

Orthodromic Atrioventricular Reciprocating Tachycardia in a Dalmatian

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Abstract

Supraventricular tachyarrhythmias may be caused by macroreentry circuits involving the AV node and accessory pathways. This paper reports a case of suspected orthodromic atrioventricular reciprocating tachycardia in an 18-month-old Dalmatian admitted with dyspnea and a lifelong history of fatigue. Cardiac auscultation documented a regular fast pace with no heart murmurs. The electrocardiogram characteristics were consistent with supraventricular tachycardia, with very regular RR interval and narrow QRS complexes. At lead II, we identified negative P waves buried within the ST segment, which resulted in a RP-to-PR ratio of 0.60, but in aVR these P waves were positive, suggesting a retrograde conduction of electrical impulses throughout the atrial myocardium. The echocardiographic study showed volume overload, and a decreased fractional shortening was calculated when SVT was sustained, highlighting its impact on systolic function. This is likely the first description of an orthodromic atrioventricular reciprocating tachycardia in a Dalmatian, and although cardiac mapping was not available to confirm this suspicion, all electrocardiographic features were supportive of such arrhythmia.

Keywords

Arrhythmia, Tachycardiomyopathy, Accessory Pathway, Reentrant Arrhythmia

1. Introduction

Supraventricular tachycardias (SVT) include all forms of tachycardia that arise above the bifurcation of the bundle of His or have dependent mechanisms of it [1]. These rhythm disturbances are attributable to structural changes within the heart, which may be either associated or not with an accessory electrical conduc-

tion pathway [2]. Different SVT classifications have been reported, including the automatic supraventricular tachycardia (ASVT), atrioventricular nodal reentrant tachycardia (AVNRT), orthodromic atrioventricular reciprocating tachycardia (OAVRT), intraatrial reentry tachycardia (IART), sinoatrial nodal reentry tachycardia (SANRT), atrial flutter, and atrial fibrillation (AF) [3].

To occur, the OAVRT requires a reentrant circuit containing an atrioventricular accessory pathway [3], which is constituted by muscle fibers capable of generating action potentials and electrical conduction parallel to the atrioventricular node and His-Purkinje system between the atrium and ventricles [4]. These accessory pathways are either single or multiple, usually have bidirectional conduction capability and can be classified according to its location over the atrioventricular sulcus. In patients with OAVRT, the accessory pathway is located predominantly in the posteroseptal region and conducts electrical impulses in a retrograde fashion. This is different from the classical Wolff-Parkinson-White syndrome, in which the accessory pathway is solely used to conduct impulses antegradely [3].

This type of SVT was first described in 1967 by Coumel [5]; therefore it is also called a Coumel type tachycardia [6]. In people, OAVRT is considered a common arrhythmia, affecting mainly infants and children [3]. In dogs, however, OAVRT has only been reported in a few breeds, including Boxers, Labrador Retrievers and Beagles, which aged from 4 months to 11 years [4]. Also, a greater predisposition for the occurrence of this arrhythmia has been recognized in patients with tricuspid dysplasia [3].

The surface electrocardiogram (ECG) has a high sensitivity and specificity for the diagnosis of SVT [7]. However, studies suggest that misclassifications are likely to occur in 40% of cases when other diagnostic methods are not used [8]. In patients with OAVRT, the ECG is usually characterized by ventricular pre-excitation, retrograde P waves located at the end of the ST segment, RP-to-PR ratio below 1, and a high incidence of QRS alternans attributable to the fluctuating refractory period of the normal conduction system [3]. Also, this arrhythmia may occur either continuously, leading to a tachycardia-induced cardiomyopathy, or intermittently, resulting in signs of weakness and syncope [4]. The unceasing tachycardia is more likely to produce dilation of cardiac chambers and ventricular dysfunction, with signs ascribed to heart failure arising as cardiac output and tissue perfusion decrease [9] [10].

Because supraventricular arrhythmias are difficult to differentiate in clinical practice without cardiac mapping, the purpose of this report is twofold: to report an unusual case of macroreentry atrioventricular tachycardia in a young dog whose breed was never diagnosed with OAVRT and to discuss its clinical and electrocardiographic aspects.

2. Case Report

A male Dalmatian (18 months; 21.6 kg) was brought to a Veterinary Teaching

facility presenting respiratory distress. The medical history of the animal indicated a lifelong history of exercise intolerance, with successive episodes of severe fatigue over the past 12 months, and intensification of symptoms and worsening of the breathing pattern for a week. Auscultation documented a regular fast pace (>250 bpm—normal ranges are between 70 and 180 bpm [11]) without heart murmurs, as well as pulmonary crackles in the dorsal caudal lung fields. Also, mixed dyspnea and weak femoral pulses were noticed.

After the animal was given intravenous furosemide (3 mg/kg), an electrocardiogram was recorded, which documented supraventricular tachycardia (283 bpm), with regular RR intervals (210 ms). Also, a detailed analysis of tracings showed negative P waves buried within the ST segments (Figure 1), QRS alternans with narrow complexes, and positive P waves in aVR. The RP interval (77 ms) was shorter than the PR interval (127 ms), resulting in a RP-to-PR ratio of 0.60. Isolated and occasional ventricular premature complexes were observed on ECG tracings. Eyeball compression immediately restored sinus rhythm with first degree AV block, which persisted transiently until SVT resumed. All these findings substantiated the suspicion of orthodromic atrioventricular reciprocating tachycardia by accessory pathway.

The echocardiographic study showed an increased end-diastolic left ventricular diameter (Figure 2). Again, compressing the animal's eyeballs interrupted

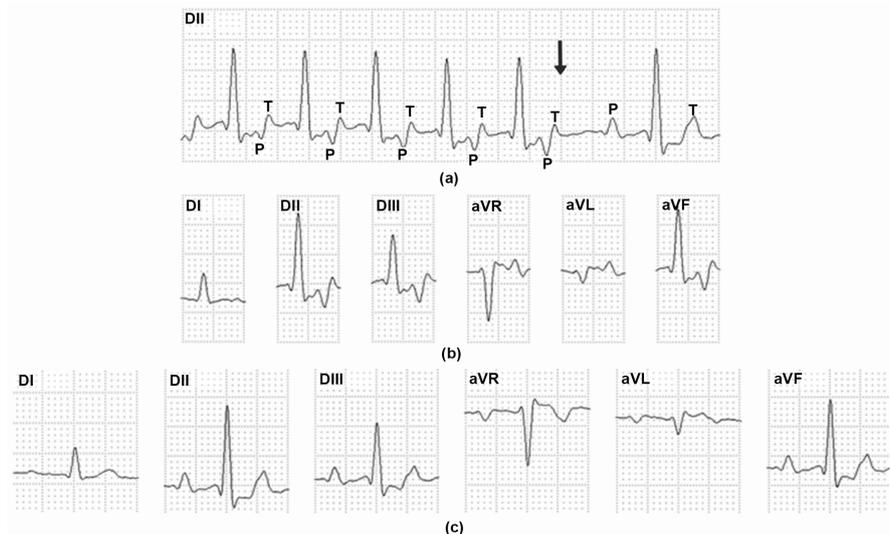


Figure 1. Electrocardiogram (50 mm/s, 1 cm = 1 mV) recorded in a young Dalmatian presenting respiratory distress and a history of chronic fatigue. (a) Supraventricular tachycardia (>280 bpm), with negative P waves buried within the ST segments, which resulted in RP becoming shorter than PR intervals. The arrow indicates the precise moment of eyeball compression, which resulted in a transient restoration of sinus rhythm with first degree AV block. Notice the prolonged PR interval and the T wave exhibiting a different morphology in comparison with preceding T waves during SVT; (b) Morphology of waves documented during SVT. A positive P wave in between the QRS and the T wave may be appreciated in aVR, indicating a retrograde electrical conduction within the atrial myocardium; (c) Wave morphology during sinus rhythm. First degree AV block is present (PR > 130 ms).

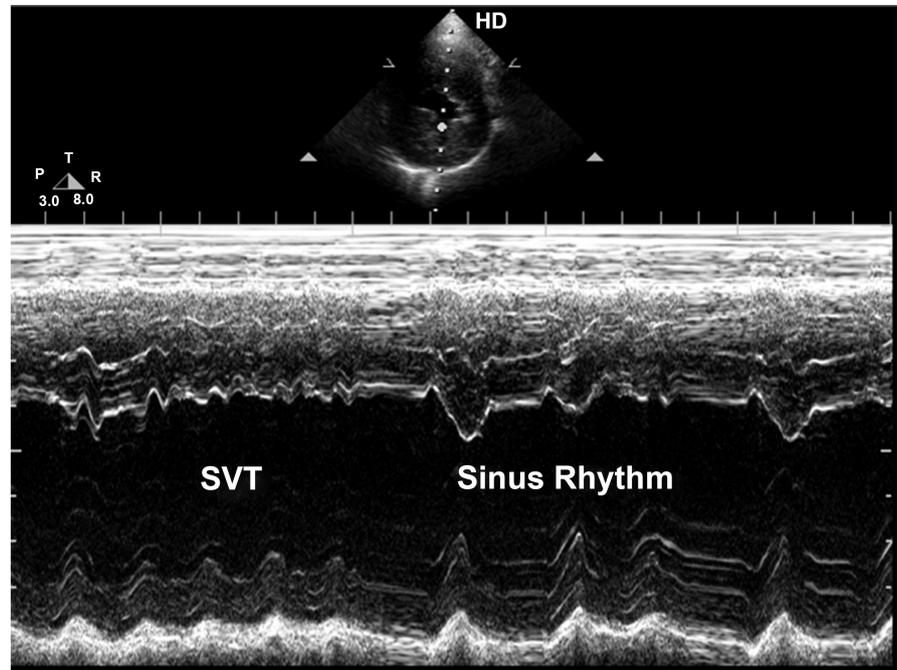


Figure 2. M-mode image obtained from a transverse view of the left ventricle, showing an increased end-diastolic left ventricular diameter, and two different contractile scenarios. During supraventricular tachycardia (SVT), there is an overt impairment of contractility, as demonstrated by the minimal change in ventricular chamber along the cardiac cycle. When sinus rhythm was transiently restored, the systolic displacement of both interventricular septum and left-ventricular free wall produce an evident reduction in ventricular lumen, therefore increasing stroke volume. The calculated shortening fraction was 23% during SVT and 28% under sinus rhythm.

the supraventricular tachycardia transiently, which allowed the calculation of the fractional shortening during arrhythmia (23%) and under sinus rhythm (28%), highlighting its impact on contractile activity. The heart valves were unchanged and competent.

Diltiazem was initiated at 0.5 mg/kg three times a day. Another electrocardiogram recorded 48 hours later documented sinus rhythm (134 bpm) with very regular RR intervals (450 ms), and spiked T waves (**Figure 3**). At that time the owner reported a more active animal and it remained that way for up to 3 weeks. There was no further contact with the owner after that.

3. Discussion

In this paper, we report a suspected OAVRT in a young dog with a lifelong history of exercise intolerance. This arrhythmia had only been previously documented in Labrador Retrievers, Beagles, Boxers and English Bulldogs [3] [4] [12] [13] [14], so this appears to be the first report of OAVRT in a Dalmatian. Also, this arrhythmia has been identified in dogs with age ranging from 4 months to 11 years [4], which is consistent with the patient that we report.

The prevalence of this arrhythmia in dogs is yet to be determined. In people there are contrasting opinions regarding the prevalence of OAVRT. While some



Figure 3. Lead II electrocardiogram (50 mm/s, 1 cm = 1 mV) recorded approximately 48 hours after oral diltiazem was initiated. A marked reduction in heart rate (140 bpm) was documented, and sinus rhythm with spiked T waves was observed throughout the examination.

studies included OAVRT among the most common atrioventricular reentrant tachycardias in the human population [3]-[10], other authors believe that such SVT represents an unusual diagnosis in people [6].

The clinical presentation of this animal included signs ascribed to heart failure. Patients with incessant SVT may present similar manifestations in less than 24 hours when the degree of ventricular dysfunction is important enough [9]. Thus, OAVRT can be incriminated as the primary cause of heart failure in this dog since this arrhythmia has the potential to produce a tachycardiomyopathy [6]. The same clinical presentation was previously documented in a Boxer dog supposedly diagnosed with OAVRT [3], besides a Labrador Retriever and an English Bulldog who carried out an electrophysiological study for diagnostic confirmation [12] [13] [14].

The SVT is characterized by increased heart rate and an abrupt start and end [7]. This arrhythmia is initiated and terminated spontaneously by atrial or ventricular ectopic beats, stimulated by ventricular pre-excitation [8]. In this report, the electrocardiographic evaluation demonstrated a SVT with a heart rate of 283 bpm and the presence of isolated and occasional premature ventricular complexes, representing the ventricular ectopic beats found together with OAVRT. Another feature of this arrhythmia is the rapid ventricular response consistent with the increased heart rate of this patient [3].

Although SVT are normally classified in accordance with their mechanism of action, the RP interval can also be used to categorize them in long RP tachycardias and short RP tachycardias. In this dog, we found the RP interval to be shorter than the PR interval, producing a RP-to-PR ratio lower than 1, which was previously shown to occur in OAVRT [1] [3] [6] [14]. The short RP depends on rapid retrograde atrioventricular electrical conduction, which is highly suggestive of an accessory atrioventricular pathway that induces reentrant circuit [14]. The negative P waves buried within the ST segment, as seen in lead II, are related to the posteroseptal location of the accessory pathway that is usually found in OAVRT, allowing each ventricular depolarization to spread toward the atrium [6] [7] [8]. In a study that investigated paroxysmal supraventricular tachycardias in dogs, a simultaneous 12-lead surface ECG was used and was identified significant differences between the polarity of P waves in leads II, III, aVR,

aVF, and V3 to V6 when OAVRT and focal atrial tachycardia were compared [15]. In this case, although the standard 6-lead ECG system was used instead, P wave polarities were similar to the most common findings of the aforementioned study. Finally, we found irregular QRS complexes that are likely attributable to changes in the electrical action potential of the His-Purkinje system [16].

The echocardiographic findings of this study are similar to those reported elsewhere, including increased left-ventricular diameter, decreased fractional shortening during OAVRT and a greater value after the restoration of sinus rhythm [3]. In people, SVT usually persists throughout the patient's life and the prolonged tachycardia is associated with impairment of cardiac contractile activity, causing a cardiomyopathy that might be reversible if SVT is to be effectively controlled [9].

When treating this condition, the main goal is to reduce the tachycardia zone, either to match the velocity of the normal conduction system and the accessory pathway or to interrupt conduction either way [3]. The initial strategy to stop SVT involves vagal maneuvers, which increase the vagal tone and, in turn, prolong the atrioventricular node refractory period [1]. In our case, this allowed the “normal” P waves to be visualized and favored the identification of the mechanism triggering the SVT.

Radiofrequency catheter ablation has been recognized as a safe and effective long-term therapy in human beings, especially those with hemodynamic compromise. Once the tachycardia zone is located, a specific catheter is used to ablate the myocardial tissue over that area, therefore reducing or stopping the arrhythmia completely [2]. In a study that recruited 35 people to undergo radiofrequency ablation, it was documented satisfactory results in 97% of the cases, which resulted in improved ejection fraction as compared to baseline data [17].

It was reported the use of successful radiofrequency catheter ablation in two dogs diagnosed with reciprocal atrioventricular tachycardia. After 8 months of the procedure, the animals remained asymptomatic and without recurrence of the arrhythmia. Although it is probably the most appropriate long term treatment in similar cases in dogs, such technique is hardly available to veterinary patients, therefore requiring medical treatment aimed at controlling AV conduction in the majority of cases [4].

Compared with other drugs, calcium channels blockers have advantages. Diltiazem, which was prescribed to this patient, has been recognized as the most effective drug to reduce the automaticity of the conduction bundles, besides having a longer acting duration [10].

The classification of SVT requires a detailed ECG assessment. It is also emphasized the importance of correlating arrhythmia, echocardiographic findings, and clinical signs. Finally, it is recommended the assessment of the patient's ECG during sinus rhythm whenever possible, since a wrong diagnosis might result in inappropriate treatment, therefore worsening the clinical condition of the patient [14]. All these recommendations were strictly followed in our patient and apparently contributed to substantiate the suspicion of OAVRT.

4. Conclusion

The diagnosis of OAVRT is challenging when standard ECG is to be used, which perhaps explains why it has been rarely described in the veterinary literature. It is a condition that clearly illustrates the role played by tachyarrhythmias in the development of congestive heart failure, even in the absence of morphological disarray of heart muscle and valves. Further studies are necessary so that its mechanisms of action, electrocardiographic features, treatment methods and real prognosis can be fully comprehended in dogs.

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