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Case Report

Mitral Valve Dysplasia Leading to Congestive Heart Failure in a Male Squirrel Monkey (*Saimiri boliviensis boliviensis*)

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Simple Summary

Squirrel monkeys (*Saimiri* species) are commonly housed in zoological collections and research facilities, where heart disease is considered an important cause of illness and death, especially in older animals. Heart disease in this species is most commonly related to aging, while congenital heart defects are rarely described. This report describes the case of an 11-year-old male black-capped squirrel monkey (*Saimiri boliviensis boliviensis*) that developed severe respiratory distress, abdominal enlargement caused by fluid accumulation, limb swelling, and collapse episodes. Clinical examination and advanced cardiac ultrasound revealed a severe malformation of the mitral valve. The abnormal valve structure caused severe mitral regurgitation, leading to congestive heart failure. Despite medical treatment, the animal's condition progressively worsened and euthanasia was elected. Post-mortem examination confirmed a congenital malformation of the mitral valve characterized by abnormal or absent supporting structures of the valve. To the authors' knowledge, this is the first report of this congenital heart defect in a squirrel monkey. This case expands the current knowledge of cardiovascular diseases affecting non-human primates and highlights the importance of cardiac imaging for the diagnosis of rare heart disorders in exotic species.

Abstract

Acquired cardiovascular diseases are recognized as a major age-related cause of morbidity and mortality in captive squirrel monkeys (*Saimiri* spp.). An 11-year-old intact male black-capped squirrel monkey (*Saimiri boliviensis boliviensis*) was presented following a syncopal episode associated with respiratory distress, abdominal distension, and peripheral edema. Upon presentation, the animal appeared thin and debilitated. Physical examination revealed respiratory distress with paradoxical breathing, abdominal distension, peripheral edema of the hind limbs, and hypothermia. Thoracic auscultation identified a loud systolic murmur over the left hemithorax and diffuse pulmonary crackles. Thoracic radiographs demonstrated severe cardiomegaly and perihilar pulmonary interstitial pattern consistent with cardiogenic pulmonary edema. Two-dimensional echocardiography revealed hyperechoic papillary muscles, thickened and restricted mitral valve leaflets, and shortened *chordae tendineae* with secondary severe left atrial and ventricular dilation. Doppler echocardiography demonstrated severe mitral regurgitation. Congestive heart failure secondary to mitral valve dysplasia was diagnosed. Treatment with diuretics and vasodilators resulted in transient clinical improvement followed by rapid deterioration of the patient's condition;

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therefore, euthanasia was elected. Gross pathological and histopathological examinations revealed thickened and irregular mitral valve leaflets with hypoplastic *chordae tendineae* associated with severe left-sided cardiac enlargement. To the authors' knowledge, this is the first report describing mitral valve dysplasia in an adult squirrel monkey. This report expands the spectrum of cardiovascular diseases described in squirrel monkeys and highlights the importance of advanced cardiac imaging in the diagnosis of rare congenital cardiac defects in non-human primates.

Keywords: atrioventricular valve dysplasia; congenital heart disease; echocardiography; non-human primates

1. Introduction

Squirrel monkeys (*Saimiri* spp.) are small neotropical primates belonging to the family Cebidae and are widely distributed throughout Central and South America. Due to their manageable size, social behavior, and physiological similarities to humans, squirrel monkeys (*Saimiri boliviensis*), particularly the subspecies *Saimiri boliviensis boliviensis*, are commonly housed in zoological collections and have historically been used as animal models in biomedical research [1].

Several diseases affecting captive squirrel monkeys have been described, including infectious, metabolic, degenerative, and cardiovascular disorders [2–4]. A squirrel monkey typically lives about 15–20 years in captivity, and age-related diseases are particularly common in captive populations due to increased life expectancy under human care. Frequently reported conditions include chronic renal disease, diabetes mellitus, obesity, degenerative joint disease, and cardiovascular pathology [5–8]. In aged squirrel monkeys, cardiovascular diseases is a leading cause of illness and death, involving degenerative conditions, while congenital heart diseases are rarely reported [9–11].

Mitral valve dysplasia (MVD) is a rare congenital heart defect characterized by the abnormal development of the mitral valve structures that can lead to mitral regurgitation and congestive heart failure (CHF) [12], and although well documented in dogs and cats [13–15], it is infrequently reported in non-human primates [16].

The present report describes the clinical, radiographic, echocardiographic, gross pathological, and histopathological findings of MVD leading to CHF in an adult black-capped squirrel monkey (*Saimiri boliviensis boliviensis*). To the authors' knowledge, this represents the first documented case of MVD in this species.

2. Case Presentation

2.1. Clinical Report

An 11-year-old intact male squirrel monkey (*Saimiri boliviensis boliviensis*) weighing 0.77 kg, housed at the Parco Faunistico Valcorba (a zoological park, Italy), was referred to the Veterinary Teaching Hospital of the University of Padua with a two-week history of respiratory distress, abdominal distension, peripheral edema and a syncopal episode.

On physical examination, the animal was debilitated and underconditioned (body condition score = 2/9). Coat quality was poor, with alopecic areas involving the tail and multiple cutaneous lesions. Mucous membranes were pink with a normal capillary refill time. The monkey was polypneic (50 breaths/min) and exhibited paradoxical breathing. Abdominal palpation revealed marked distension with a positive fluid wave consistent with ascites. Bilateral hind limb edema and hypothermia (T = 34 °C) were also evident.

Thoracic auscultation identified a loud left apical systolic murmur (grade IV/VI), with a heart rate of 130 bpm and diffuse pulmonary crackles.

Right lateral and dorsoventral thoracic radiographs were obtained in the awake animal (Figure 1). Both projections revealed generalized moderate-to-severe cardiomegaly, with a vertebral heart score of 10.6 vertebrae (v) (reference interval for right lateral vertebral heart score: 8.5–9.3 v [17]). Prominent pulmonary vessels and perihilar pulmonary interstitial pattern were also observed. Furthermore, increased soft tissue opacity ventrally and mild retraction of the caudal lung lobe, as well as decreased abdominal serosal contrast, suggested mild pleural effusion and severe abdominal effusion, respectively.

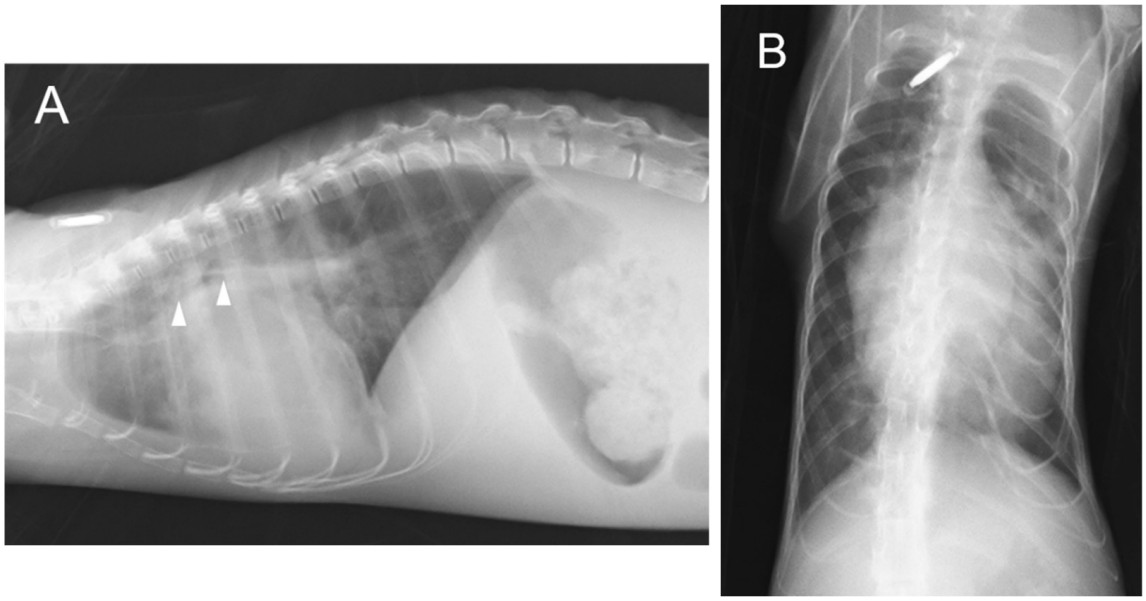

Figure 1. Thoracic radiographies: right lateral view (**A**) and dorsoventral view (**B**). Left-sided cardiomegaly is evident (vertebral heart score = 10.6 v, reference range = 8.5–9.3 v) resulting in dorsal displacement of the trachea and left mainstem bronchus (arrowheads). Prominent pulmonary vessels and a moderate perihilar interstitial pulmonary pattern are suggestive of cardiogenic pulmonary edema. Increased soft tissue opacity ventrally and mild retraction of the caudal lung lobe suggest the presence of a small volume of pleural effusion. Decreased abdominal serosal detail is consistent with a large volume of abdominal effusion.

Transthoracic echocardiography, including two-dimensional, M-mode, color Doppler, and spectral Doppler imaging, was performed following mask induction with sevoflurane in 100% oxygen at a fresh gas flow rate of 0.7 L/min. Anesthesia was maintained with sevoflurane in 100% oxygen, and physiological parameters were continuously monitored, with peripheral oxygen saturation (SpO_2) remaining consistently high (97–100%). The monkey was positioned in right and left lateral recumbency. Simultaneous electrocardiographic monitoring was recorded throughout the examination.

Two-dimensional echocardiography revealed the restricted motion of the mitral valve leaflets during both systole and diastole, consistent with Carpentier type IIIa classification [18], reduced diastolic excursion, shortened *chordae tendineae*, and hyperechoic papillary muscles with secondary severe LA and left ventricular enlargement (Figure 2).

Subjective right atrial and right ventricular dilation were also observed. The left atrial diameter (LA), measured from a right parasternal short-axis view at the heart base, was 17.2 mm (reference interval: 5.60–7.62 mm [10], with a left atrial-to-aortic root ratio (LA:Ao) of 4.1 (reference interval: 1.28–1.50)). M-mode echocardiography demonstrated increased left ventricular internal diameter in diastole (LVIDd = 13.6 mm; reference interval: 7.59–10.41 mm [10]).

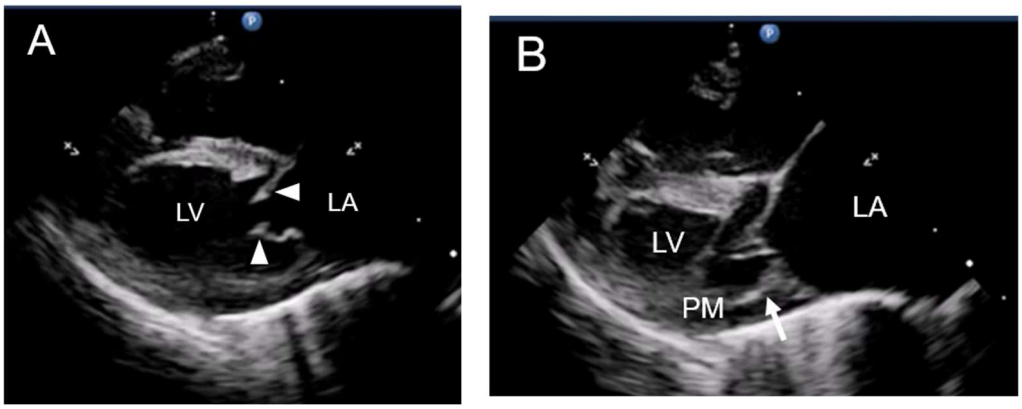

Figure 2. Two-dimensional echocardiographic images obtained from the right parasternal long axis view showing the left cardiac chambers and mitral valve in early diastole (**A**) and systole (**B**). (**A**) There is thickening and reduced opening of the mitral valve leaflets (arrowheads) in early diastole and (**B**) direct attachment of the posterior mitral valve leaflet to the papillary muscle (arrow). Severe left atrial enlargement is also evident. LA: left atrium; LV: left ventricle; PM: posterior papillary muscle.

Color flow and spectral Doppler interrogation demonstrated moderate-to-severe mitral regurgitation associated with increased early diastolic transmitral flow velocity (mitral valve E-wave velocity = 1.91 m/s; reference interval: 0.52–0.89 m/s). This finding supports the presence of a severe volume overload and elevated LA pressure associated with a peak pressure gradient of 14.7 mmHg, suggesting concurrent hemodynamically significant mitral stenosis. Moderate tricuspid regurgitation was also detected, with a peak systolic velocity of 2.97 m/s corresponding to an estimated systolic pressure gradient of 35 mmHg. Color and spectral Doppler interrogation of the Left Ventricle Outflow Tract (LVOT) and aorta evidenced laminar flow with a peak velocity of 1.25 m/s, and a peak gradient of 6.29 mmHg. Additional findings included mild pleural effusion and caudal vena cava distension without appreciable respiratory variation in diameter. Frequent ventricular premature complexes were observed during echocardiography and subsequently confirmed by six-lead electrocardiography.

Based on the clinical and echocardiographic findings, MVD and CHF were suspected. Medical treatment consisting of furosemide (2.4 mg PO q12h), benazepril (0.3 mg PO q12h), and spironolactone (2.5 mg PO q24h) was initiated. An initial improvement in respiratory rate and peripheral edema was observed; however, the clinical condition progressively deteriorated after 41 days, and euthanasia was ultimately elected for by the owner.

2.2. Gross and Histopathological Findings

At necropsy, the heart exhibited marked left atrial enlargement with severe dilation of the pulmonary veins. Moderate enlargement of both ventricles and mild-to-moderate dilation of the right auricle were also observed. Gross examination revealed diffusely reddish, congested and edematous lungs with the presence of pink frothy fluid within the tracheobronchial lumen, consistent with pulmonary edema. On longitudinal sectioning of the LA and left ventricle through the mitral valve and the left ventricular outflow tract, severe hypoplasia of the mitral *chordae tendineae* was evident, with the thickened and irregular mitral valve leaflets appearing almost directly attached to recognizable papillary muscles (Figure 3A). A focal whitish fibrous thickening was observed along the left ventricular endocardial surface, extending from the interventricular septum toward the region below the aortic valve. This lesion was associated with abnormal muscular trabeculations extending from the papillary muscle region to the interventricular septum (Figure 3B). No significant gross abnormalities were detected in the tricuspid, pulmonic, or aortic valves.

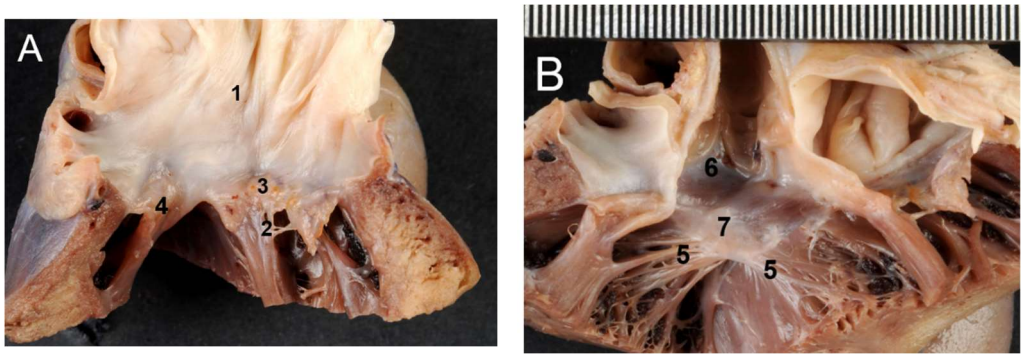

Figure 3. Internal view of the left heart in a squirrel monkey with mitral valve dysplasia, caudal view. (**A**) Severe left atrial dilatation (1); severe hypoplasia and aplasia of the mitral *chordae tendinae* (2), with irregular and thickened mitral valve leaflets (3) appearing to be directly attached to identifiable papillary muscles (4). (**B**) A fan-like arrangement of muscular trabeculae (5), arising from the endocardial surface of the lateral wall and interventricular septum and extending toward the interventricular septum just below the aortic valve, associated with a coarse whitish fibrous thickening (7), located ventral to the aortic valve (6).

Gross examination revealed mild hepatomegaly and moderate abdominal effusion, with no other gross abnormalities identified.

Histopathological examination confirmed severe hypoplasia and aplasia of the mitral *chordae tendineae*, associated with the persistence of immature myxoid connective tissue within the valve leaflet (Figure 4A). Moderate diffuse cardiomyocyte hypertrophy affecting both ventricles and the interventricular septum was also present, consistent with chronic cardiac remodeling. The whitish subaortic fan-like structure was composed of focal fibrous endocardial thickening (Figure 4B). Pulmonary histological findings included severe diffuse alveolar edema, multifocal moderate non-suppurative lymphoplasmacytic interstitial pneumonia, and mild multifocal alveolar emphysema. Additional findings included mild diffuse hepatic congestion and multifocal marked mineralization of the renal cortical tubular epithelium.

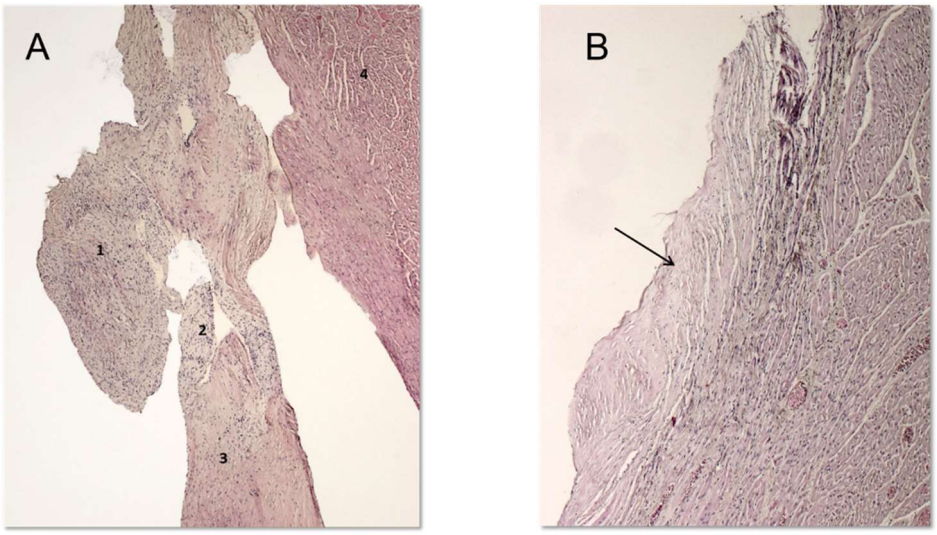

Figure 4. Photomicrographs of section of the left ventricle. (**A**) Histological examination revealed marked leaflet thickening associated with failure of normal valvular stratification and persistence of immature myxoid connective tissue within the valve leaflet. There are extensive myxoid changes in the posterior mitral valve leaflet (1), severe hypoplasia of the chordae tendineae (2), papillary muscle (3), and the lateral wall of the left ventricle (4). (**B**) Histological examination of the fan-like structure along the endocardial surface of the left ventricular interventricular septum. The structures were composed of well-differentiated fibrous tissue (arrow), shown using H&E staining.

Based on gross and histopathological findings, a diagnosis of MVD characterized by hypoplasia and aplasia of the chordae tendineae with secondary eccentric left ventricular hypertrophy, LA dilation, and CHF was established.

3. Discussion

Mitral valve dysplasia is a congenital malformation resulting from abnormal embryological development of the mitral valve apparatus, including the valve leaflets, *chordae tendineae*, papillary muscles, and annulus. This developmental abnormality is thought to occur during embryogenesis, potentially due to alterations in the normal delamination process involved in atrioventricular valve formation [19]. These structural abnormalities result in varying degrees of mitral regurgitation, volume overload, cardiac remodeling, and, in severe cases, CHF. Although MVD has been extensively described in dogs and, less frequently, in cats, reports in non-human primates are exceedingly rare [20].

Cardiovascular disease represents a major cause of morbidity and mortality in captive monkeys, particularly in aged individuals. Previous studies have described predominantly acquired and degenerative lesions, including myocardial fibrosis, arteriosclerosis, cardiomyopathy, valvular degeneration, and heart failure in several monkey species, including squirrel monkeys [9,21–25]. In contrast, congenital cardiac disorders have only sporadically been reported in non-human primates, and descriptions of congenital valvular dysplasia are exceptionally uncommon.

The present case demonstrated a strong correlation between the clinical, echocardiographic, and pathological findings. The initial clinical presentation of the animal was consistent with decompensated cardiogenic shock. The normal resting heart rate for a squirrel monkey typically ranges between 200 and 300 bpm [11], and tachycardia is the expected compensatory response in the early stage of CHF. On initial physical examination, the concurrent presence of hypothermia and bradycardia may indicate a failure of compensatory mechanism and a progression toward a decompensated stage of cardiogenic shock, reflecting a severe cardiovascular compromise.

Echocardiography identified hallmark features consistent with congenital MVD, including shortened *chordae tendineae*, thickened and restricted mitral leaflets, papillary muscle abnormalities, and severe mitral regurgitation with marked left-sided cardiac enlargement. These findings were subsequently confirmed at necropsy and by histopathological examination, which additionally revealed the persistence of immature myxoid connective tissue throughout the valve leaflet.

The near-direct insertion of the mitral valve leaflets onto the papillary muscles observed in this case is consistent with a severe developmental malformation of the subvalvular apparatus and likely represented the principal anatomical substrate for valvular incompetence. Similar lesions have been described in dogs with congenital MVD and are associated with restricted leaflet mobility and severe regurgitation [26]. The marked left atrial enlargement and eccentric left ventricular hypertrophy documented, both echocardiographically and at post-mortem examination, were consistent with chronic volume overload secondary to longstanding mitral insufficiency [13,15,26]. Likewise, the presence of ventricular premature complexes may have reflected myocardial remodeling and electrical instability associated with chronic cardiac dilation and hypertrophy [27].

Another noteworthy finding was the presence of abnormal muscular trabeculations associated with focal subaortic fibrous endocardial thickening. Although echocardiographic findings suggesting fixed or dynamic LVOT obstruction were not detected echocardiographically, these lesions may represent primary congenital malformations or secondary remodeling changes induced by chronic abnormal traction forces within the malformed mi-

tral valve apparatus. Similar subvalvular fibrous lesions have occasionally been reported in association with congenital mitral valve disease in domestic animals and humans [28–30].

The pulmonary edema, pleural effusion, ascites, and peripheral edema observed in this monkey were consistent with advanced CHF involving both the left and right sides of the heart. Right-sided involvement was further supported by the echocardiographic detection of moderate tricuspid regurgitation associated with right atrial and ventricular dilation and caudal vena cava distension. Similar sequelae, secondary to chronic severe mitral regurgitation resulting from either MVD or acquired myxomatous mitral valve disease, are commonly reported in companion animals [31]. Thus, the severe mitral valve dysfunction and marked left atrial enlargement observed in the present case support the hypothesis that post-capillary pulmonary hypertension developed secondary to chronic left-sided heart disease and contributed to the observed right-sided cardiac remodeling and clinical manifestations [32]. However, the concurrent presence of interstitial pneumonia and alveolar emphysema also suggests the possibility of a concomitant pre-capillary component contributing to pulmonary hypertension [31].

In human medicine, medical treatment of primary mitral regurgitation plays a limited role and is mainly aimed at managing complications such as CHF and arrhythmias, particularly atrial fibrillation. Surgical intervention is generally recommended for symptomatic patients and for asymptomatic individuals who develop left ventricular dysfunction [33]. In small animal practice, surgical mitral valve repair is available only in specialized centers and is therefore infrequently performed, whereas medical therapy is commonly used to stabilize animals with CHF [34]. In the present case, medical treatment was instituted following the diagnosis of CHF; however, it failed to achieve long-term control of the clinical signs.

Interestingly, despite the severity of the anatomical abnormalities, the animal survived until adulthood before developing overt CHF. This finding suggests the presence of prolonged hemodynamic compensation followed by progressive cardiac decompensation later in life. A similarly delayed clinical presentation has occasionally been reported in dogs affected by congenital mitral valve abnormalities, particularly in cases characterized by slowly progressive volume overload [15]. In the present case, concurrent myxomatous degeneration of the mitral valve leaflets may have further exacerbated valvular dysfunction and contributed to the eventual onset of clinical heart failure.

This case highlights the diagnostic value of transthoracic echocardiography for the ante-mortem identification of congenital cardiac defects in non-human primates. Despite the limited availability of species-specific reference data for exotic mammals, echocardiographic examination allowed accurate characterization of the mitral valve abnormalities and their hemodynamic consequences, findings that were subsequently confirmed by pathological examination.

4. Conclusions

This report expands the spectrum of congenital cardiovascular diseases recognized in squirrel monkeys and documents the description of MVD in a squirrel monkey (*Saimiri boliviensis boliviensis*). Congenital MVD should therefore be considered among the differential diagnoses in captive non-human primates presenting with cardiac murmurs, cardiomegaly, or signs of CHF. Furthermore, this case emphasizes the value of integrating clinical, echocardiographic, and pathological findings to achieve an accurate diagnosis and comprehensive characterization of rare cardiac disorders in exotic species.

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Institutional Review Board Statement: In accordance with the European regulations (Directive 2010/63/EU) on the protection of animals used for scientific purposes and with the Italian Legislative Decree no. 26/2014 (D.Lgs. 26/2014), non-experimental clinical veterinary practice is excluded from the scope of legislation and therefore ethical approval was not required for this study.

Informed Consent Statement: Written informed consent was obtained from the owner for clinical and diagnostic procedures.

Data Availability Statement: The original contributions presented in this study are included in the article. The data presented in this study are available on request from the corresponding author, in compliance with privacy and ethical requirements.

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Conflicts of Interest: The authors declare no conflicts of interest.

Abbreviations

The following abbreviations are used in this manuscript:

CHF	Congestive heart failure
LA	Left atrial
LVIDd	Left ventricular internal diameter in diastole
LVOT	Left ventricle outflow tract
MVD	Mitral valve dysplasia

References

1. Brady, A.G. Research techniques for the squirrel monkey (*Saimiri* sp.). *ILAR J.* **2000**, *41*, 10–18. [CrossRef] [PubMed]
2. Rogers, D.L.; McClure, G.B.; Ruiz, J.C.; Abee, C.R.; Vanchiere, J.A. Endemic Viruses of Squirrel Monkeys (*Saimiri* spp.). *Comp. Med.* **2015**, *65*, 232–240. [PubMed]
3. Lieberman, M.T.; Wachtman, L.M.; Marini, R.P.; Bakthavatchalu, V.; Fox, J.G. Spontaneous Cholelithiasis in a Squirrel Monkey (*Saimiri sciureus*). *Comp. Med.* **2016**, *66*, 63–67. [PubMed]
4. Daggett, G.J.; Wood, J.S.; Gumber, S.; Pinelli, C.J. Spontaneous Pituitary Adenomas in Squirrel Monkeys (*Saimiri sciureus*). *Comp. Med.* **2020**, *70*, 83–86. [CrossRef] [PubMed]
5. Gozalo, A.; Dagle, G.E.; Montoya, E.; Weller, R.E.; Málaga, C.A. Spontaneous cardiomyopathy and nephropathy in the owl monkey (*Aotus* sp.) in captivity. *J. Med. Primatol.* **1992**, *21*, 279–284. [CrossRef]
6. Stills, H.F., Jr.; Bullock, B.C. Renal disease in squirrel monkeys (*Saimiri sciureus*). *Vet. Pathol.* **1981**, *18*, 38–44. [CrossRef] [PubMed]
7. Ausman, L.M.; Rasmussen, K.M.; Gallina, D.L. Spontaneous obesity in maturing squirrel monkeys fed semipurified diets. *Am. J. Physiol.* **1981**, *241*, R316–R321. [CrossRef] [PubMed]
8. Portal, C.G.R.; Imbeloni, A.A.; Rahal, S.C.; da Silva, W.B.; Kano, W.T.; Yamashita, S.; Mayor, P.; Monteiro, F.O.B. Radiographic Assessment of Spinal Degeneration in Vervet Monkeys (*Chlorocebus aethiops*): Prevalence, Patterns, and Relevance to Primate Aging Models. *Am. J. Primatol.* **2026**, *88*, e70136. [CrossRef] [PubMed]
9. Brady, A.G.; Watford, J.W.; Massey, C.V.; Rodning, K.J.; Gibson, S.V.; Williams, L.E.; Abee, C.R. Studies of heart disease and failure in aged female squirrel monkeys (*Saimiri* sp.). *Comp. Med.* **2003**, *53*, 657–662. [PubMed]
10. Locquet, L.; Houdellier, B.; Broeckx, B.J.G.; Bouts, T.; Liekens, V.; Saunders, J.H.; Smets, P. Transthoracic echocardiography and cardiac biomarkers in healthy captive male and female squirrel monkeys (*Saimiri* spp.). *BMC Vet. Res.* **2020**, *16*, 217. [CrossRef] [PubMed]
11. Huss, M.K.; Ikeno, F.; Buckmaster, C.L.; Albertelli, M.A. Echocardiographic and electrocardiographic characteristics of male and female squirrel monkeys (*Saimiri* spp.). *J. Am. Assoc. Lab. Anim. Sci.* **2015**, *54*, 25–28. [PubMed]

12. MacDonald, K.A. Congenital heart diseases of puppies and kittens. *Vet. Clin. Small Anim. Pract.* **2006**, *36*, 503–531. [CrossRef] [PubMed]
13. Tidholm, A. Retrospective study of congenital heart defects in 151 dogs. *J. Small Anim. Pract.* **1997**, *38*, 94–98. [CrossRef] [PubMed]
14. Tidholm, A.; Ljungvall, I.; Michal, J.; Häggström, J.; Höglund, K. Congenital heart defects in cats: A retrospective study of 162 cats (1996–2013). *J. Vet. Cardiol.* **2015**, *17*, S215–S219. [CrossRef] [PubMed]
15. Oliveira, P.; Domenech, O.; Silva, J.; Vannini, S.; Bussadori, R.; Bussadori, C. Retrospective review of congenital heart disease in 976 dogs. *J. Vet. Intern. Med.* **2011**, *25*, 477–483. [CrossRef] [PubMed]
16. Sao-Ling, L.; Jane-Fang, Y.; Chian-Ren, C.; Shih-Chien, C. Coexisting tricuspid valve dysplasia and ventricular septal defect in a young patas monkey (*Erythrocebus patas*). *J. Zoo Wildl. Med.* **2004**, *35*, 576–579. [CrossRef] [PubMed]
17. Houdellier, B.; Liekens, V.; Smets, P.; Bouts, T.; Saunders, J.H. Thoracic radiography of healthy captive male and female Squirrel monkey (*Saimiri* spp.). *PLoS ONE* **2018**, *13*, e0201646. [CrossRef] [PubMed]
18. de Groot-de Laat, L.E.; McGhie, J.; Ren, B.; Frowijn, R.; Oei, F.B.; Geleijnse, M.L. A Modified Echocardiographic Classification of Mitral Valve Regurgitation Mechanism: The Role of Three-dimensional Echocardiography. *J. Cardiovasc. Imaging* **2019**, *27*, 187–199. [CrossRef] [PubMed]
19. Asante-Korang, A.; O'Leary, P.W.; Anderson, R.H. Anatomy and echocardiography of the normal and abnormal mitral valve. *Cardiol. Young* **2006**, *3*, 27–34. [CrossRef] [PubMed]
20. Schrope, D.P. Prevalence of congenital heart disease in 76,301 mixed-breed dogs and 57,025 mixed-breed cats. *J. Vet. Cardiol.* **2015**, *17*, 192–202. [CrossRef] [PubMed]
21. Tolwani, R.J.; Waggie, K.S.; Green, S.L.; Tolwani, A.J.; Lyons, D.M.; Schatzberg, A.F. Dilative cardiomyopathy leading to congestive heart failure in a male squirrel monkey (*Saimiri sciureus*). *J. Med. Primatol.* **2000**, *29*, 42–45. [CrossRef] [PubMed]
22. Lammey, M.L.; Baskin, G.B.; Gigliotti, A.P.; Lee, D.R.; Ely, J.J.; Sleeper, M.M. Interstitial myocardial fibrosis in a captive chimpanzee (*Pan troglodytes*) population. *Comp. Med.* **2008**, *58*, 389–394. [PubMed]
23. Kanthaswamy, S.; Reader, R.; Tarara, R.; Oslund, K.; Allen, M.; Ng, J.; Grinberg, C.; Hyde, D.; Glenn, D.G.; Lerche, N. Large scale pedigree analysis leads to evidence for founder effects of Hypertrophic Cardiomyopathy in Rhesus Macaques (*Macaca mulatta*). *J. Med. Primatol.* **2014**, *43*, 288–291. [CrossRef] [PubMed]
24. Baldessari, A.; Snyder, J.; Ahrens, J.; Murnane, R. Fatal myocardial fibrosis in an aged chimpanzee (*Pan troglodytes*). *Pathobiol. Aging Age Relat. Dis.* **2013**, *3*, 21073. [CrossRef] [PubMed]
25. Rishniw, M.; Schiavetta, A.M.; Johnson, T.O.; Erb, H.N. Cardiomyopathy in captive owl monkeys (*Aotus nancymae*). *Comp. Med.* **2005**, *55*, 162–168. [PubMed]
26. Sudunagunta, S.; Hamilton-Elliott, J.; Dukes-McEwan, J. Mitral valve dysplasia in eight English Springer Spaniels. *J. Vet. Cardiol.* **2021**, *33*, 52–60. [CrossRef] [PubMed]
27. Marcus, G.M. Evaluation and Management of Premature Ventricular Complexes. *Circulation* **2020**, *141*, 1404–1418. [CrossRef] [PubMed]
28. Otoni, C.; Abbott, J.A. Mitral valve dysplasia characterized by isolated cleft of the anterior leaflet resulting in fixed left ventricular outflow tract obstruction. *J. Vet. Cardiol.* **2012**, *14*, 301–305. [CrossRef] [PubMed]
29. Connolly, D.J.; Boswood, A. Dynamic obstruction of the left ventricular outflow tract in four young dogs. *J. Small Anim. Pract.* **2003**, *44*, 319–325. [CrossRef] [PubMed]
30. Carpentier, A.; Branchini, B.; Cour, J.C.; Asfaou, E.; Villani, M.; Deloche, A.; Relland, J.; D'Allaines, C.; Blondeau, P.; Piwnica, A.; et al. Congenital malformations of the mitral valve in children. Pathology and surgical treatment. *J. Thorac. Cardiovasc. Surg.* **1976**, *72*, 854–866. [PubMed]
31. Reinero, C.; Visser, L.C.; Kellihan, H.B.; Masseau, I.; Rozanski, E.; Clercx, C.; Williams, K.; Abbott, J.; Borgarelli, M.; Scansen, B.A. ACVIM consensus statement guidelines for the diagnosis, classification, treatment, and monitoring of pulmonary hypertension in dogs. *J. Vet. Intern. Med.* **2020**, *34*, 549–573. [CrossRef] [PubMed]
32. Borgarelli, M.; Bussadori, C.M. Pulmonary hypertension in dogs with myxomatous mitral valve disease. *J. Vet. Cardiol.* **2026**, *in press*. [CrossRef] [PubMed]
33. Otto, C.M.; Nishimura, R.A.; Bonow, R.O.; Carabello, B.A.; Erwin, J.P., 3rd; Gentile, F.; Jneid, H.; Krieger, E.V.; Mack, M.; McLeod, C.; et al. 2020 ACC/AHA Guideline for the Management of Patients With Valvular Heart Disease: A Report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. *J. Am. Coll. Cardiol.* **2021**, *77*, e25–197.
34. Saunders, A.B. Key considerations in the approach to congenital heart disease in dogs and cats. *J. Small Anim. Pract.* **2021**, *62*, 613–623. [CrossRef] [PubMed]