A 5-year-old 12-kg (26.5-lb) sexually intact male American Cocker Spaniel had been evaluated because of a 15-day history of worsening exercise intolerance that included coughing, dyspnea, and abdominal distension. The dog was treated with prednisone (1 mg/kg [0.45 mg/lb], PO, q 24 h) and enrofloxacin (5 mg/kg [2.3 mg/lb], PO, q 24 h) for 10 days.

The dog was referred to the cardiology service at the University of Perugia because of a failure to improve after the 10-day treatment period. Physical examination revealed cyanotic oral mucous membranes, cold extremities, an irregular and weak femoral pulse, dyspnea, and ascites. During thoracic auscultation, crackles and wheezes were evident over the lung fields and a soft (grade II/VI) systolic murmur was detected over the mitral and tricuspid valve regions. Neutrophilic leukocytosis was detected on CBC. Serum biochemical analysis revealed high BUN and creatinine concentrations and high activities of aspartate aminotransferase, alanine aminotransferase, alkaline phosphatase, and creatine kinase. Serum cardiac troponin I concentration was high (4.23 ng/mL; upper reference limit, < 0.5 ng/mL). Thoracic radiography was not performed because the dog was dyspneic. A sinus tachycardia (170 beats/min) with sporadic ventricular premature complexes (8 single ventricular premature complexes in a 2-minute recording) was detected on ECG. An echocardiogram was obtained (Figure 1).

Determine whether additional imaging studies are required, or make your diagnosis from Figure 1—then turn the page →

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Diagnosis

Biventricular enlargement is evident. The left ventricle is hypokinetic (left ventricular internal dimension in systole, 40.5 mm; shortening fraction, 9%; ejection fraction, 19%), and a hyperechoic bilobed mass adherent to the interventricular septum is evident in the left ventricular lumen (Figure 2).

Further imaging revealed a second spherical hyperechoic mass in the auricle of the left atrium (Figure 3). Moderate mitral and mild tricuspid valve regurgitation was demonstrated by color Doppler echocardiography. Pleural and abdominal effusions were also identified on ultrasonography.

Differential diagnoses for the intracardiac masses included thrombosis, tumor, or nonvalvular endocardial vegetations. Because of localization (lack of adherence to valvular cusps) and echocardiographic appearance (regular in shape, not cauliflower-like, and without complex echogenicity), endocardial infectious vegetation was considered unlikely, leaving neoplasia and thrombosis as the 2 most likely differential diagnoses. The presence of profound myocardial dysfunction increased the likelihood of thrombosis. Differential diagnoses for biventricular congestive heart failure associated with severe myocardial failure in an American Cocker Spaniel were cardiomyopathy secondary to taurine deficiency, dilated cardiomyopathy unrelated to taurine deficiency, or myocarditis.1,2

Treatment and Outcome

The dog was treated with oxygen therapy, benazepril (0.5 mg/kg [0.23 mg/lb], PO, q 24 h), furosemide (2 mg/kg [0.9 mg/lb], PO, q 12 h), pimobendan (0.25 mg/kg [0.11 mg/lb], PO, q 12 h), aspirin (5 mg/kg, PO, q 24 h), taurine (500 mg/kg [34.1 mg/lb], PO, q 12 h), and carnitine (75 mg/kg [34.1 mg/lb], PO, q 12 h). The dog was reexamined after 30 days of treatment, at which time the clinical condition of the dog had improved and echocardiography revealed that the suspected thrombus in the auricle of the left atrium was no longer apparent. The suspected thrombus in the left ventricle was smaller and no longer bilobed. Left ventricular contractility was marginally improved (left ventricular internal dimension in systole, 38 mm; shortening fraction, 12%; ejection fraction, 27%). Treatment was continued. After 60 days, thrombi could not be detected in either chamber, but left ventricular contractility was not substantially improved (left ventricular internal dimension in systole, 38 mm; shortening fraction, 12%; ejection fraction, 27%). The pleural and abdominal effusions had resolved, and cardiac troponin I concentration had decreased to 0.2 ng/mL. Treatment was continued.

At 21 months after the initial diagnosis and onset of treatment, the dog was still alive and without clinical signs of congestive heart failure, but myocardial function remained compromised. The high serum cardiac troponin I concentration at initial evaluation (15 days after the onset of clinical signs), which normalized 60 days later, and lack of substantial improvement in echo-
cardiographic measures of contractility after treatment with taurine and carnitine were suggestive of myocardial failure secondary to myocarditis.

**Comments**

Intracardiac thrombus is a common complication of cardiac diseases in cats but is uncommon in dogs, especially in the left side of the heart. Thrombus formation in the left atrium has been reported as a complication of atrial fibrillation in 3 dogs. Intra-cardiac thrombi development has been described in cases of ischemic heart disease, dilated cardiomyopathy, myocarditis, and left ventricle dysfunction secondary to valvular heart disease in humans. Myocarditis predisposes to ventricular and atrial thrombi as a result of endocardial injury, procoagulant effects of cytokines, and blood flow stasis.

In the dog of the present report, dissolution of the thrombus in the left atrium and partially in the left ventricle was observed 30 days after the onset of treatment, whereas complete dissolution of the thrombus in the left ventricle was observed 60 days after the initial treatment. This difference in time to dissolution can probably be related to the larger size of the thrombus in the left ventricle than in the left atrium.

No clinical signs of systemic embolization were observed before or after the onset of treatment in the dog of the present report. Intracardiac thrombus formation in the left side of the heart and embolization of the thrombus to the systemic circulation are rare in dogs, compared with the incidence in cats and people. In the case described here, the thrombi in the left ventricle and atrium can be considered secondary to myocarditis or other causes of dilated cardiomyopathy, and their dissolution was observed after antiplatelet treatment (ie, aspirin) and conventional treatment for cardiac disease.