Acta Scientiae Veterinariae, 2025. 53(Suppl 1): 1016.

CASE REPORT
Pub. 1016

ISSN 1679-9216

Arrhythmogenic Right Ventricular Cardiomyopathy in English Bulldog with Brachycephalic Obstructive Airway Syndrome (BOAS)

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ABSTRACT

***Background*:** Arrhythmogenic right ventricular cardiomyopathy (ARVC) is a hereditary disorder where right ventricular cardiomyocytes are replaced by fibrofatty tissue, disrupting myocardial electrical continuity. Causes may include mutations in the striatin gene or cardiac ryanodine receptor gene. Brachycephalic Obstructive Airway Syndrome (BOAS) involves congenital abnormalities in brachycephalic dogs, such as stenotic nares and elongated soft palate, which can lead to right-sided cardiac remodeling and heart failure. This report aimed to present a case of category III ARVC in an English Bulldog with BOAS.

***Case*:** A 9-year-old male English Bulldog, presented with episodes of syncope, postprandial cyanosis, and respiratory difficulty. The dog exhibited severe stenosis of the nares, characteristic of BOAS, along with signs of heart failure. Physical and complementary exams revealed chronic respiratory acidosis, hyponatremia, hypochloremia, and anemia. Echocardiography indicated significant remodeling of the cardiac chambers, particularly in the right ventricle, with tricuspid and pulmonary valve insufficiency, suggesting ARVC. Myocardial biopsy could not be performed. The electrocardiogram showed sinus tachycardia and other irregularities. Thoracic radiographs revealed cardiomegaly and signs of congestive heart failure (CHF). Analysis of abdominal transudate revealed significant peritoneal effusion. The dog succumbed 1 month after initial presentation, without a definitive *ante mortem* diagnosis and without authorization for necropsy.

***Discussion*:** The definitive diagnosis of arrhythmogenic right ventricular cardiomyopathy (ARVC) is made through myocardial biopsy, a procedure often performed *post mortem* due to its difficulty in living animals. ARVC presents in 3 clinical forms: Category I is asymptomatic with few arrhythmias; Category II involves syncope and easy fatigue with monomorphic extrasystoles; and Category III, which is rare, includes signs of congestive heart failure (CHF), ventricular tachyarrhythmias, and occasionally atrial fibrillation. In English Bulldogs, ARVC is more likely to cause supraventricular arrhythmias. This breed often shows CHF signs such as edema and syncope, with a high prevalence of arrhythmias and CHF symptoms. Most ARVC cases have normal left ventricular function, but any deviations can significantly impact survival. Echocardiography typically reveals right heart chamber enlargement and tricuspid valve insufficiency. Conventional ECG detects premature ventricular complexes but may miss some arrhythmias, making Holter monitoring a better tool for long-term assessment. Thoracic radiography may show cardiomegaly and CHF signs, while abdominal imaging can reveal effusion indicating right-sided CHF. Although definitive diagnosis usually requires myocardial biopsy, complementary tests and clinical findings in this case suggest Category III ARVC. Treatment involves managing arrhythmias with antiarrhythmics like sotalol or mexiletine and addressing any CHF with standard protocols. Prognosis varies based on disease presentation, with a generally reserved to unfavorable outlook due to risks of sudden death or worsening heart function. This case underscores the complexity of diagnosing and managing ARVC in brachycephalic dogs with BOAS. Early and accurate identification of these conditions is crucial for effective interventions, although achieving a definitive *in vivo* diagnosis remains challenging. The case highlights the necessity for continuous monitoring and a multidisciplinary approach in managing dogs with multiple cardiac and respiratory comorbidities.

Keywords: cardiac remodeling, congestive heart failure, cyanosis, echocardiography, electrocardiogram, peritoneal effusion.

DOI: 10.22456/1679-9216.142097

Received: 20 September 2024 Accepted: 17 December 2024 Published: 23 January 2025

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INTRODUCTION

Arrhythmogenic right ventricular cardiomyopathy (ARVC) is a hereditary disease characterized by the replacement of right ventricular cardiomyocytes with fibrofatty tissue, leading to a disruption in the electrical continuity of myocardial fibers [15,22]. It is believed that potential causes of ARVC include a mutation in the striatin gene or in the gene encoding the cardiac ryanodine receptor [1,9,20].

Brachycephalic obstructive airway syndrome (BOAS) is characterized by multiple congenital anatomical abnormalities of the cranial structures and soft tissues of the upper airways in brachycephalic dogs and cats [26]. Stenotic nares, elongated soft palate, and hypoplastic trachea are primary alterations that characterize the syndrome and can be found either in isolation or in combination [11]. The stenosis of the nares increases the resistance of airflow through the muzzle, consequently raising pulmonary pressure, which can lead to right-sided cardiac remodeling and potentially result in heart failure [4]. Additionally, it is believed that increased blood pressure, hypoxia, and surges in sympathetic activity may cause myocardial injury [2].

This study aims to present a case report and findings from complementary examinations compatible with category III ARVC in an English Bulldog affected by BOAS.

CASE

A 9-year-old male English Bulldog, weighing 32 kg, was presented at the Veterinary Clinics Hospital of the Federal University of Pelotas (UFPel), Pelotas, RS, Southern Brazil, with a history of syncope episodes accompanied by cyanosis and postprandial cyanosis. On physical examination, the dog exhibited severe nasal stenosis indicative of brachycephalic syndrome, a body condition score of 5/5, lethargy, pale mucous membranes, a heart rate of 64 bpm, muffled heart sounds, a respiratory rate of 60 bpm with labored breathing, a distended abdomen, and tracheal stertor. Given the case, complementary examinations such as blood gas analysis, echocardiography, and ultrasonography were requested.

In the blood gas analysis performed on venous blood from the patient, the pH was 7.327, the oxygen pressure was 31.7 mmHg (ref. 47.9-56.3 mmHg [5]), the base excess (BE) was -5.8 mmol/L (ref. 1 to -7 mmol/L), and the hematocrit was 33.6% (ref. 35-54%).

In the echocardiography, tachycardia was observed at 164 bpm (ref. 60-100 bpm) with an irregular rhythm. The left ventricle showed a slight increase in diastolic diameter (5.26 cm), reduced fractional shortening (18%), and reduced ejection fraction (0.38). The right ventricle exhibited a significant increase in the infundibular cavity (3.31 cm) [Figure 1A], and the right atrium was also enlarged. The mitral valve displayed thickening and mild insufficiency (Figure 1B); the tricuspid valve showed significant insufficiency (Figure 1C), while the pulmonary valve had moderate insufficiency (Figure 1D). These findings indicate significant remodeling of the right heart chambers and marked tricuspid valve insufficiency, suggesting arrhythmogenic right ventricular cardiomyopathy (ARVC). The involvement of the left ventricle and valvular insufficiencies are likely associated with the progression of ARVC. These findings were consistent with category III ARVC, which includes myocardial dysfunction and ventricular dilation in a minority of patients.

The electrocardiogram (ECG) was performed over a continuous duration of 4 min, during which the patient remained calm. Considering that normal values ranging from 60 to 130 ms [25], the results revealed predominant sinus tachycardia, with a PR interval at the upper limit of normal. Isolated atrial ectopics, both blocked and conducted, as well as isolated and monomorphic ventricular ectopics with left bundle branch block morphology originating from the right ventricle, were observed. There was an increase in QRS complex duration, suggestive of left ventricular overload. The other values for wave amplitude and duration were within the normal range for the species and size of the animal.

The thoracic radiograph revealed an enlarged, rounded heart displaced towards the left hemithorax (Figure 2A) and a cranially displaced diaphragm. The Vertebral Heart Score (VHS) was not performed due to hemivertebrae. The trachea maintained its lumen but was dorsally displaced at the cardiac base, and a mild perihilar broncho-interstitial pattern was observed, along with increased diffuse opacity in the cranial lobes, likely related to hypoinflation due to abdominal pressure (Figure 2B), consistent with cardiomegaly. Abdominal volume was increased, with soft tissue

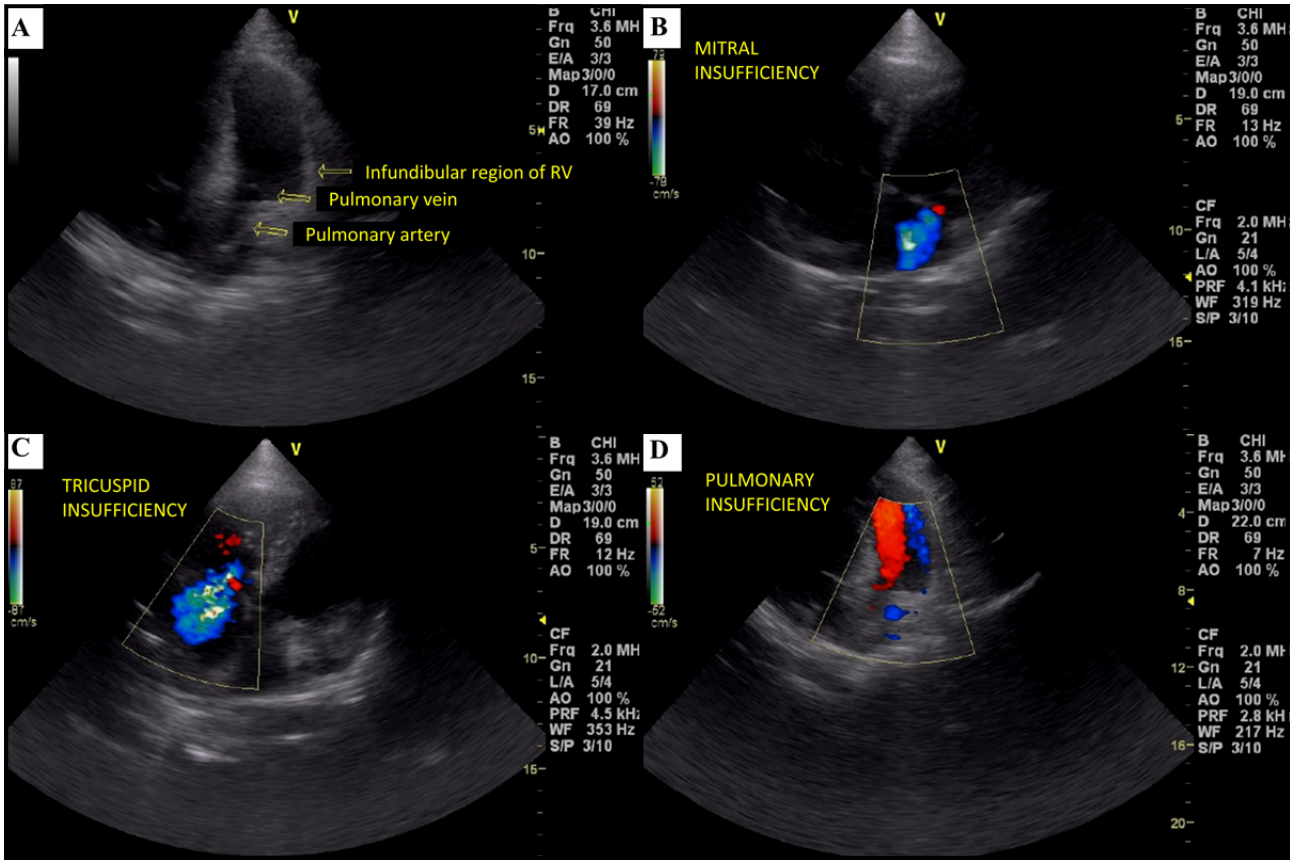

Figure 1. Echocardiography of a 9-year-old male English Bulldog with a history of syncope and postprandial cyanosis. With tachycardia (164 bpm) and an irregular rhythm, the exam revealed a slight increase in the diastolic diameter of the left ventricle (LV), a significant enlargement of the cavity in the infundibular region of the right ventricle (RV, A), and an enlarged right atrium (RA). The mitral valve was thickened with mild insufficiency (B), the tricuspid valve showed significant insufficiency (C), and the pulmonary valve demonstrated moderate insufficiency (D). These findings indicated significant remodeling of the right heart chambers and important tricuspid valve insufficiency, suggesting category III arrhythmogenic right ventricular cardiomyopathy (ARVC).

opacity and loss of intra-abdominal definition, indicating peritoneal effusion. The radiographic findings of cardiomegaly, dorsal displacement of the trachea, broncho-interstitial pattern, and diffuse pulmonary opacity suggested left-sided congestive heart failure (CHF). The increased abdominal volume and peritoneal effusion indicated right-sided CHF, prompting the performance of an Abdominal Fast Scan and abdominocentesis for confirmation.

The Abdominal Fast Scan was performed at 2 different times (0 and 16 h), monitoring 4 distinct areas: hepato-diaphragmatic, spleno-renal, cisto-colic, and hepato-renal. At both times, there was a significant amount of fluid accumulation in all 4 assessed areas, predominantly in the caudal abdominal region. Ultrasound-guided abdominocentesis at both time points revealed the fluid to be translucent with a reddish tint, suggestive of modified transudate.

Unfortunately, the patient passed away at home within 1 month, and a diagnosis was not made in time to institute an appropriate therapeutic protocol. The owners did not authorize *in vivo* myocardial biopsy or necropsy and histopathology. Given the case, the laboratory findings were suggestive of category III arrhythmogenic right ventricular cardiomyopathy.

DISCUSSION

The definitive diagnosis of arrhythmogenic right ventricular cardiomyopathy (ARVC) is established through myocardial biopsy [9]. However, this procedure is challenging to perform *in vivo* [3], and confirmation is often achieved *post mortem* through histopathological examination of fibrofatty infiltration in the right ventricular myocardium [6,14].

ARVC has three clinical forms of presentation. In Category I, dogs are asymptomatic and a small number of ventricular arrhythmias may be identified. In Category II, dogs exhibit syncope and easy fatigue, also accompanied by the presence of monomorphic extrasystoles. In the rare Category III, the patient

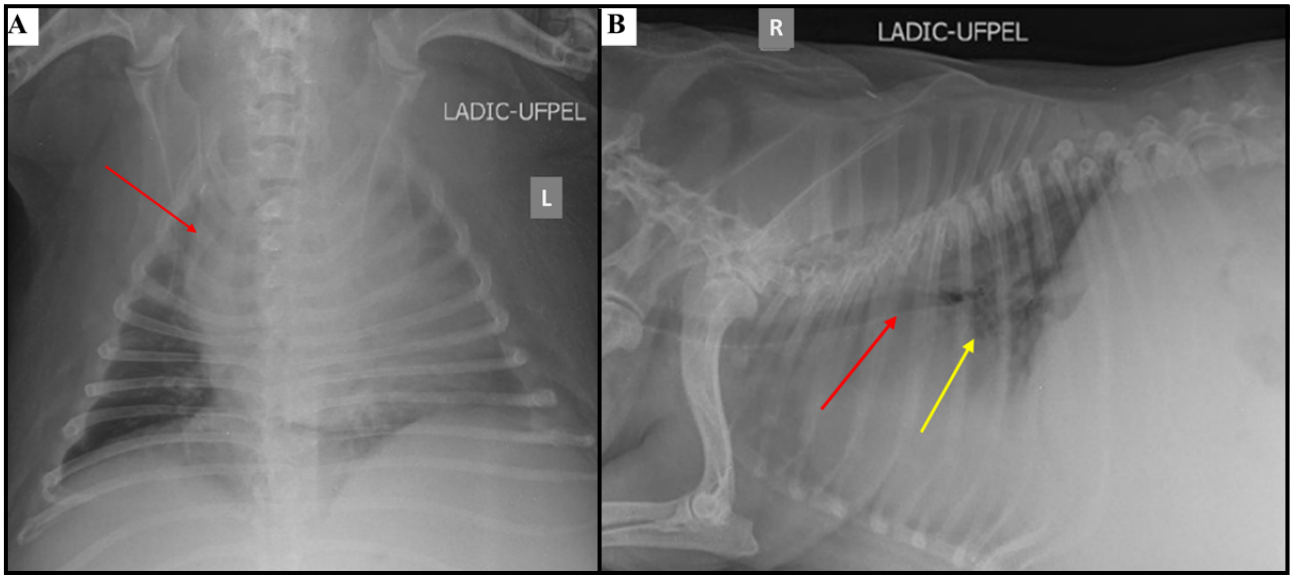

Figure 2. Ventrodorsal thoracic radiography revealed cardiomegaly with displacement towards the left hemithorax (A, red arrow; L, Left) and right latero-lateral view (R) demonstrating dorsal displacement of the trachea (B, red arrow) and a mild peri-hilar broncho-interstitial pattern (B, yellow arrow).

shows signs of congestive heart failure (CHF), and also presents with ventricular tachyarrhythmias and occasionally supraventricular tachyarrhythmias, such as atrial fibrillation [6,13]. In Boxer dogs, ARVC is an important cause of sudden death [8].

English Bulldogs are more prone to exhibit supraventricular arrhythmias than ventricular tachycardia [10], as demonstrated in this case report, where the patient presented with both supraventricular and ventricular ectopies. In a study of dogs of this breed, 38% showed signs of left-sided CHF with pulmonary edema, 33% had right-sided CHF, and 19% had biventricular CHF, while 38% had a history of syncope or collapse, 69% exhibited dyspnea, and 44% had a cough [10]. The patient reported here presented with CHF, generalized edema (anasarca), syncope, cough, easy fatigue, cardiogenic pulmonary edema, and cavitary effusion.

Most animals affected by ARVC have ventricular dimensions and function within normal ranges; however, changes in these parameters can be associated with significantly reduced survival [6,7,19]. In echocardiography, key findings that suggest the presence of ARVC include enlargement of the right heart chambers, tricuspid valve insufficiency, decreased right ventricular wall thickness due to dilation, and aneurysms [3,18]. It's important to note that only a minority of patients exhibit myocardial dysfunction and ventricular dilation, which align with category III ARVC. Most animals with ARVC show normal patterns for left ventricular dimensions and function. When deviations are present, they are often associated with significantly reduced survival [6,7,19].

In the echocardiographic findings of this patient, significant remodeling of the right heart chambers, particularly in the infundibular region of the right ventricle, along with severe tricuspid valve insufficiency and rhythm disturbances, are indicative of ARVC. The slight increase in left ventricular systolic diameter, mild reduction in systolic function, mild mitral valve insufficiency, and moderate pulmonary valve insufficiency are likely linked to the progression and chronicity of ARVC. These subtle changes, while initially minor, can have significant impacts over time, affecting the overall prognosis and highlighting the importance of early detection and management.

Conventional electrocardiography primarily identifies premature ventricular complexes, often showing left bundle branch block morphology. In cases classified as category III, additional findings may include tachyarrhythmias, atrial fibrillation, atrial extrasystoles, and isolated right or left bundle branch blocks. However, these arrhythmias may not be recorded during the short duration of the ECG exam, and their absence does not rule out the diagnosis of ARVC, as the examination period is much shorter compared to the full 24 h of a day. Therefore, ambulatory electrocardiography (Holter) is recommended [6,19,22,23]. Holter monitoring provides similar results to conventional electrocardiography but measures cardiac

electrical activity over a longer period - 24 h - allowing for a more comprehensive assessment of arrhythmia frequency and complexity [6,15,22].

Thoracic radiographic changes are not typically found, but when present, they may include myocardial dysfunction, ventricular dilation, dilation of the right chambers, generalized cardiomegaly, and signs of congestive heart failure (CHF), such as pleural effusion and/or pulmonary edema [3,18,19]. In the thoracic radiograph of the patient, cardiomegaly was observed, characterized by global enlargement of the heart and dorsal displacement of the trachea. Signs of CHF were also visible, including a mild perihilar broncho-interstitial pattern with increased diffuse opacity, primarily in the cranial lung lobes, and a significant increase in abdominal volume with soft tissue opacity and loss of intra-abdominal definition, indicating peritoneal effusion. These findings are suggestive of left-sided and right-sided CHF, respectively. Abdominal effusion, which can also indicate right-sided CHF [3,12], was observed on the abdominal FAST scan performed on the patient. A large amount of fluid was detected at all 4 assessed points, and when drained via ultrasound-guided abdominocentesis, the fluid was translucent with a reddish tint, suggestive of modified transudate.

The definitive diagnosis of ARVC is established through myocardial biopsy [9], but this procedure is challenging to perform *in vivo* [3], and confirmation is often achieved post mortem through histopathological examination of fibrofatty infiltration in the right ventricular myocardium [4,17]. A definitive diagnosis via intracardiac biopsy or necropsy could not be performed due to a lack of authorization from the owners. However, all findings from the other complementary tests, along with the patient's clinical presentation, are suggestive of category III arrhythmogenic right ventricular cardiomyopathy in this English Bulldog.

The treatment of ARVC focuses on controlling ventricular arrhythmias and, if present, managing systolic dysfunction and congestive heart failure (which are less common). Treatment with antiarrhythmics is based on assessing the number of premature ventricular complexes (PVCs) over 24 h (typically more than 1,000 PVCs/24 h, though there is no consensus), presence of ventricular tachycardia, or any repeated patterns of PVCs. If the patient exhibits any of these abnormalities, even if asymptomatic (Category I), a therapeutic protocol is established. Antiarrhythmics used can include sotalol (at an initial dose of 1.5 to 2 mg/kg orally BID, or as needed, 2.5 to 3 mg/kg BID); mexiletine (at a dose of 5 to 6 mg/kg orally TID) or a combination therapy with both sotalol and mexiletine may also be employed.

It is crucial to evaluate the response to therapy [4,19]. A study demonstrated that adjunctive therapy with omega-3 (780 mg/d EPA and 497 mg/d DHA, both orally) in the diet of Boxer dogs with ARVC significantly reduced the number of ventricular extrasystoles, although this study was limited to only 6 weeks [24]. For the treatment of systolic dysfunction, L-carnitine supplementation at a dose of 50 mg/kg orally BID or TID is recommended, though only a small fraction of patients show improvement in cardiac function [17]. For congestive heart failure, the standard protocol is recommended, based on the degree of CHF, including a combination of ACE inhibitors, diuretics, and positive inotropes [16,17,21,23].

The prognosis for ARVC is variable due to the diverse presentations of the disease, ranging from reserved to unfavorable. This variability arises from the risk of sudden death in asymptomatic cases or the development of ventricular dilation, systolic dysfunction, or congestive heart failure, which generally have a worse prognosis [22,23].

This case of an English Bulldog with BOAS and clinical signs of category III ARVC illustrates the complexity of diagnosing and treating cardiac and respiratory comorbidities in brachycephalic dogs. Clinical history and complementary examination findings were suggestive of category III ARVC. The combination of BOAS with heart failure makes the clinical management of these patients particularly challenging, requiring a multidisciplinary approach and constant vigilance. This report emphasizes the importance of early and accurate diagnosis, as well as the need for further research to improve management and intervention strategies in dogs with multiple comorbidities.

Acknowledgements. The authors thank Veterinary Cardiology Study Group ("VetCor", UFPEL) and the "Projeto Focinho Curto" (Short Snout Project, UFPEL) for the support and for promoting research on brachycephalic syndrome in Brazil.

Declaration of interest. The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

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