

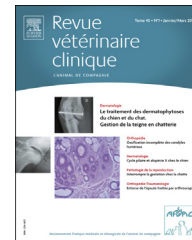


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CLINICAL CASE

Interstitial idiopathic pulmonary fibrosis and Sick-Sinus-Syndrome in a West Highland white terrier dog[☆]



Fibrose pulmonaire interstitielle idiopathique et Sick-Sinus-Syndrome chez un chien West Highland white terrier

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KEYWORDS

Pulmonary hypertension;
Dog;
Sick-Sinus-Syndrome;
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Lung

Summary An eleven-year-old West Highland white terrier treated for a Sick-Sinus-Syndrome since 2 years with a VVIR pacemaker was presented for exercise intolerance and panting due to interstitial idiopathic pulmonary fibrosis. Diagnosis was made before death by radiography, echocardiography and histology of lung biopsies. The follow-up was done before the dog died, 95 days after diagnosis. Necropsy was performed on heart and histology on sinus node also.
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MOTS CLÉS

Hypertension artérielle pulmonaire ;

Résumé Un chien West Highland *white terrier* mâle de 11 ans, traité auparavant pour un Sick-Sinus-Syndrome avec un pacemaker de type VVIR, est présenté pour une intolérance à l'effort et un essoufflement. Une fibrose interstitielle pulmonaire idiopathique du Westy est diagnostiquée du vivant de l'animal à l'aide de radiographies, d'une échographie cardiaque et

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Chien ;
Sick-Sinus-Syndrome ;
Pacemaker ;
Poumon

d'un examen histologique sur des prélèvements biopsiques pulmonaires. Le suivi est effectué jusqu'au décès du chien, 95 jours après le diagnostic. Un examen nécropsique est réalisé sur le cœur, ainsi qu'un examen histologique de la région du nœud sinusal.
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Description and case history

Nash, an 11-year-old male West Highland white terrier (WHWT) dog, weighing 9.5 kg, was presented for exercise intolerance and panting when at rest. The dog had been followed for 2 years (since 2011) for Sick-Sinus-Syndrome which had been diagnosed after episodic weakness associated with marked irregular bradycardia, and treated by implanting a VVIR pacemaker (ventricular pacing, ventricular sensing, inhibition response and rate-adaptive) with a unipolar epicardial pacing wire. Arrhythmia was confirmed and a complete pre-operative evaluation was performed at the time of diagnosis (Holter examination, echocardiography, biological examination, thoracic radiographs) before implanting the pacemaker. A control echocardiogram was obtained one year later (in 2012). The animal no longer presented any symptoms at this time (complete disappearance of syncope).

Clinical examination

The animal was alert on admission. Mucous membranes were pink, with a capillary refill time of less than 2 seconds. Respiratory auscultation revealed inspiratory crackles, most audible at end-inspiration, of great intensity, bilateral and diffuse ("Velcro Crackles"), associated with moderate polypnea (respiratory rate of 45 cycles per minute). Cardiac auscultation demonstrated a left-sided apical grade 2/6 systolic murmur and a right-sided apical grade 2/6 systolic murmur. The cardiac rhythm was generally regular. Some occasional irregularities were noted, probably secondary to rhythm changes linked to pacemaker triggering. The heart rate was between 70 and 80 beats per minute. Abdominal palpation was normal, together with the rest of the clinical examination.

Clinical history

An 11-year old male West Highland white terrier, already being followed for Sick-Sinus-Syndrome, treated by pacemaker implantation two years earlier, was presented for exercise intolerance and moderate polypnea at rest. The only anomalies detected in the clinical examination were moderate polypnea, left and right-sided apical grade 2/6 systolic murmurs and "Velcro" type pulmonary crackles.

Hypotheses

The hypotheses were as follow:

- breathlessness during effort, moderate polypnea, associated with left-sided apical systolic murmur of grade 2/6 and respiratory noises most audible at the end of inspiration, of strong intensity, bilateral and diffuse, maybe secondary to a pulmonary edema (left heart failure) secondary to a degenerative mitral valve disease;
- in view of the epidemiological context (age, predisposition of terrier breed), an interstitial pulmonary fibrosis was also possible, which would explain the "Velcro Crackles". The grade 2/6 right-sided apical systolic murmur could, in this case, correspond to tricuspid regurgitation, which would suggest pulmonary arterial hypertension (PAH) or a degenerative tricuspid valve disease;
- other pulmonary conditions (bacterial pneumonia, respiratory disorder, parasites, etc.) can also be responsible for respiratory symptoms. A pulmonary hemorrhage was unlikely due to the reported absence of injury or ingestion of anticoagulant substances;
- finally, endocarditis could be responsible for the heart murmurs but seemed unlikely.

Complementary examinations

Thoracic radiographs

The principal hypotheses were explored by thoracic radiography (Fig. 1). A right cardiomegaly was noted (possibly secondary to PAH). A fine, smooth, interstitial pattern of the entire lung field was demonstrated. These lesions were more visible on the mid-thoracic region, causing a blurring of the vascular structures and indicative of damage to the pulmonary interstitium. There were no signs of left congestive cardiac failure.

Echocardiographic and Doppler examination

The two murmurs and the right cardiomegaly highlighted on the thoracic radiographs were explored using ultrasonic echocardiography (Table 1).

The two-dimensional echocardiographic examination showed normal valve morphology. Doppler color and continuous modes revealed very mild mitral insufficiency of weak velocity. Tricuspid regurgitation was noted. This had already been detected in the previous echocardiograms but this time the tricuspid flowrate was significantly

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