Case Report

Possible intervertebral disc disease related to ventricular tachycardia in a Labrador Retriever

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Abstract

Ventricular arrhythmia (VA) is a common arrhythmia in dogs. Several extracardiac causes such as pain may contribute this condition. This case report describes a 9-year-old neutered male Labrador Retriever with ventricular tachycardia (VT) potentially related to severe neck pain due to intervertebral disc disease (IVDD). The dog was referred with a sign of inability to walk, tachypnea and an irregular heartbeat. Electrocardiography confirmed VT at 160-200 bpm and an increased cardiac troponin I level (35.22 ng/ml). Despite antiarrhythmic treatment, the VT persisted. Cardiac evaluation revealed no structural abnormalities. Interestingly, VT episodes were provoked by cervical palpation and resolved following analgesic treatment. MRI revealed disc extrusion at C6–C7 with spinal cord compression. Surgical stabilization and pain management led to complete resolution of the arrhythmia. These findings suggest that severe cervical pain from IVDD may be associated with VA in this dog.

Keywords: dogs, intervertebral disc disease, pain, ventricular arrhythmia

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Received: October 6, 2025
Accepted: January 4, 2026
https://doi.org/10.56808/2985-1130.3949

Introduction

Ventricular arrhythmia (VA) is one of the second most common types of arrhythmia in dogs. It can be caused by both cardiogenic and non-cardiogenic factors. VA includes ventricular fibrillation and ventricular tachycardia (VT), which occurs in approximately 2% of dogs overall and in 16% of dogs diagnosed with VA (Williams and Viswanathan, 2013; Noszczyk-Nowak *et al*., 2014). Cardiogenic causes include congenital heart disease and acquire heart disease such as valvular disease, cardiomyopathy and myocarditis. Non-cardiogenic causes include inflammatory disease, trauma, sepsis, electrolyte imbalances, drugs, myocardial hypoxia, neoplasia and pain (Santili *et al*., 2018). For effective management, it is necessary to determine the underlying cause of VA. A complete history and physical examination, along with electrocardiography, echocardiography and potentially measurement of cardiac troponin I levels, should be performed to assess for cardiac involvement.

To appropriately manage VA, it is essential to evaluate both the severity of the arrhythmia and any associated hemodynamic alterations. Identifying the primary cause is also crucial for successful treatment. Severe pain and the resulting surge in catecholamine release can increase the likelihood of VA. Dogs experiencing significant pain from several conditions including trauma, splenectomy, pancreatitis, gastric dilatation-valvulus may develop VA (Hayashi *et al*., 1997; Mackenzie *et al*., 2010; Pastarapatee *et al*., 2017; Santilini *et al*., 2018). However, to our knowledge, there are no reports VA associated with orthopedic conditions such as intervertebral disc disease (IVDD). Therefore, this case report aims to describe a dog presenting with VA possibly related to IVDD.

Case Description

A 9-year-old neutered male Labrador Retriever weighing 34 kg was presented to the Emergency Unit of the Small Animal Teaching Hospital, Faculty of Veterinary Science, Chulalongkorn University, Thailand for further investigation. The dog was referred from a private clinic with a history of hyperthermia (103.10 °F), tachypnea when restrained, hindlimb for 2 years, and inability to walk since the previous week. A cardiovascular problem was suspected by the referring veterinarian; therefore, furosemide was administered at a dosage of 2 mg/kg IV every hour, followed by twice- daily dosing. However, the dog's condition did not improve, and it was referred for further investigation.

During the initial visit, the dog appeared depressed. Physical examination revealed difficulty breathing, normal heart sounds, a heart rate of 160 beats per minute with an irregular rhythm, and pulse deficits. Focused ultrasonography showed no evidence of pleural effusion or ascites. Thoracic radiographs revealed a normal cardiac shape and size, with no evidence of left atrial enlargement or pulmonary vascular enlargement. Bronchointerstitial lung infiltrates, suspected to be due to age-related, were observed. Multilevel spondylosis with disc space collapse at C6 and C7 was also identified (Fig. 1). Electrocardiography revealed VT with a heart rate of 160 beats per minute (Fig. 2).

The serum cardiac troponin I level was 35.22 ng/ml. A 2% lidocaine HCl bolus was administered intravenously at a dose of 2 mg/kg, followed by a three additional bolus doses of 1 mg/kg. Subsequently, a continuous rate infusion (CRI) of lidocaine was initiated at a rate 30 mcg/kg/min for 17 hours. Following this, the VT transitioned to ventricular premature complex beats (VPCs) (Fig. 3).

On the second day of the visit, an echocardiography was performed, revealing no significant structural abnormalities in the heart. Both the left atrium and left ventricle were of normal size and left ventricular systolic and diastolic functions were within normal ranges. However, during the echocardiographic examination, the dog showed clinical signs of neurologic deficits and severe neck pain. To address these symptoms, tramadol was administered subcutaneously at a dose of 3.9 mg/kg, with a 12-hour dosing interval. Notably, no VPC were observed 10 hours after the initial tramadol administration. On the third day, a comprehensive neurological examination was conducted. Simultaneously, electrocardiography was used to assess the cause of the previously observed VPC. Remarkably, VPC were triggered instantaneously by neck examination and palpation (Fig. 4). In addition, abdominal ultrasonography was performed, revealing no abnormalities in the abdominal organs.

Based on the clinical findings and treatment trials, the most probable diagnosis for the induced VT was severe neck pain. Differential diagnoses, including heart disease as a trigger for VT and other extracardiac causes such as splenic or hepatic masses, were deemed less likely due to absence of lesions in the heart and abdominal organs assessed by echocardiography and abdominal ultrasonography.

The tentative diagnosis was cervical spondylomyelopathy, prompting the scheduling of magnetic resonance imaging (MRI) for further investigation. The medication regimen was adjusted to include gabapentin at a dose of 8.8 mg/kg every 8 hours, methocarbamol at 44 mg/kg every 8 hours, a combination of vitamin B1 (100 mg), B6 (200 mg) and B12 (200 mcg) (Neurobion®) at 1 tablet twice daily, and omega-3 fatty acid (Antinol®) at 2 capsules twice daily. Additionally, subcutaneous administration of meloxicam at a dose of 0.1 mg/kg once daily and morphine at 0.3 mg/kg every 8 hours was initiated. The MRI results indicated (i) intervertebral disc extrusion at the C6-C7 level, resulting in spinal cord compression and contusion, and (ii) hemorrhage, and intervertebral disc degeneration spanning from C5-T1; and (iii) intervertebral disc extrusion C5-C6 (Fig. 5). Subsequently, a procedure involving the distraction of the C6-C7 vertebrae and fusion with a locking plate was carried out.

The VA was completely resolved throughout the entire course of treatment.

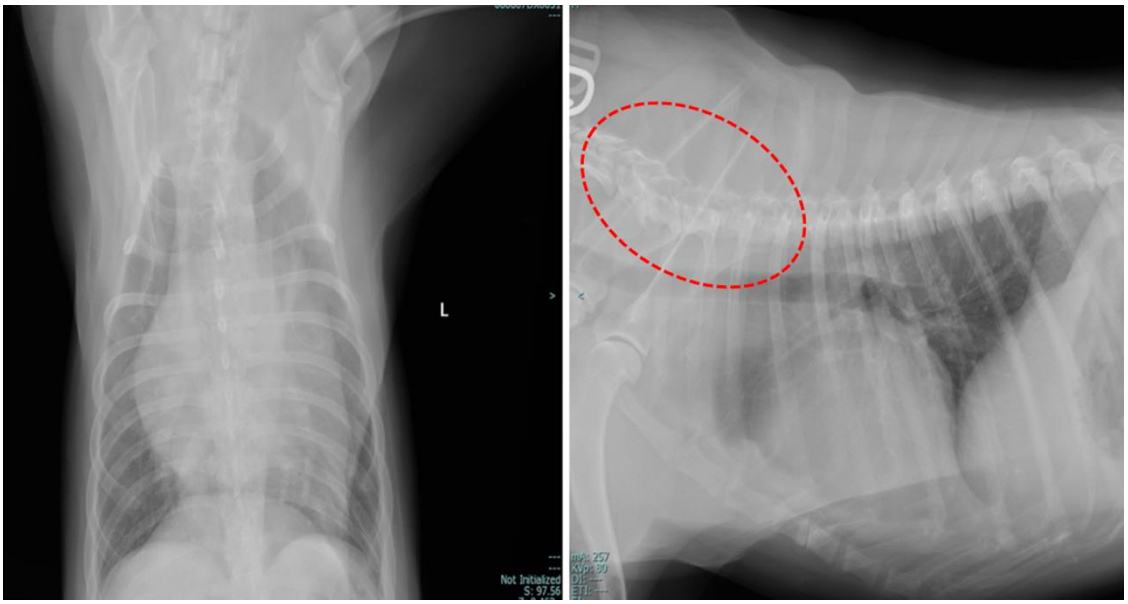

Figure 1 Thoracic radiographs in ventrodosal (left) and right lateral (right) views show an enlarged cardiac silhouette without evidence of left atrial enlargement, small pulmonary vessels, bronchointerstitial lung infiltrates, and multilevel spondylosis with collapse of the C6-C7 intervertebral disc space (oval).

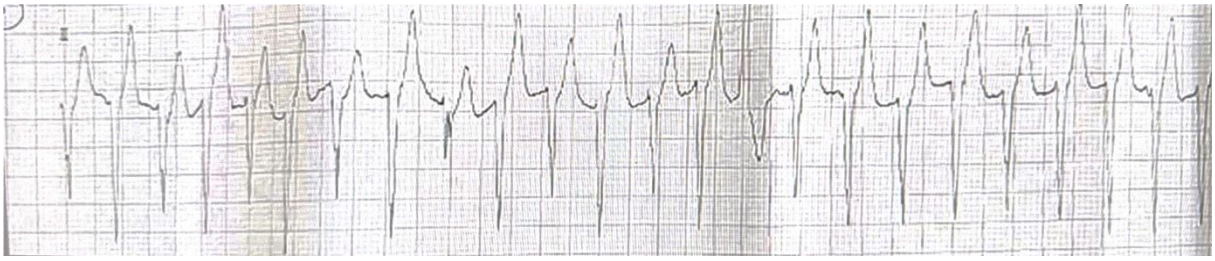

Figure 2 Electrocardiography on the first day of the dog's presentation reveals sustained ventricular tachycardia with a heart rate of 160 beats/minute (paper speed: 25 mm/sec).

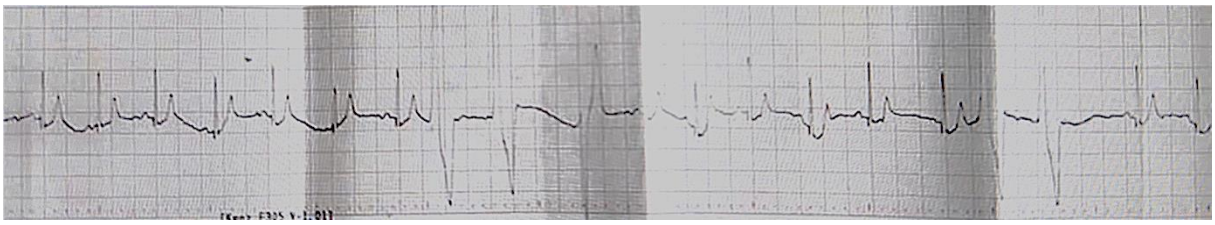

Figure 3 Electrocardiography of the dog after a 17-hour constant-rate infusion of lidocaine (30 mcg/kg/min) shows sinus rhythm with occasional ventricular premature complex (VPCs) and a heart rate of 140 beats/minute (paper speed: 25 mm/sec).

Figure 4 Echocardiography during the neck examination reveals multiple ventricular premature complexes, with a heart rate of 140 beats/minute during the ECG recording over 10 seconds. (paper speed: 25 mm/sec).

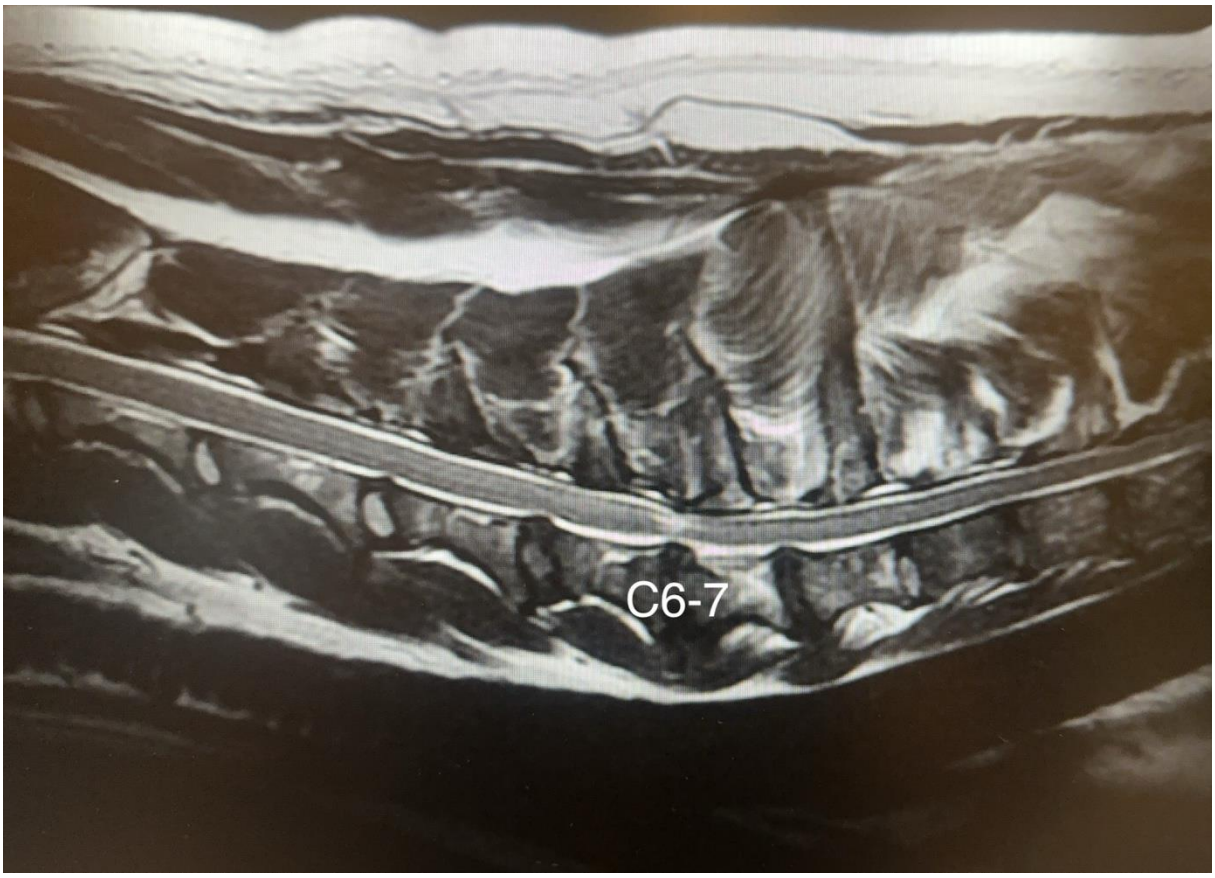

Figure 5 Longitudinal magnetic resonance imaging reveals intervertebral disc extrusion at the C6-C7 level.

Discussion

Ventricular tachycardia constitutes a subset of the second most common arrhythmia observed in dogs (Noszczyk-Nowak *et al*., 2017). In humans, VT can cause sudden cardiac death and is a significant contributor to cardiac mortality. Dogs with VT may have either an underlying structural cardiac abnormality or extracardiac abnormalities (Santilini *et al*., 2018). Several studies in humans have suggested an association between stimulation of the sympathetic nervous system (SNS) and the pathogenesis of VT triggered by extracardiac abnormalities (Hayashi *et al*., 1997; Wang *et al*., 2017). Activation of the SNS can be influenced by various factors, with pain being a significant contributor. Initially, SNS activation can help alleviate pain by inhibiting nociceptive transmission in the spinal cord. However, in cases of chronic pain, sympathetic disturbances may occur, leading to amplification and prolongation of the pain response (Schlereth *et al*., 2008). Here, we describe a case of VT related to severe pain due to intervertebral disc disease.

Lidocaine is the drug of choice for treating VA, as it acts on inactivated sodium channels, reducing action potential duration and increasing the refractory period. This mechanism raises the ventricular fibrillatory threshold and disrupts the re-entry mechanism (Güler *et al*., 2023). Additionally, lidocaine has analgesic properties in human patients with neuropathic pain (McCleane, 2007; Dunn and Durieux, 2017; Estebe, 2017). In dogs, lidocaine administration has been shown to be beneficial in both intraoperative and postoperative pain management (Ortega and Cruz, 2011; Alves *et al*., 2014). Taken together, the dual effects of lidocaine may help diminish VT induced by pain. In this dog, VT was partially suppressed by a low-dose constant rate infusion (CRI) of lidocaine; however, it completely disappeared after tramadol was administered for pain control. Tramadol, a synthetic analogue of codeine, is commonly used in veterinary practice to manage pain. Many studies have demonstrated that tramadol has effective analgesic properties, with some suggesting its equivalence to morphine in human studies (Mastrocinque and Fantoni, 2003; Vettorato *et al*., 2010; Trettene *et al*., 2023). However, the effectiveness of tramadol can vary among individuals due to differences in liver function and genetic factors (Domínguez-Oliva *et al*., 2021). Based on the treatment outcomes observed in this case, tramadol was effective in reducing pain, potentially leading to a decrease in sympathetic stimulation that may have triggered the VPC.

Although cardiac troponin I was markedly increased, the dog's echocardiographic findings were structurally within normal limits. Myocardial injury may occur acutely, during which structural abnormalities are not yet detectable on echocardiography. In addition, cardiac function could not be accurately assessed due to the presence of arrhythmias. The elevated cardiac troponin I level may have been associated with arrhythmia-induced myocardial injury (Ramito *et al*., 2024); however, the degree of injury may not have been sufficient to produce detectable echocardiographic changes.

Myocardial injury can induce arrhythmias, and arrhythmias can conversely lead to myocardial injury. In this case, no definitive cause of myocardial injury

other than the arrhythmia was identified. Therefore, it is more likely that the arrhythmia precipitated the myocardial injury rather than myocardial injury being the primary cause of the arrhythmia.

The ventricular tachycardia (VT) observed in this dog may have been triggered by sympathetic overdrive secondary to severe neck pain, which was the only notable clinical finding. This is supported by two observations: 1) Complete resolution of arrhythmia following analgesic administration. 2) Recurrence of ventricular premature complexes (VPCs) upon palpation of the neck.

In conclusion, the dog likely experienced VT of extracardiac origin, triggered by neck pain associated with intervertebral disc disease at the C6 and C7 level.

Acknowledgements

We would like to express our sincere gratitude to the Small Animal Teaching Hospital, Chulalongkorn University, Thailand, for their valuable support in providing the data used in this case report.

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