaining ABG samples. Approximately 80% of dogs with PTE show hypoxemia on blood gas analysis. Again, care must be taken when restraining patients for blood gases. Blood gas draws are clearly more stressful and require more restraint than a regular venous blood draw. Ideally, patients should be off oxygen and breathing room air for 5 minutes prior to obtaining an arterial blood gas sample; however, in this population of patients, that may not be possible. Patients may have normal blood gas values if they have not have previous pulmonary disease, or if the thrombus is small. Respiratory alkalosis may be seen with patients who are mildly hypoxic since tachypnea can result in hypocarbia. As hypoxemia progresses, a metabolic acidosis will occur which will further progress to concurrent respiratory acidosis as hypoxemia becomes severe.

Obtaining an echocardiogram may be useful in animals suspected of PTE. Echocardiograms do not provide a definitive diagnosis of PTE; however, they may provide indications of pulmonary hypertension or dilation of the right-sided chambers of the heart.

Arrhythmias may be found in patients with suspected PTE and may be due to acid-base disorders, electrolyte derangements, myocardial hypoxia, and increased levels of circulating catecholamines.

Testing for PTE remains challenging in veterinary patients. Ventilation-Perfusion (V/Q) radionuclide scanning can be used to detect PTE. This test involves two nuclear scans which may be done separately or together. With the perfusion scan, radiomarked albumin contrast is injected IV into the patient. An absence of contrast agent in a portion of the lung can indicate a PTE. With the ventilation scan, radiomarked gas is inhaled and images taken with a gamma camera. If the lungs are working properly, the air flow seen on the ventilation scan will match the blood flow seen on the perfusion scan.

CT Pulmonary Angiography (CTPA) is now considered to be the first line imaging test for PTE diagnosis. CTPA uses a multi-slice technique and has a high sensitivity and specificity. Sensitivity and specificity may vary somewhat depending upon the location of the emboli.

Other testing includes D-dimer measurements and thromboelastography (TEG).

Treatment of PTE is challenging as it involves attempting to reverse the pulmonary and hemodynamic changes caused by the PTE itself, as well as treatment of the initial underlying disease(s), which caused the hypercoagulable state of the patient. In dogs, thrombi undergo a 50% reduction in clot volume within a few hours due to fibrinolytic dissolution; however, if a hypercoagulable state still exists with the patient, new thrombi may form. It is also important to remember that thrombosis of additional organs is also possible. Supportive care with excellent nursing care is essential. Oxygen therapy is indicated either via an oxygen cage or nasal cannula. In patients who can tolerate it, passive range of motion is indicated to prevent blood stasis. Recumbent patients should be turned frequently to prevent further lung atelectasis.

Pharmacotherapies include heparin and warfarin as anticoagulant therapies. These agents prevent new clots from forming but do not dissolve clots already existing. Bleeding is a risk of using these therapies. Thrombolytic agents, such as streptokinase, TPA, and urokinase have also been used in patients with PTE.

Streptokinase is derived from streptococci species. Enzymes secreted by these streptococci bind and activate plasminogen in an effort to lyse the clot formation. The use of streptokinase in veterinary patients is often cost prohibitive. Tissue plasminogen activator (tPA) is a thrombolytic agent that can be used which acts mostly on fibrin. Mortality rates were shown to be about the same as treatment with heparin and was associated with an increase in hemorrhagic complications. tPA is extremely expensive with the estimated cost for a 20 kg dog being approximately \$21,000. Reperfusion injury may occur when existing clots dissolve causing further patient compromise.

Improvements and advances in early detection of PTE in veterinary patients are greatly needed. Since supportive care is often the only option, the veterinary technician provides a critical link to the attempt to decrease the morbidity and mortality rate of this complex disease. As with any critical patient, a fine balance must be achieved between what monitoring is essential in order to provide adequate support and intervention versus stressing a patient to the point where intervention is detrimental or even fatal. Respiratory distress patients are especially prone to succumbing to stress with manipulation, diagnostics, monitoring and radiographs. There is often no way to tell just what will tip the balance and cause a patient to no longer be able to compensate. Often these patients must be treated with a 'less is more' approach.

References available upon request.

Keywords: blood gas, dyspnea, pulmonary thromboembolism, PTE, Virchow's triad