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# An Intriguing Case of Ventricular Arrhythmia in A Dog with Intervertebral Disk Disease

*Case Report*

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## Case Report

A four-year-old, 10.8 kg, neutered female mongrel dog with signs of intervertebral disk disease (IVDD) and history of recurrent gastritis, was presented at a veterinary referral hospital. The owner reported that the dog had had a tense abdomen over the past two days, which led him to take it to a private clinic where bloodwork and ultrasound scans were carried out. The only abnormality on the CBC was a slight erythrocytosis. Similarly, there were no alterations in total protein and fractions, ALT, ALP, BUN and creatinine measured in plasma biochemistry. Abdominal ultrasonography was compatible with gastritis and enteritis although the owner had noted neither vomiting nor diarrhea in his pet. After one day of hospitalization to receive a treatment for gastritis, the patient was discharged. A few hours later an abnormal gait developed. The day after, the dog became non-ambulatory, which forced the owner to return to the clinic; where a thoracolumbar radiograph revealed signs of intervertebral disk disease (IVDD).

On physical examination, the dog exhibited complete paralysis of the hind limbs. Pulmonary auscultation was unremarkable. Cardiac auscultation revealed a heart rate of 140 bpm, femoral pulse was strong, mucous membranes were pink, capillary refill time was two seconds, rectal temperature 38.6°C (101.5°F), hydration status was normal, and body condition was overweight. A contrast CT scan of T12-L5 vertebrae confirmed a lesion between T12-T13. Therefore, a thoracolumbar hemilaminectomy was scheduled for the next day. Of note, the patient produced bland and orange-colored feces while still sedated shortly after CT was completed.

Before surgery, lactated's ringer fluid therapy at a rate of 50mL/kg/24h (22.7mL/lb/24h) was administered. Premedication was dexmedetomidine (2µg/kg [0.91µg/lb] IV) and methadone (0.2mg/kg [0.09 mg/lb] IV), followed by induction with propofol (1mg/kg [0.05mg/lb]), and anesthesia maintained with continuous rate infusion of remifentanil (10µg/kg/h [4.54µg/lb/h]), dexmedetomidine (1µg/kg/h [0.45µg/lb/h]), propofol (12mg/kg/h [5.44mg/lb/h]) and ketamine (0.6mg/kg/h [0.27mg/lb/h]). In the last third of surgery, isolated ventricular premature complexes together with a sinus rhythm of 87bpm developed, and systolic arterial blood pressure was 118mmHg. A continuous rate infusion of lidocaine was initiated (1mg/kg/h [0.05mg/lb/h]). After completion of surgery, the continuous rate infusion of lidocaine was reduced to half of the initial dose (0.5mg/kg/h [0.025mg/lb/h]) and methadone was continued for pain relief at 0.2mg/kg [0.09mg/lb] IV every six hours, plus a continuous rate infusion of ketamine (0.6mg/kg/h [0.27mg/lb/h]).

Six hours after surgery, the patient had the first episode of ventricular tachycardia (VT), so a bolus of lidocaine (2mg/kg IV [0.9mg/lb]) was given intravenously. After this, ventricular premature complexes reduced to ten per minute. Another episode of VT was documented five hours later, which required another bolus of lidocaine, followed by CRI lidocaine 1mg/kg/h (0.05mg/lb/h). VT recurred within 15 minutes and the bolus of lidocaine was repeated. During one of the episodes of VT, the heart rate increased up to 166 bpm, but systolic blood pressure was still satisfactory at 140mmHg.

## ECG Interpretation

The following morning, a standard 10-lead electrocardiogram was recorded for 5 minutes, which documented sinus rhythm (90bpm) with isolated ectopic polymorphic ventricular complexes. The ventricular complexes had right bundle branch block morphology, indicating left ventricular origin, and there were paroxysmal episodes of ventricular tachycardia (240bpm) lasting more than seven seconds. The coupling interval (283-300ms) of the ventricular tachycardia was regular. The mean electrical axis and all electrocardiographic measurements of the sinus complexes were within the normal reference ranges for dogs.

To rule out supraventricular tachycardia with aberrant conduction, a manual ocular vagal stimulus was performed. The absence of response confirmed the ventricular origin of the tachyarrhythmia. An echocardiogram was also performed, which was unremarkable. The chron-

ic antecedents of diarrhea and vomiting with the identification of ventricular tachycardia, raised suspicion of pancreatitis and abdominal ultrasonography showed fat tissue accumulation within the pancreas, suggesting chronic pancreatitis. A cPLi test was positive.

Based on this diagnosis, a transversus abdominis plane block with bupivacaine (0.3mL/kg/side [0.14ml/lb/side]) was added to the treatment, after which there were no more episodes of ventricular tachycardia. At the end of the next day, CRI lidocaine was discontinued, and no more ventricular premature complexes were recorded. Four days after, the patient was finally discharged. The dog was re-examined two weeks later, including a 3-minute ECG. Sinus rhythm (140bpm) was documented (Figure 1-3).

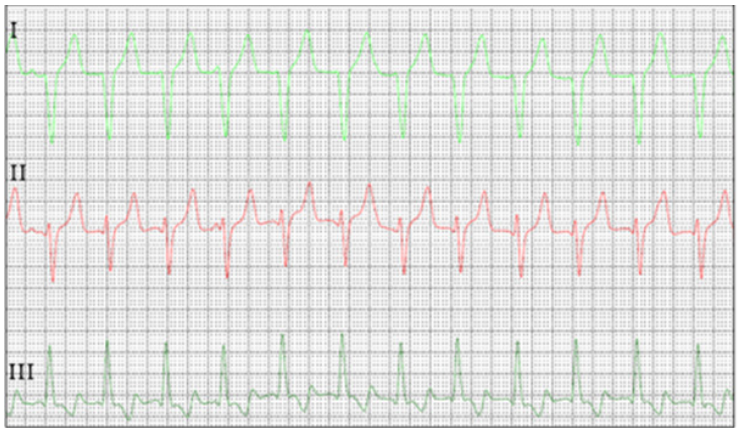


**Figure 1:** Segment of a 10-lead electrocardiogram showing a paroxysmal episode of monomorphic ventricular tachycardia (200bpm) in a mongrel dog with IVDD and pancreatitis. The morphology of the VT in lead II is compatible with left ventricular origin. Paper speed: 50mm/s; 1 cm = 1mV.

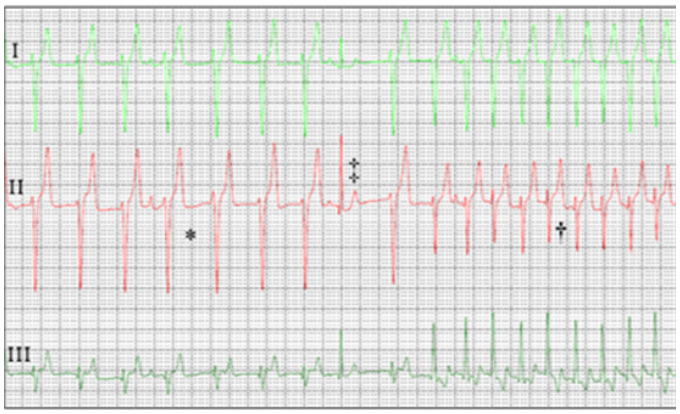


**Figure 2:** Segment of a 10-lead electrocardiogram illustrating two different morphologies (* and †) of ventricular tachycardia alternating with a single sinus complex (‡). Mean heart rate 160bpm. Paper speed: 25mm/s; 1cm = 1mV.

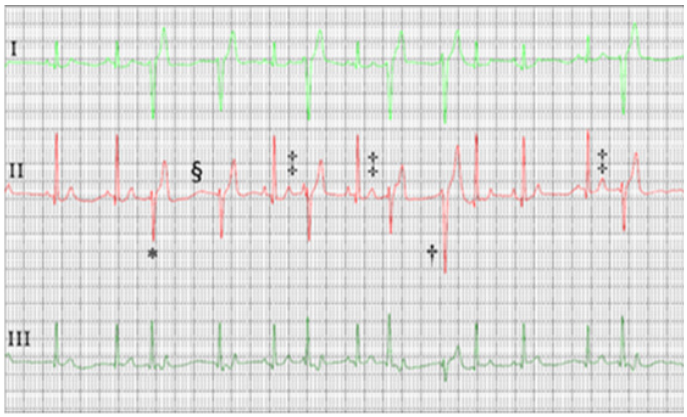


**Figure 3:** Segment of a 10-lead electrocardiogram in which sinus complexes are alternated with polymorphic ectopic premature beats (* and †). Three episodes of bigeminy (‡) and a couplet (§) are also recorded. Paper speed: 25mm/s; 1cm = 1mV.

## Discussion

Ventricular tachycardia is the sequence of more than three ectopic ventricular complexes at a rate above that determined by the auxiliary intrinsic ventricular pacemaker cells [1]. One or more ectopic foci within the ventricular myocardium may be the source of these premature depolarizations. Ectopic ventricular beats that possess similar morphology are called monomorphic and usually originate from the same ectopic focus. Conversely, ectopic ventricular beats of different morphologies most likely come from distinct ventricular foci and are named polymorphic

Typically, ectopic ventricular complexes have a prolonged duration (>70ms in the dog) as the electrical impulse is conducted at a slower speed by cardiomyocytes [2]. In addition to response to vagal maneuvers, the coupling interval between wide QRS complexes and the compensatory pause at the end of the tachycardia help to differentiate ventricular tachycardia from aberrant conduction. While VT has a constant coupling interval and a compensatory pause after the last wide QRS complex, aberrant conductions show the opposite [3]. The etiologies of VT are highly varied, ranging from structural heart disease to anemia [4].

Pancreatitis results in enzymatic autodigestion of the pancreas, leading to inflammation and, in severe cases, to multi-organic failure. There are a wide array of nonspecific signs associated with chronic pancreatitis, including inappetence, prostration, and behavioral changes [5]. In both human and veterinary medicine, pancreatitis is a well-known trigger for cardiac arrhythmias. In medicine, it is an important differential for myocardial infarction, as it mimics the classical S-T segment elevation in the absence of coronary obstruction [6-8]. In dogs, pancreatitis can cause supraventricular and ventricular arrhythmias [9].

In people, spinal cord injury has been associated with pancreatitis [10-12]. Animals with IVDD commonly present with signs of gastrointestinal disturbance (inappetence, diarrhea, emesis, abdominal pain). A recent study found significant association between elevated serum cPLI concentrations (>200µg/L) in 38/84 (45%) of dogs with confirmed IVDD [13]. One explanation for this finding is an autonomic input imbalance secondary to selective compression of spinal cord segments resulting in overstimulation of the sphincter of Oddi. Pancreatic exogenous secretion accumulates, permitting absorption of pancreatic enzymes into the blood stream and parenchymal autodigestion [10].

Similarly, pancreatic perfusion would be affected, leading to vasoconstriction. The resulting hypoxia would trigger cellular alterations that predispose the animal to pancreatitis [13]. Pancreatitis may cause arrhythmias through three mechanisms, which are the release of myocardial depressant factor, electrolytic disturbances and presence of pancreatic enzymes in the blood [9]. Myocardial depressant factor (MDF) is a peptide synthesized by pancreatic lysosomes in response to the ischemic states caused by hypoperfusion. This molecule has a marked negative chronotropic effect that reduces cardiac output, and may ultimately lead to cardiac arrest. Also, MDF causes vasoconstriction of splanchnic vasculature and decreases reticuloendothelial activity. These two mechanisms together worsen and perpetuate MDF action since further vasoconstriction exacerbates pancreatic ischemia, and reticuloendothelial cells decrease the clearance of MDF [14-16].

Since this patient was initially presented for IVDD, and pancreatitis was discovered later, electrolytes were not measured. However, electrolyte imbalances including hypocalcemia, along with decreased potassium, sodium, and chloride levels [5] may occur in dogs with pancreatitis. Hypocalcemia is known to increase QT interval by lengthening phase 2 of the cardiomyocyte action potential. A prolonged QT interval is a recognized predictive value for ventricular tachycardias, including *torsades de pointes*. Likewise, hypokalemia hyperpolarizes the resting membrane potential, which can also prolong QT interval and enhance Purkinje fiber automaticity. Both effects create a significant predisposition to ventricular tachycardia, especially in patients with preexisting structural heart disease [17].

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