P’ wave axis analysis for the identification of sustained focal atrial tachycardia from low atrial origin - case report

Análise do eixo da onda P para identificação de taquicardia atrial sustentada com origem atrial baixo- relato de caso

DOI: 10.34188/bjaerv3n3-135

Recebimento dos originais: 20/05/2020
Aceitação para publicação: 20/06/2020

Pedro Pablo Martínez Padua
PhD em medicina veterinária pela Unesp/Jaboticabal, Brazil
Instituição: Betel Clínica de especialidades veterinárias
Endereço: Cll 140 # 13-66, Bogota
E-mail: pedromartinezpadua@hotmail.com

ABSTRACT
Anamnese: A 8-year-old female poodle was presented by a clinical examination, being evidenced tachycardia over the auscultation. The patient was referred to cardiac assessment. Clinical and laboratory findings: At admission the surface ECG showed sustained runs of complex atrial tachycardia, with a ventricular cycle length (R-R interval) of 240 ms. According to the leads ECG system to the P wave axis on the frontal plane, it present an inferior-to-superior, right-to-left axis; these characteristics were indicative of a Focal Atrial Tachycardia (FAT) from low atrial origin, arising from the Coronary Sinus Ostium (CSO). Treatment approach: Radiofrequency catheter ablation is the recommended treatment from FAT, but in the absence must to be used pharmacological treatment. The drugs of choice were a β-blocker and calcium channel blockers. Conclusion: The combination and the dosages used in the pharmacological treatment of this casewere effective, provided a good control on heart rate in dogs with FAT arising from low atrial origin.

Keywords: Canine, Arrhythmia, Holter, FAT.

RESUMO
Anamnese: Uma fêmea Poodle de 8 anos de idade, foi apresentada à nossa instituição para uma consulta, mostrando taquicardia na auscultação. O paciente foi encaminhado para avaliação cardíaca. Achados clínicos e laboratoriais: Na admissão, o ECG mostrou episódios de taquicardia atrial sustentada, com duração do ciclo ventricular (intervalo RR) de 240 ms. De acordo com o sistema eletrocardiográfico (ECG) do eixo da onda P no plano frontal, apresentava um eixo de inferior a superior, da direita para a esquerda, onde foram encontradas características indicativas de taquicardia atrial focal (TAF) com origem em foco baixo, decorrente do óstio do seio coronário (OSC). Tratamento: a ablação por cateter de radiofreqüência é o tratamento recomendado para FAT, mas, na sua ausência, deve ser utilizado tratamento farmacológico, sendo os fármacos de escolha os betabloqueadores e bloqueadores de canais de cálcio. Conclusão: A combinação e as doses utilizadas no tratamento farmacológico deste caso foram eficazes, proporcionando um bom controle sobre a frequência cardíaca neste cão com FAT de baixa origem atrial.

Palavras-chave: Canino, arritmia, Holter, TAF.
1 INTRODUCTION
ANAMNESIS AND MEDICAL HISTORY

Anamnesis

An 8 year old female poodle was presented to our institution by clinical examination, the dog was presented to the cardiology service for evaluation of an irregular heart rhythm identified on routine physical examination by the regular veterinarian.

Clinical findings

Blood work revealed normal complete blood count as well normal serum chemistry. On presentation, the dog was alert, responsive, with normal body condition score. The heart rate was approximately 220 beats per minute (bpm) with regular rhythm and tachycardia. A II/VI heart murmur was auscultated and the pulses were intensive. The rest of the physical examination was unremarkable.

Diagnostic data

An ECG was performed to evaluate the cardiac rhythm. The underlying ECG surface showed sustained runs of complex atrial tachycardia alternating with periods of sinus rhythm, with a ventricular cycle length (R-R interval) of 240 ms, negative P waves in the inferior leads (II, III and aVF), the arrhythmia at times is dominated by a sinus rhythm (Figure 1), the electrical axis in the frontal plane was of -40°, with PR duration in the normal limit of the reference range (80 ms), and a normal QRS configuration (Figure 2). When we have P-wave inversion in the inferior leads indicates a non-sinus origin of the P waves. when the PR interval is below of normality parameters, the origin suggest to be in the Atrio Ventricular junction (AVJ); When the PR interval is in normal parameters, the origin is within the atria as in this case (1, 2); Vagal maneuver were used by ocular pressure, to know if the arrhythmia was dependent or independent of atrioventricular node (AVN), suggesting an ectopic supraventricular origin independent of AVN (1, 2). (Figure 3). Thoracic radiographs and echocardiography were within normal limits, systolic blood pressure (SBP) was measured by vascular doppler, with average of 7 measurements, with an average value of 140 mmHg. “According to the electrocardiographic characteristics of the supraventricular tachycardia (SVT), four possible diagnoses were considered: focal atrial tachycardia (FAT) or supra ventricular tachycardia (SVT) originating from the CSO, atypical (fast-slow or slow-slow variant) atrio-ventricular reentrant nodal tachycardia (AVRNT), permanent junctional reciprocating tachycardia (PJRT) and orthodromic atrioventricular reciprocating tachycardia (OAVRT)”(1). Whereas, Previous studies
showed that atrial depolarization (AD) wave position may differentiate focal atrial tachycardia (FAT) from orthodromic atrioventricular reciprocating tachycardia (OAVRT), although at multivariate analysis only QRS complex alternans and P’ wave axis on the frontal plane resulted independent predictors of the arrhythmia type(1, 2).

Figure 1. Lead II: SVT from low atrial origin in the blue P, which at times is dominated by a sinus rhythm indicated in the red P.

Figure 2. Electrical P’ wave axis in the frontal plane was - 40°.
Figure 3. Vagal maneuver on ECG by ocular pressure, showing second degree AV block during tachycardia; suggesting independence from AVN.

The focal atrial tachycardia is an arrhythmia that is a part of the group of supraventricular tachycardia, and could be associated with underlying heart disease, but long-standing SVT could induce heart failure by systolic and diastolic dysfunction (2).

24 hours Holter monitoring was performed at his home; owners reported usual activities without exposure to physical exercise and habitual behavior was reported during Holter monitoring time; The Holter interpretation evidenced many episodes of sustained supraventricular tachycardia, HR
variability: 29.23%, Minimum HR (56-143 bpm) reported with deep sleep and resting without sleep. Middle HR (86-198 bpm), and maximum HR (207 - 250 bpm) (Figure 4). As the identification of the arrhythmia was a clinical finding in this patient, the fact that there were not manifestations of clinical signs strongly suggests that the patient had very short time of present the arrhythmia.

Treatment

After Holter evaluation, treatment was instituted with atenolol\(^1\) at a dose of 0.5 mg/kg/12hrs hours and diltiazem\(^2\) at 2.0 mg/kg/12 horas, and reassessment in one week.

One week later, the owner reported that the dog was doing well. The ECG was repeated which showed a predominant sinus rhythm with 132 bpm. Again, 24 hours Holter monitorization was performed at his home, the analysis showed, predominant sinus rhythm with few episodes of paroxysmal supraventricular tachycardia, HR variability: 41.82%, Minimum HR (78-112 bpm). Middle HR (98-123 bpm), and maximum HR (108-145 bpm) (Figure 5). Owners reported that during the examination the animal remained normal to them, SBP was measure with an average value of 130 mmHg.

At this time (four months later) the dog is going well with the medication, with values mean of SBP in 115 mmHg, and considered normal ECG (Figure 6).

\(^{1}\) Atenolol 50 mg. Lab Procaps S.A. Bogotá, Colombia.
\(^{2}\) Clorhidrato de diltiazem 60 mg. Lab Genfar S.A. Bogotá, Colombia.
Figure 4. Holter monitorization showing many episodes of SVT, HR variability: 29.23%, Minimum HR (54-143 bpm). Middle HR (198-96 bpm), and maximum HR (207-250 bpm).
Figure 5. Holter monitorization, showing: Predominant sinus rhythm with few episodes of SVT, HR variability: 41.82%, Minimum HR (78-112 bpm). Middle HR (98-123 bpm), and maximum HR (108-145 bpm).
2 DISCUSSION

Previous studies showed that atrial depolarization (AD) wave position may differentiate focal atrial tachycardia (1). The CSO is oblong in shape and located posterior and inferiorly on the interatrial septum, between the orifices of the inferior vena cava and the tricuspid valve, it is considered the major venous drainage vessel of the heart, commonly described as a short and wide venous channel, running from left to right, in the posterior part of the atrioventricular groove (3, 4, 5).

Since this tachycardia can present with the earliest site of atrial activation at the CSO area, only detailed electrophysiologic maneuvers can determine the exact underlying electrogenic mechanism (1, 6). To distinguish AVRT from AVNRT and FAT, must be effected electrophysiologic maneuvers during tachycardia, a timed ventricular extrastimulus could be introduced to analyze refractory period (7, 8).

Both, animals and humans have similarities on Heart’s irrigation; The characteristics on surface ECG were indicative of a Focal Atrial Tachycardia (FAT) from low atrial origin, arising from CSO. This type of tachycardia has been already described in veterinary medicine with the aid of endocardial mapping (9), there for, analyzing the leads system, the morphology of the P wave, and using vagal maneuvers, could be estimated the foci origin. Vagal maneuvers increase the refractory
period of the AV node, reduce the tachycardia zone, and blocking the AV node resulting in Wenckebach conduction (2, 10). In the diagnosis with vagal maneuvers is important to describe that, since we have second-degree AV block during tachycardia we can exclude any reciprocating tachycardias.

The diagnosis of the electrogenic mechanism of a supraventricular tachycardia can be only made by endocardial mapping (5, 9, 11). The use of surface ECG and Holter monitoring provide only a suspicious of diagnosis. Whereas, on depth analysis of the arrhythmia on the surface of ECG and according to the P' wave axis on the frontal plane it present an inferior-to-superior, right-to-left axis, we could suggest, this focal atrial tachycardia form low atrial origin is arising from the CSO (7, 11). The characteristics of FAT arising from CSO on the ECG surface, very similar like this arrhythmia have been described (1, 7, 9, 11).

In humans and dogs, have been described similars FAT, these tend to occur along the long axis of the crista terminalis, in the para-hisian region, and the CSO (5, 9, 12, 13). Tachycardias arising from the CSO have characteristics on P wave morphology on ECG surface, as were shown in this case. The P-wave must be deeply negative in leads II, III, and aVF, and positive, in Leads aVL and aVR. The P-wave in the precordial leads generally shows a uniphasic upright P-wave in rV2, which remains upright across most precordial leads (5). All these characteristics were evidenced on ECG surface from this dog.

The recommended treatment from FAT is radiofrequency catheter ablation, but in the absence must be used pharmacological treatment, to this patient, the drugs of choice were b-blocker and calcium channel blockers. Calcium channel blockers, and beta-blockers are drugs which increase the refractory period of the AV node, reduce the tachycardia zone, blocking the AV node. As well, beta-blockers have an action on potential duration (2, 10). Collateral effects are reported for both medications, not evidenced at the dosage to this dog, whereas; diastolic dysfunction with tachycardia and fast HR could produce sudden death. At the dosages used in this study, beta-blocker and calcium channel blockers therapy, provided a good control on heart rate in this dog with SVT arising from CSO. It is recommended to do ECG monitoring and SBP measurement every month for this type of patient, at this time our service does not consider appropriate to withdraw the medication; in such case, the SBP and ECG remain stable for a period of 6 months, it is suggested to gradually withdraw the medication for a period of 3 months until leaving without medication, always with the monthly accompaniment in mind.
CONFLICTS OF INTEREST

None.

REFERENCES

1. Santilli RA, Santos LF, Perego M. Permanent junctional reciprocating tachycardia in a dog. J Vet Cardiol. 2013; 15: 225-30.
2. Santilli RA, Bussadori C. Orthodromic incessant atrioventricular reciprocating tachycardia in a dog. J Vet Cardiol. 2000; 2: 25-9.
3. Dyce KM, Sach WO, Wensing CJG. Textbook of Veterinary Anatomy. WB Philadelphia: Saunders; 1996.p. 219–220.
4. Hamlin RL. Normal cardiovascular physiology. In: Sisson D, Fox PR, Moise NS, editors. Textbook of feline and canine cardiology. 2nd ed. Philadelphia: Saunders; 1999. p. 25–37.
5. Kistler PM, Fynn SP, Haqqani H, Stevenson IH, Vohra JK, Morton JB, Sparks PB, Kalman JM. Focal atrial tachycardia from the ostium of the coronary sinus. Electrocardiographic and electrophysiological characterization and radiofrequency ablation. J Am Coll Cardiol. 2005; 45: 1488-93.
6. Bensler JM, Frank CM, Razavi M, Rasekh A, Saeed M, Haas PC, Nazeri A, Massumi A. Tachycardia-mediated cardiomyopathy and the permanent form of junctional reciprocating tachycardia. Tex Heart Inst J. 2010; 37: 695 - 8.
7. Santilli RA, Spadacini G, Moretti P, Perego M, Perini A, Tarducci A, Crosasa S, Salemo-Uriarte JA. Radiofrequency catheter ablation of concealed accessory pathways in two dogs with symptomatic atrioventricular reciprocating tachycardia. J Vet Cardiol. 2006; 8: 157-65.
8. Santilli RA, Spadacini G, Moretti P, Perego M, Perini A, Crosara S, Tarducci A. Anatomic distribution and electrophysiologic properties of accessory atrioventricular pathways in dogs. J Am Vet Med Assoc. 2007; 231: 393-8.
9. Santilli RA, Perego M, Perini A, Moretti P, Spadacini G. Electrophysiologic characteristics and topographic distribution of focal atrial tachycardias in dogs. J Vet Intern Med. 2010; 24: 539-45.
10. Wright KN, Atkins CE, Kanter R. Supraventricular tachycardia in four young dogs. J Am Vet Med Assoc. 1996; 208: 75-9.
11. Santilli RA, Perego M, Crosara S, Gardini F, Bellino C, Moretti P, Spadacini G. Utility of 12-lead electrocardiogram in differentiating paroxysmal supraventricular tachycardias in the dogs. J Vet Intern Med. 2008; 22: 915-23.
12. Kalman JM, Olgin JE, Karch MR, Hamdan M, Lee RJ, Lesh MD. “Cristal tachycardias”: origin of right atrial tachycardias from the crista terminalis identified by intracardiac echocardiography. J Am Coll Cardiol. 1998; 31: 451–9.
13. Morton JB, Sanders P, Das A, Vohra JK, Sparks PB, Kalman JM. Focal atrial tachycardia arising from the tricuspid annulus: electrophysiologic and electrocardiographic characteristics. J Cardiovasc Electrophysiol. 2001; 12: 653–9.