Acute and Chronic Pulmonary Thromboembolism in Dogs: A Prospective Clinical Study

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Pulmonary thromboembolism (PTE) in dogs lacks an evidence-based diagnosis. In humans acute and chronic PTE is diagnosed based on clinical probability, D-dimers and thoracic imaging. This study describes history, clinical, laboratory and echocardiographic findings in dogs with necropsy-confirmed PTE. Thoracic imaging findings are discussed in a separate study.

Dogs with clinical suspicion of PTE were prospectively included from 2019 until 2023. Physical examination, echocardiography, D-dimers, cardiac troponin I (cTNi), and viscoelastography were performed. Only dogs with confirmed PTE on necropsy after fatal outcome remained included. Data are expressed as median and range. Acute PTE was histopathologically characterized by few organized thrombi in pulmonary arteries and secondary surrounding acute lung lesions, whereas chronic PTE displayed chronic, fibrotic, organized thrombi within the pulmonary vasculature.

Twenty-four dogs were included, of which 9 were excluded (8 unconfirmed necropsy; 1 still alive). Ten had a histopathological diagnosis of acute PTE, 4 acute and chronic, 1 chronic. Chihuahuas (3) and shih tzus (3) were most commonly represented, median age and weight were 12 years [7–16] and 8.8 kg [2.60–31.4]. Eight had predisposing factors identified: hyperadrenocorticism (3), and severe pulmonic stenosis, pulmonary carcinoma, splenectomized hemangiosarcoma, protein losing enteropathy receiving prednisolone and acute kidney injury in individual dogs.

All dogs presented lethargy and dyspnea/tachypnea. Forty-three percent demonstrated low cardiac output (syncope and/or systemic blood pressure ≤90 mm Hg). All dogs had increased D-dimers (2273 µg/L [388–>6000]), whilst cTNi concentrations were increased in 7/10 dogs (443 ng/L [273.70–3542]), yet normal in 3/10 (24,8 ng/L [11.7–49]). Viscoelastography indicated hypercoagulability (5/8), hypocoagulability (3/8) and hyperfibrinolysis in a dog with hypercoagulability. Echocardiographic data were available for 13, of which 1 dog with pulmonary stenosis was excluded. Severe (6/12), moderate (4/12), mild (1/12) and absence of pulmonary hypertension (1/12) were identified based on pressure gradient and indirect echocardiographic changes. Right ventricular free wall appeared thicker and right ventricular normalized systolic area appeared enlarged in chronic PTE, yet these values did not differ significantly from acute PTE cases. Eleven dogs died during the initial PTE episode and 4 dogs died later with a median survival time of 62.5 days [12–279] (other causes [3] or PTE relapse [1]).

This study demonstrates two coexisting forms of PTE, which appear to be associated with a high incidence of fatality. In this population, D-Dimers were uniformly elevated whereas a majority of dogs demonstrated increased cTNi concentrations, abnormalities on viscoelastography and moderate to severe pulmonary hypertension on echocardiography.

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Speaker Information

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