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CASE REPORT

Resolution of sustained ventricular tachycardia in a horse presenting with colic with magnesium sulfate

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Summary

Case reports demonstrating a return to sinus rhythm from sustained ventricular tachycardia (VT) are limited. VT is uncommon in horses but can be life threatening and has been reported in horses with primary gastrointestinal disease. Treatment is recommended if there is poor perfusion, if heart rate exceeds 100 beats/min, if multiform/polymorphic complexes or torsades des pointes is present. Lidocaine or magnesium sulfate is the first-line medication. In this case, a 19-year-old Warmblood gelding with a history of exploratory laparotomy presented with an irregularly irregular cardiac rhythm and heart rate of 80 beats/min. ECG demonstrated VT with a heart rate of 75 beats/min. As the horse was already receiving a lidocaine bolus, the VT was treated with multiple boluses of intravenous magnesium sulfate over a period of several hours. This converted the VT to normal sinus rhythm (NSR) with heart rate of 44 beats/min and the horse remained in NSR until discharge 8 days later.

KEYWORDS
horse, arrhythmia, colic, magnesium sulfate, ventricular tachycardia

INTRODUCTION

There are few published reports demonstrating a return to normal sinus rhythm (NSR) from sustained ventricular tachycardia (VT) in horses (De Clercq et al., 2007; Kovács et al., 2019; Reimer et al., 1992; Stern et al., 2012). VT is a rapid dysrhythmia originating within the ventricles resulting in wide and bizarre QRS complexes (House & Giguère, 2009). VT is defined as more than three ventricular complexes in sequence (de Solis, 2020; van Loon, 2019), while fewer are isolated ventricular premature complexes (VPCs) or couplets of VPCs. It is accepted that VT is uncommon in horses but can be life threatening when it reduces cardiac output or acts as a substrate for ventricular fibrillation (de Solis, 2020). It is reported in horses with primary gastrointestinal (GI) disease, primary myocardial disease, electrolyte imbalance, acidosis, sepsis or systemic inflammatory response syndrome (SIRS), anaesthetic drug use and hypoxia (Coudry et al., 2007; de Solis, 2020; Mitchell, 2017; Morgan et al., 2011; van Loon, 2019).

Treatment requires identification and resolution of the underlying cause in addition to antiarrhythmic drugs (van Loon, 2019). Some antiarrhythmic drugs have proarrhythmic effects and therefore VT is only treated in cases with clinical signs or where rapid VT is detected (van Loon, 2019). Treatment is recommended if there is evidence of poor perfusion, HR is greater than 100 beats/min, there are multiform/polymorphic complexes or *torsades des pointes* is present (de Solis, 2020; House & Giguère, 2009). When treating, lidocaine and magnesium sulfate are first-line medications (Coudry et al., 2007). Other drugs such as quinidine, amiodarone, procainamide and phenytoin have been used for refractory cases (De Clercq et al., 2007; Garber et al., 1992; Wijnberg & Ververs, 2004) however, all have potential adverse effects. Quinidine has proarrhythmic effects and can cause colic and diarrhoea. Procainamide can be proarrhythmic

at higher doses and causes hypotension. Amiodarone affects multiple organ systems although no cardiac effects have been reported. Phenytoin has a narrow therapeutic index and can cause sedation or excitement (Redpath & Bowen, 2019). Recently a case series has demonstrated successful treatment of three cases of VT with oral propranolol (Kovács et al., 2019).

Intravenous magnesium sulfate was first used to treat spontaneous VT in the horse in 1935 (Zwillinger, 1935). Since then, magnesium sulfate use for this purpose has only been anecdotally reported. van Loon (2013) reported that magnesium sulfate rarely converts sustained VT, however, it decreases the risk of developing *torsades des pointes* during other antiarrhythmic therapies. In human medicine magnesium sulfate is the therapy of choice for *torsades des pointes* (Manz et al., 1997) and is recommended in quinidine-induced VT in horses (Reef et al., 1995).

This case demonstrates the safe and effective use of magnesium sulfate, in addition to lidocaine CRI, in a horse with VT secondary to GI disease.

CASE HISTORY

On a routine post-operative check 36 h following exploratory laparotomy a 19-year-old Warmblood gelding with no history of cardiac disease was identified as having an irregularly irregular cardiac rhythm and HR of 80 beats/min. All post-operative checks until this time had revealed a regular cardiac rhythm with a heart rate between 40 and 60 beats/min. The exploratory laparotomy performed 36 h earlier had revealed an epiploic foramen entrapment with small intestine which was non-ischaemic. At the time the irregular rhythm was detected the horse was showing signs of abdominal pain and had gastric reflux (net volume 40 L in 12 h). The horse was already being treated with a CRI of 0.05 mg/kg/min lidocaine following 1.3 mg/kg bwt as a bolus (Bova Specials Ltd) when he developed the VT. Additionally, intravenous fluid therapy (IVFT) with Hartmanns (4 ml/kg/h; Vetivex 11; Dechra Veterinary Products), along with procaine penicillin (25,000 iu/kg bwt; Depocillin; MSD Animal Health) twice daily, gentamicin (8 mg/kg bwt; Genta-Equine; Dechra Veterinary Products) once daily and flunixin meglumine (1.1 mg/kg bwt; Flunixin Injection; Norbrook Laboratories) twice daily were administered.

CLINICAL FINDINGS

The horse was quiet and dull and showing signs of abdominal pain including pawing at the ground and flank watching. There was an irregularly irregular heart rhythm with a HR of 80 beats/min but no cardiac murmurs were detected. Arterial pulse quality was variable and weak and there was no jugular distension. Capillary refill time (CRT) was 2 s and mucous membranes were pale pink and moist. Respiratory rate was 16 breaths/min with normal respiratory effort. GI borborygmi were reduced but present in all quarters. Rectal temperature was 37.7°C. Urination was considered normal, however faecal output was reduced.

Serum biochemistry showed marked hypomagnesaemia (0.47 mmol/L; 0.71–1.01 mmol/L), reduced total protein (53 g/L; 56–79 g/L), increased activities of aspartate aminotransferase (861 U/L; 100–600 U/L), creatine kinase (692 U/L; 10–350 U/L) and lactate dehydrogenase (2782 U/L; 250–2070 U/L). Total bilirubin concentrations were increased (105 µmol/L; 0–60 µmol/L). Glucose, creatinine, urea, total calcium, sodium, potassium, chloride, albumin, globulin, alkaline phosphatase, gamma-glutamyl transferase and lactate were within normal limits.

A base apex ECG showed runs of wide and bizarre QRS complexes at a regular fast rhythm at a rate of 76 beats/min (Figure 1). There was no associated P wave, and the T wave was large and followed immediately after the QRS complex. This rhythm was infrequently interspersed with normal smaller QRS complexes at a rate of 52 beats/min creating an irregular rhythm. This ECG is typical of paroxysms of VT interspersed with periods of NSR.

DIAGNOSIS

A diagnosis of paroxysmal VT was made based on the ECG findings. This may have been caused by electrolyte imbalances secondary to GI losses, or dilutional effects following resuscitation fluids (Hartmann's), primary myocardial disease, bacterial endocarditis, sepsis or hypoxia. Aetiology of VT was not required to begin treatment in this case, therefore, no more diagnostic tests were completed.

TREATMENT

Boluses of undiluted 25% magnesium sulfate (10 mL; Magniject; Norbrook Laboratories) were administered slowly intravenously to effect (total dose 20 g) with continuous ECG monitoring. VT converted to NSR with a rate of 44 beats/min within 20 min (Figure 2).

Following conversion, a second exploratory laparotomy was performed under general anaesthesia due to the ongoing signs of abdominal pain and increasing volume of gastric reflux.

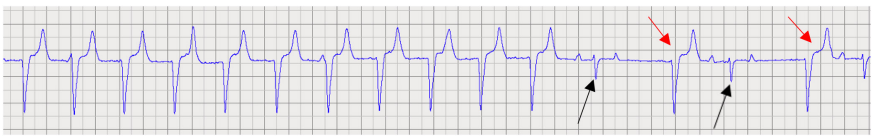

FIGURE 1 ECG (25 div/s) showing ventricular tachycardia with heart rate of 76 beats/min followed by normal sinus rhythm at a rate of 52 beats/min (sinus complexes shown with black arrows) with two isolated ventricular premature complexes (red arrows).

Anaesthetic monitoring included a continuous ECG, capnograph and invasive arterial blood pressure. Due to the potential cardiovascular depressive effects of isoflurane which include a reduction in blood pressure and cardiac output (Steffey et al., 1987), the lowest possible dose was used. Therefore, a CRI of ketamine (5 mg/kg/h; Anesketin; Dechra Veterinary Products) was added. A lidocaine CRI was administered at 0.05 mg/kg/min following a 1.3 mg/kg bwt bolus until 20 min before recovery. Shortly after induction, a relapse of VT was detected which was abolished by a bolus of magnesium sulfate (Figure 3). The horse stayed in NSR until the ECG was removed for recovery.

The surgery identified ileus, therefore the SI was decompressed. Recovery was uneventful and a Holter ECG was used postoperatively. Initially the occasional VPC couplet was detected, no further treatment was given and this resolved within 1 h of recovery and the horse remained in NSR. Following surgery, the horse received IVFT with Hartmanns supplemented with 0.14% potassium chloride (Potassium chloride 20%; Hameln Pharmaceuticals), 0.1% magnesium sulfate (Magniject 25%; Norbrook Laboratories) and 0.4% calcium gluconate (Calciject 40%; Norbrook Laboratories) at 4 ml/kg/h.

OUTCOME

The horse was settled overnight and a Holter ECG showed NSR with a heart rate of 48 beats/min. Repeat electrolyte analysis the following morning showed an increase in total magnesium concentrations (0.61 mmol/L; 0.72–1.01 mmol/L). IVFT was discontinued 2 days later following normal clinical examinations, no ongoing losses and normal blood analysis which included a normal total magnesium (0.86 mmol/L). Injectable antibiotic therapy with penicillin and gentamicin was discontinued 5 days later and he was placed onto trimethoprim potentiated sulfadiazine (30 mg/kg bwt; Trimediazine; Vetoquinol) twice daily orally for a further 14 days due to the increased risk of an incisional infection as two exploratory laparotomies had been performed. Injectable pain relief with flunixin meglumine was continued at 1.1 mg/kg bwt twice daily for 4 days and then changed to oral phenylbutazone (1.75 mg/kg bwt; Equipalazone; Dechra) twice daily for a further 14 days; 48 h after the second exploratory laparotomy, food was reintroduced, and the horse was discharged on Day 8 with detailed exercise instructions for the following 6 months. At the time of discharge the horse remained in NSR. A resting and an exercising ECG was recommended prior to commencement of ridden exercise.

DISCUSSION

The primary cause of the VT was thought to be hypomagnesaemia secondary to nasogastric reflux, however SIRS could not be excluded. No further investigations were done following conversion to NSR as once electrolyte imbalances were corrected, no further VT was detected. Diagnostics could have included echocardiography to investigate primary myocardial disease and blood analysis to quantify cardiac troponin I along with the cardiac isoenzymes hydroxybutyrate dehydrogenase and lactate dehydrogenase.

Current guidelines would suggest that this VT would not require treatment as there was no evidence of poor perfusion, HR was less than 100 beats/min and there were no multiform/polymorphic complexes or *torsades des pointes* present (de Solis, 2020; House & Giguère, 2009). However, in this case, the VT was treated due to the requirement for a second exploratory laparotomy. The presence of VT prior to anaesthesia was considered a significant risk factor for peri-anaesthetic morbidities. A lidocaine CRI was already being used as an analgesic and potential GI prokinetic (Malone et al., 2006), therefore, magnesium sulfate was chosen as it is the second-line choice for therapy (Coudry et al., 2007). This, along with the presence of hypomagnesaemia, was the reason magnesium sulfate was used. Magnesium sulfate has no proarrythmic properties unlike other antiarrhythmic drugs (Redpath & Bowen, 2019).

Assessing magnesium status is difficult as only 1% of the body's magnesium is found extracellularly. It is present in three fractions; protein bound, complex bound and ionised, of which only ionised magnesium is biologically active (Jahnen-Dechent & Ketteler, 2012). The relationship between ionised and total magnesium in serum do not correlate well particularly when hypomagnesaemia is present. Ionised magnesium is affected by acid–base status. For example, nasogastric reflux can produce an alkalosis lowering ionised magnesium and causing clinical signs of hypomagnesaemia, with normal total magnesium. Therefore, measurement of the total and ionised fractions is important. We only measured total serum magnesium since no ion-specific electrode for magnesium was available; this is a limitation of this case report.

The association between magnesium and cardiac electrical abnormalities is long established (Dyckner & Wester, 1982). Deficiency in magnesium potentiates cellular depolarisation by decreasing cellular potassium and increasing cellular calcium (Iseri & French, 1984). This effect, along with shortening the absolute refractory period due to magnesium's effect on the SA node, increases ventricular vulnerability. This increases the risk of arrhythmias, especially VPCs and VT (Berkelhammer & Bear, 1985). Therefore, magnesium sulfate may be effective for treating VT by preventing this effect

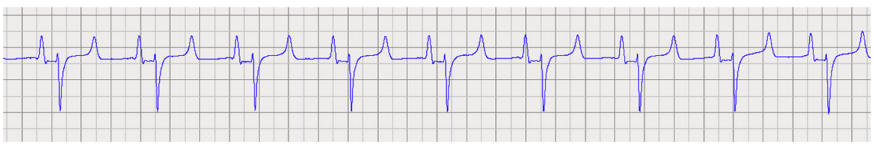

FIGURE 2 ECG (25 div/s) showing normal sinus rhythm with a heart rate of 44 beats/min.

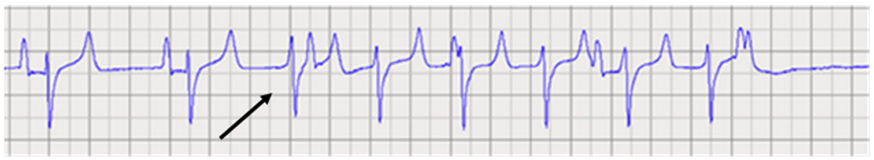

FIGURE 3 ECG (25 div/s) showing normal sinus rhythm with a relapse of ventricular tachycardia (black arrow) post induction of anaesthesia.

in hypomagnesaemic horses. In normomagnesaemic horses it may block calcium channels therefore restricting increases in cellular calcium (Redpath & Bowen, 2019).

Previous literature discusses using magnesium sulfate to convert VT with limited success, making this case unusual (van Loon, 2013). It is likely the VT responded to magnesium sulfate as it was caused by hypomagnesaemia. Electrolyte disorders are common in horses with acute abdominal crises. The prevalence of preoperative hypomagnesaemia in horses with surgically managed colic is 54%, and those which developed post-operative ileus had significant hypomagnesaemia post-surgery (Garcia-Lopez et al., 2001). This correlates with the presence of ileus and hypomagnesaemia seen here. Additionally, lidocaine treatment was initiated prior to VT development, as this is a routine post-operative treatment in horses with small intestinal lesions at exploratory laparotomy, increasing suspicion the VT was caused by hypomagnesaemia.

High magnesium concentrations can lead to bradycardia and when anaesthetised, hypotension. During anaesthesia blood pressure was monitored and remained above 70 mmHg. To further improve monitoring, we could have measured central venous pressure to more accurately monitor cardiac output. Additionally, VT can lower blood pressure due to decreased ventricular filling. Therefore, a blood pressure measurement could have helped to determine if the initial rhythm required treatment.

In this case, we demonstrate that magnesium sulfate, in addition to a pre-existing CRI of lidocaine, is a safe and effective treatment for VT in the horse especially where hypomagnesaemia is present. Additionally, it can be considered a first-line treatment for VT and can be used in conjunction with other antiarrhythmic drugs.

AUTHOR CONTRIBUTIONS

All authors contributed to the case management and written report.

CONFLICT OF INTEREST

No conflicts of interest have been declared.

ETHICS STATEMENT

This case report involves a client-owned horse. Informed client consent was obtained to enable submission and publication of this report.

SOURCE OF FUNDING

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