

Tricuspid Valve Dysplasia Associated with Atrial Septal Defect in a Sphynx Cat

Sabrinne Peglow dos Santos^{†1}, Bruna Pioner de Jesus^{②2}, Nathalia Franco^{③3}, Anelise Bonilla Trindade Gerardi^{④1},
Isabella Michels Carvalho^{⑤2}, Marilia Avila Valandro^{⑥2} & Rochelle Gorczak^{⑦2}

ABSTRACT

Background: Congenital heart diseases are rare in cats, accounting for less than 5% of cardiac cases, with tricuspid valve dysplasia (TVD) and atrial septal defect (ASD) being common types. TVD causes structural valve abnormalities and right heart dilation, while ASD creates abnormal atrial blood flow, enlarging the right heart and pulmonary artery. Diagnosis relies on echocardiography, and treatment includes symptomatic management and ACE inhibitors. Prognosis varies with severity and diagnosis timing. The aim is to report a case of tricuspid valve dysplasia associated with atrial septal defect in a Sphynx cat.

Case: A 60-day-old male Sphynx kitten, and weighing 0.5 kg, was presented with respiratory distress, nasal discharge, epiphora, and decreased appetite. Physical examination revealed dehydration, fever (39.9°C), and a grade III/VI holosystolic murmur. Blood tests were within normal limits. Thoracic radiographs showed a globoid cardiac silhouette with enlargement of the right heart chambers and pulmonary edema suggestive of bronchopneumonia. Echocardiography diagnosed tricuspid valve dysplasia (TVD) characterized by right atrial and ventricular enlargement and significant tricuspid regurgitation. Contrast echocardiography identified an atrial septal defect. The kitten was treated with furosemide and lisinopril. Follow-up echocardiograms demonstrated progressive reduction and eventual closure of the atrial septal defect, accompanied by improvement in the severity of TVD. After 2 years of clinical and echocardiographic monitoring, the cat remains asymptomatic without signs of heart failure, and ACE inhibitor therapy was discontinued. This case highlights the potential for spontaneous closure of atrial septal defects in feline patients with congenital tricuspid valve dysplasia and the benefits of medical management in such cases.

Discussion: Congenital heart diseases in cats are rare and challenging due to small patient size and frequent lack of symptoms at presentation. In this case, a Sphynx kitten was brought in with hyporexia and diarrhea, without obvious cardiac signs. Tricuspid valve dysplasia (TVD), a rare congenital defect more studied in dogs and humans, is scarcely reported in cats, especially in breeds like Sphynx. Atrial septal defects (ASDs) are common in humans and increasingly recognized in veterinary patients, mainly dogs. Auscultation revealed a grade 3 holosystolic murmur on the right, matching the tricuspid valve location. Radiographs showed right atrial and ventricular enlargement and pulmonary edema, linked to TVD, ASD, and bronchopneumonia. Echocardiography confirmed significant TVD; contrast echo suggested a left-to-right shunt without microbubble passage. Treatment with ACE inhibitors and furosemide led to clinical improvement. After 24 months, echocardiography showed reduction or closure of the atrial defect and decreased tricuspid regurgitation, which is unusual. The initial septal defect might have been a remnant embryonic membrane, as it was not confirmed by microbubble contrast and disappeared over time. Spontaneous closure of small interatrial communications is common. Surgical correction is not feasible in many settings due to cost and technical limitations. The cat remains asymptomatic with regular monitoring and a favorable prognosis. Early diagnosis and treatment improved quality of life, but ongoing surveillance is essential. Given limited feline data, it's unclear if therapy alone caused defect regression, as spontaneous remission occurs in kittens over months to years.

Keywords: atrioventricular hypertrophy, atrium, cardiology, cat.

DOI: 10.22456/1679-9216.151120

Received: 10 January 2026

Accepted: 12 June 2026

Published: 18 July 2026

[†]In memoriam. ¹Faculdade de Veterinária, Universidade Federal do Rio Grande do Sul (UFRGS), Porto Alegre, RS, Brazil. ²Faculdade de Medicina Veterinária, Centro Universitário Ritter dos Reis (UniRitter), Porto Alegre. ³Departamento de Medicina Veterinária, Universidade Federal de Santa Maria (UFSM), Santa Maria, RS. CORRESPONDENCE: R. Gorczak [r.gorczak@yahoo.com.br]. UniRitter - Campus FAPA. Av. Manoel Elias n. 2001. CEP 91240-261 Porto Alegre, RS, Brazil.

INTRODUCTION

Congenital heart diseases (CHDs) represent less than 5% of cardiac cases in felines, unlike acquired heart diseases, such as cardiomyopathies, which are more common in cats [8]. These congenital abnormalities are defined by morphological changes observed immediately after birth, stemming from embryonic origins and may involve the heart or major vessels [6,7]

The etiology of the vast majority of CHDs varies and remains uncertain, potentially arising from hereditary causes, spontaneous mutations, exposure to maternal drugs or toxins during gestation [6,9]. Tricuspid valve dysplasia (TVD) and atrial septal defect (ASD) are significant congenital abnormalities in clinical practice, often leading to severe heart failure [6].

Animals with CHDs may remain asymptomatic during juvenile and adult stages, with clinicians primarily focusing on detecting heart murmurs during physical exams [6]. Depending on the disease, symptoms may include lethargy, exercise intolerance, episodes of syncope, and cyanosis [6,9]. Coughing, dyspnea, and rapid breathing due to CHDs may also be observed. Comparing the patient to littermates is crucial to identify growth deficits and behavioral changes [6].

TVD is considered one of the most common congenital heart anomalies in felines [6,7]. It is characterized by structural changes in the tricuspid valve, including thickened leaflets, absence, shortening, or fusion of chordae tendineae, papillary muscle fusion and hypertrophy, inadequate insertion of papillary muscles into the leaflets, incomplete valve development, and subsequent dilation of the right atrium (RA) and right ventricle (RV) [6,9]. Abnormal blood flow, with direct reverse flow from the RV to the RA, can lead to pleural or pericardial effusion [12].

ASD, on the other hand, is a congenital defect characterized by a persistent communication between the RA and left atrium (LA), considered a rare anomaly [7,9]. It may occur alone or in association with other congenital dysfunctions in various cardiac regions [6].

The presence of an atrial septal anomaly allows blood to flow from the LA to the RA, increasing blood volume in the right heart and consequently elevating pulmonary artery blood flow [9,11]. The flow volume between atria is influenced by individual conditions such as defect size, pulmonary artery resistance, and ventricular compliance [4]. Felines with moderate to

severe ASD tend to develop RA and RV enlargement and pulmonary artery distension [9].

Echocardiography, with or without contrast enhancement, is the standard diagnostic test for CHDs [6,7] and guides therapeutic interventions. Treatment typically involves symptomatic management and angiotensin-converting enzyme inhibitors (ACEIs), possibly combined with pimobendan. Prognosis depends on the degree of valve involvement and the age at diagnosis [9].

Due to the limited number of cases reported in national veterinary medicine, this study aims to report a case of a Sphynx cat presenting with TVD and ASD, including diagnosis and instituted therapy.

CASE

A 60-day-old male Sphynx kitten, and weighing 0.5 kg, was brought to a veterinary clinic exhibiting respiratory signs such as dyspnea, purulent nasal discharge, epiphora, and hyporexia. Physical examination revealed moderate dehydration and fever, with temperature of 39.9°C. Cardiopulmonary auscultation detected a grade III/VI holosystolic murmur in the right hemithorax. Further diagnostic tests including complete blood count, total proteins, platelets, and albumin were within normal reference values for the species. Additionally, thoracic radiography and echocardiography were performed. The kitten was hospitalized and received treatment including fluid therapy with Ringer lactate¹ [Solução de Ringer com Lactato - 2 mL/h infusion pump], subcutaneous amoxicillin-clavulanic acid² [Agemoxi® CL - 16 mg/kg BID], oral acetylcysteine³ [Mucomucil® Xarope Pet - 15 mg/kg TID], tobramycin⁴ eye drops [Tobrasyn® - mg/mL, 1 drop every 4 h] and diclofenac sodium⁴ eye drops [DiclofenacoSyntec® 0.1% - 1 drop BID].

Thoracic radiography revealed a globoid cardiac silhouette, prominently affecting the right ventricular and atrial chambers with elevation of the terminal tracheal path, indicative of cardiopathy. The lung fields showed areas of dispersed interstitial pattern in the pulmonary parenchyma, suggestive of pulmonary edema with associated peribronchial infiltration, indicating bronchopneumonia. The major vessels appeared slightly dilated (Figure 1).

The echocardiogram revealed tricuspid valve dysplasia associated with enlargement of the right atrium and ventricle. Doppler evaluation showed turbulent systolic flow in the right atrium due to significant

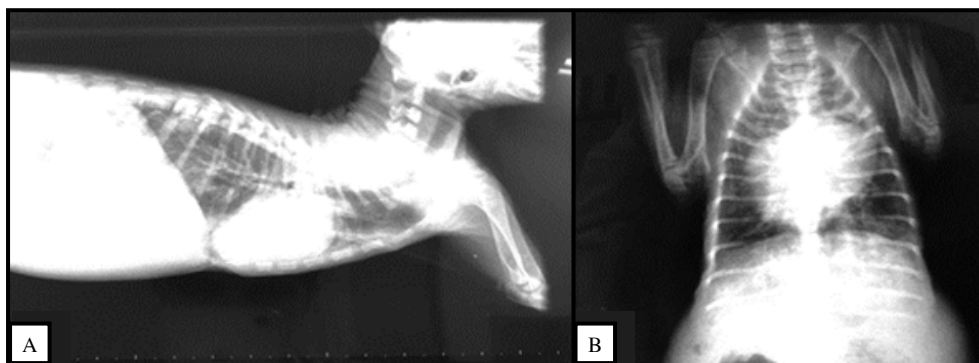


Figure 1. Thoracic radiographs in right lateral and ventrodorsal projections of a feline presenting with a holosystolic murmur. A- Right lateral projection: showing lung fields with areas of dispersed interstitial pattern in the pulmonary parenchyma, suggestive of pulmonary edema and associated peribronchial infiltration, indicating bronchopneumonia. The major vessels were slightly dilated, with right ventricular and atrial chambers showing elevation of the terminal tracheal path, indicative of cardiopathy. B- Ventrodorsal projection: demonstrating a globoid cardiac silhouette with prominence in the right ventricular and atrial chambers.

tricuspid valve insufficiency. Contrast echocardiography was performed by puncturing the right femoral vein with a 24G peripheral intravenous catheter. A 0.9% NaCl solution¹ [Cloreto de sódio 0.9% - 10 mL, IV bolus, 3 repetitions] was administered to evaluate for cardiac shunts, but no bubbles were observed flowing from the right ventricle to the left. However, a hypoechoic area measuring 0.2 cm consistent with an atrial communication was visualized.

Therapy was initiated with furosemide³ [Furoresin® - 2 mg/kg, IV, single dose] and lisinopril⁵ [Lisinopril Teuto - 0.5 mg/kg, PO, SID], with follow-up echocardiography recommended every 6 months. In the 2nd echocardiographic evaluation, performed 120 days after the 1st, there was a reduction in the diameters of the right atrium and ventricle, and measurement of the pressure gradient confirmed a reduction in the atrial communication.

One year later, on a subsequent echocardiographic evaluation, complete restoration of the atrial communication was observed, with no hypoechoic area between the atria. There was mild thickening of the tricuspid valve leaflets, with 1 leaflet shortened and the other elongated, indicating regression of the tricuspid valve dysplasia. Based on these results, therapy with ACE inhibitors was discontinued. The animal continues to undergo clinical follow-up every 6 months for the past 2 years, showing no clinical signs or radiographic evidence of heart failure (Figure 2).

DISCUSSION

Congenital heart diseases in felines, although rare, pose a challenge to clinicians, primarily due to the small size of animals at initial presentation, typically weighing less than 500 g, and the high number of initially asymptomatic cases [9]. In the case at hand,

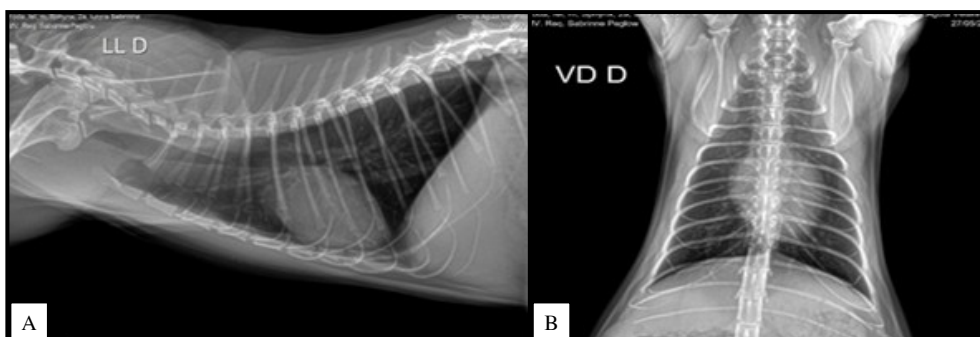


Figure 2. Thoracic radiographic image of a feline with congenital tricuspid valve dysplasia and atrial septal defect, taken 24 months after diagnosis and medical therapy. A- Right lateral projection: the cardiac silhouette measures 2.5 vertebral spaces (normal range 2.0 to 2.5). Vertebral heart score (VHS) is 7.5 vertebral bodies (normal range 7.5 ± 0.3). The tracheal path is intact with no deviations. The pulmonary image shows a bronchial pattern, suggestive of allergic process or bronchitis. No other significant changes noted. B- Ventrodorsal projection: showing a cardiac silhouette with physiological dimensions for the species.

the animal was brought to the clinic with complaints of hyporexia and diarrhea, without clinical signs suggestive of cardiac origin, consistent with literature indicating frequent absence of symptomatic presentation in cardiopathy.

Tricuspid valve dysplasia (TVD) is recognized as a rare congenital heart condition in human medicine, affecting neonates with a postnatal mortality rate potentially exceeding 50% [5,8]. In veterinary medicine, most studies pertain to dogs, with higher occurrence in Labrador Retrievers, Golden Retrievers, and German Shepherds [6,10]. Reports in feline medicine are sparse, and the incidence in breeds such as Sphynx has not been reliably estimated, nor have cases specifically been documented in this breed from the present report.

Atrial septal defects (ASDs), on the other hand, are commonly observed in human medicine, including cases where patients remain asymptomatic for years, accounting for approximately 25% of diagnoses made in adulthood [3,8]. With advances in diagnostic technologies in veterinary medicine, there is an increasing description of such anomalies, particularly in dogs [4]. The patient in this report, initially evaluated at 2 months of age, originated from a Sphynx cattery. Literature review revealed only sporadic case reports of congenital heart diseases, specifically TVD and ASD, commonly observed in domestic cats without specific breed associations, including Persian, Chartreux, Siamese, Maine Coon, and Burmese [4,9].

Cardiac auscultation revealed a grade 3 holosystolic murmur on the right side, consistent with the location of the tricuspid valve. This observation helped narrow down the differential diagnosis [6]. The enlargement of the right atrium and ventricle noted on the initial thoracic radiograph is explained by morphological changes stemming from both ASD and TVD, as increased pressure within the right heart leads to cardiac hypertrophy and dilation of the caudal vena cava, although the latter was not evident on the initial radiographic examination. The pulmonary edema-like image can be attributed to the systemic condition, exacerbated by bronchopneumonia. Given the overall clinical presentation, age, and degree of dehydration, a single dose of furosemide was administered, yielding a favorable response in the animal.

Echocardiography is the gold standard for diagnosing congenital heart conditions, confirming

the suspicion of significant TVD. For ASD diagnosis, contrast echocardiography was chosen, capable of demonstrating the presence of shunts and abnormal blood flow through the formation of microbubbles using 0.9% NaCl solution [6,9]. In this case, microbubble formation did not occur, suggesting a left-to-right shunt, which can explain the finding of right-sided cardiomegaly due to pressure and turbulent flow resulting from the combination of TVD and ASD.

The treatment regimen aligns with literature recommendations, involving ACE inhibitor administration [9]. Due to limited case reports, pimobendan was not included in the therapy; however, recent studies indicate its efficacy as an adjunct in managing congenital heart disease in animals [1,9,12]. Surgical intervention, considered for correcting congenital defects, particularly ASD, is not currently a viable option in Brazilian feline medicine due to high costs and the need for extracorporeal bypass for cardiac manipulation, equipment unavailable in most Brazilian veterinary clinics and hospitals [11]. Despite receiving therapy solely with an ACE inhibitor and furosemide, the patient showed clinical improvement, with resolution of the feline's symptoms.

During the 2nd echocardiogram, performed 24 months after diagnosis, a reduction in atrial defect size was observed, along with decreased severity of tricuspid valve regurgitation. Such outcomes are uncommon, given that the regurgitation level from TVD was initially considered moderate to severe. Additionally, as the atrial defect dimensions were no longer measurable, it is believed to have been a small defect, where no direct surgical intervention was needed for correction. Typically, such patients may experience exacerbations of clinical signs and may become refractory to medical treatment [9,11].

Based on all data and examinations reported, it cannot be definitively concluded whether the septal defect truly existed, as the small hypoechoic area seen in the 1st echocardiogram, suggestive of a defect, was not confirmed by microbubble testing and was subsequently not visible over time. This small area may have been a thin remnant membrane from embryogenesis, which can create an impression of a defect; furthermore, the animal's size posed challenges for examination. However, the spontaneous closure of an interatrial communication is common as animals grow. In this case report, more frequent echocardiographic

evaluations were needed to confirm the defect's presence, which was not possible due to the owner's circumstances. Indications for surgical intervention include extensive septal defects and the presence of clinical signs [2].

After 24 months from the definitive diagnosis of TVD and ASD, the feline continues to receive regular veterinary monitoring and currently shows no clinical signs consistent with cardiopathy. Animals without clinical symptoms of heart failure in adulthood have a favorable to excellent prognosis and can live to an advanced age without complications, even with congenital conditions like TVD and ASD [9].

Concluding that early diagnosis and appropriate therapy initiation in a feline with congenital tricuspid valve disease and atrial dysplasia provided an excellent quality of life, with reduction in defects observed on echocardiography and thoracic radiography. However,

animals with these conditions should remain under constant monitoring.

Due to the limited published data in feline medicine, it cannot be definitively stated that the instituted treatment was the sole reason for the regression of the atrial septal defect. It is common to observe such defects in kittens, with remission occurring months to years after diagnosis.

MANUFACTURERS

¹Farsenius KabiBrasil Ltda. Barueri, SP, Brazil.

²Agener UniãoDistribuidora de Medicamentos Ltda. São Paulo, SP, Brazil.

³VetnilIndústria e Comércio de Produtos Veterinários Ltda. Louveira, SP, Brazil.

⁴Synteco Brasil Ltda. Santana de Parnaíba, SP, Brazil.

⁵LaboratórioTeuto Brasileiro. Anápolis, GO, Brazil.

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

REFERENCES

- 1 **Adamany J.L. & Dhumeaux M.P.J. 2014.** Pimobendan: its place in managing feline heart failure. *Companion Animal*. 19(2): 102-105. DOI: 10.12968/coan.2014.19.2.102.
- 2 **Birchard S.J. & Sherding R.G. 2008.** *Manual Saunders de Clínica de Pequenos Animais*. 3.ed. São Paulo: Roca, pp. 2048-2048.
- 3 **Brida M., Diller G.P., Kempny A., Drakopoulou M., Shore D. & Gatzoulis M.A. 2019.** Atrial septal defect closure in adulthood is associated with normal survival in the mid to longer term. *Heart*. 105(13): 1014-1019. DOI: 10.1136/heartjnl-2018-314380.
- 4 **Chetboul V., Charles V., Nicolle A., Sampedrano C.C., Gouni V., Pouchelon J.L. & Tissier R. 2006.** Retrospective study of 156 atrial septal defects in dogs and cats (2001-2005). *Journal of Veterinary Medicine. A, Physiology, Pathology, Clinical Medicine*. 3(4): 179-184. DOI: 10.1111/j.1439-0442.2006.00813.x
- 5 **Freud L.R., Escobar-Diaz M.C., Kalish B.T., Komarlu R., Puchalski M.D., Jaeggi E.T., Szwast A.L., Freire G., Levasseur S.M., Kavanaugh-McHugh A., Michelfelder E.C., Moon-Grady A.J., Donofrio M.T., Howley L.W., Tierney E.S.S., Cuneo B.F., Morris S.A., Pruetz J.D., Van Del Velde M.E., Kovalchin J.P., Ikemba C.M., Vernon M.M., Samay C., Satou G.M., Gitteiner N.L., Phoon C.K., Silverman N.H., McElhinney D.B. & Tworetzky W. 2015.** Outcomes and predictors of perinatal mortality in fetuses with Ebstein anomaly or tricuspid valve dysplasia in the current era: a multicenter study. *Circulation*. 132(6): 481-489. DOI: 10.1161/CIRCULATIONAHA.115.015839.
- 6 **MacDonald K.A. 2006.** Congenital heart diseases of puppies and kittens. *Veterinary Clinics Small Animal Practice*. 36(3): 503-531. DOI: 10.1016/j.cvsm.2005.12.006.
- 7 **Pereira G.G. & Larsson M.H.M.A. 2015.** Cardiopatias congênitas em cães e gatos. In: Jericó M.M., Andrade Neto J.P. & Kogika M.M. (Eds). *Tratado de Medicina Interna de Cães e Gatos*. Cap. 14. Rio de Janeiro: Roca, pp.1119-1136.
- 8 **Prat V., Rozec B., Gauthier C. & Lauzier B. 2017.** Human heart failure with preserved ejection versus feline cardiomyopathy: what can we learn from both veterinary and human medicine? *Heart Failure Reviews*. 22(6): 783-794. DOI: 10.1016/j.cvsm.2005.12.006.
- 9 **Scansen B.A., Schneider M. & Bonagura J.D. 2015.** Sequential segmental classification of feline congenital heart disease. *Journal of Veterinary Cardiology*. 17(Suppl 1): S10-S52. DOI: 10.1016/j.jvc.2015.04.005.
- 10 **Scurtu I., Tabaran F., Mircean M., Giurgiu G., Nagy A., Catoi C. & Ohad D.G. 2017.** Combined double chambered right ventricle, tricuspid valve dysplasia, ventricular septal defect and subaortic stenosis in a dog. *BMC Veterinary Research*. 13(1): 367. DOI: 10.1186/s12917-017-1275-1.

- 11 Uechi M., Harada K., Mizukoshi T., Mizuno T., Mizuno M., Ebisawa T. & Ohta Y. 2011.** Surgical closure of an atrial septal defect using cardiopulmonary bypass in a cat. *Veterinary Surgery*. 40(4): 413-417. DOI: 10.1111/j.1532-950X.2011.00798.x
- 12 Wainberg S. 2013.** Use of pimobendan in feline congenital heart failure. *Canadian Veterinary Journal*. 54(12): 1164-1166. ISSN: 0008-5286.