J. Vet. Med. A **54,** 618–623 (2007)

ISSN 0931–184X
doi: 10.1111/j.1439-0442.2007.00972.x

Case Report

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Third Degree Atrioventricular Block and Accelerated Idioventricular Rhythm Associated with A Heart Base Chemodectoma in A Syncopal Rottweiler

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With 4 figures and 4 tables

Received for publication March 25, 2007

Summary

A 7-year-old male intact Rottweiler was presented with a 1-week history of lethargy, anorexia, vomiting and multiple syncopal events. The results of the clinical examination and electrocardiography were consistent with a third degree atrioventricular block and an intermittent accelerated idioventricular rhythm. Haematology, serum biochemistry, serology for *Borrelia burgdorferi*, blood culture, total T_4, thoracic radiography and echocardiography did not reveal the cause of the arrhythmia. Response to medical treatment with isoproterenol was poor. Pacemaker placement was declined by the owners and the dog was euthanized at their request. Histopathological examination of the heart revealed a chemodectoma at the base of the heart. There was no neoplastic infiltration of the conduction tissue. Potential mechanisms explaining the association of the arrhythmias and the tumour, such as vagal stimulation and neuroendocrine factors are discussed.

Introduction

Third degree atrioventricular (AV) block is a complete interruption of conduction from the atria to the ventricles, leading to dissociation of atrial and ventricular depolarization. Depolarization of the atria is controlled by the sinus node, whereas ventricular depolarization is generated by a subsidiary pacemaker, which is located below the area of the block and typically produces a life saving, slow, regular, autonomic rhythm, called escape rhythm (Tilley, 1992). Clinical signs of third degree AV block typically include exercise intolerance, weakness and syncope. Syncope can occur following prolonged asystole, the inability to increase heart rate in periods of excitement or exercise, or accelerated idioventricular tachyarrhythmias originating from an ectopic ventricular focus, leading to circulatory arrest and overdrive suppression of the subsidiary pacemaker activity. However, some animals may be asymptomatic (Kittleson and Kienle, 1998).

In the dog, third degree AV block has been associated with a variety of conditions including bacterial endocarditis (Robertson and Giles, 1972; Chomel et al., 2001), Lyme myocarditis (Levy and Duray, 1988), myasthenia gravis (Hackett et al., 1995), systemic lupus erythematousus (Malik et al., 2003), myocardial infarction (Jaffe and Bolton, 1974), hypokalemia (Musselman and Hartsfield, 1976), severe digitalis intoxication (Kovacevic and Zvorc, 1999) and chest trauma (Abbott and King, 1993; Nicholls and Watson, 1995). Other conditions, such as congenital abnormalities (isolated congenital AV block, aortic stenosis, ventricular septal defect), hyperkalemia, infiltrative cardiomyopathy (amyloidosis, fibrosis), hypertrophic cardiomyopathy and arteriosclerosis have also been cited (Tilley, 1992). In addition, hypothyroidism has been associated with various degrees of AV block (Panciera, 2001). However, in most cases, no underlying cause can be identified (Kittleson and Kienle, 1998). To the author's knowledge, there is only one case report describing a complete AV block associated with an aortic body tumour in a dog. The tumour in that case was highly invasive and had extensively infiltrated the right atrium (Patterson et al., 1961). The present case report describes the clinical and histopathological features of a dog with third degree AV block associated with a non-infiltrative aortic body tumour and discusses the possible pathomechanisms.

Case presentation

A 7-year-old intact male Rottweiler weighing 39.2 kg was presented as an emergency at the teaching hospital of the Veterinary Faculty of the University of Liege (Belgium) with a 1-week history of lethargy, partial anorexia and three episodes of alimentary and bilious vomiting. Since 2 days previously the dog had suffered a total of six syncopal events, characterized by stiffening of the forelimbs and opisthotonus followed by loss of consciousness and collapse. The syncopes were not related to meals or exercise and the dog recovered rapidly, without any post-ictal period.

On presentation, the dog was in good general condition but very lethargic. Body temperature, mucous membranes and capillary refill time were within normal limits. Respiratory rate

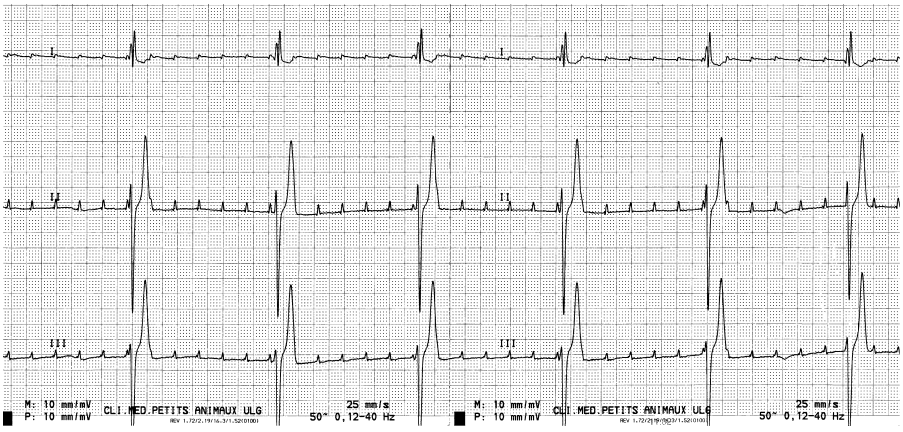

Fig. 1. ECG, right lateral recumbency (speed 25 mm/s, amplitude 1 cm = 1 mV). Complete atrioventricular block, P wave rate 200 BPM, ventricular escape rate 30 BPM.

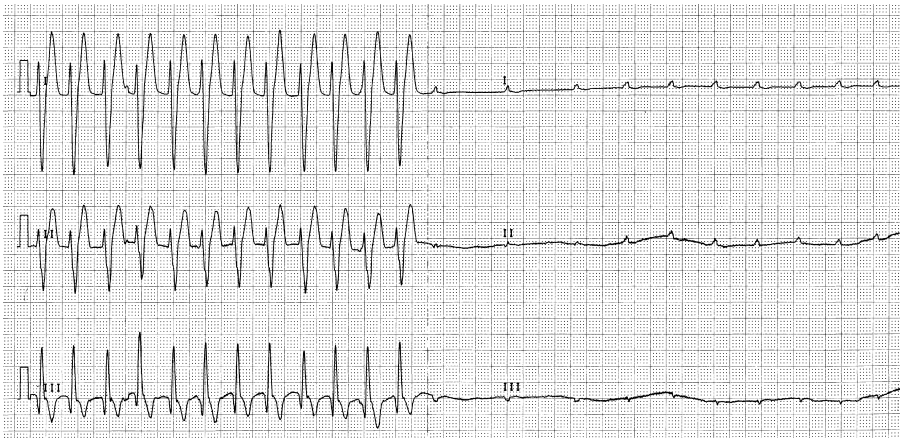

Fig. 2. ECG, right lateral recumbency (speed 25 mm/s, amplitude 1 cm = 1 mV). Intermittent accelerated idioventricular rhythm (150 BPM) with subsequent asystole due to overdrive suppression of the subsidiary pacemaker.

was 30 breaths/min. Thoracic auscultation revealed normal heart sounds with a regular rhythm at a rate of 32 beats per minute (BPM) associated with occasional runs of a regular tachycardia. Femoral pulses were strong, symmetrical and synchronous to the heartbeats. No jugular pulse was observed. The rest of the physical, including neurological examination, was within normal limits. During the initial examination the dog had two syncopal episodes.

Electrocardiography showed a third-degree AV block with a regular idioventricular escape rhythm at 30 BPM, a P wave rate of 150 BPM (Fig. 1) and an intermittent accelerated idioventricular rhythm at 150 BPM (Fig. 2). Telemetry confirmed that the syncopal episodes were secondary to prolonged ventricular asystole (15 s), which occurred on its own, and occasionally secondary to overdrive suppression of the subsidiary pacemaker by the accelerated idioventricular rhythm (Fig. 2). Blood was taken for a complete blood count and biochemistry profile. Samples for blood culture, total T_4 measurement and Borrelia antibody titre were also submitted. The results of the haematological and biochemical analysis are presented in Tables 1–3. A mild leukocytosis associated with a mature neutrophilia, eosinopenia and monocytosis were present, being consistent with a stress leukogram. Serum activities for ALT, AST and GGT were mildly increased. Alkaline phosphatase was moderately raised. A mild pre-renal azotaemia was noted. Serum potassium was in the high normal range and a mild hyperphosphataemia was present. Aerobic and anaerobic blood cultures and the Borrelia antibody titre were negative, total T_4 was in the low end of the reference range. Thoracic radiography showed mild generalized cardiomegaly, abdominal radiographs were unremarkable.

Table 1. Haematological data

Parameter	Day 2	Reference interval
Haematocrit (%)	38	37–55
Haemoglobin (g/dl)	13.1	12–18
RBC ($\times 10^6/\mu$l)	5.5	5.5–8.5
MCV (fl)	69	66–77
MCH (pg)	24	20–25 pg
MCHC (%)	35	31–34
WBC/μl	18 700	6000–15 000
Neutrophils/μl	16 456	3000–11 500
Monocytes/μl	1122	200–1000
Lymphocytes/μl	1122	1000–4800
Eosinophils/μl	0	< 750
Basophils/μl	0	Rare
Platelets ($\times 10^3/\mu$l)	312	200–500

Comment on blood smear cytology: normal, red and white cell morphology, mature neutrophilia.
RBC, red blood cell count; MCV, mean corpuscular volume; MCH, mean corpuscular haemoglobin; MCHC, mean corpuscular haemoglobin concentration; WBC, white blood cell count.

Echocardiography showed an increase in the left ventricular end-diastolic (LVDd), end-systolic diameters (LVDs) and E-point to septal separation (EPSS). The atria measured within normal limits. Fractional shortening was mildly

Table 2. Serum biochemistry

Parameter	Day 2	Reference interval
ALT (U/l)	216	11–58
AP (U/l)	461	14–39
Amylase (U/l)	382	0–1800
AST (U/l)	81	5–66
Cholesterol (g/l)	2.2	0.8–3.0
Creatinine (mg/l)	13.5	5–15
GGT (U/l)	34	5–8
Lipase (U/l)	83	< 60
Phosphorous (mg/l)	75	23–66
Potassium (mEq/l)	5.1	3.4–5.1
Sodium (mEq/l)	147	138–152
Total bilirubin (mg/l)	3.0	1–6
Total calcium (mg/l)	100	88–113
Total protein (g/l)	56	60–80
Urea (ctgr/l)	70	13–51

ALT, alanine aminotransferase; AP, alkaline phosphatase; AST, aspartate aminotransferase; GGT, gammaglutmamyltransferase.

Table 3. Miscellaneous

Parameter		Reference value
Aerobic and anaerobic blood culture	Negative	–
Borrelia burgdorferi antibody titre	< 1/64	< 1/64
Total T_4 (μg/l)	10	10–40

T_4, Thyroxin.

Table 4. Echocardiographic parameters

Parameter		Reference interval
IVSd (mm)	12.3	10–12
IVSs (mm)	14.4	16.3–17.8
LVd (mm)	57.0	43–47
LVs (mm)	43.8	26.8–29.7
PWd (mm)	15.6	8.6–9.8
PWs (mm)	16.2	13.9–15.3
EPSS (mm)	0.78	< 0.6
Ao/LA	WNL	< 1.5
FS (%)	23	25–45
Ao (m/s)	1.44	< 1.5
PA (m/s)	0.84	< 1.2
AI	–	–
PI	–	–
MR (m/s)	–	–
TR (m/s)	–	–
MV E/A	> 1	> 1
TV E/A	> 1	> 1

IVSd, interventricular septum at end diastole; IVSs, interventricular septum at end systole; LVd, left ventricular internal diameter at end diastole; LVs, left ventricular internal diameter at end systole; PWD, posterior wall at end diastole; PWs, posterior wall at end systole; EPSS, E-point to septal separation; Ao/LA, aortic diameter to left atrial diameter (short axis); FS, left ventricular fractional shortening; Ao, aortic flow; PA, pulmonic flow; AI, aortic insufficiency; PI, pulmonic insufficiency; MR, mitral regurgitation; TR; tricuspid regurgitation; MV, mitral valve; TV, tricuspid valve; E/A, E-to-A ratio.

decreased (Table 4). The echocardiographic findings were consistent with eccentric hypertrophy, most likely secondary to prolonged bradycardia.

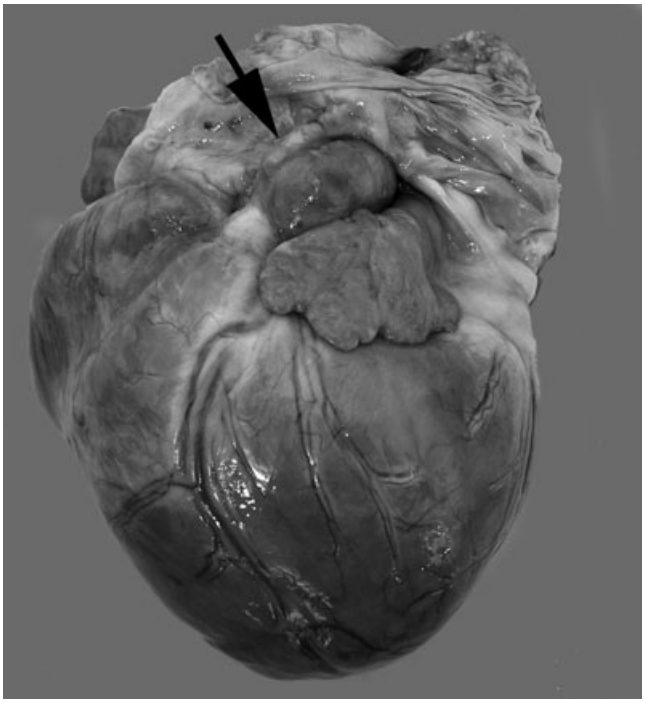

Fig. 3. Chemodectoma (4 × 2 cm) at the heart base. The mass was located between the medial aspect of the left atrial appendage and the ascending aorta and was attached to the external surface of the pulmonary artery.

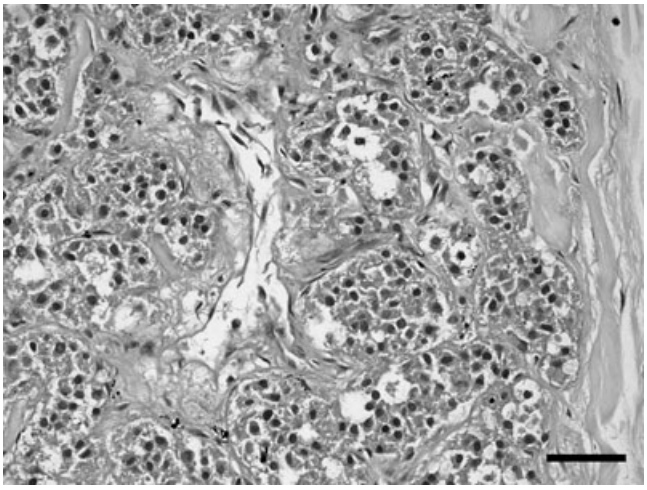

Fig. 4. Haematoxylin and Eosin stain, chemodectoma consisting of packets of ovoid or polyhedral cells with finely granular cytoplasm and central dark nuclei, separated by fibrous trabeculi.

The dog was treated with cage rest. To increase ventricular escape rate and inotropism, a continuous rate infusion of isoproterenol 0.05 μg/kg/min (Isoprel®; Abbott, Ottignies, Belgium.) was started. Broad-spectrum anti-biotherapy using ampicillin 30 mg/kg IV q8 h (Pentrexyl®; Bristol Myers, Brussels, Belgium.) and enrofloxacin 5 mg/kg IV q24 h (Baytril®; Bayer, Leverkusen, Germany) was commenced given that endocarditis or infectious myocarditis could not be excluded at this point. There was no sustained response to

isoproterenol treatment and the dog had five more syncopes during the night.

The next morning an atropine response test was performed to evaluate the vagal influence on the bradyarrhythmia. Following intravenous injection of 0.04 mg/kg of atropine sulphate (Atropine®; Sigma, Bornem, Belgium), the P wave rate increased from 160/min to 200/min but the rate of the ventricular escape rhythm did not alter (20 BPM). Another short run of the accelerated idioventricular rhythm was also observed. As the patient was now showing an increasing frequency of syncopal attacks and a poor response to the medical treatment, the placement of a pacemaker was advised to the owners, who opted for euthanasia. Autopsy of the heart showed mild right and left ventricular hypertrophy and the presence of a firm pale grey encapsulated ellipsoidal tumour (4×2 cm) at the heart base (Fig. 3). The mass was located between the medial aspect of the left atrial appendage and the ascending aorta and was attached to the external surface of the pulmonary artery. The mitral valve showed mild nodular thickening. Histopathology of the mass was consistent with a chemodectoma consisting of packets of ovoid or polyhedral cells with finely granular cytoplasm and central dark nuclei, separated by fibrous trabeculi (Fig. 4). Mitoses were not a feature. The nearby sinoatrial node tissue was normal in appearance. There were no significant changes in the atrioventricular node region. The histopathologist commented that there was no inflammatory or neoplastic infiltration of the cardiac conduction system in the AV node and the ventricular sections. The tumour cells were well-circumscribed and closely associated with the arterial walls rather than the cardiac muscle. Nerves were present in the connective tissue alongside the neoplasia but there was no direct association of nerves and tumour.

Discussion

Chemodectomas (non-chromaffin paragangliomas) are neoplasms of chemoreceptor cells. Chemoreceptor cells are situated near the aortic root (aortic bodies), carotid bifurcations (carotid bodies) and in other locations (glomus pulmonale, glomus jugulare), where they monitor the blood levels of oxygen, carbon dioxide tensions, blood pH and temperature, and participate via sympathetic and parasympathetic nerves in the regulation of respiration and heart rate (Hayes, 1975). In dogs, chemodectomas arise most often from chemoreceptor cells within the aortic bodies. The term aortic body tumour (ABT) is, therefore, used synonymously (Ware, 1994).

Aortic body tumours have been reported to be the second most common primary heart tumour in dogs, with brachycephalic breeds, especially Boxers and Boston Terriers, being predisposed (Ware and Hopper, 1999). Overstimulation of the chemoreceptors secondary to chronic hypoxia has been proposed as a possible aetiology of ATBs in humans (Edwards et al., 1970) and may offer an explanation for the increased occurrence of ABTs in brachycephalic breeds (Hayes, 1975). Clinical symptoms associated with ABTs are either a consequence of the local invasiveness of the tumour or pericardial effusion, which is usually, but not invariably present (Tobias, 2005). Various arrhythmias, such as premature ventricular contractions and ventricular tachycardia, have been reported secondary to heart base tumours (Owen et al., 1996) but, to the author's knowledge, third degree AV block has only been described once (Patterson et al., 1961). In the present case, syncope due to third degree AV block was the predominant clinical sign.

The results of the general blood panel were very unspecific. Elevations of the liver enzymes can be explained by hepatic hypoxia and congestion secondary to the bradyarrhythmia. Fatty degeneration of hepatocytes and liver enzyme elevations have been described in dogs with right sided heart failure (Glinska et al., 2005). However, a full postmortem was not performed in this case and a hepathopathy, therefore, cannot be excluded. Mildly increased urea with normal creatinine is consistent with pre-renal azotaemia, which can be explained by a decreased renal perfusion due to low cardiac output. Possible causes of a complete AV block, such as hypothyroidism and borreliosis, were ruled out by the normal test results. Bacterial endocarditis was excluded on the basis of absence of its major and minor diagnostic criteria (DeFrancesco, 2000). Mild generalized cardiomegaly observed on thoracic radiographs was considered to be secondary to bradycardia. Despite thorough echocardiographic examination by a board certified cardiologist, the mass at the heart base was only detected postmortem. Imaging of the heart base was difficult because of the broad-chested nature of the dog, the presence of a large amounts of intrapericardial fat and the absence of a pericardial effusion. This finding highlights the fact that heart base tumours can be extremely difficult to image and should, therefore, not be ruled out on the basis of a negative echocardiographic examination. The echocardiographic findings were interpreted as changes secondary to a chronic bradycardia: bradycardia induces a rise in ventricular diastolic pressures with a small increase in ventricular volume (Alboni et al., 1999). This could eventually lead to the development of overt congestive heart failure but this had fortunately not happened in our case.

Histopathology of the heart showed no abnormalities of the conduction system and also excluded myocarditis as a possible cause. The complete AV block, therefore, cannot be explained by an infiltrative lesion. Two different pathomechanisms for the development of a complete AV block associated with an ABT can be hypothesized.

First, ABTs are part of a tumour family called APUDomas (amine precursor uptake and decarboxilation). APUDomas are tumours derived from cells that have the ability to produce and secrete biogenic amines, such as adrenaline, noradrenaline, dopamine and serotonin (Feldman and Nelson, 2004). While ABTs in dogs have never been reported to secrete vasoactive substances (Morrison, 2002), they share many characteristics of neuroendocrine tumours, such as immunohistochemical staining for chromogranin A (Doss et al., 1998) and the presence of somatostatin receptors (Buchanan et al., 1998). Secretion of vasoactive substances, such as noradrenaline, adrenaline (Schwaber, 1984), dopamine (Levin et al., 1998) and serotonin (Jensen, 1994) – is a rare but well recognized feature in human chemodectomas. Symptoms caused by the tumour depend on the hormones secreted and their respective direct and indirect effects on the autonomous nervous system (Schwaber, 1984). Although in the present case, there are few clinical signs to confirm this hypothesis, hormone secretion by the tumour could be a possible explanation for the development of complete AV

block in the absence of a structural lesion within the conduction system. Means of investigation for this hypothesis would have been blood pressure and urinary catecholamine/creatinine ratio measurement, scintigraphy and immunohistochemical studies.

Another hypothesis could be based around the fact that stimulation of chemoreceptor cells, from which ABTs derive, are implicated in the regulation of respiration and heart rate via sympathetic and parasympathetic nerves (Hayes, 1975). Early investigators showed that stimulation of the carotid bodies in dogs caused cardiac slowing as part of the vagal depressor reflex (Heymans et al., 1931). In association with bradycardia, varying degrees of heart block have been observed as a consequence of an increased vagal efferent drive (Downing et al., 1963). An autonomic vagal stimulation by the tumour could be possible. However, a more recent study showed that, in contrast to the results of carotid body stimulation, experimental stimulation of the aortic body in dogs evoked a sympathetically rather than a parasympathetically mediated response, characterized by positive chronotropism and inotropism (Karim et al., 1980).

To determine a possible vagal origin of the complete AV block in this case an atropine response test was performed. In dogs with vagally mediated AV block, an increased P wave rate followed by an improved AV conduction and a normal sinus rhythm can be expected after atropine injection (Rishniw and Thomas, 2000). The lack of improvement of the AV conduction after atropine injection suggested that an increased vagal tone was unlikely to be the cause of the AV block in the present case.

A part from these hypotheses, it cannot be ruled out that the AV block was unrelated to the presence of the heart base tumour and that its association is purely coincidental.

Regardless of the underlying pathomechanism, treatment of the dog would have required pacemaker placement and continuous monitoring. To increase ventricular escape rate and inotropism, medical treatment with isoproterenol was attempted. However, response to isoproterenol in dogs is known to be poor (Rishniw and Thomas, 2000).

ABTs are usually slow growing and expansive (Ettinger, 2004), thus the tumour would eventually have become visible on echocardiography. Complete surgical resection of the ABTs is rarely achieved, but palliative pericardectomy has been shown to prolong survival greatly. Dog which had pericardectomy performed survived longer (median survival, 730 days; range, 1–1621 days) compared with dog that did not have pericardectomy (median survival, 42 days; range, 1–180 days) and had a good quality of life (Ehrhart et al., 2002).

Limitations of this case report include the lack of a full postmortem to rule out other concurrent diseases and further investigations, such as blood pressure and urinary catecholamine/creatinine ratio measurement, scintigraphy and immunohistochemical studies, which could have confirmed an underlying neuroendocrine pathomechanism.

In conclusion, a connection between the presence of a heart base chemodectoma and a third degree AV block in the present case, albeit unproven, appears very likely. In the absence of a structural lesion within the conduction system and a negative response to atropine administration, hormone secretion seems the most likely explanation for the development of complete AV block in this case.

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