https://doi.org/10.15446/rfmvz.v71n2.109980

Aortic stenosis and mitral valve dysplasia in a miniature Bull Terrier

Piero Vargas-Pinto[1], *J. Delgado*[2]

Recibido: 06/07/2023 Aprobado: 05/03/2024

ABSTRACT

The clinical case of a one-year-old miniature Bull Terrier is presented. The patient was brought to the cardiology service of the Small Animal Clinic at the Universidad Nacional de Colombia due to historical syncopal episodes during exercise or moments of excitement, as well as at rest. Additionally, these syncopal episodes have increased in frequency to the point of daily occurrence. Echocardiography reveals thickening of the interventricular septum and the free wall of both ventricles. Additionally, irregular enlargement is evidenced in the mass of the papillary muscles of the left ventricle, which are distributed concentrically to the ventricular cavity, as well as a thickened mitral valve, which neither closes nor opens correctly causing mitral insufficiency and regurgitant flow to the left atrium. Similarly, narrowing of the left ventricular outflow tract and increased echogenicity in the aortic valve are observed, which, similarly to the mitral valve, is unable to open properly. The findings suggest congenital mitral valve dysplasia leading to stenosis, accompanied by aortic stenosis. Due to the absence of surgical intervention options, therapy aims to control existing clinical signs and halt the progression of cardiac enlargement using beta-blockers.

Keywords: congenital defect, cardiomyopathy, canine, Bull Terrier.

Estenosis aórtica y displasia de la válvula mitral en un Bull Terrier miniatura

RESUMEN

Se presenta el caso clínico de un Bull Terrier miniatura de un año. El paciente es llevado al servicio de cardiología de la Clínica para Pequeños Animales de la Universidad Nacional de Colombia debido a que presenta síncopes históricos durante el ejercicio o momentos de excitación, así como en reposo. Adicionalmente, estos síncopes han aumentado su frecuencia al punto de tener un episodio diario. La ecocardiografía revela engrosamiento del septo interventricular y de la pared libre de ambos ventrículos. Se evidencia adicionalmente un agrandamiento de forma irregular en la masa de los músculos papilares del ventrículo izquierdo, los cuales se distribuyen de manera concéntrica a la cavidad ventricular, así como una válvula mitral engrosada que no cierra ni abre correctamente, lo cual ocasiona insuficiencia mitral y un flujo regurgitante al atrio izquierdo. De igual manera, se observa un estrechamiento en el tracto de salida del ventrículo izquierdo y

[1] Universidad Nacional de Colombia, Facultad de Medicina Veterinaria y Zootecnia. Clínica para pequeños animales, Bogotá, Colombia.
[2] Universidad Nacional de Colombia, Facultad de Medicina Veterinaria y Zootecnia. Calle 38 sur #52c 25. Bogotá, Colombia.
* Correo electrónico: jdelgadov@unal.edu.co

un aumento de la ecogenicidad en la válvula aórtica que, de manera similar a la válvula mitral, no es capaz de abrirse adecuadamente. Los hallazgos sugieren displasia congénita de la válvula mitral que ocasiona una estenosis de esta, acompañada de estenosis aórtica. Debido a la ausencia de posibilidades de intervenciones quirúrgicas, la terapia se dirige a controlar los signos clínicos existentes y frenar la progresión del agrandamiento cardíaco mediante el uso de beta bloqueadores.

Palabras clave: defecto congénito, cardiomiopatía, canino, Bull Terrier.

INTRODUCTION

Aortic stenosis (AS) is reported as the third most common congenital cardiac anomaly in dogs (Oliveira *et al.* 2011). There are three forms of the disease: subvalvular, valvular, and supravalvular, with the subvalvular form being the most common. The valvular presentation is infrequent, while the supravalvular presentation is very rare (Atkins *et al.* 2009). Subvalvular aortic stenosis occurs most frequently in Newfoundlands, Golden Retrievers, German Shepherds and Boxers, with a recent increase in incidence observed in Bull Terriers and Rottweilers (Atkins *et al.* 2009; Oliveira *et al.* 2011).

Most cases of aortic stenosis show mild lesions, discovered incidentally, that do not compromise the quality of life. However, when the stenosis is moderate or severe, it results in weakness, exercise intolerance, syncope, and even sudden death. Signs of congestive heart failure are rare unless there is concomitant mitral insufficiency (Ettinger & Sutter 2023). Mitral valve dysplasia (MVD) is a rare congenital malformation of the mitral valve (MV) complex in dogs, characterized by inadequate closure of the valve resulting in systolic regurgitation into the left atrium (LA) (Komtebedde *et al.* 1993). It is most prevalent in Bull Terriers, German Shepherds, Dalmatians and Great Danes (Litu & Tilley 1975; Lucina *et al.* 2016). In this cardiac condition, any component of the mitral valve complex can be affected (chordae tendineae, papillary muscles, or leaflets), either individually or collectively. Severe mitral insufficiency caused by the malformation(s) leads to volume overload in the left atrium (LA) and left ventricle (LV), resulting in pulmonary venous congestion and subsequent left-sided congestive heart failure (Komtebedde *et al.* 1993).

This report describes the case of a Bull Terrier presenting with mitral valve dysplasia associated with mitral stenosis, as well as valvular aortic stenosis with a possible subvalvular component due to the mispositioning of the papillary muscles of the mitral apparatus in the left ventricular chamber, and interventricular septal hypertrophy resulting from the high afterload the ventricle was subjected to in order to maintain adequate cardiac output. Additionally, the steps followed, and the methods used to reach the definitive diagnosis through transthoracic two-dimensional echocardiography and color Doppler are described, along with their respective results. Finally, the available therapeutic options for the case are discussed. The congenital heart diseases described represent clinical occurrences that are very rare. Therefore, this report is presented to serve as a reference for colleagues who may encounter similar cases in the future, and to highlight the increasing incidence of congenital heart diseases in this breed of dogs (Atkins *et al.* 2009;

Litu & Tilley 1975; Lucina *et al.* 2016; Oliveira *et al.* 2011).

MATERIALS AND METHODS

Echocardiography was performed using a General Electric LOGIC V5 ultrasound machine, which features color Doppler, pulsed wave, continuous wave, and tissue Doppler functions, as well as an appropriate sector probe for obtaining echocardiographic images, utilizing frequencies in the range of 1–5 MHz. Additionally, the simple electrocardiographic measurement function of the ultrasound machine was used to monitor the cardiac electrical activity of the animal and to complement the associated findings.

Case

A one-year-old male Bull Terrier patient, with no prior illnesses and unknown family history, was brought to consultation due to presenting *seizures* for approximately 6 months, occurring once a month. The owner describes these episodes as moments when the animal loses consciousness for a couple of seconds and then faints, only to regain consciousness afterward. However, the episodes have been increasing in frequency to once a week, eventually reaching multiple episodes daily. Based on the description of the owner, it is suspected that these episodes of loss of consciousness are syncopal episodes, as the owner denies the presence of muscle movements or any signs or behavioral changes prior to the loss of consciousness.

During the clinical examination, the patient was found to be alert, with a docile temperament, pale pink moist mucous membranes, normal capillary refill time (2 seconds), immediate skin fold return time, tachycardia (184 bpm), panting respiratory rate, and normal temperature (38.6 °C). Upon auscultation, a grade 6/6 holosystolic murmur was detected, radiating to all cardiac auscultation points, making it impossible to determine its possible origin. Based on the clinical findings, it is deduced that the *seizures* reported by the owner are syncopal episodes, confirming the initial suspicion.

Electrocardiography (ECG) was performed, revealing a severe increase in P wave amplitude, indicating left atrial enlargement (figure 1). Echocardiography confirmed the diagnosis obtained in the ECG, with severe dilation of the left atrium observed (figure 2). Additionally, the interventricular septum adjacent to the free wall of the right ventricle (RV) was found to be thickened, resulting in a visible reduction of the right ventricular chamber space. However, the most notable finding is the malformation of the papillary muscles of the left ventricle (LV), which are irregularly distributed in the center of the left ventricular chamber, along with thickening of the mitral valve (MV) with impaired ability to open or close adequately (figure 2). In addition to the above, there is narrowing of the left ventricular outflow tract and an increase in echogenicity of the aortic valve (AV), along with impaired opening (figure 2 and figure 3). Additional two-dimensional echocardiographic findings are summarized in table 1. At this point, two clarifications are important: firstly, since this Bull Terrier belongs to the miniature variety, there are insufficient studies to establish specific echocardiographic parameters for this breed, and secondly, due to the absence of an established reference point, comparisons are made based on ranges described for healthy Bull Terriers (O'Leary *et al.* 2003) and dogs of different breeds but with similar weight and size.

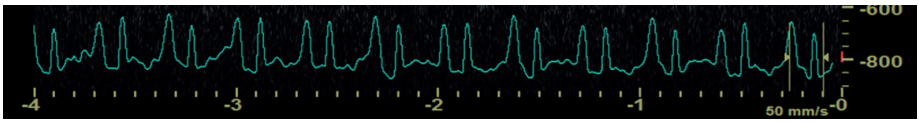

FIGURE 1. Electrocardiogram, Lead II, P wave increased size and length.
Source: own elaboration.

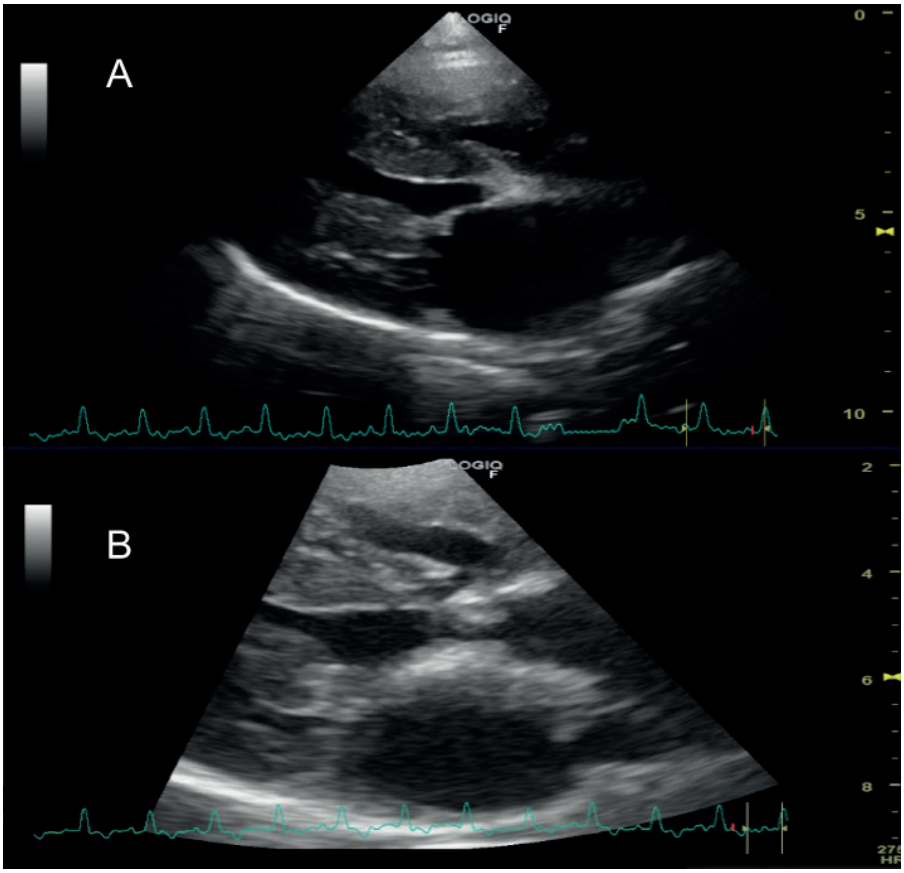

FIGURE 2. Right parasternal window, long axis. A. Interventricular septum and right ventricle thickening; subsequent reduction of the internal ventricular volume. Abnormal distribution of the papillary muscles concentrically to the internal left ventricle chamber, as well as narrowing of its respective outflow tract and augmentation of the aortic valve's echogenicity. B. Left atriomegaly, thickening and loss of the normal left ventricle's architecture. Deformation of the papillary muscles.
Source: own elaboration.

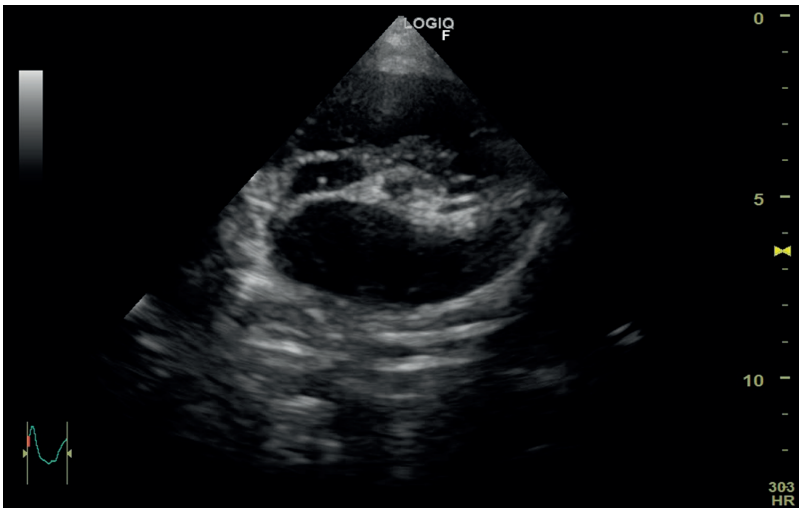

FIGURE 3. Right parasternal window, short oblique axis. Severe atriomegaly, Mitral valve hiperechogenicity and architecture loss.
Source: own elaboration.

TABLE 1. Echocardiographic values obtained in long and short axis

Parameter	Value obtained	Reference value	Standard deviation
Long axis			
LVd	3.8 cm	1.7 cm	0.2
LVIDd	3.1 cm	3.8 cm	0.3
Ao	1.1 cm	1.9 cm	0.2
LA/Ao	3.4	0.9	0.2
LA/LV	1.2	1	0.1
Short axis			
LVIDd	3.2 cm	3.8 cm	0.3 cm
LVIDs	1.8 cm	1.5 cm	0.3 cm
IVSd	1.05 cm	1 cm	0.2 cm
IVSs	1.3 cm	1.3 cm	0.2 cm
LVFWd	0.9 cm	1 cm	0.1 cm
LVFWs	1.2 cm	1.2 cm	0.1 cm
FE (Teichz)	66%	70%	5%
FS (Teichz)	41.9%	32%	10%

LA: Left Atrium. LVIDd: Left Ventricle Internal Diameter in diastole. Ao: Aorta. LVIDs: Left Ventricle Internal Diameter in systole. IVS: Interventricular Septum, LVFW: Left Ventricle Free Wall. FE: Ejection Fraction. FS: Shortening Fraction. Summary of the obtained values in this case and its comparison with the reported normal values for the Bull Terrier and other dogs of similar weight.

Source: O'Leary *et al.* 2003; Rosas & Salazar 2008; Sthrom *et al.* 2018.

The patient exhibits severe dilation in its left atrium compared to standardized measurements (Rosas & Salazar 2008; Strohm *et al.* 2018), caused by constant mitral regurgitation secondary to aortic stenosis, which is the most notable finding. Similarly, the dog presents a decreased diameter of the aortic root, and although there is no standardized measurement for this parameter, it is assumed to likely be caused by severe valvular stenosis (Bellumori *et al.* 2013). Another important finding is the internal diameter of the left ventricle in diastole (LVIDd), which for this animal measures 3.1 cm, compared to the average length of 3.8 cm for a Bull Terrier (O'Leary *et al.* 2003), giving the impression of no myocardial dilation or growth. However, when compared to the length of a dog of approximately the weight of the patient, which is 2.3 cm to 2.5 cm (Rosas & Salazar 2008), it is concluded that the animal does indeed have moderate ventricular dilation. Nevertheless, the malposition of the papillary muscles should be considered, as they could be influencing the atypical distribution of the ventricular chamber (Sudunagunta *et al.* 2021).

Due to the observed alterations in the MV and AV, a color Doppler study was performed to determine the presence of mitral regurgitation flows, as well as to observe the characteristics of the LV flow. The obtained images confirm the suspicion of moderate to severe mitral regurgitation (figure 2A and 2B, and figure 4B). Additionally, turbulent LV outflow and severe aortic regurgitation were found (figure 5).

Upon establishing the presence of aortic and mitral regurgitation, measurements of the regurgitation jet velocity and LV outflow were conducted. These yielded peak velocities of 5 m/s and 6 m/s, respectively (figure 5A and 5B). Regarding the morphology of transmitral ventricular filling waves, there is no clear distinction between the E wave and the A wave. Additionally, the filling wave consistent with atrial contraction (A) reaches very high peak velocities, up to 5 m/s, and its morphology tends to be biphasic (image 3). The abnormal findings could be explained based on the alteration

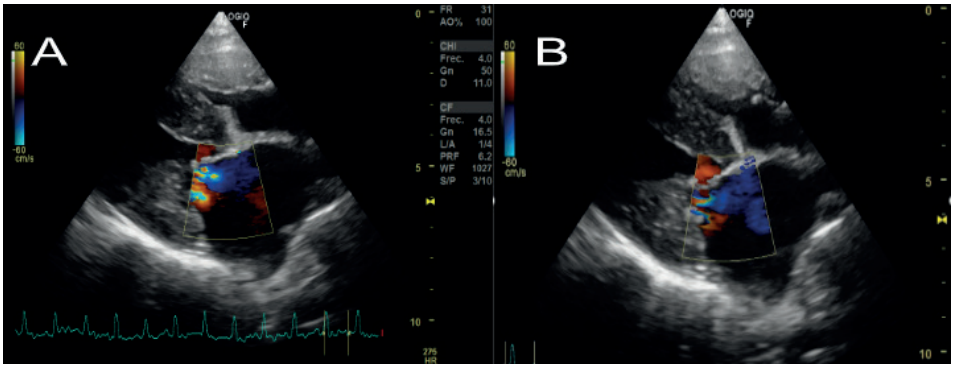

FIGURE 3. Right parasternal window, long axis. A & B: Mitral regurgitation consequence of the mitral complex malformations. Turbulent regurgitation jet, represented by the mix of red and blue tonalities at the valve's level.
Source: own elaboration.

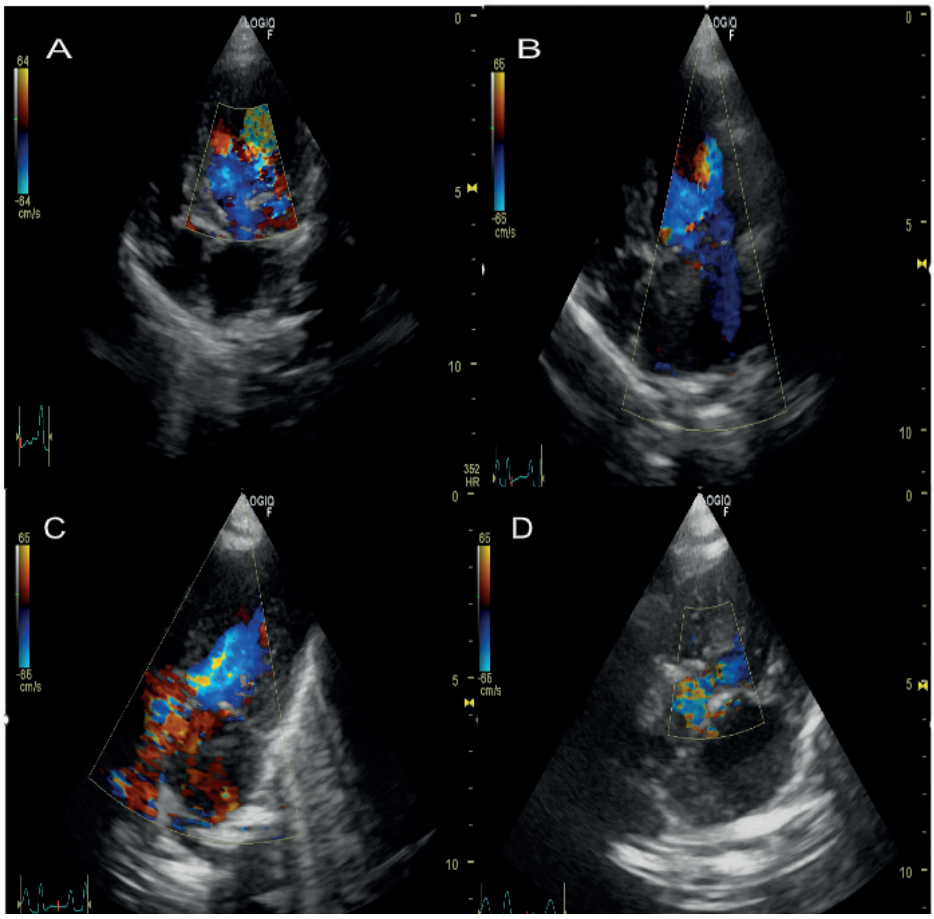

FIGURE 4. Left parasternal window, apical two chamber view. A: LV turbulent ejection flow and its respective outflow tract. B: Severe mitral regurgitation. C & D: Turbulent aortic systolic flow due to the aortic stenosis, respective aortic regurgitation caused by the aortic insufficiency secondary to the inadequate closure of the valve.
Source: own elaboration.

of valvular architecture; where the valves, not opening correctly, cause an increase in the duration of flow and the velocity of blood passage through them. By basing on Poiseuille's law: The velocity of a fluid is proportional to its flow rate and inversely proportional to the diameter of the pipe. In this case, the flow rate is determined by the pressure difference, where the intra-atrial pressure is much higher than the intraventricular pressure (causing atrial dilation), and the pipe diameter is determined by the stenosis of the mitral valve, which narrows the diameter. By applying these physical concepts to intracardiac hemodynamics, a justification for the phenomenon of blood acceleration through the mitral valve is obtained (Secomb & Pries 2007).

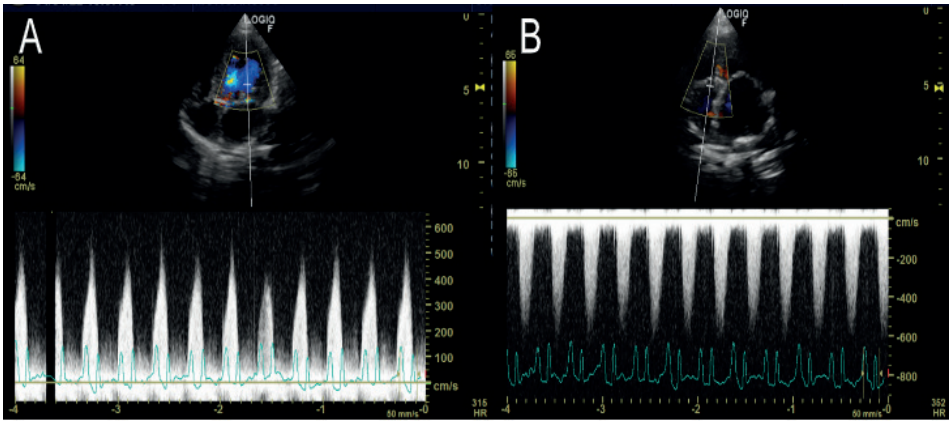

FIGURE 5. A: Mitral regurgitation flow; 5 m/s peak velocity. B: LV outflow tract; 6 m/s velocity peak.
Source: own elaboration.

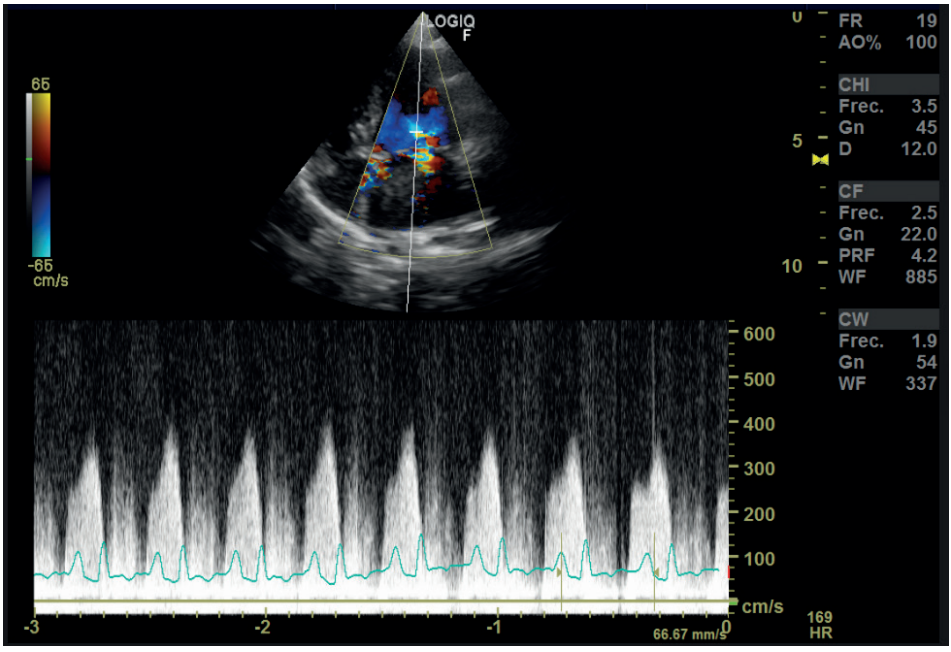

FIGURE 6. Atrial filling wave (A) related to ECG. Biphasic morphology and increased length. 4 m/s peak velocity.
Source: own elaboration.

The malformation of the papillary muscles of the mitral valve apparatus and the inability of its leaflets to open or close properly suggest a differential diagnosis of mitral dysplasia, which, in this case, leads to mitral stenosis as the main

hemodynamic consequence. Similarly, the increased echogenicity of the aortic valve and its inability to open or close as it occurs in the case of the mitral valve suggest a valvular aortic stenosis process. However, the narrowing of the outflow tract secondary to the hypertrophy of the interventricular septum and the presence of deformed papillary muscles occupying space in the ventricular chamber could indicate that aortic stenosis also has a subvalvular component.

Due to the unavailability of surgical therapeutics for correcting these defects, the initial approach for treating the dog involves administering atenolol at a dose of 0.25 mg/kg BID, PO, with the animal being kept under constant supervision.

Following the initial medication, it is reported that the frequency of syncopal episodes decreased to approximately 1-2 episodes per month. Therefore, it is decided to continue with the therapy, and echocardiographic monitoring every 3 months is recommended to adjust the atenolol dose as needed or explore new pharmacological alternatives.

After the second follow-up, the animal did not attend further appointments, which prevented proper traceability of the therapy. It is suspected that it may have died suddenly due to the high predisposition caused by the presence of both heart diseases.

DISCUSSION

The clinical consequence of MV dysplasia can be either mitral insufficiency or mitral stenosis, depending on the components of the MV affected (Jin *et al.* 2016; Litu & Tilley 1975; Sudunagunta *et al.* 2021). Furthermore, the clinical signs of dysplasia correlate with the severity of the defect and the presence of congestive heart failure (CHF) (Otoni & Abbott 2012). This dog presented mitral insufficiency with concomitant mitral stenosis. Additionally, the papillary muscles exhibited abnormal morphology and a concentric location to the LV, hindering their proper function. Furthermore, the MV showed morphological abnormalities in its leaflets, preventing both opening and closing adequately. The combination of these factors leads to the manifestation of symptoms of congestive heart failure (CHF) (Sudunagunta *et al.* 2021). Mitral insufficiency caused by inadequate closure of the MV resulted in progressive mitral regurgitation, which, in turn, led to left atrial (LA) overload and its consequent dilation. The volume overload, coupled with LA dilation, would eventually predispose this animal to develop pulmonary edema at any time, potentially resulting in sudden death (Lucina *et al.* 2021). However, to date, there is no clinical evidence of pulmonary edema.

The appearance of aortic stenosis is equally important (valvular type in this case, with a possible subvalvular component). Aortic stenosis generally leads to mild or absent clinical signs, and its discovery is usually incidental (Crofton *et al.* 2023). Any of the three types of aortic stenosis leads to concentric hypertrophy of the LV, and as its severity increases so does the degree of the latter, and in turn, concentric hypertrophy leads to myocardial ischemia, resulting in ventricular arrhythmias (Ettinger *et al.* 2016; Taniguchi *et al.* 2018).

The obstruction of the left ventricular outflow tract leads to a decrease in systolic volume, even under sympathetic stimulation (Bellumori *et al.* 2013). This results in weakness (due to muscular hypoxia), syncope (due to cerebral hypoxia), or ventricular arrhythmias (due to myocardial

hypoxia) (Otoni & Abbott 2012; Bellumori *et al.* 2013; Brambilla *et al.* 2020). Ventricular arrhythmias, although not currently present in the animal, can lead to a progression in weakness or the occurrence of syncope, or even sudden death if they result in ventricular fibrillation (Taniguchi *et al.* 2018).

In summary, the heart of this dog was unable to pump blood adequately through the aortic valve due to its stenosis, and additionally, a significant portion of the systolic volume is regurgitated through an insufficient mitral valve to the left atrium, which is dilated due to volume overload. Tissue hypoxia caused by decreased cardiac output results in sympathetic stimulation of the heart, which explains the elevated resting heart rate and concentric hypertrophy of the left ventricle (Bellumori *et al.* 2013). Furthermore, these physiological responses paradoxically worsen the situation, as the increase in heart rate further reduces the systolic volume, and moreover, concentric hypertrophy increases the degree of mitral regurgitation due to the impaired transit of blood through a stenotic aortic valve (Brambilla *et al.* 2020).

The ideal therapy for valvular aortic stenosis is surgical, either through forced valve opening with a balloon catheter, which is positioned in the damaged valve and inflated, or through total valve replacement (Brambilla *et al.* 2020; Schrope 2015). Regarding the treatment of mitral dysplasia, it is also exclusively surgical. Nonetheless, very few successful attempts of surgical mitral valve replacement in dogs are reported (White *et al.* 1995).

Unfortunately, in Colombia neither of the two options is a possibility, so therapy is limited to pharmacological options that ensure the best possible remaining quality of life. For this reason, atenolol, a selective Beta-1 antagonist, is used, which binds to the Beta-1 adrenergic receptors of the heart, blocking the positive chronotropic and inotropic action of endogenous catecholamines and, therefore, sympathetic stimulation. This results in reduced heart rate, blood pressure and cardiac contractility. Beta blockers are also known to increase the refractory period of the AV node, making them useful in the management of supraventricular tachycardia or atrial fibrillation (Wadworth *et al.* 1991; White *et al.* 1995).

In conclusion, the prognosis for the animal is poor due to the severity of the malformations in the mitral valve complex and valvular stenosis (Ettinger & Sutter 2023; Jin *et al.* 2016; Sudunagunta *et al.* 2021), and it is expected to rapidly progress to pulmonary edema or sudden death. However, the response to atenolol is positive, and the animal is expected to undergo quarterly monitoring for any clinical progression to pulmonary edema or the need for adjustment or change in pharmacological therapy.

CONCLUSIONS

The occurrence of congenital heart diseases in dogs is a rare clinical finding; therefore, the manifestation of multiple conditions in a single animal is a phenomenon worthy of being a case study. For this reason, it was considered pertinent to report the case. Another factor to consider is that the literature mentioning mitral valve dysplasia is extremely scarce and appears in the form of isolated case reports and a couple of literature reviews, due to its very low incidence as a congenital heart disease, which adds value to this report.

This case report highlights the lack of technical advancements and training of personnel for cardiac interventions, as well as open-heart surgery in the country. This should be a cause of concern for educational and veterinary centers, as the implementation of cutting-edge methods and proper training is needed to maintain animal cardiovascular health in Colombia.

Regarding the case of the animal, the coexistence of mitral dysplasia and aortic stenosis foreshadows a very poor prognosis, as both heart diseases pose a threat of sudden, severe, and aggressive decrease in cardiac output manifested by recurrent syncope episodes and the imminent risk of sudden death. Similarly, considering that the ideal therapeutic approach involves interventional and surgical treatment of the diseases, pharmacological therapy alone will only provide mild and temporary improvement in symptoms and disease progression.

CONFLICT OF INTEREST

None of the authors of this report declare any conflicts of interest.

SOURCES OF FUNDING/ETHICAL COMMITTEE DECLARATION

Not applicable.

ACKNOWLEDGMENTS

This case report served as part of a graduation project. Therefore, we would like to express our gratitude to Dr. Piero Ardani Vargas for his guidance and mentorship throughout this process, as well as for allowing the dissemination of this academic material authored by him.

REFERENCES

Atkins C, Bonagura J, Ettinger S, Fox P, Gordon S, Haggstrom J, Stepien R. 2009. Guidelines for the diagnosis and treatment of canine chronic valvular heart disease. Journal of Veterinary Internal Medicine. 23(6):1142-1150. https://doi.org/10.1111/j.1939-1676.2009.0392.x

Bellumori TP, Famula TR, Bannasch DL, Belanger JM, Oberbauer AM. 2013. Prevalence of inherited disorders among mixed-breed and purebred dogs: 27,254 cases (1995-2010). Journal of the American Veterinary Medical Association. 242(11):1549-1555. https://doi.org/10.2460/javma.242.11.1549

Brambilla PG, Polli M, Pradelli D, Papa M, Rizzi R, Bagardi M, Bussadori C. 2020. Epidemiological study of congenital heart diseases in dogs: Prevalence, popularity, and volatility throughout twenty years of clinical practice. PloS one. 15(7):e0230160. https://doi.org/10.1371/journal.pone.0230160

Crofton AE, Kovacs SL, Stern JA. 2023. Subvalvular aortic stenosis: learning from human and canine clinical research. Cardiology Research. 14(5):319. https://doi.org/10.14740/cr1547

Ettinger SJ, Edward C, Cote E. 2016. Congenital heart diseases. Textbook of veterinary internal medicine. 8th edition. Philadelphia: Saunders.

Ettinger SJ, Suter PF. 2023. Canine cardiology. 9th edition. Saunders Company.

Jin YS, Kim SJ, Hyun C. 2016. Mitral valve dysplasia in a Maltese dog. Journal of Veterinary Clinics. 33(6):368-371. https://doi.org/10.17555/jvc.2016.12.33.6.368.

Komtebedde J, Ilkiw E, Follette D, Breznock M, Tobias H. 1993. Resection of subvalvular aortic stenosis surgical and perioperative management in seven dogs. Veterinary Surgery. 22(6):419-430. https://doi.org/10.1111/j.1532-950X.1993.tb00417.x

Litu SK, Tilley LP. 1975. Malformation of the canine mitral valve complex. Journal of the American Veterinary Medical Association. 167(6):465-471.

Lucina SB, Sarraff AP, Wolf M, Silva VB, Sousa MG, Froes TR. 2021. Congenital heart disease in dogs: a retrospective study of 95 cases. Topics

in Companion Animal Medicine. 43:100505. https://doi.org/10.1016/j.tcam.2020.100505.

O'Leary C, Mackay B, Taplin R, Atwell R. 2003. Echocardiographic parameters in 14 healthy English Bull Terriers. Australian Veterinary Journal. 81(9):535-542. https://doi.org/10.1111/j.1751-0813.2003.tb12881.x

Oliveira P, Domenech O, Silva J, Vannini S, Bussadori R, Bussadori C. 2011. Retrospective review of congenital heart disease in 976 dogs. Journal of Veterinary Internal Medicine. 25(3):477-483. https://doi.org/10.1111/j.1939-1676.2011.0711.x

Otoni C, Abbott JA. 2012. Mitral valve dysplasia characterized by isolated cleft of the anterior leaflet resulting in fixed left ventricular outflow tract obstruction. Journal of Veterinary Cardiology. 14(1):301-305. https://doi.org/10.1016/j.jvc.2012.01.010.

Rosas J, Salazar M. 2008. Determinación de los valores ecocardiográficos normales en caninos adultos sanos a la altura de Bogotá 2600 m s. n. m. Bogotá: Universidad de La Salle. Recuperado de: https://ciencia.lasalle.edu.co/medicina_veterinaria/117

Schrope DP. 2015. Prevalence of congenital heart disease in 76,301 mixed-breed dogs and 57,025 mixed-breed cats. Journal of Veterinary Cardiology. 17(3):192-202. https://doi.org/10.1016/j.jvc.2015.06.001.

Secomb TW, Pries AR. 2007. Basic principles of hemodynamics. Biomedical and Health Research-Commission of the European Communities Then IOS Press. 69:28.

Strohm L, Visser L, Chapel E, Drost W, Bonagura J. (2018). Two-dimensional, long-axis echocardiographic ratios for assessment of left atrial and ventricular size in dogs. Journal of Veterinary Cardiology. 20(5):330-342. https://doi.org/10.1016/j.jvc.2018.07.008

Sudunagunta S, Hamilton-Elliott J, Dukes-McEwan J. 2021. Mitral valve dysplasia in eight English Springer Spaniels. Journal of Veterinary Cardiology: the official journal of the European Society of Veterinary Cardiology. 33:52-60. https://doi.org/10.1016/j.jvc.2020.11.003

Taniguchi T, Morimoto T, Shiomi H, Ando K, Kanamori N, Murata K. (2018). Sudden death in patients with severe aortic stenosis: observations from the CURRENT AS registry. Journal of the American Heart Association. 7(11):e008397. https://doi.org/10.1161/JAHA.117.008397

Wadworth AN, Murdoch D, Brogden RN. 1991. Atenolol. A reappraisal of its pharmacological properties and therapeutic use in cardiovascular disorders. Drugs. 42(3):468-510. https://doi.org/10.2165/00003495-199142030-00007.

White RN, Stepien RL, Hammond RA, Holden DJ, Torrington AM, Milner HR, Cobb MA, Hellens SH. 1995. Mitral valve replacement for the treatment of congenital mitral dysplasia in a bull terrier. Journal of Small Animal Practice. 36(9):407-410. https://doi.org/10.1111/j.1748-5827.1995.tb02968.x.

Forma de citación del artículo:

Vargas-Pinto, P. y Delgado, J. (2024). Aortic stenosis and mitral valve dysplasia in a miniature Bull Terrier. Rev Med Vet Zoot. 71(2): e109980. https://doi.org/10.15446/rfmvz.v71n2.109980