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Sinus Node Dysfunction in A Dog with Mast Cell Tumor

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Abstract

12-year-old, 24.5kg, neutered male mongrel dog was admitted to the Oncology section of a referral veterinary hospital. The owner reported nodules in the right hind limb, perianal region and scrotum three months before. A local veterinarian performed nodulectomy of the limb's nodule and orquiectomy. After histopathological examination of the limb mass, the diagnosis was mastocytoma. The two remaining nodules exhibited a steady growth rate. No major complaints apart from sporadic lethargy and difficulty to stand up were reported. Since articular pain was noted at physical examination, carprofen was prescribed, after which the owner reported the dog became more active. Cytologic evaluation of the scrotal nodule was compatible with mastocytoma as well. During physical examination by the oncology veterinarian, a heart rate of 104bpm, rectal temperature 37.9°C, capillary refill time of two seconds, pink mucous membranes, strong femoral pulse, normal hydration status and normal body condition were documented. No abnormalities in cardiac or pulmonary auscultation were noted. Bloodwork revealed normal values of leukocytes, aggregated platelets and mild anemia (RBC 4.7 million/µL, PCV 33%, hemoglobin 11.3 g/dL, mild anisocytosis and mild hypochromia and anisocytosis). A few toxic neutrophils were also observed. The serum concentrations of creatinine, BUN, alkaline phosphatase and total plasma protein were within normal reference ranges. Alanine aminotransferase was slightly increased. Abdominal ultrasound revealed a reactive right medial iliac lymph node and urinary bladder microuroliths. However, the most remarkable finding was an oval hypoechoic structure (0.52cm x 0.50cm) within the splenic body and a heterogeneous structure (1.24cm x 1.00cm) located in the splenic tail. The most important differentials for these structures were metastasis, hematoma and nodular hyperplasia. Surgery for scrotal ablation was programmed for a few days later. The extirpated scrotal nodule was sent to histopathological examination, which confirmed the diagnosis of type II mastocytoma.

ECG Interpretation

A standard 10-lead electrocardiogram recorded for three minutes showed sinus arrhythmia (73 - 117bpm) alternating with junctional escape beats. Mean electrical axis had a marked left shift (-44°), QRS complex duration was 60ms, there was qR pattern in lead aVL and rS pattern in inferior leads (II, III and aVF). Together, these characteristics are diagnostic for left anterior fascicular block. In lead II, sinus beats had a P-wave of increased duration (63ms) and normal amplitude (0.20mV) with P-Q interval of 113ms. Junctional escape beats emerged after 843 to 1057ms of absence of sinus depolarizations. A maximum of three continuous escape beats were observed between sinus beats. The frequency set by the junctional ectopic focus ranged from 69 to 72bpm. Interestingly, junctional beats exhibited various P'-wave morphologies, including merged with the ascending portion of the R-wave, embedded on the S-T segment or on the T-wave [Figure 1], as sometimes they were absent. Additionally, four premature atrial complexes were documented [Figure 2]. Two weeks after extirpation of the scrotal mastocytoma, the dog was reevaluated. Another electrocardiogram showed sinus arrhythmia (93-163bpm) interspersed with a maximum of three continuous junctional beats (75-81bpm) and QRS complexes with left anterior fascicular block morphology [Figure 3]. Sinus rate increased notoriously after each junctional rhythm episode, which generated some bradycardia-tachycardia patterns [Figure 4]. No atrial premature complexes were noticed. Unfortunately, the owner declined chemotherapy protocol suggested by the oncology team. Almost two months after scrotal surgery, the dog presented sudden death at home.

Discussion

In this report, we present a case of sinus arrhythmia alternating with junctional escape rhythm conducted in both cases with left anterior fascicular block in a 12-year-old dog. A sinus node dysfunction is a likely diagnosis due to the age of the patient. Sinus node dysfunction is characterized by abnormal firing of the action potential from the sinus node, which causes an altered atrial rate unable to respond normally to physiological requirements [1]. This condition is more common in the elderly population in both dogs [2] and humans [1,3]. Electrocardiographic patterns compatible with sinus node dysfunction include sinus bradycardia, sinus arrest that lasts between 2 to 8 seconds and is ended by an escape beat, sinus standstill with junctional escape rhythm, varying degrees of sinoatrial block and periods of tachycardia followed by long sinus pauses (tachycardia-bradycardia pattern) [4].

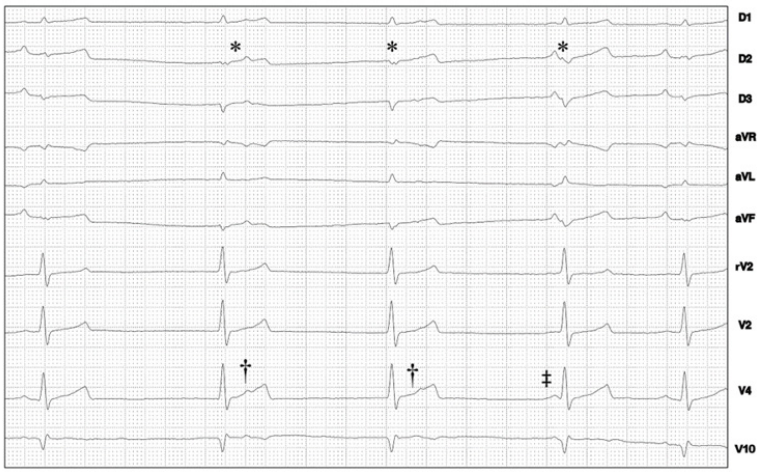

Figure 1: A ten-lead electrocardiogram shows a sinus standstill episode of 2.8 seconds with three junctional escape beats (rate 70 bpm) (*) that comes after a sinus beat. The first and second junctional beats have the P´-wave on the ascendant portion of the T-wave (†), and the third junctional beat has the P´-wave attached to the ascending portion of the R wave (‡). Paper speed: 50mm/s; 1cm = 1 mV.

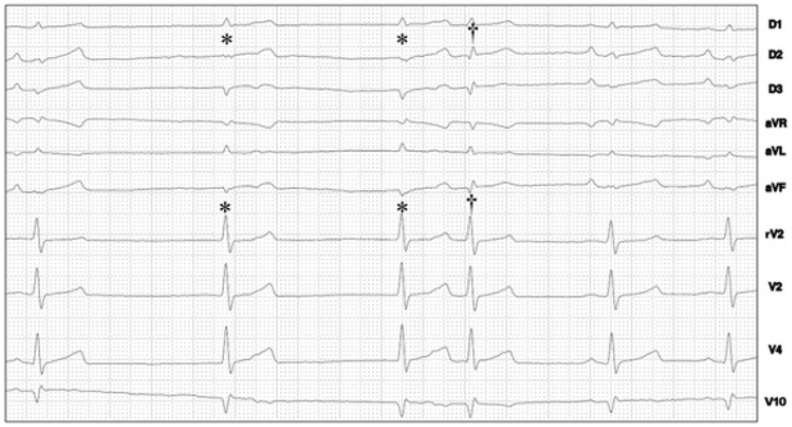

Figure 2: A ten-lead electrocardiogram shows how a sinus beat is followed by 643ms pause, which give rise to two junctional escape beats (71bpm) (*). After 333ms of the second junctional beat, an atrial premature beat emerges(†).Sinushythm resumes after 567ms. Paper speed: 50 mm/s; 1cm = 1 mV.

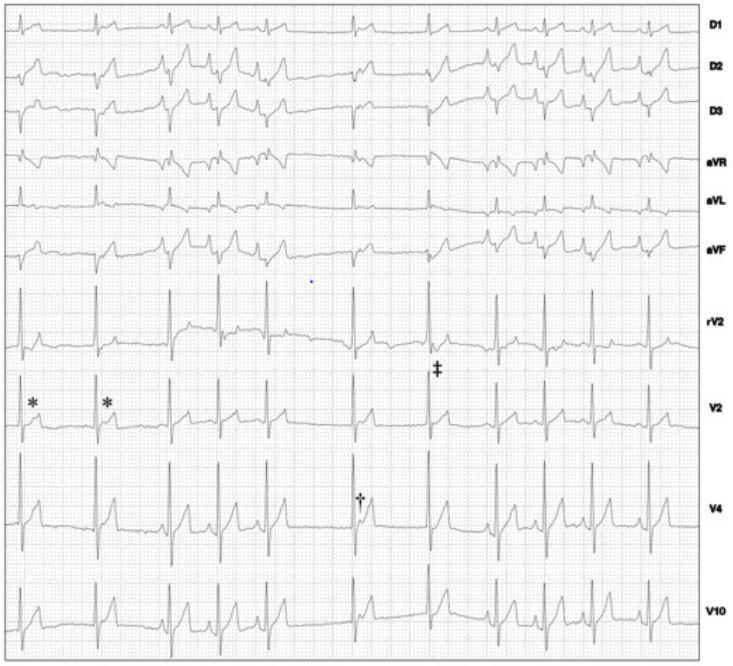

Figure 3: A ten-lead electrocardiogram illustrates a pause of 660ms after a sinus beat. Then, two junctional escape beats (76bpm) with the P'-wave embedded on the T wave (*) emerge. There is a pause of 673 ms after the second junctional beat, and sinus rhythm resumes (118bpm). Again, a pause of 643 ms gives rise to two junctional escape beats (76 bpm) with the P'-wave on the T wave (†) and on the S-T segment (‡). After 577ms of the second junctional beat, sinus rhythm resumes (120bpm). Paper speed: 25mm/s; 2cm = 1mV.

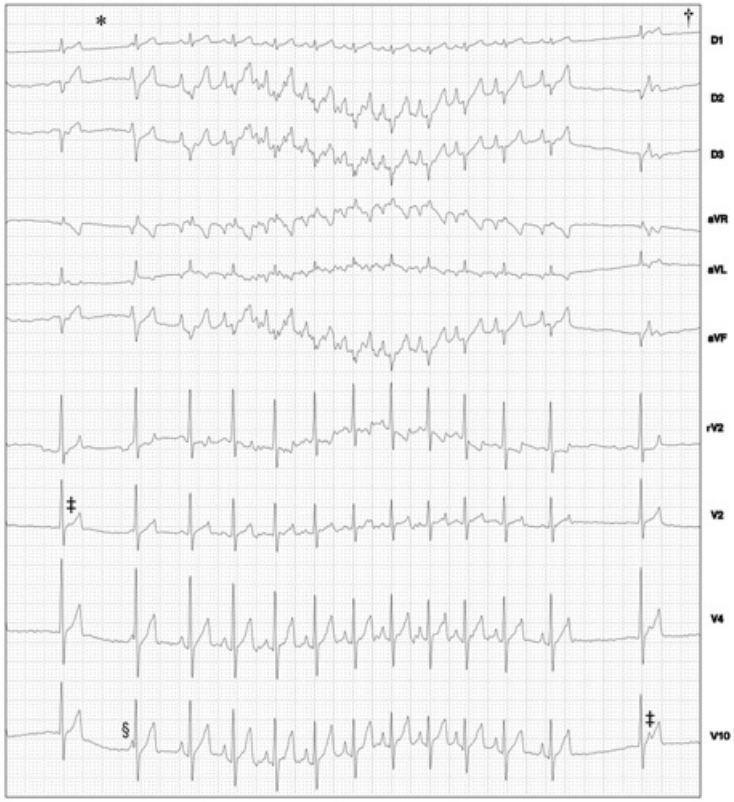

Figure 4: A ten-lead electrocardiogram shows two junctional escape beats (77bpm) (*) followed by a sinus rhythm that reaches a maximum of 163bpm. Suddenly, the sinus rhythm stops and comes a pause of 710ms. Then, two junctional escape beats emerge (75 bpm) (†). P'-waves due to concentric retrograde atrial activation are visible on the ascending portion of the T wave (‡) and attached to the ascending portion of the R wave (§). Paper speed: 25mm/s; 2cm = 1mV.

The electrocardiographic tracings we present are compatible with a case of sinus standstill as an early manifestation of sinus node dysfunction. Sinus standstill is a persistent or prolonged sinus arrest. The lack of depolarization coming from the sinus node is replaced by an escape junctional rhythm, or seldom, by atrial or ventricular escape rhythm. In dogs, the expected rate of firing of junctional subsidiary pacemakers is between 60 to 100bpm. A junctional ectopic beat that occurs after a pause is called junctional escape beat. More than three junctional escape beats with a rate closer to 60bpm is known as junctional escape rhythm [5]. The presence of concentric retrograde atrial activation is a typical feature of sinus standstill, as there is ventriculo-atrial conduction ratio of 1:1 evidenced by P'-waves in the first part of the S-T segment. In sinus standstill, P-waves are absent, QRS complexes have normal duration when originated from the junctional region (<70ms in dogs) and R-R intervals are regular [4].

Interestingly, our electrocardiographic tracings of sinus standstill had not only junctional P'-waves in the S-T segment. Accordingly, P-waves of junctional origin may be observed at different places or be absent, which depends on the site of origin of the junctional beat and the velocity of retrograde propagation towards the atria. Commonly, P'-wave is present before the QRS complex with a normal or short P'-Q interval when the ectopic beat originated at a superior area of the junctional region and/or the impulse traveled faster retrogradely than anterogradely. When both retrograde and anterograde velocities are similar, ventricles and atria become activated at the same time, therefore burying the P' wave within the QRS complex. Whenever anterograde velocity of propagation be faster than the retrograde one, P'-wave may be seen bulging in the S-T segment or in the T-wave [5].

Left anterior fascicular block (left anterior hemiblock) was present in all QRS complexes as a conduction abnormality. In this blockade, the anterosuperior portion of the left ventricle is depolarized through the Purkinje fibers that come out of the posterior fascicle and by cell-to-cell conduction of the functional myocytes [6]. The QRS complex duration is not altered importantly due to the dense Purkinje fibers that interconnect both fascicles [7]. However, there is left deviation to -30°/-60° of the mean electrical axis in the frontal plane. As a direct consequence of the precocious activation of the left ventricular posterior wall, the inferior leads show a rS pattern and aVL has a qR pattern. Left anterior fascicular block is one of the most frequently reported intraventricular conduction alterations in dogs and cats [6]. Fascicular blocks are commonly associated with left ventricular hypertrophy and fibrosis. Hypercalemia and ischemia might be associated as well. Although this intraventricular disturbance is not associated with hemodynamic disturbances [7], the cause of such alteration should be investigated [8]. In this patient, echocardiographic examination showed thickened mitral and tricuspid valves causing insufficiency, and an abnormal left ventricular filling pattern.

Both sinus node dysfunction and left anterior fascicular block may develop secondary to the increase in myocardial infiltration of fibrous and fatty tissue related to aging in elderly individuals [9,10]. However, mast cell tumors may also enhance the development of fibrosis in various tissues, including the myocardium [11]. This type of white blood cell cancer account for approximately 20% of all cutaneous canine neoplasia and are usually reported in older dogs [12]. Mast cell tumors release both pro- and anti-fibrotic substances when they degranulate. Some degranulation products known to promote myocardial fibrosis include chymase, triptase and TGF-β1, which trigger fibroblast activation, myofibroblast differentiation and collagen synthesis. Also, chymase is an independent angiotensin converting enzyme that generates angiotensin II, a known fibrosis activator [11].

Thyroid hormones status was not assessed in this patient, which makes thyroid disorders an additional differential. Subclinical hypothyroidism is reported to cause intraventricular conduction abnormalities secondary to atherosclerosis and myocardial fibrosis and bradycardia in humans [13]. Elevated serum concentrations of thyroid-stimulating hormone (TSH) with normal serum levels of free thyroxine are diagnostic of this condition [14].

Additional to clinical end electrocardiographic examinations, a thorough evaluation to diagnose sinus node dysfunction includes Holter examination and pharmacologic tests [2]. Unfortunately, we could not fulfill those two requirements due to sudden death of the patient. However, the presence of concentric retrograde atrial activation characteristic of sinus standstill, the presence of tachycardia-bradycardia pattern, and the markedly increased P-wave duration advocate for the presence of sinus node dysfunction and intra-atrial conduction problems.

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