A 15-month-old 23-kg sexually intact male Vizsla was evaluated because of regurgitation and sialorrhea of 5 days’ duration. The referring veterinarian detected a tachyarrhythmia, and 3-lead ECG was performed. The dog was then referred for further investigation; the ECG findings were forwarded to the referral hospital. At the referral evaluation, the dog was bright, alert, and responsive. Evidence of sialorrhea was noted, and the dog’s mucous membranes were pink and moist with a normal capillary refill time. The dog’s heart rate was 88 beats/min with a regularly irregular heart rhythm; pulses were synchronous with the heartbeat. Respiratory rate was 24 breaths/min with no increased effort. Cardiac auscultation revealed no evidence of murmur or gallop sounds, and bronchovesicular sounds were unremarkable. Abdominal palpation revealed no abnormalities. The dog’s rectal temperature was 38.0 °C (100.4 °F). A neurologic examination revealed mild masticatory muscle atrophy and equivocally reduced jaw tone and gag reflex. Mild right pelvic limb lameness was also observed.

Hematologic and venous blood gas and electrolyte analyses were performed, and results were unremarkable. Serum biochemical analysis revealed high creatine kinase activity (2,334 U/L; reference interval, 67 to 446 U/L), mildly high alanine aminotransferase activity (146.9 U/L; reference interval, 19.8 to 124 U/L), and mild hyperbilirubinemia (4.9 μmol/L; 0.1 to 4.2 μmol/L). Serum cardiac troponin I concentration was 2.78 ng/mL (reference interval, < 0.03 ng/mL). Results of an immunofluorescence assay for Toxoplasma spp and Neospora spp were negative.

The dog underwent Doppler echocardiography, which revealed mild dilation of the cardiac chambers with reduced systolic function, consistent with a dilated cardiomyopathy phenotype. At this time, 6-lead ECG revealed sinus arrhythmia with a mean heart rate of 80 beats/min.

Thoracic radiography revealed mild changes compatible with aspiration pneumonia. Magnetic resonance imaging revealed multifocal intramuscular lesions supportive of myositis. Histologic examination of a temporal muscle biopsy specimen revealed inflammatory and necrotizing myopathy without obvious fiber loss or fibrosis, supportive of idiopathic inflammatory polymyopathy.

**ECG Interpretation**

The initial 3-lead (leads I, II, and III) ECG recordings (Figures 1 and 2) obtained by the referring veterinarian prior to referral of the dog were assessed. At the initial examination, the dog had narrow-complex tachycardia with a variable instantaneous ventricular rate, which ranged between 180 and 200 beats/min, and an atrial rate of approximately 460 depolarizations/min. No distinct P waves were observed. Instead, sawtooth deflections (also termed F waves or flutter waves) with a variable atrioventricular (AV) conduction ratio were present. The QRS-complex amplitude (R-wave amplitude, 2.2 mV; upper reference limit, 3.0 mV) and duration (QRS-complex duration, 0.04 seconds; upper reference limit, 0.06 seconds) were within reference limits. An ECG diagnosis of atrial flutter with an AV conduction ratio that varied from 2:1 to 5:1 was made. In transit to the referral evaluation, the dog spontaneously converted to sinus arrhythmia; during hospitalization and 2 subsequent recheck examinations, the dog had no recurrence of the tachyarrhythmia.

Figure 1—Three-lead ECG tracing obtained from a 15-month-old Vizsla that was initially evaluated because of regurgitation and sialorrhea of 5 days’ duration. Because the referring veterinarian detected a tachyarrhythmia during the initial examination, this ECG tracing was obtained at that time. Notice the broad, positive sawtooth waves (F waves) characteristic of atrial flutter. Paper speed = 50 mm/s; 1 cm = 1 mV.
Atrial flutter is a form of supraventricular tachycardia that occurs as a result of a macroreentrant circuit within the atria. The different conduction properties within the circuit help to maintain the constant electrical activity. Atrial flutter is rare in dogs, and it is most commonly associated with advanced structural heart diseases.²,³

There are different types of atrial flutter, which are broadly classified as typical atrial flutter or atypical atrial flutter. A subtype of typical atrial flutter is the reverse typical atrial flutter. The anatomic location responsible for the reentry mechanism in both types of typical atrial flutter is the cavotricuspid isthmus, formed by the caudal vena cava, Eustachian ridge, and tricuspid valve.²,⁵ Conduction can therefore occur in 1 of 2 directions. Atypical atrial flutter involves a different and more variable substrate that is not the cavotricuspid isthmus. In dogs, 2 isthmic locations have been described: one in the right septal wall and another in the right atrial free wall.⁴

The characteristic ECG appearance of a typical atrial flutter consists of characteristic sawtooth deflections, which are evident in tracings from leads II, III, and aVF.² With typical atrial flutter, absence of an isoelectric baseline in ECG tracings is common but not always evident. Atrial rates vary between 350 and 600 beats/min, whereas ventricular rates vary between 160 and 330 beats/min,²,⁴,⁵ depending on the AV nodal conduction ratio. The ratio may vary from 1:1 to 6:1 but is more commonly 2:1; such variation in AV nodal conduction, even within the same animal, can make the ventricular rhythm regular or irregular.² However, the different types of atrial flutter can have different clinical presentations, the features of which can overlap and mimic each other. These challenges regarding atrial flutter classification are common with surface ECG data, which leads to the frequent need for electrophysiological investigations to characterize the precise anatomic location of the macroreentrant circuit.¹,⁶–⁸

In dogs, radiofrequency ablation is the treatment of choice for persistent atrial flutter.⁴ Alternatively, medical management (including administration of lidocaine,⁹ sotalol,¹⁰ or a combination of quinidine and digitalis¹¹) can result in conversion to sinus rhythm. For the case described in the present report, the dog converted spontaneously to sinus rhythm before arrival at the referral hospital; therefore, antiarrhythmic treatment was not necessary. Paroxysmal atrial flutter was not evident during the period of hospitalization.

Inflammatory myopathies are a heterogeneous group of disorders that have previously been described in dogs. Depending on the extent of the disorder, inflammatory myopathies can be classified as either focal (most often affecting the masticatory muscles or the extraocular muscles) or generalized.¹²,¹³ The most common causes include immune-mediated processes, infectious conditions (including infection with Toxoplasma gondii, Neospora caninum, Borrelia burgdorferi, Rickettsia rickettsia, Ehrlichia canis, or Hepatozoon americanum), and paraneoplastic processes.¹² The diagnosis for the dog of the present report was idiopathic inflammatory polymyopathy, a condition to which Vizslas are genetically predisposed.¹⁴,¹⁵

This condition is an immune-mediated inflammatory process that primarily involves skeletal muscle. It is characterized by cellular infiltration, with masticatory and pharyngeal-esophageal muscles being particularly affected.¹⁶ A recent review¹⁶ described si-alorrhea, dysphagia, regurgitation, and masticatory muscle atrophy as the most common clinical findings associated with idiopathic inflammatory polymyopathy; all of those signs were evident in the dog of the present report.

Humans with idiopathic inflammatory myopathies may have cardiac involvement, the prevalence of which varies depending on patient selection characteristics and methods used to detect the cardiac involvement.¹⁷–¹⁹ Clinical signs of cardiac involvement are infrequent, with heart failure being the most common clinical problem.¹⁷,²⁰,²¹ On the other hand, subclinical heart abnormalities in association with idiopathic inflammatory myopathies seem to occur frequently; more than half of patients with idiopathic inflammatory myopathies and cardiac involvement have electrical disturbances.²⁰–²² Histologic examina-
tion of cardiac specimens from humans with a confirmed diagnosis of polymyositis may reveal changes consistent with myocarditis, including diffuse interstitial and perivascular mononuclear cell infiltrates and replacement fibrosis; these changes are similar to those detected in skeletal muscle.15,16

To the authors’ knowledge, clinically evident structural or electrophysiological cardiac abnormalities in Vizslas with idiopathic inflammatory polymyopathy have not been reported.15,16 Interestingly, a reported case series15 documented 1 dog that had severe, chronic, multifocal, lymphoplasmacytic, and histiocytic myocarditis with fibrosis and fiber loss identified during postmortem examination of the myocardium; however, no antemortem cardiac-related signs were evident. Echocardiographic findings for another dog from the same case series were unremarkable.15

The dog of the present report had systolic dysfunction. This could have been associated with myocardial involvement of the systemic disease (idiopathic inflammatory polymyopathy) or could have been secondary to the reported tachyarrhythmia (tachycardia-induced cardiomyopathy). Unfortunately, the exact cause of the systolic dysfunction could not be established.

References


