Pulmonary Edema

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Pulmonary edema is the accumulation of fluids in the interstitium and alveoli of the lung. There are two main basic mechanisms for edema development: increased hydrostatic pressure in the lung capillaries (“high-pressure edema”) and increase vascular permeability (“low-pressure edema”). This classification helps understand the basic pathophysiological differences between the two types of pulmonary edema, but has limitations. Disruption of some or all layers of the alveolar-capillary unit occurs during elevated capillary hydrostatic pressures, a phenomenon termed “pulmonary capillary stress failure”. Pulmonary capillary stress failure represents a process that blurs the distinction between high-pressure and low-pressure pulmonary edema, as the disruption of the alveolar-capillary membrane by high hydrostatic pressures may render it more permeable to fluid and proteins. The resulting edema fluid has a higher concentration of protein than would be expected in conventional high-pressure pulmonary edema. These observations may explain some features seen in high-altitude pulmonary edema and neurogenic pulmonary edema.

High-pressure edema is usually secondary to left-sided congestive heart failure and many times called “cardiogenic pulmonary edema”, whereas low-pressure pulmonary edema are termed “noncardiogenic”. Compensatory mechanisms activated during heart failure cause sodium and water retention. Sodium and water retention increases circulating volume and venous pressure leading to transudation of fluids in body cavities (effusion) or interstitium (edema). These signs develop preferentially in the capillary beds drained by the failing ventricle. Thus, elevated pulmonary venous and capillary hydrostatic pressures lead to pulmonary edema and can be manifested as dyspnea, cough, pulmonary crackles, and exercise intolerance. Fluid in noncardiogenic pulmonary has a higher concentration of proteins and the edema occurs with normal capillary wedge pressure. The increased vascular permeability can occur with a wide variety of pulmonary and systemic disorders including vasculitis, acute respiratory distress syndrome, electric shock, neurogenic edema and uremic pneumonitis. Patients with pulmonary edema are usually presented with expiratory or mixed dyspnea with normal to increased lung sounds and presence of abnormal sounds (e.g.; crackles). A restrictive pattern of respiration is typical. In patients with restrictive respiratory disease, expansion of the lungs is restricted. Their lungs operate at smaller volumes and the patient has a rapid shallow breath. Radiographs are helpful in the diagnosis and differentiation between cardiogenic and noncardiogenic edema based on the distribution of the edema.

The initial goals of therapy in cardiogenic pulmonary edema include increasing arterial PO₂, reducing oxygen demand, establishing a diuresis, and unloading the ventricles while supporting blood pressure, tissue perfusion and renal function. Supplemental oxygen therapy and sedation are used as needed to reduce distress or air hunger. Pulmonary edema sufficient to cause respiratory failure and respiratory muscle fatigue is an indication for artificial ventilation. Diuresis is established with furosemide at an initial IV bolus of 2–5 mg/kg that can be followed by serial IV or IM boluses of 1-4 mg/kg every 6 to 8 hours or more frequently when necessitated by insufficient clinical response. The use of constant rate infusion (CRI) of furosemide also may be used to treat dogs and cats with life-threatening pulmonary edema. In healthy dogs and in human patients with CHF, furosemide CRI increases urine output and minimizes electrolyte disturbances when compared to repeated bolus injections. After the initial IV bolus of furosemide, the furosemide dosage required for the next 24 hours is estimated, and then infused by syringe pump. Supplemental boluses also can be given if required during the CRI. Nitroglycerin ointment can be used to decrease preload, whereas nitroprusside can be used to decrease afterload in dogs with florid pulmonary
edema. Two percent nitroglycerin ointment (¼ to 1 inch of the 2% ointment, topically q12h) acts primarily as a systemic venodilator, and this treatment is well-tolerated by both dogs and cats. Although some question exists about the efficacy of topically-administered nitroglycerin, the anticipated venodilation should work in concert with furosemide to lower venous and capillary hydrostatic pressures. The need for arteriolar dilators in the hospital setting depends on the cause and severity of CHF. Although vasodilator therapy has the potential to induce systemic hypotension, such treatment generally is safe in dogs when baseline ABP exceeds 95 mm Hg. For dogs with severe CHF, sodium nitroprusside (1–5 μg/kg/min IV by constant rate infusion), enalapril (0.5 mg/kg PO q12h), and hydralazine (1–2 mg/kg PO q12h) are effective vasodilators in the hospital setting. Each drug can increase stroke volume and reduce pulmonary edema. Afterload reduction is particularly useful in the treatment of severe mitral regurgitation arising from canine endocardiosis or when left ventricular dysfunction is evident, as in dogs with dilated cardiomyopathy.

Cardiac output, arterial blood pressure, and tissue perfusion are supported when necessary by providing inotropic support. In dogs or cats with severe systemic hypotension (ABP <80 mm Hg), inotropic support with dobutamine (2.5–10 μg/kg/min) or dopamine (2–10 μg/kg/min) is indicated. Catecholamines most often are administered to dogs in CHF caused by dilated cardiomyopathy. Occasionally, this approach is used in patients with severe mitral regurgitation or pulmonary embolism. Cats with any form of cardiomyopathy may develop cardiogenic shock characterized by bradycardia, hypothermia, and hypotension. Treatment with dobutamine can be life-saving in affected cats. Infusions should be titrated to a systolic ABP of 90 to 120 mm Hg and can be combined with slow external warming in an oxygen incubator. When treatment with catecholamines is impractical, oral administration of the calcium sensitizer pimobendan should be considered once this drug is available for general use.

There is no specific pharmacological treatment for noncardiogenic pulmonary edema. Diuretics are often ineffective and, despite widespread use, there is no evidence that corticosteroids are helpful. Support therapies include: controlling the causative factor, ventilatory support and maintaining the patient well hydrated.