Thoracic Trauma Causing an Acquired Gerbode Defect, Aortic Sinus Rupture, and Third-Degree Atrioventricular Block With Secondary Endocarditis in a Dog

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INTRODUCTION

The Gerbode defect is a unique presentation of a ventricular septal defect (VSD) that results in a left ventricle (LV) to right atrium (RA) shunt. This is a rare defect, although it is becoming increasingly recognized as a complication of cardiac interventional procedures in human patients.[1-3] Untreated, Gerbode defects can lead to complications including heart failure, infective endocarditis (IE), and death.[3] For that reason, recognition is crucial to allow for appropriate treatment and management. Here we present a case that illustrates the diagnosis and progression of an acquired Gerbode defect with concurrent ruptured aortic sinus and third-degree atrioventricular (AV) block in an adult dog that experienced blunt thoracic trauma. Secondary IE of the tricuspid valve (TV) was also suspected.

CASE PRESENTATION

A previously healthy 3-year-old female spayed English setter with no history of a heart murmur was referred to the cardiology service of a veterinary teaching hospital for evaluation of third-degree AV block. One week prior to presentation to the teaching hospital, the dog was running at full speed and hit a stationary object, resulting in blunt thoracic trauma and a relatively superficial but penetrating wound in the left axilla that did not require sutures. The dog collapsed following the episode and continued to have collapsing episodes, prompting evaluation by the primary veterinarian. No wound care was provided, and third-degree AV block was suspected, prompting referral.

On presentation to the veterinary teaching hospital, the dog was bradycardic (56 bpm; reference range: 60-160 bpm) with a grade IV/VI right apical continuous heart murmur present. An approximately 1 cm sanguinous crust was noted in the left axillary region covering a recent puncture wound, and the patient had moderate generalized muscle wasting with peripheral lymphadenopathy. An electrocardiogram (ECG; Figure 1A) was performed and was consistent with third-degree AV block with a ventricular escape rate of 55 bpm and an atrial rate of 210 bpm. No ventricular premature complexes were observed.

Transthoracic echocardiography (TTE) revealed mild dilation of all heart chambers (Figure 2; Video 1).

There was a focal region of hyperechoic tissue on the right atrial side of the interatrial septum, which was thought to represent traumatic intramural hemorrhage or, less likely, interatrial endocarditis (Figure 2, Video 1). The septal tricuspid leaflet was hyperechoic and thickened, raising concern for possible endocarditis (Video 2). A defect was observed within the membranous portion of the interventricular septum, which connected the LV outflow tract to the RA. Left-to-right, high-velocity systolic flow (maximum velocity, 6.5 m/sec) was noted through the defect. These findings were consistent with a Gerbode defect (Figure 3, Video 2).

Additionally, high-velocity flow throughout diastole (velocities at end diastole were 3.0 m/sec) was observed, and this finding could not be explained by the Gerbode defect alone (Figure 3B). This prompted further evaluation of the defect with color-flow Doppler interrogation, and an aortic sinus rupture with diastolic shunt flow from the right coronary sinus into the RA was identified (Figure 4, Videos 3 and 4). The echocardiographic findings indicated a Gerbode defect, complicated by concurrent aortic sinus rupture.

During this initial TTE, a nonsustained run of ventricular tachycardia was observed that resulted in a 10-second period of ventricular asystole attributed to overdrive suppression. A precordial thump was administered that produced the reemergence of ventricular escape beats. Emergent pacemaker implantation was performed due to the perceived risk of sudden death, and an epicardial approach was elected given the concern for endocarditic lesions. Epicardial pacemaker implantation was performed without complication.

Diagnostic testing prior to pacemaker implantation revealed severe elevation in cardiac troponin I (2.5 ng/mL; reference range, <0.2 ng/mL), which was considered indicative of myocardial damage from the patient's traumatic event. Three peripheral sites were chosen, and each had a sterile preparation performed on the skin immediately prior to blood sampling. Each blood sample was placed in a separate blood culture tube, and all 3 samples were ultimately positive for *Staphylococcus pseudointermedius* growth. The patient was hospitalized for 5 days and received intravenous cefazolin (22 mg/kg every 6 hours) and oral clopidogrel (2 mg/kg every 12 hours) until discharge.

Serial TTEs were performed each day of hospitalization, which revealed progressive right atrial and right ventricular dilation and subjectively increased shunting of flow through the Gerbode defect (Figure 5, Videos 5-7).

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Keywords: Arrhythmia, Infection, Shunt, Veterinary

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2468-6441
https://doi.org/10.1016/j.case.2024.02.001

VIDEO HIGHLIGHTS

Video 1: Two-dimensional TTE, right parasternal long-axis view during third-degree AV block, demonstrates cardiac chamber dilation, normal global LV systolic function, and a focal, hyperechoic lesion on the right atrial side of the interatrial septum.

Video 2: Two-dimensional TTE, right parasternal long-axis view, without (*left*) and with (*right*) color-flow Doppler, demonstrates an atypical intermediate Gerbode defect, turbulent, high-velocity flow shunting into the RA, and a hyperechoic, thickened septal TV leaflet.

Video 3: Two-dimensional TTE, right parasternal basal short-axis view, without (*left*) and with (*right*) color-flow Doppler, demonstrates an aortic sinus rupture with turbulent blood flow originating at the level of the right coronary sinus and shunting into the RA throughout systole and diastole.

Video 4: Two-dimensional TTE, right parasternal long-axis view, without (*left*) and with (*right*) color-flow Doppler, demonstrates an aortic sinus rupture with turbulent, high-velocity flow shunting into the RA and RV throughout systole and diastole. The color-flow signal from the aortic sinus rupture and the Gerbode defect cannot definitively be separated.

Video 5: Two-dimensional TTE, right parasternal long-axis view, performed 1 day after pacemaker implantation, demonstrates a Gerbode defect and associated mass lesion (suspected vegetation) near the exit of the shunt within the RA.

Video 6: Serial 2D TTE, right parasternal long-axis view, performed days later, demonstrates the Gerbode defect and a larger mass lesion at the same RA location.

Video 7: Two-dimensional TTE, right parasternal long-axis view, without (*left*) and with (*right*) color-flow Doppler, performed on hospital day 4, demonstrates a subjective increase in left-to-right shunting through the Gerbode defect compared to the initial echocardiogram on hospital day 1.

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The septal leaflet of the TV became progressively hyperechoic over this time frame, and a discrete lesion suspected to be endocarditic in origin developed near the septal tricuspid leaflet in association with the shunt exit while the patient was hospitalized. Additionally, serial thoracic radiographs were performed, which documented the development of progressive interstitial to alveolar infiltrates within the left cranial and right middle lung lobes. The patient eventually developed mild tachypnea at the end of hospitalization. These developments were attributed to septic pulmonary thromboemboli from the suspected TV endocarditic lesion and hematogenous spread of bronchopneumonia from the documented bacteremia on blood cultures. Aspiration pneumonia was considered less likely based on the clinical events. The dog was discharged 5 days after initially presenting to the hospital and was prescribed oral clopidogrel (2 mg/kg every 12 hours) and amoxicillin-clavulanic acid (22 mg/kg every 12 hours). Thoracic radiography performed 1 week after discharge showed significant improvement of the pulmonary infiltrates and static pacemaker lead placement.

Thirty days later, the dog experienced 2 syncopal events. An ECG was performed and revealed bipolar pacing spikes without associated ventricular depolarization (pacing spike rate of 100/minute, ventricular escape rate of 55/minute). When the patient was moved from the right to the left lateral recumbency position, pacing spikes preceded each ventricular depolarization, indicating appropriate pacing (Figure 1B). Neither pacemaker interrogation nor thoracic radiographs revealed a cause for intermittent failure to capture. Paroxysms of accelerated idioventricular rhythm and ventricular bigeminy were observed throughout interrogation (Figure 1C). A TTE revealed a reduction in size of the previously observed suspected TV endocarditic lesions. Additionally, cardiac troponin I concentrations were decreased compared to the previous exam (0.7 ng/mL). Progressive right atrial and right ventricular dilation were observed, and peritoneal effusion was present. These findings were consistent with right-sided heart failure. Adjustments to pacemaker function were made (primarily increased amplitude of generator output), and proper pacemaker function was verified prior to discharge. Furosemide (1 mg/kg every 24 hours) was also prescribed.

Two weeks later, the dog developed anorexia and progressive ascites. An ECG confirmed appropriate pacemaker function with no ventricular ectopy, and spironolactone (2 mg/kg every 12 hours) and pimobendan (0.3 mg/kg every12 hours) were initiated. Nine days later, the dog experienced sudden cardiac death at home. Postmortem pacemaker interrogation revealed a significant increase in the number of tachycardia events in the 2 days prior to death, compared with only rare tachycardia events previously recorded. The last recorded pacemaker activity was 20 minutes of high rate (>180/minute) tachycardia.

A gross postmortem evaluation of the heart was performed. The epicardial pacemaker lead was well adhered to the epicardium. A discolored, well-demarcated, slightly depressed, wedge-shaped region of myocardium was observed near the LV apex at the point of pacemaker lead insertion. These findings suggested an infarction had occurred (Figure 6). Residual pericardium was observed around the insertion of the epicardial lead to the epicardium, possibly in response to local inflammation.

The right atrial appendage and RA were both severely enlarged. Upon incising the RA, a focal region of pale tissue was noted on the right atrial free wall, most consistent with a jet lesion. The septal leaflet of the TV was thickened and irregular and had vegetative-like growths surrounding it. A focal, circular area of red discoloration was noted on this leaflet, and the Gerbode defect allowing direct communication between the RA and LV was confirmed (Figures 6 and 7). Direct communication between the aortic root and the RA via a defect in the aortic sinus was also confirmed (Figure 7).

DISCUSSION

The patient described in this report had a suspected traumatic origin for a Gerbode defect as well as an aortic sinus rupture, a combination not commonly encountered. The Gerbode defect is a unique type of VSD, which results in LV-RA shunting. Each Gerbode defect can be classified based on the position of the defect in relationship to the TV.[1] Supravalvular defects, also known as direct or type I defects, exist superior to the TV at the level of the AV septum and result in direct LV-RA shunting.[1-3] Infravalvular defects, also known as indirect or type II defects, consist of a VSD ventral to the septal leaflet of the TV with a concurrent defect in the tricuspid leaflet that allows

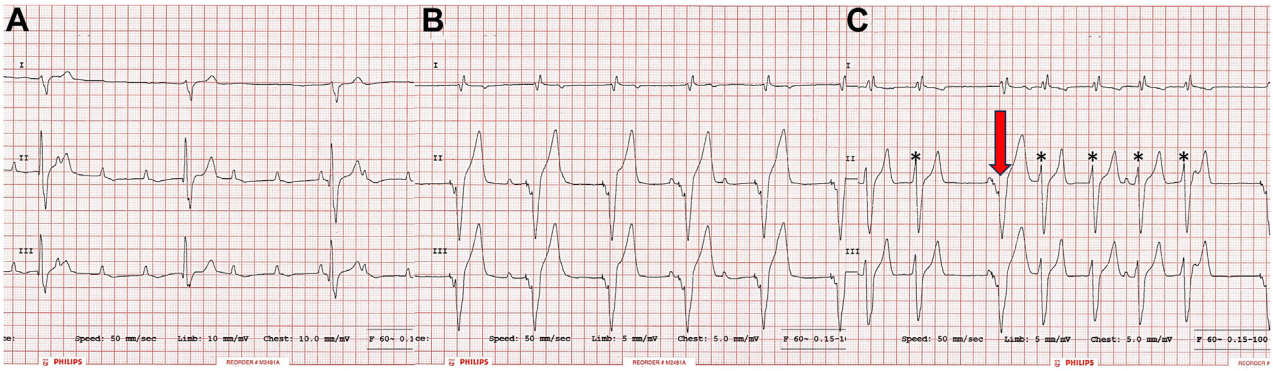

Figure 1 An ECG with leads I, II, and III at initial examination **(A)**, on the day following pacemaker implantation **(B)**, and at recheck examination **(C)** 30 days later. **(A)** Third-degree AV block is observed. **(B)** Bipolar VVI pacing via an epicardial pacemaker at 100 bpm is observed. **(C)** Bipolar VVIR pacing (*arrow*) with rate response settings to give a range of 65 to 150 bpm and frequent ventricular ectopy (*) is observed. 50 mm/sec, 1 cm = 1 mV.

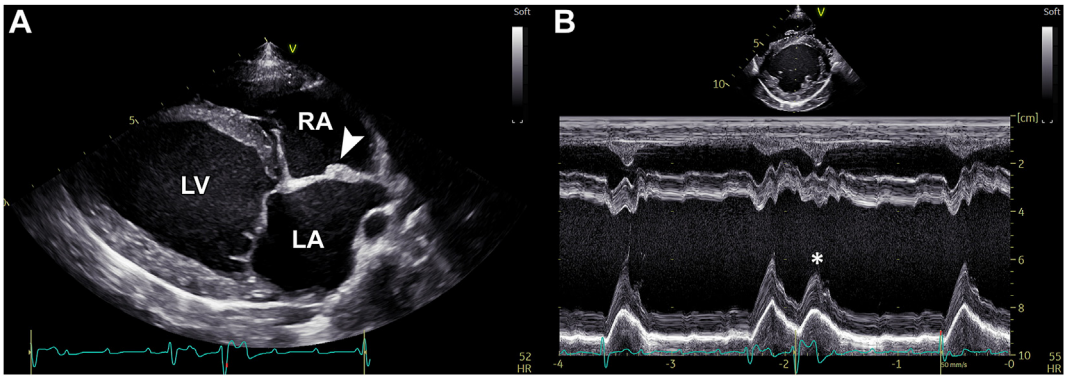

Figure 2 **(A)** Two-dimensional TTE, right parasternal long-axis view, demonstrates moderate dilation of all cardiac chambers and a hyperechoic interatrial septum with a focal, nodular thickening (*arrowhead*). **(B)** M-mode TTE of the LV and RV, short-axis view, demonstrates appropriate systolic function, third-degree AV block, and occasional single ventricular ectopic complexes (*asterisk*). *LA*, Left atrium.

regurgitant blood to enter the RA from the right ventricle (RV).[1-3] Intermediate, type III, defects have both supravalvular and infravalvular components.[1-3] This patient's Gerbode defect was in a supravalvular location; however, color-flow Doppler interrogation demonstrated abnormal blood flow into both the RA and RV. The typical classification schemes of Gerbode defects do not account for abnormal shunting originating from the aortic sinus, as this is not a common concurrent lesion. The presence of the aortic sinus rupture is challenging, as it is likely that some of the blood observed shunting into the RV originated at the aortic sinus.

Congenital Gerbode defects are considered rare, occurring in <1% of human patients with congenital heart disease and only 0.08% of all patients with congenital cardiac shunts.[1,3] These defects are similarly rare in veterinary medicine, with only 3 case reports in veterinary medicine, and all were speculated to be congenital in origin; 2 kittens[4,5] and one 9-year-old Yorkshire terrier.[6] As interventional cardiology has progressed, acquired iatrogenic Gerbode defects (AIGDs) are increasingly recognized as the predominant etiology in human patients.[1-3] In a review of 237 patients that had Gerbode defects, 121 (51.1%) were AIGDs.[3] The most common causes for AIGD include cardiac surgery near the membranous AV septum, with aortic and mitral valve replacements being the most frequently implicated procedures, as well as percutaneous tricuspid annuloplasty.[1,3] In contrast, there are no reports of AIGDs in veterinary medicine to date. However, this may be an important consideration in veterinary medicine as interventional approaches to the mitral and aortic valve become more prevalent.

Acquired noniatrogenic Gerbode defects (ANIGDs) are less common in human patients, with IE responsible for 36.7% of defects and trauma responsible for only 9.3%.[3] This is in sharp contrast to veterinary medicine, in which these are responsible for the majority of Gerbode defects, including in the dog of this report.[7-11] Based on the acute onset of the Gerbode defect and the aortic sinus rupture, in addition to the gross appearance of these lesions, a traumatic etiology for both of these lesions was believed to be the most likely. There are 3 previously reported dogs that suffered blunt trauma leading to ANIGD, and all of these dogs developed arrhythmias.[7-9] One dog had a ventricular tachyarrhythmia and first-degree AV block,[7] while 2 dogs had third-degree AV block.[8,9] All of these patients survived to discharge; 2 patients were asymptomatic on cardiac medications at the time of publication,[7,8] and 1 patient had spontaneous closure of the Gerbode defect after 2 months.[9] Similarly, our patient suffered blunt thoracic trauma and acutely developed third-degree AV block.

In contrast to the relatively positive outcomes associated with ANIGD due to trauma, ANIGD secondary to IE is associated with

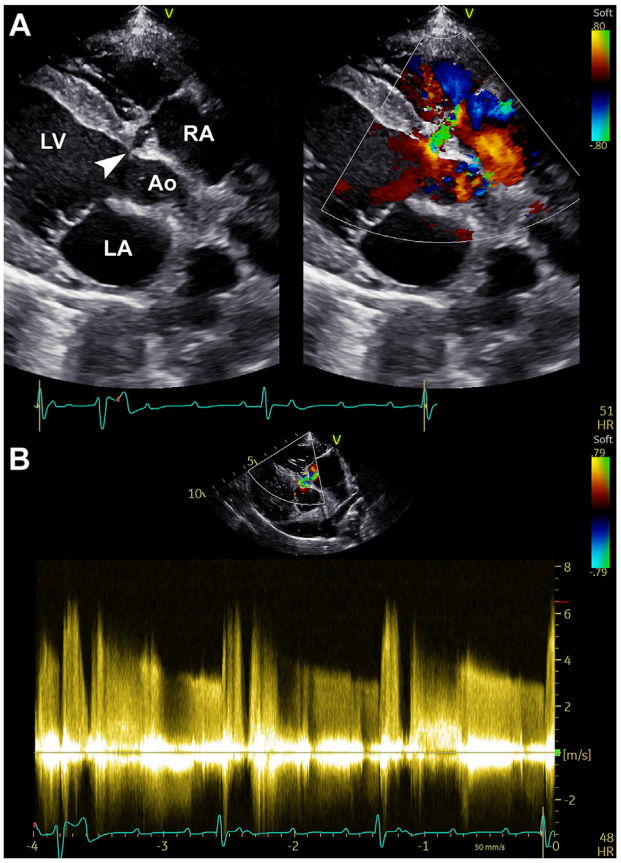

Figure 3 **(A)** Two-dimensional TTE, right parasternal long-axis view in systole, without (*left*) and with (*right*) color-flow Doppler, demonstrates a septal defect (*arrowhead*) connecting the LV and the RA, and turbulent blood flow moving from the LV to the RA. **(B)** Continuous-wave Doppler interrogation of the turbulent blood flow signal entering the RA reveals high-velocity, left-to-right shunting during both systole and diastole. *Ao*, Aortic sinus; *LA*, left atrium.

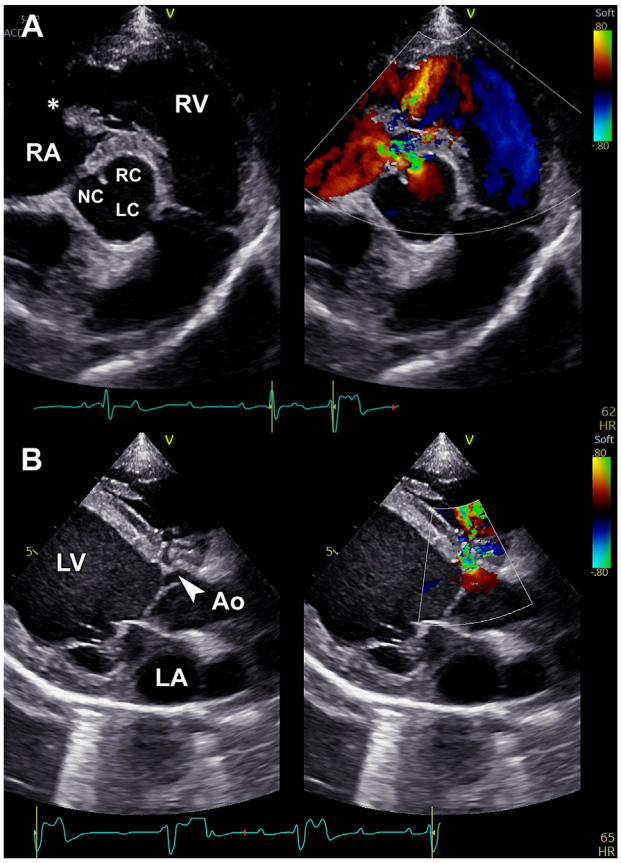

Figure 4 **(A)** Two-dimensional TTE, right parasternal long-axis view in diastole, without (*left*) and with (*right*) color-flow Doppler, demonstrates a rupture of the right coronary (RC) sinus with turbulent blood flow from the aorta to the RA. The septal leaflet of the TV (*) is thickened, and a single ventricular premature complex is present. **(B)** Two-dimensional TTE, right parasternal long-axis view in diastole, without (*left*) and with (*right*) color-flow Doppler, demonstrates a defect (*arrowhead*) allowing turbulent blood to flow from the aortic sinus (Ao) into the RA. *LA*, Left atrium; *LC*, left coronary sinus; *NC*, non-coronary sinus.

a poorer prognosis in dogs.[10,11] Of the 2 reports of dogs with ANIGD caused by IE, neither survived, and, importantly, both dogs had evidence of aortic valve involvement.[10,11] It is well documented in veterinary medicine that aortic IE carries a worse prognosis than IE affecting other valves.[12] In contrast, the patient in this report was diagnosed with suspected TV endocarditis, which was presumed to be a complication of the Gerbode defect and penetrating wound sustained initially, as opposed to the causative agent. This conclusion was made based on the short time frame between the trauma and the shunt diagnosis, as well as the progression of lesions attributed to endocarditis, including the hematogenous spread of bronchopneumonia, while the patient was hospitalized. However, histopathology of the TV was not available to verify the etiology of these changes to the TV. Other differentials such as torn tissue and associated thrombosis could also be considered. The dog of this report also had aortic sinus rupture, but based on the echocardiographic and postmortem examination, this lesion was most consistent with traumatic damage. However, an endocarditic etiology cannot be ruled out. Aortic sinus rupture is rare in veterinary medicine. Spontaneous rupture leading to an aorta–pulmonary artery fistula has been documented in a young Dalmatian.[13]

Without intervention to occlude an LV-RA or aortic sinus–RA shunt, patients may be at risk of progressive cardiac volume overload, IE, and death. Due to the risk of IE, some argue for intervention, even if the patient is asymptomatic with a hemodynamically insignificant defect.[1,2] In reviewing 40 patients who did not undergo correction, 14 (35%) had prophylactic treatment for IE including antibiotic therapy, and 1 of them developed IE despite prophylaxis.[3]

Intervention has traditionally involved a surgical patch repair, which remains the treatment of choice in cases of ANIGD caused by IE, as well as in patients who develop IE as a complication of their Gerbode defect.[1-3] Although percutaneous closure is becoming increasingly pursued for Gerbode defects in human patients, it remains rare in veterinary medicine. A percutaneous closure was attempted in one dog with a traumatic ANIGD but was unsuccessful due to proximity of the device to the aortic valve and likelihood of aortic valve leaflet entrapment when deployed.[8] A similar approach has also been described in a horse with aortocardiac fistula.[14] In our patient, percutaneous occlusion was recommended following

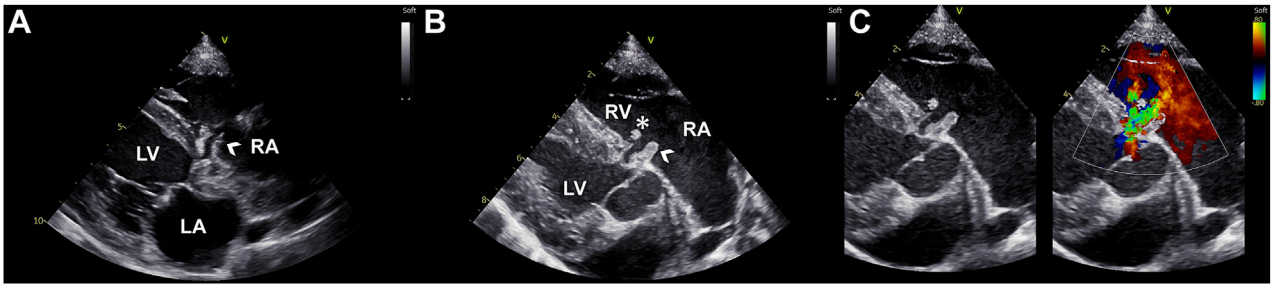

Figure 5 Two-dimensional TTE, right parasternal long-axis view, demonstrates a hyperechoic, mobile, suspected endocarditic lesion (*arrowhead*) near the Gerbode defect and septal tricuspid leaflet (*) on day 3 of hospitalization **(A)**, which became progressively larger on hospital day 5 **(B)**. Color-flow Doppler on hospital day 5 **(C)** revealed subjectively worsened shunting through the Gerbode defect. *LA*, Left atrium.

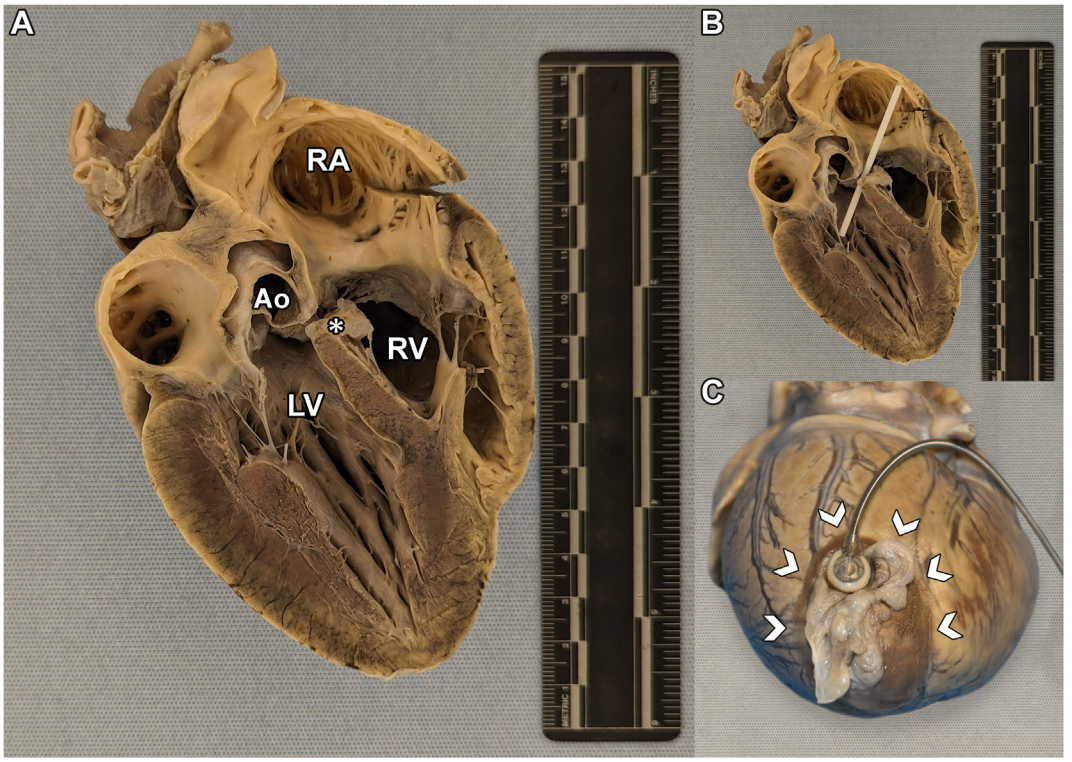

Figure 6 Photograph of the dog's heart transected in a 4-chamber section. The defect connecting the LV to the RA is observed in close proximity to the aortic sinus (Ao). Note that the defect is observed just above the attachment of the septal tricuspid leaflet (*) to the septum. **(B)** A plastic applicator is observed traversing the Gerbode defect. **(C)** The pacemaker lead is observed with the electrode secured to the epicardial surface of the LV, and a wedge-shaped discoloration is observed (*arrowheads*). Residual pericardium is present surrounding the pacemaker lead.

resolution of the patient's IE. Occlusion of the aortic sinus rupture and Gerbode defect was planned, but the patient died suddenly before intervention could be attempted.

This case illustrates an atypical intermediate ANIGD with concurrent aortic sinus rupture caused by blunt force trauma to the thorax. Although echocardiography was not performed earlier in the patient's life, it is considered unlikely that the diagnosed shunts were congenital in origin based on the lack of a cardiac murmur auscultated prior to presentation, the acute onset of third-degree AV block with the associated blunt thoracic trauma, and the progression of right heart dilation and occurrence of right-sided heart failure observed during serial TTEs. A rupture of the aortic sinus, which communicated with the Gerbode defect, was also observed after the blunt thoracic trauma, which is critical for understanding why this patient had continuous, high-velocity shunting of blood into the RA throughout systole and diastole. A Gerbode defect alone should only have systolic LV-RA shunting due to the minimal pressure gradient between these chambers in diastole, whereas an aortic sinus rupture, leading to left-to-right shunting from the aorta to the RA, will have continuous flow driven by the large pressure gradient between these structures during both

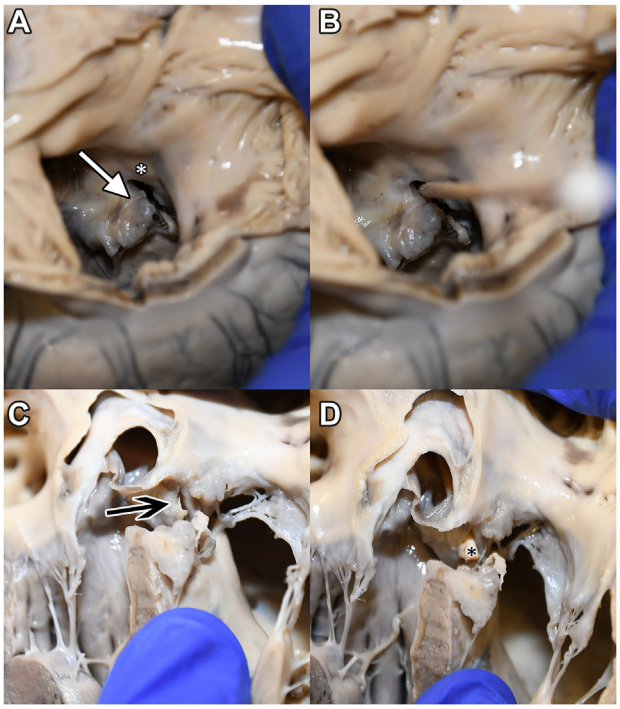

Figure 7 **(A, B)** The dorsal right atrial wall has been incised to reveal a surgeon's view of the TV (*white arrow*). The defect (*white asterisk*) is observed just dorsal to the TV, and a wooden applicator is seen emerging through the defect as it traverses from the LV to the RA. **(C, D)** A defect in the aortic sinus (*black arrow*) is visible, and its connection to the RA is demonstrated by the course of a wooden applicator that originated in the ascending aorta and emerged through the defect (*black asterisk*).

systole and diastole. Damage to the endothelium during traumatic shunt formation possibly predisposed this patient to develop *Staphylococcus pseudointermedius* endocarditis of the TV. Ultimately, despite management of AV block via pacemaker implantation and improvement of IE, this patient experienced sudden cardiac death, attributed to a suspected ventricular tachyarrhythmia.

CONCLUSION

Blunt thoracic trauma may lead to both ANIGD and aortic sinus rupture, and careful evaluation of echocardiographic images is necessary to make these diagnoses. Although uncommon in the dog, documented complications from ANIGD and aortic sinus rupture in the dog of this report included acute development of third-degree AV block as well as IE of the TV. Serial echocardiographic evaluation of patients with Gerbode defects with or without concurrent aortic sinus rupture may be indicated in order to observe whether progressive right heart dilation, right-sided heart failure, endocarditic lesions, or life-threatening arrhythmias develop.

ETHICS STATEMENT

The authors declare that the work described has been carried out in accordance with the ARRIVE guidelines and with the U.K. Animals (Scientific Procedures) Act, 1986 and associated guidelines, EU Directive 2010/63/EU for animal experiments, or the National Research Council's Guide for the Care and Use of Laboratory Animals.

CONSENT STATEMENT

The authors declare that since this was a noninterventional, retrospective, observational study utilizing de-identified data, informed consent was not required from the patient under IRB exemption status.

FUNDING STATEMENT

The authors declare that this report did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

DISCLOSURE STATEMENT

The authors report no conflict of interest.

ACKNOWLEDGMENTS

We thank Silas Zee and Dr. Jey W. Koehler, DVM, PhD, DACVP, for their assistance in figure and video preparation.

SUPPLEMENTARY DATA

Supplementary data related to this article can be found online at https://doi.org/10.1016/j.case.2024.02.001.

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