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Case Report

Pericardial effusion associated with systemic inflammatory disease in seven dogs (January 2006 – January 2012)

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Abstract Pericardial effusion (PE) is reported in dogs as a consequence of neoplasia, primary cardiac disease or as an idiopathic condition. We describe seven dogs with systemic inflammatory disease, PE without tamponade and increased cardiac troponin I concentrations. Echocardiographic findings and adjunctive testing did not identify other known causes of PE. Resolution of the PE was documented in five of seven dogs in which follow-up echocardiography was performed, often after anti-inflammatory therapy. Resolution of PE was associated with normalisation of cardiac troponin I levels. Clinical signs had not recurred in six dogs with follow-up for more than 12 months and up to 7 years. These findings suggest an association between systemic inflammation and PE in dogs.

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Case 1

A 4-year 8-month-old female neutered border collie was referred with a 3-week history of pyrexia. Physical examination revealed neck pain, temperature 40.1 °C, heart rate (HR) 160 min⁻¹, respiratory rate (RR) 48 min⁻¹ and free fluid scanning had documented the presence of

pericardial effusion (PE), without evidence of cardiac tamponade. Routine diagnostics documented a stress leukogram (neutrophilia 17.86 × 10⁹/L; reference interval (RI) 3–11.5 × 10⁹/L) and hypoalbuminaemia (20.9 g/L; RI 28–39 g/L). Cardiac troponin I (cTnI)^a was increased (3 µg/L; RI 0–0.23 µg/L).

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^a Cardiac troponin I measured by chemiluminescent assay (Euro/DPC, then from 2008 became Siemens, Llanberis, North Wales).

Abbreviations

cTnl	cardiac troponin I
FS	fractional shortening
HR	heart rate
LA:Ao	ratio of the left atrial dimension to the aortic annulus dimension
LVIDd	left ventricular internal dimension at end-diastole
LVIDs	left ventricular internal dimension at end-systole
PE	pericardial effusion
RI	reference interval
RR	respiratory rate
SIRS	systemic inflammatory response syndrome
SRMA	steroid responsive meningitis arteritis

Echocardiographic measurements were made in M-mode for left ventricular internal dimension at end-diastole (LVIDd), left ventricular internal dimension at end-systole (LVIDs) and fractional shortening (FS) [1] and in two-dimensional mode for the ratio of the left atrial dimension to the aortic annulus dimension [2]. Echocardiographic measurements were consistent with systolic dysfunction (FS 8.8%; RI 23–47%, normalised LVIDd 2.01; RI 1.27–1.85, normalised LVIDs 1.72; RI 0.71–1.26). Pericardial effusion and mild mitral insufficiency were noted. Diagnostic pericardiocentesis revealed a neutrophilic exudate with no bacterial growth.

Further investigations by computed tomography revealed pleural fluid and an area of hyperintensity in the cervical musculature. Pleocytosis (protein content 0.53 g/L; RI 0–0.3 g/L) was identified on cerebrospinal fluid analysis. Cervical muscle biopsy revealed panniculitis and myositis with no bacterial growth. A diagnosis of steroid-responsive meningitis-arteritis (SRMA) was reached.

Treatment with prednisolone (3.22 mg/kg PO q 24 h) was instituted on day 2 and repeat echocardiography after four days revealed resolution of the PE, with subjectively similar systolic dysfunction. Prednisolone therapy was tapered by 25% every 4 weeks, for a complete course duration of 8 months, and telephone follow-up revealed her to be alive and asymptomatic 3 years after presentation.

Case 2

An 8-year-old male neutered whippet presented with an 11-day history of pyrexia. At presentation,

his temperature was 39.9 °C, HR 120 min⁻¹, RR 24 min⁻¹, and a grade I/VI systolic murmur was auscultated. Physical evidence of cardiac tamponade was not evident. Hypoalbuminaemia (22 g/L) and neutrophilia (23.52 × 10⁹/L) were present, and canine-specific pancreatic lipase immunoreactivity was increased (772 µg/L; RI < 200 µg/L). Cardiac troponin I was not measured. Echocardiography revealed decreased FS (12.7%) and increased normalised LVIDd (2.1; RI 1.27–1.85) and normalised LVIDs (1.71; RI 0.71–1.26). Pericardial effusion was present with mild diastolic collapse of the right atrial free wall that did not affect cardiac filling or chamber dimensions. Diagnostic thoracocentesis revealed a neutrophilic exudate (total nucleated cell count 45 × 10⁹/L and protein 33.5 g/L) with negative bacterial culture. Diagnostic ultrasound revealed abdominal lymph node enlargement and mild ascites but was otherwise unremarkable. Computed tomography confirmed PE, pleural effusion, and small volume ascites. Urinalysis was unremarkable. He was hospitalised with a diagnosis of systemic inflammatory response syndrome (SIRS) of unknown aetiology and treated with intravenous fluid therapy. Repeat echocardiography two days later revealed the resolution of the PE however fractional shortening remained reduced at 13%. Clinical signs, including abdominal effusion, resolved, and he was discharged with no medication. Follow-up confirmed continued survival 4 years after presentation.

Case 3

A 2-year-old female neutered mixed breed was presented for investigation of a 1 week history of pyrexia. At presentation, physical examination revealed evidence of neck pain, with a temperature of 39.1 °C, HR 120 min⁻¹, and RR 30 min⁻¹. Blood testing revealed hypoalbuminaemia (25.2 g/L) and neutrophilia (14.4 × 10⁹/L). Cardiac troponin I was increased (31.2 µg/L). Echocardiography confirmed small volume PE without tamponade, and no structural cardiac abnormalities. Fractional shortening was decreased (16%), but normalised LVIDd (1.8) and normalised LVIDs (1.42) were within reference intervals. Abdominal ultrasonography revealed minimal peritoneal effusion but was otherwise unremarkable. Cerebrospinal fluid analysis revealed mononuclear pleocytosis. Serology for *Toxoplasma sp.*, *Neospora sp.*, *Bartonella sp.*, and *Ehrlichia sp.*, were negative. Canine-specific pancreatic lipase immunoreactivity and urinalysis were within

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