UDC 636.3.09:616.04-005

CARDIOGENIC PULMONARY EDEMA IN SMALL PETS. A MODERN APPROACH

Kolosov S.O., higher education student of the program "Veterinary Medicine"
Scientific supervisor – **Tsymerman O.O.,** cand. of vet. sc., ass. prof.
State Biotechnology University, Kharkiv, Ukraine

Introduction: Cardiogenic pulmonary edema is a common and urgent condition that is frequently encountered in veterinary practice and can lead to respiratory distress, lethargy, and other life-threatening symptoms in affected animals. An understanding of the underlying pathophysiology and current approaches to diagnosis and treatment is crucial for veterinarians caring for these patients. The current clinical approaches to the treatment of cardiogenic pulmonary edema in small pets will be reviewed. This will include an analysis of common diagnostic tests such as chest radiography.

Despite the availability of a variety of treatment protocols aimed at eliminating underlying cardiac dysfunction and alleviating pulmonary congestion, there is still no consensus on optimal management strategies. Existing treatment protocols will be critically analyzed, including the use of diuretics, vasodilators, inotropes, and ventilatory support. Critical analysis of existing treatments, from pharmacologic interventions to more invasive procedures, provides an opportunity to identify potential areas for improvement.

Objective: To conduct a thorough analysis of existing protocols for the treatment and diagnosis of cardiogenic pulmonary edema in small pets, scrutinizing their effectiveness and compliance with evidence-based practice. To critically evaluate the diagnostic methods currently used to detect and evaluate cardiogenic pulmonary edema, including their respective advantages, including the use of vasodilators.

Methods:

1. A systematic review of the literature to comprehensively study current scientific research and publications on the pathophysiology, diagnosis and treatment of cardiogenic pulmonary edema in small pets.

2. Meta-analysis of relevant clinical studies and trials to quantify the effectiveness of various diagnostic tests and therapeutic interventions.

3. Retrospective analysis of clinical cases (cats n=18) (dogs n=13) to evaluate the results of different diagnostic and therapeutic approaches in real clinical settings

4. Expert interviews with leading veterinary specialists in the field of cardiology and pulmonology to obtain valuable clinical observations and opinions.

Clinical picture: based on a retrospective study of clinical cases, the mean age of patients presenting to the emergency department with cardiogenic pulmonary edema (CPE) associated with heart failure (HF), hypertrophic cardiomyopathy (HCM) is 10.1 years for cats (range 2.5-19.0 years) and 8.6 years for dogs (range 2.0-20.0 years). (The statistics were collected from veterinary clinics and hospitals Zoodiac, Emergency Veterinary Care, Druzhochok, HCV, Clinica Veterinaria la Fuensnata). A similar age at presentation was reported in a prospective study by Smith Dukes-McEwan, who reported a mean age of 9.0 years in cats (range 0.75-18 years (n=103))[1] . In both of the above studies, dyspnea and/or tachypnea were the main signs of acute heart failure (AHF). Tachypnea, apnea, dyspnea, forced, abdominal breathing in HF can have different causes, the most obvious of which is pulmonary edema or pleural effusion. These symptoms of respiratory distress are complicated by the pain syndrome that

certainly accompanies this condition, and can also be complicated by arterial thromboembolism (ATE)4 . Rectal temperature in patients with CPE was lower than normal, which may be associated with localized hypothermia in the case of ATE. Approximately half of cats with HF do not have auscultation signs associated with underlying heart disease, such as audible heart

murmurs, galloping sounds, or arrhythmias, which explains the difficulty of diagnosing HF in cats during physical examination. [5]

Unlike cats, significant tachycardia is observed in dogs with HF. However, sinus bradycardia may also be present, and its mechanism may potentially be related to concomitant hypothermia, current or previous pharmacologic treatment with beta-receptor antagonists (e.g., atenolol or propranolol), downregulation of myocardial beta-receptors, altered arterial baroreflex sensitivity, or any other dysfunction affecting the cardiac autonomic nervous system[2] . Pallor of the mucous membranes, weak pulse in the thighs, lethargy, and abdominal distension are other common signs, although none of these signs can be considered direct clinical evidence.[3]

Diagnosis: It should be noted that most patients with PNL demonstrate rapid manifestation of symptoms and complications of the condition when stress is induced. Therefore, all manipulations should be performed under conditions of reduced stress. The patient should be in a comfortable position on the back or sternum, which facilitates respiratory movements, and in no case should forced immobilization be used.[2]

Chest radiography is considered the "gold standard" for confirming the presence of IPF. This method can also detect signs of heart disease that have caused the manifestation of IPF. Radiologically, IPF initially appears as an interstitial opacification and due to the perivascular nature of the initial effusion, the blood vessels have less distinct walls. With the progression of congestion, the fluid tends to invade the lumen of the alveoli, creating an image of an alveolar type of opacification with ill-defined borders ("like cotton wool"), and this is usually the picture usually seen in the acute clinical picture. An enlarged left atrium and pulmonary veins larger than their paired arteries are typical signs of left ventricular heart failure. Diuresis can reduce the size of the heart and blood vessels, making them look normal or even small on radiographs. The anatomical distribution of alveolar pulmonary edema in cats is quite peculiar and may reflect different stages of the lesion at the time of radiographic examination. cats' PLE is often more random, patchy, and asymmetrical. The central and peripheral regions are usually more severely affected. In dogs, CPE usually begins in the larynx and spreads bilaterally and symmetrically to the central lungs. The peripheral lungs usually remain unaffected[7] .

Treatment: Sedation. Despite the minimal risk of respiratory depression, sedation is generally associated with reduced metabolic requirements, reduced anxiety and neurohumoral stress response, which in turn leads to improved respiratory muscle function respiratory muscle function, heart rate and blood pressure[5] . According to the authors, butorphanol is an effective sedative for patients with respiratory distress4 (at a dose of 0.05- 0.30 mg/kg IV, IM)[6] . Oxygen therapy: Supplemental oxygen therapy is recommended to reduce respiratory effort. In doing so, oxygen may temporarily help during manipulation or may provide a calm environment while waiting for medications to take effect[5] . Supplemental oxygen will be most useful for a patient who is hypoventilating due to respiratory muscle fatigue. Oxygen can be delivered by "flow-by", face mask, nasal clamps, or an oxygen cage[4] . Diuresis: Regardless of the underlying etiology of the condition, forced diuresis plays a key role in the pharmacologic management of heart failure although it does not directly improve cardiac output and can potentially lead to decreased renal perfusion and electrolyte disturbances[2] . The drug of choice is fursemide (1-2 mg/kg IV, IM every 0.5-4 hours, if necessary, depending on improvement in respiratory rate and respiratory effort. After improvement of clinical signs, increase the dosing interval to 6-12 hours, monitor urea, creatinine and

Electrolytes, and start oral therapy if tolerated)[6] . Use the lower end of the dose range for cats and observe for response[6] . Vasodilation: Reducing afterload with vasodilators increases cardiac output because afterload is often elevated in HF[5] . Nitroglycerin expands and redistributes blood to the abdominal cavity, away from the heart and lungs. It is available in various forms, but 2% ointment is most commonly used for small animals, usually applied topically (6-50 mm applied topically to the skin every 6-8 h)[6] . Inotropes: Currently the drug of choice is pimobendan[2] . The drug has the property of increasing calcium sensitivity, which improves contractility with minimal effects on myocardial oxygen consumption. Another mechanism of action is inhibition of

phosphodiesterase, which mainly leads to balanced vasodilation (arterial and venous) and possibly improved relaxation. (0.1-0.3 mg/kg orally, every 12 hours, 1 hour before meals)[6]

Conclusion: Cardiogenic pulmonary edema is a common, life-threatening disease in small animals. The speed of patient diagnosis and stabilization has a direct impact on the mortality rate of patients admitted with CPE. Further research through meta-analysis, case studies, and expert input may help improve diagnosis and treatment. A comprehensive, evidence-based approach, tailored to each patient, is crucial to achieve the best possible outcomes.

References:

1. Smith, S. and Dukes-McEwan, J. (2012), Clinical signs and left atrial size in cats with cardiovascular disease in general practice. Journal of Small Animal Practice, 53: 27-33.
2. Ferasin, Luca & Defrancesco, Teresa. (2015). Management of acute heart failure in cats. Journal of Veterinary Cardiology. 17. S173-S189. 10.1016/j.jvc.2015.09.007.
3. Fox PR, Sisson D, Moïse NS. Textbook of canine and feline cardiology : principles and clinical practice, 96-22328
4. 2nd ed. Philadelphia: W.B. Saunders; 1999;xvi, 955 p. Lesley G. King, Amanda Boag BSAVA Manual of Canine and Feline Emergency and Critical Care third edition; 93-123
5. Ferasin, Luca. How i treat congestive heart failure in dogs SEVC Barcelona 9-11 November 2023 4346PUBS18
6. Fergus Allerton BSAVA Small Animal Formulary 10th edition – Part A: Canine and Feline 2020 ISBN 978-1-910443-70-5
7. Muhlbauer, Mike C., Kneller, Steve Radiography of the dog and cat : guide to making and interpreting radiographs 2024 LCCN 2022058868