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Anesthesia/Analgesia Case Assessment

In collaboration with the American College of Veterinary Anesthesia and Analgesia (ACVAA)

Ventricular arrhythmias during anesthesia in a juvenile German Shepherd Dog

Keywords: German Shepherd, VPC, anesthesia, ventricular, arrhythmia

History

A 10-month-old 27-kg intact female German Shepherd Dog (GSD) presented to the North Carolina State University College of Veterinary Medicine for abdominal CT evaluation of a suspected intrahepatic portosystemic shunt.

Recent serum biochemistry analysis revealed the following relevant abnormalities: hypoglycemia (66 mg/dL; reference range, 75 to 126 mg/dL), decreased BUN (5 mg/dL; reference range, 11 to 27 mg/dL), decreased creatinine (0.4 mg/dL; reference range, 0.5 to 1.4 mg/dL), hypoproteinemia (4.0 g/dL; reference range, 5.3 to 7.2 g/dL), hypoalbuminemia (2.4 g/dL; reference range, 3.2 to 4.3 g/dL); hypocholesterolemia (71 mg/dL; reference range, 151 to 348 mg/dL), increased ALP (153 IU/L; reference range, 9 to 88 IU/L); increased serum ALT (89 IU/L; reference range, 17 to 78 IU/L), and increased serum AST (80 IU/L; reference range, 16 to 42 IU/L).

Physical examination on the day of anesthesia revealed no abnormalities and no overt neurologic deficits or behavioral abnormalities. The patient was appropriately fasted and received trazodone (5.5 mg/kg, PO) 90 minutes prior to anesthesia for anxiolysis. Premedication included butorphanol (0.2 mg/kg, IV) and dexmedetomidine (0.002 mg/kg, IV). During preoxygenation, 3-lead ECG and noninvasive oscillometric blood pressure monitoring were instituted. Ventricular bigeminy was noted, with a right bundle branch block morphology and a ventricular rate of 80 beats/min **(Figure 1)**. Noninvasive blood pressure was also normal, with an average systolic arterial pressure of 135 mm Hg, an average diastolic arterial pressure of 78 mm Hg, and an average mean arterial pressure of 90 mm Hg. No pulse deficits were noted, and the arrhythmia did not appear to be affecting perfusion as evidenced by the patient's normal mentation, capillary refill time, and adequate pulse quality. Induction of anesthesia was briefly delayed while the supervising anesthesiologist discussed the potential consequences of the arrhythmia with the surgical

Tyne Hovda, DVM*; Lynelle Graham, DVM, MS, DACVAA; Lydia Love, DVM, DACVAA

Department of Molecular Biomedical Sciences, College of Veterinary Medicine, North Carolina State University, Raleigh, NC

*Corresponding author: Dr. Hovda (thovda@ncsu.edu)

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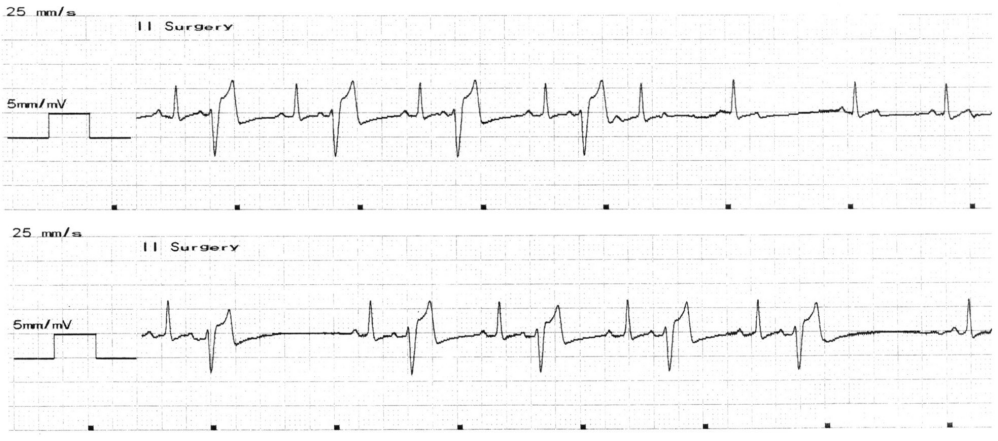

Figure 1—Pre-induction ECG from a 10-month-old intact female German Shepherd Dog presenting for advanced imaging for surgical planning of a presumed portosystemic shunt after administration of butorphanol (0.2 mg/kg, IV) and dexmedetomidine (0.002 mg/kg, IV).

service; the decision was made to continue and obtain the desired diagnostics.

Anesthesia was induced with propofol (2 mg/kg, IV), a 12mm cuffed silicone endotracheal tube was placed, and general anesthesia was maintained with 1% to 1.5% isoflurane in 100% oxygen using a rebreathing circuit. Capnography and pulse oximetry monitoring were initiated, and the patient was kept in sternal recumbency throughout the procedure. Intermittent ventricular bigeminy continued but no cardiovascular or respiratory parameters were different from preinduction values at this time. Atipamezole (0.02 mg/kg, IM) was administered after induction to avoid potential excitability during intubation and to reverse the cardiovascular effects of the α-2 agonist.

The patient was transported to CT, where monitoring and anesthesia circuitry were briefly disconnected to allow for positioning in the gantry. All monitoring was reestablished, and mechanical ventilation was initiated with a peak inspiratory pressure of 12 cm H_2O. Several couplets of R-on-T complexes were noted that quickly progressed into short runs of ventricular tachycardia **(Figure 2)**. A lidocaine bolus (2 mg/kg, IV) was administered, and a normal sinus rhythm with a heart rate of 70 bpm was readily obtained. A lidocaine infusion was started (3 mg/kg/h, IV) and a normal sinus rhythm predominated throughout the rest of the anesthetic procedure; however, several brief episodes of bigeminy and isolated monomorphic ventricular premature complexes (VPCs) were noted intermittently. Lactated ringers solution was administered at a rate of 5 mL/kg/h throughout the anesthetic event, lasting 65 minutes.

Following cessation of anesthesia, lidocaine was discontinued and naloxone (0.025 mg/kg, IV) was administered to-effect, to facilitate extubation and discharge of the patient from the hospital. Recovery was uneventful, and the patient remained in a primarily normal sinus rhythm with several couplets of VPCs noted, and a heart rate ranging between 60 and 80 bpm. Post-recovery, the patient was referred to the cardiology service for further work up.

What is your diagnosis and intervention plan?

Case Management and Outcome

Ventricular arrhythmias may occur secondary to cardiac disease or a variety of systemic issues including electrolyte derangements, hypoxemia, and intraabdominal disease. In addition, some sedatives and anesthetic agents can be proarrhythmic. Of the drugs used for premedication, the α-2 adrenergic receptor agonist dexmedetomidine is arrhythmogenic due to the development of sinus bradycardia and occasional second-degree atrioventricular block. Dexmedetomidine can reduce the arrhythmogenic dose of epinephrine in dogs, in which the arrhythmogenic dose of epinephrine mimics periods of increased sympathetic tone.[1] Prolongation of the QT interval can be pharmacologically induced in dogs with opioids and sevoflurane, but the clinical relevance is uncertain.[2] To the authors' knowledge, the impact of trazodone on cardiac conduction in dogs has not been published. Propofol is substantially less arrhythmogenic than other induction agents (eg, thiobarbiturates), but has been shown to prolong canine QT intervals.[3]

Diagnosis of a portosystemic shunt was confirmed on CT in this patient. There are reports in human literature regarding the concurrent diagnosis of cardiovascular disorders in patients with portosystemic shunts. While this is not specifically reported in the veterinary literature, the influence of abnormal abdominal pathology on cardiac rhythm has been well documented, particularly in emergency settings.

In this case, a tentative diagnosis of Inherited Ventricular Arrhythmia (IVA) in the juvenile GSD was made, based on the patient's age, breed, and type of arrhythmia.

Comments

Inherited Ventricular Arrhythmia is an uncommon primary electrical disorder first described in 1994, the key features of which are ventricular arrhythmias and sudden death in juvenile GSDs.[4] IVA is thought to be either a simple autosomal dominant trait with incomplete penetrance or a polygenic

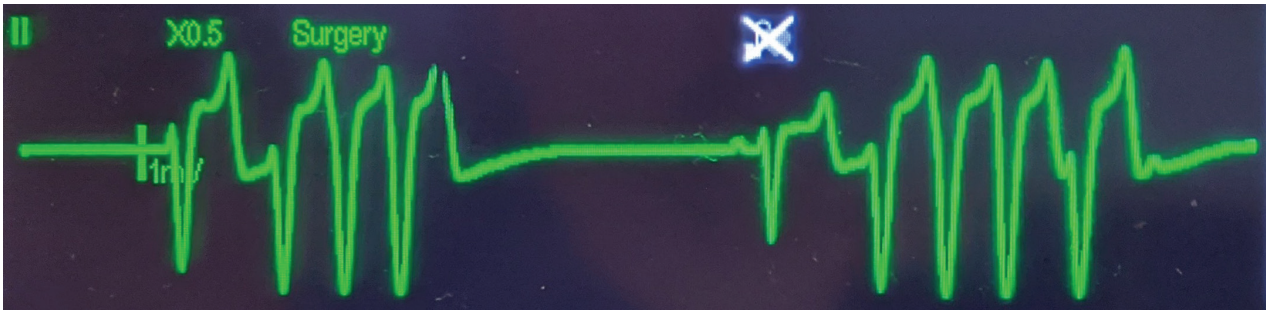

Figure 2—ECG of an anesthetized 10-month-old intact female German Shepherd Dog presenting for advanced imaging for surgical planning of a presumed portosystemic shunt prior to treatment with lidocaine (2 mg/kg, IV). The patient had received butorphanol (0.2 mg/kg, IV) and dexmedetomidine (0.002 mg/kg, IV) for premedication, and propofol (2 mg/kg, IV) for induction. Atipamezole (0.02 mg/kg, IM) was also given approximately 10 minutes prior to collection of this ECG.

trait although its exact mode of inheritance is still unknown.[4]

While rare in the overall population, the incidence of sudden death in a litter of affected puppies can be 15% to 20%.[5] Puppies typically first develop spontaneous ventricular arrhythmias around 3 to 4 months with worsening severity seen between 5 and 7 months. Peaks in the incidence of tachycardia have been noted as late as 18-20 months, although the incidence of arrhythmias usually decreases by this age.[5] These dogs are usually asymptomatic with a distinctive lack of clinical signs (no exercise intolerance, lethargy, or syncope). IVA is exacerbated by bradycardia, often secondary to high vagal tone or drug administration, which may confound diagnosis on a routine puppy examination or in a new environment.

Due to the association with increased vagal tone and the propensity for arrhythmias to develop around 6 to 18 months of age, IVA often first becomes apparent during the initial anesthetic event for castration or ovariohysterectomy.[5] Dogs that survive to approximately 2 years of age experience either stabilization of arrhythmias or spontaneous regression and the risk of sudden death diminishes.[5] Interestingly, death often occurs during times when parasympathetic vagal tone predominates (eg, rest, sleep, sedation, and anesthesia), leading to an overall lowered heart rate.

The arrhythmic mechanism is thought to be related to inappropriate sympathetic innervation to the left ventricle and delayed repolarization of the left ventricle secondary to abnormalities in transmembrane potassium and calcium currents.[5] No structural abnormalities or identifiable lesions are identified on necropsy of affected puppies, and echocardiography is often unremarkable.[4] Holter monitoring is considered the gold standard for diagnosis of IVA, but is not traditionally recommended as routine screening due to an overall low prevalence of this age-dependent disease.[4] A diagnosis is made when there are an average of 10 VPCs per 60-minute period during a 24-hour Holter, or if an episode of pause-dependent ventricular tachycardia is identified at any time (typically lasting for at least several seconds). Currently, there is no widely available genetic screening test for IVA.

In an acute setting, lidocaine (2 mg/kg, IV) shortens the action potential duration and is effective at terminating the acute arrhythmia.[5] Prevention of overall bradycardia, through the appropriate use of anticholinergics, has been suggested for short term management during anesthetic episodes.[5]

Long term therapy consisting of sotalol and mexiletine during the juvenile period is controversial as both drugs independently do not reduce the number of runs of ventricular tachycardia, although a synergistic effect may occur if used concurrently.[5] Even with appropriate medical management, there is no literature supporting a reduction in the risk of sudden death with this protocol. It is unknown if the benefits of medical intervention would persist in the anesthetized dog.

Ultimately, the diagnosis of IVA in this juvenile GSD is presumptive and based on breed, type of arrhythmia, patient age, lack of pre-existing clinical signs, and occurrence of the arrhythmia during times of increased vagal tone. Anesthetists should be aware of the existence of IVA, since affected dogs are usually asymptomatic when awake but can experience profound arrhythmias during anesthesia when parasympathetic tone is increased.

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References

1. Hayashi Y, Sumikawa K, Maze M, et al. Dexmedetomidine prevents epinephrine-induced arrhythmias through stimulation of central alpha 2 adrenoceptors in halothane-anesthetized dogs. *Anesthesiology*. 1991;75(1):113–117. doi:10.1097/00000542-199107000-00018
2. Keating S, Fries R, Kling K, Graham L, Clark-Price S, Schaeffer DJ. Effects of methadone or hydromorphone on cardiac conductivity in dogs before and during sevoflurane anesthesia. *Front Vet Sci*. 2020;7:573706. doi:10.3389/fvets.2020.573706
3. Dennis SG, Wotton PR, Boswood A, Flaherty D. Comparison of the effects of thiopentone and propofol on the electrocardiogram of dogs. *Vet Rec*. 2007;160(20):681–686. doi:10.1136/vr.160.20.681
4. Moise NS, Meyers-Wallen V, Flahive WJ, et al. Inherited ventricular arrhythmias and sudden death in German Shepherd Dogs. *J Am Coll Cardiol*. 1994;24(1):233–243. doi:10.1016/0735-1097(94)90568-1
5. Ribas T, Pariaut R. Inherited ventricular arrhythmias in German Shepherd Dogs. In: Willis R, Oliveira P, Mavropoulou A, eds. *Guide to Canine and Feline Electrocardiography*. Blackwell Publishing; 2018:315–318. doi:10.1002/9781119254355.ch22