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Ventricular septal defect and congestive heart failure in a common degu (*Octodon degus*)

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Abstract

We report a case of ventricular septal defect causing congestive heart failure in a two-year-old, male common degu (*Octodon degus*). The patient presented for anorexia and dental disease, and a grade 4/6 holosystolic cardiac murmur was detected on physical exam. Thoracic radiographs showed cardiomegaly and a diffuse interstitial pulmonary pattern, consistent with congestive heart failure. Echocardiography was supportive of a perimembranous ventricular septal defect exhibiting low-velocity left-to-right shunting, and biatrial enlargement. These diagnoses were confirmed on post-mortem exam, along with pulmonary edema and cardiomyocyte hypertrophy, degeneration, and regeneration. This is the first published account of a ventricular septal defect and congestive heart failure in a degu.

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Keywords

congestive heart failure; congenital heart disease; echocardiography; ventricular septal defect; degu; *Octodon degus*

A two-year-old, male, pet common degu was presented for severe dental disease and anorexia. Prior to presentation, the owner noted a decrease in timothy hay consumption and increase in pellet consumption for approximatively a week, followed by anorexia for three days or more. The patient continued to drink water and there were no obvious changes in the patient's urination and defecation. The owner also noted weight loss and incisor malocclusion within the week prior to presentation. Husbandry was overall adequate. [1]

Abnormal physical exam findings included a low body condition score (2/9) with diffuse muscle atrophy, estimated 10% dehydration, increased respiratory excursions, unkempt hair coat with urine staining around the perineum, piloerection, and a hunched posture. Cardiac auscultation revealed a 4/6 holosystolic cardiac murmur and a regular rhythm. A point of maximal intensity of the murmur could not be determined due to the small patient size and the murmur radiating so widely across both hemithorax. Lung sounds were normal. The abdomen was mildly distended and painful with palpation consistent with a gas filled stomach and cecum. Oral exam showed severe incisor malocclusion with associated superficial ulceration of the mucosa, irregular and rough lateral gliding motion of the mandible, and abnormal occlusion surfaces of premolars and molars.

Prior to additional diagnostic tests, the patient was administered subcutaneous fluids (Lactated Ringer's solution; Baxter, Deerfield, Illinois 60015 USA; 75 mL/kg SQ q 12 hr), syringe fed Critical Care Herbivore diet mixed with water to make a slurry (Oxbow Animal Health, Omaha, Nebraska 68138 USA; 3-5 mL PO q 6-12 hr), and pain management with oxymorphone (Endo Pharmaceuticals, Malvern, Pennsylvania 19355 USA; 0.13 mg/kg SQ q 4 hr) initially for the first 12 hours followed by oral tramadol (Amneal Pharmaceuticals, Glasgow, Kentucky, 42141 USA; 15 mg/kg PO q 12 hr). Bloodwork revealed a mild neutropenia (1,181 cell s/µ L; reference values: 1,340-6,320 cells/µL[1]) with leucocytes in the lower normal range (1,875 cells/µL; reference values: 1,840-11,360 cells/µL[1]), marked hypoglycemia (48 mg/dL; reference values: 93-305 mg/dL[1]) and hypophosphatemia (2.0 mg/dL; reference values: 4.5-9.9 mg/dL[1]).

The patient was sedated for diagnostic imaging procedures using ketamine 2.0 mg/kg intramuscularly (IM; Vedco, St. Joseph, Missouri, 64507, USA), oxymorphone 0.2 mg/kg IM, and midazolam 1 mg/kg IM (West-ward Pharmaceuticals, Eatontown, New Jersey 07724, USA; midazolam dose was administered twice to obtained adequate level of sedation). Radiographs showed marked cardiomegaly with dorsal elevation of the trachea and a moderate, diffuse, unstructured interstitial pulmonary pattern in all lungs (Fig. 1 A). The pulmonary vasculature and caudal vena cava were difficult to clearly delineate due to the overlying pulmonary pattern and small patient size. No radiographic abnormalities were noted in the abdomen. Given the combination of radiographic findings, left-sided cardiac decompensation with cardiogenic edema secondary to congenital or acquired cardiac disease was the primary differential diagnosis. Other differentials for the pulmonary pattern, such as

non-cardiogenic edema or infectious/non-infectious inflammatory infiltrate were considered less likely. Osteoarthritis of the pelvic limbs and multifocal spondylosis deformans affecting the vertebral column was also observed.

A cardiology consultation was performed based on the radiographic exam findings. Echocardiography with Doppler was suggestive of a perimembranous ventricular septal defect (VSD) with low-velocity left-to-right shunting of blood (Fig. 2). Biatrial enlargement was suspected but could not be confirmed due to lack of reference measurements. The right-sided heart enlargement coupled with the low-velocity left-to-right shunting was interpreted to imply some degree of pulmonary hypertension was apparent. Left atrium (LA) diameter was 0.83 cm, aortic (Ao) diameter was 0.38 cm, LA/Ao ratio was 2.20, and pulmonic valve maximum velocity was 1.15 m/sec.

Sedated computed tomography scan of the head confirmed generalized dental disease with marked elongation of the clinical crown and malocclusion of the mandibular and maxillary arches. After diagnostic procedures, the patient was reversed with flumazenil 0.02 mg/kg IM (West-ward Pharmaceuticals, Eatontown, New Jersey 07724, USA) and had a smooth recovery.

Based on the diagnostics performed, clinical interpretation was left-sided congestive heart failure (CHF). Subcutaneous fluids were immediately discontinued, and furosemide was administered at 1 mg/kg SQ q 12 hr (West-ward Pharmaceuticals, Eatontown, New Jersey 07724, USA). Furosemide administration was discontinued after three doses due to worsening dehydration, which put the patient at risk for reoccurrence of gastrointestinal stasis. Pimobendan administration was considered but not initiated because of a lack of a parenteral formulation and for fear of exacerbating gastrointestinal clinical signs (anorexia). Sildenafil administration was not considered for fear of worsening left heart volume overload and exacerbating pulmonary edema. Forty-eight hours after the initial radiographs were taken, and 24 hours after the last dose of furosemide, the patient was sedated again using the previously described protocol (with only one dose of midazolam required) and thoracic radiographs were repeated (Fig. 1B). Images showed static cardiomegaly and a mildly improved but persistent diffuse interstitial pulmonary pattern.

Due to the marked pain associated with the untreated severe dental disease and the risks related to secondary gastrointestinal stasis, the patient was placed under general anesthesia the following day and a dental occlusal adjustment was performed, knowing the increased anesthetic risks. The patient was sedated with ketamine 2.0 mg/kg IM, oxymorphone 0.2 mg/kg IM, and midazolam 1 mg/kg IM He was then induced and maintained on sevoflurane via facemask, and a 25-gauge intraosseous (IO) catheter was placed in the left femur. After the occlusal adjustment, the patient was reversed with flumazenil 0.02 mg/kg IO The patient was hypothermic and slow to recover from anesthesia, so a second dose of flumazenil 0.02 mg/kg IO was administered, and the patient was placed on supplemental oxygen in a warm incubator. Approximately one hour after anesthesia had been stopped and reversal agents administered, the patient began breathing agonally and then went into respiratory arrest. Cardiopulmonary resuscitation (CPR) was initiated, and the patient was euthanized after unsuccessful CPR due to poor prognosis for recovery.

On necropsy, the thoracic cavity contained ~1 mL of pink, watery, transparent effusion. The heart had moderate to marked dilation of the atria (right side more severely than the left; Fig. 3A) and a perimembranous ventricular septal defect (Fig. 3B) measuring 2 mm in diameter. Histology of the heart muscle showed variable (mild to severe), multifocal to coalescing cardiomyocyte hypertrophy and multifocal cardiomyocyte degeneration and regeneration (prominent and increased numbers of interstitial or satellite cells, increased numbers and rowing of cardiomyocyte nuclei, and cardiomyocyte hypertrophy and disarray), with the right side more severely affected than the left. The lungs had moderate to severe, diffuse pulmonary edema, and microscopically had alveolar histiocytosis and congestion. The liver exhibited diffuse, acute, central vein dilation and congestion with surrounding sinusoid dilation and congestion. These pathologic findings suggest that the degu had both right and left-sided cardiac dysfunction, resulting in pulmonary edema and acute, passive congestion of the liver. Concurrent pulmonary hypertension was suspected to be the cause for the right-sided pathology.

Ventricular septal defects (VSD) are a congenital abnormality of the heart that develop in the embryonic stage, and lead to an opening in the septum dividing the right and left ventricles of the heart. [2] Generally, this results in blood being shunted from the high-pressure left ventricle to the lower pressure right ventricle. [2] Left-to-right shunting VSDs lead to pulmonary overcirculation, increased pulmonary venous return, and dilation of the left atrium and ventricle. [2] The size of the VSD and the relative pressures of the systemic and pulmonary circulation are the most important factors in determining the direction and magnitude of blood shunting and the resulting clinical signs.[2] Reported cases of VSDs in wild and captive exotic species cover a wide range of taxa, including primates,[3,4] ungulates,[5,6] rodents,[7] and birds.[8,9] Although rare, anecdotal reports of congenital heart disease such as VSDs have been described in chinchillas (*Chinchilla lanigera*).[10] This is the first published report of VSD in a common degu.

There are no references to naturally occurring degu cardiac disease in the peer-reviewed literature, and no published echocardiogram measurements for degus (electronic searches for peer-reviewed manuscripts were performed in PubMed, JSTOR, Scopus, Web of Science, and Google Scholar). For this reason, data on chinchillas were used for comparison. Chinchillas have been found to have a 23% prevalence of heart murmurs, and animals with a grade ≥3 were approximatively 29 times more likely to have echocardiographic abnormalities. [10] Echocardiograms of clinically healthy, anesthetized male chinchillas showed LA diameters of 0.53 ± 0.05 cm (mean ± SD), LA/Ao diameter of 1.49 ± 0.17 cm, and pulmonary artery peak flow velocities of 0.61 ± 16 m/s. [11] Despite degus being smaller in size, our patient had a larger LA diameter and LA/Ao than the upper bound of that measured in chinchillas, perhaps as a result of his significant cardiac disease and increased volume overload to the left side of his heart.

Our patient’s maximum pulmonic valve velocity was 1.15 m/s, which is higher than that measured in chinchillas. [11] This is likely secondary to the left-to-right shunting of the VSD and subsequent pulmonary over circulation. Concurrent pulmonary hypertension (secondary to the VSD) likely resulted in elevated right ventricular pressure and a decreased pressure gradient between the ventricles, slowing the flow of blood across the VSD. In dogs

and cats, low-velocity flow (<4.5 m/s) across the VSD in combination with elevated pulmonic valve velocities are considered “unrestrictive,” and indicate that a large volume of blood is being shunted between the ventricles. [2] Compared to high-velocity, “restrictive” VSDs, these “unrestrictive” VSDs are generally more hemodynamically significant and more likely to be associated with clinical signs. [2] An alternate consideration for the low-velocity VSD flow is Doppler cursor misalignment and underestimating blood flow velocity through the defect. However, this is less likely given the overt right heart enlargement noted on gross pathology examination. In dogs and cats, VSDs are associated with exercise intolerance, syncope, cyanosis, heart murmurs, pulmonary hypertension, CHF, and endocarditis. [2] Our degu patient had a prominent heart murmur and radiographic evidence of CHF. The only overt clinical sign of cardiac disease detected was increased respiratory excursions. However, based on the severity of his cardiac disease, we expect that additional clinical signs may have been masked by his co-morbidities.

This patient had a myriad of health problems that lead to his ultimate respiratory arrest and euthanasia, including a VSD, CHF, and severe dental disease resulting in anorexia, gastrointestinal stasis, and inadequate body condition. It is unknown if the patient was in CHF prior to the administration of fluids to correct dehydration, or if the supplemental fluids caused the patient’s cardiac disease to decompensate while hospitalized. It is also unknown how large a role the patient’s underlying cardiac disease played in his anorexia and overall ill thrift, or if these were primarily caused by the patient’s dental disease. However, it is likely that the patient’s VSD and CHF played a major a role in his inability to fully recovery from anesthesia.

This case presents the first report of VSD in a common degu with CHF and describes diagnostic data that will be useful in the ante-mortem diagnosis and treatment of future patients. Research regarding radiographic evaluation of the heart size and echocardiographic evaluation in degus is needed.

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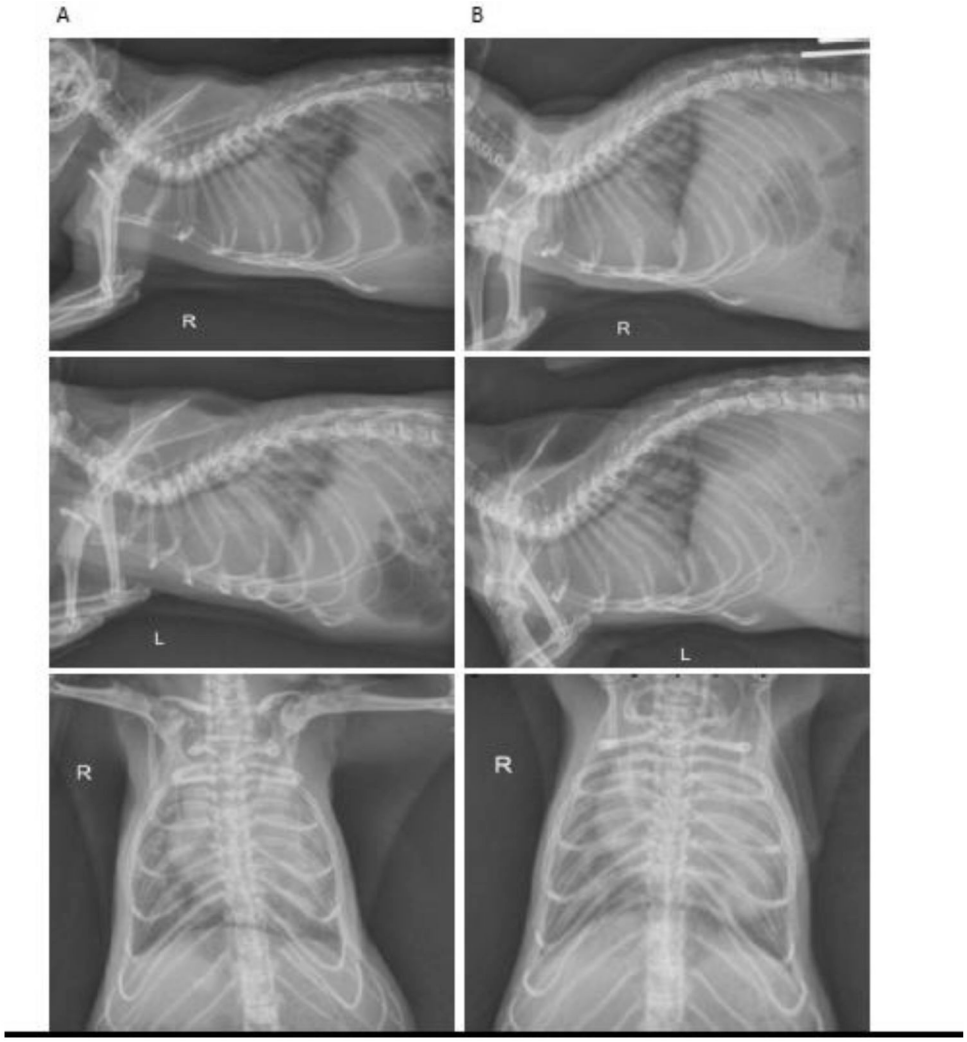

Figure 1.

Thoracic radiographs of a degu in congestive heart failure. (A) Initial radiographs, taken after patient stabilization and administration of subcutaneous fluids, demonstrated cardiomegaly with dorsal deviation of the trachea and a diffuse interstitial pulmonary pattern in all lung fields, consistent with congestive heart failure. (B) Radiographs taken 48 hours later (24 hours after last dose of furosemide) showed static cardiomegaly and mildly improved but persistent interstitial pulmonary pattern.

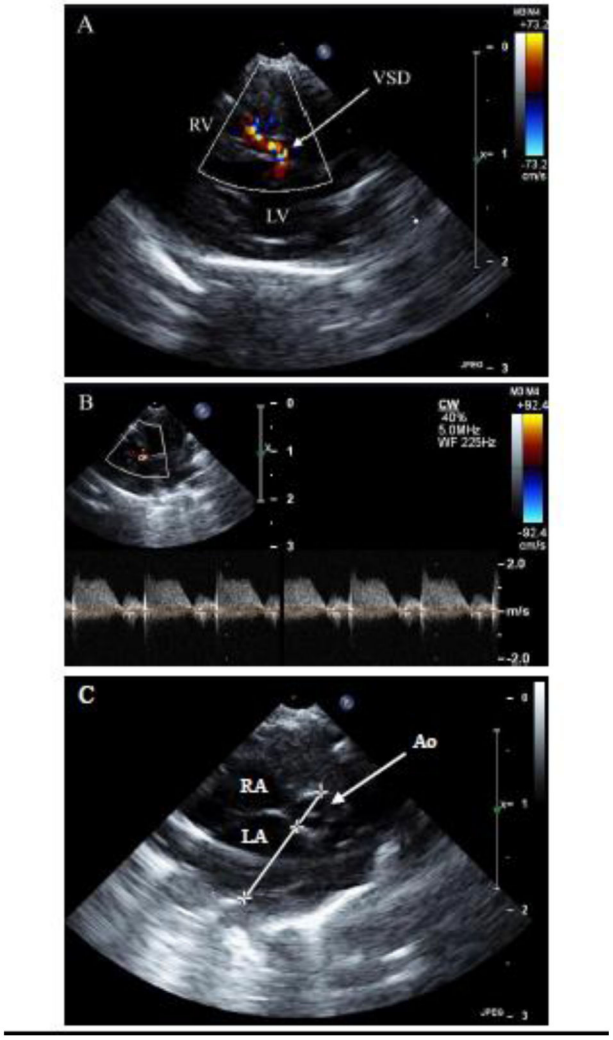

Figure 2.

Echocardiographic images of a degu heart with a ventricular septal defect. (A) Right parasternal long axis imaging plane demonstrating the perimembranous ventricular septal defect (VSD) exhibiting left-to-right shunting of blood from the left ventricle (LV) to the right ventricle (RV). (B) Spectral Doppler imaging demonstrating the blood flow across the VSD was low-velocity, peaking at ~2 m/s, suggestive of a right ventricular systolic pressure overload (e.g., pulmonary hypertension). (C) The left atrium (LA) to aorta (Ao) ratio was 2.20, suggestive of left atrial enlargement (RA = right atrium). Calipers indicate how measurements of the LA (0.83 cm) and Ao (0.38 cm) diameter were obtained.

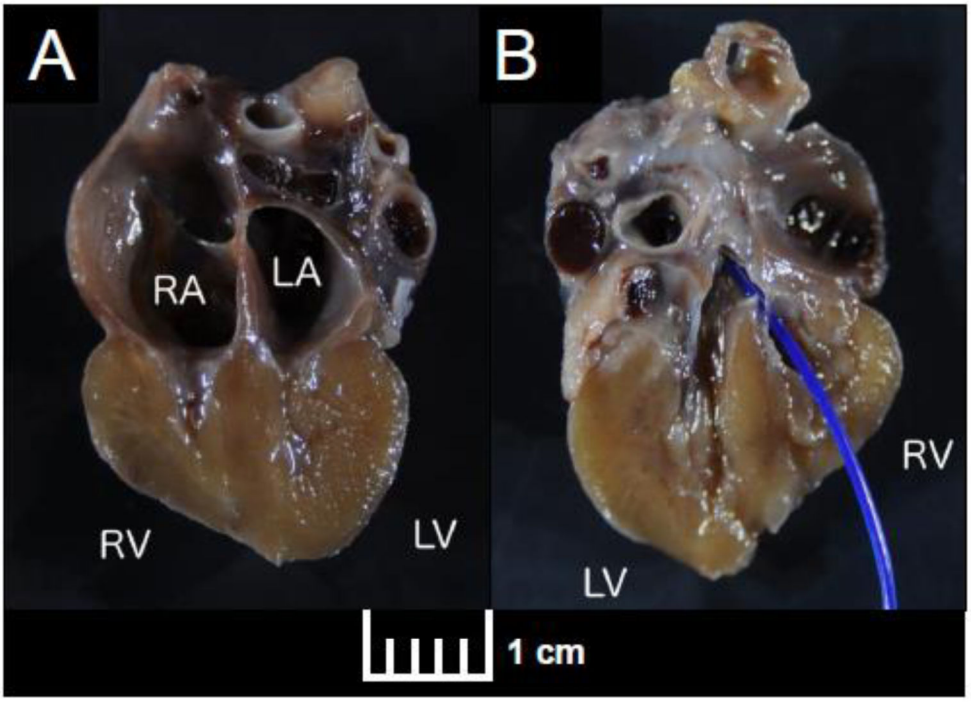

Figure 3.

Gross pathology image of a degu heart with a ventricular septal defect. (A) The right and left atria (RA and LA, respectively) were markedly dilated and the right ventricle (RV) was hypertrophied (LV = left ventricle). (B) The blue wire shows the perimembranous ventricular septal defect.