Journal of Veterinary Internal Medicine
Open Access

Case Report
J Vet Intern Med 2017;31:884–889

Third-Degree Atrioventricular Block and Collapse Associated with Eosinophilic Myocarditis in a Horse

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Third-degree atrioventricular block (AVB) and primary inflammatory myocarditis are uncommon findings in horses. The horse of this report presented for collapse at rest and was found to have multiple cardiac arrhythmias, most notably 3rd-degree AVB. The horse was subsequently diagnosed with eosinophilic myocarditis on necropsy, a rare form of myocarditis not previously reported in horses. Despite extensive testing, an etiologic agent could not be identified, illustrating the difficulty in identifying a specific cause of myocarditis in horses.

Key words: Arrhythmia; Cardiac; Heart; Syncope.

Third-degree (complete) atrioventricular block (AVB) is uncommon in the horse and often is presumed to be associated with inflammation or degenerative changes of the atrioventricular (AV) node.[1] Impaired AV conduction associated with complete AVB can result from systemic disease, electrolyte imbalances, drugs, toxins, or myocardial disease.[2] Previous occurrences of complete AV block in the horse have been associated with congenital defects,[3,4] mediastinal lymphoma,[5] and rattlesnake envenomation.[6]

Diagnosis of complete AVB is based on electrocardiographic (ECG) findings. Electrocardiography discloses regular, non-conducted P waves, with dissociation of the P waves and QRS complexes, a variable P-R interval, and a slow ventricular rate that may be junctional (narrow) or ventricular (wide) in origin.[1] Concurrent inflammation or fibrosis within the His bundle may result in conduction delay that also may affect the appearance of the QRS complexes.

Myocarditis is identified as the cause of AV block in 6% of human cases diagnosed based on right ventricular endomyocardial biopsies.[7] Eosinophilic myocarditis, a rare form of cardiac disease in humans, is most commonly associated with hypersensitivity or allergic reactions.[8] Eosinophilic myocarditis recently also has been reported in dogs.[9] Primary inflammatory myocarditis in horses is rare, and an eosinophilic component has not been previously described.[10]

Abbreviations:

AVB	atrioventricular block
CTnI	cardiac troponin I
ECG	electrocardiogram
ELISA	enzyme-linked immunosorbent assay
EM	eosinophilic myocarditis
LC-MS	liquid chromatography–mass spectrometry
MFI	median fluorescent intensity
OSP	outer surface protein
PCR	polymerase chain reaction
WBC	white blood cell

Case Report

An 11-year-old 473 kg Quarter Horse mare was referred to New Bolton Center at the University of Pennsylvania for evaluation of collapse. The horse had acute onset of multiple collapse episodes 16 hours before presentation. Collapse episodes ranged from leaning against the wall to complete recumbency and did not appear to be associated with any premonitory signs. Initial treatment by the referring veterinarian consisted of flunixin meglumine (1.1 mg/kg IV) and dexamethasone (0.1 mg/kg IV). The horse was current on vaccinations including Eastern Equine Encephalitis, Western Equine Encephalitis, tetanus, West Nile virus, influenza, equine herpesvirus, and rabies and had no previous relevant medical history. The horse lived on pasture with other horses, which were unaffected, and was fed free choice grass hay and 4 pounds of oats daily. The horse was dewormed on a regular rotational schedule and did not receive any medications. The horse was ridden 2 weeks previously with no report of exercise intolerance.

On physical examination, the mare was quiet, alert, and responsive. She had a body condition score of 6/9. Rectal temperature was 99.1°F (37.3°C). Respiratory rate was 16 breaths/min. Cardiac auscultation identified 2 simultaneous but dissociated heart rhythms: a louder, regular ventricular rhythm of 16–20 beats/min and a quieter, regular atrial (4th heart sound) rhythm of

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All work was done at the University of Pennsylvania

This study was not supported by a grant.

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Submitted November 10, 2016; Revised January 10, 2017; Accepted January 31, 2017.

DOI: 10.1111/jvim.14682

60–70 beats/min. Peripheral pulses were weak, a faint jugular pulse was evident bilaterally extending halfway up the neck with the head in a neutral position, and jugular filling was slow. Both pupils were dilated.

Laboratory abnormalities included leukocytosis (14,850 WBCs/μL; reference range, 4,900–10,300 WBCs/μL) due to neutrophilia (12,350 neutrophils/μL; reference range, 2,200–8,100 neutrophils/μL), mild hyperfibrinogenemia (392 mg/dL; reference range, 150–375 mg/dL), hyperglycemia (313 mg/dL; reference range, 72–114 mg/dL), hyponatremia (128 mmol/L; reference range, 132–141 mmol/L), hypochloremia (86 mmol/L; reference range, 94–102 mmol/L), and azotemia (serum creatinine concentration, 3.6 mg/dL; reference range, 0.6–1.8 mg/dL). Cardiac troponin I (cTnI)[a] was increased at 0.59 ng/mL (reference range, 0.00–0.07 ng/mL).

A base-apex ECG identified 3rd-degree AVB with a junctional escape rhythm (Fig 1). A continuous ECG monitor with radiotelemetry[b] recorded over 4 hours disclosed persistent 3rd-degree AVB with periods of ventricular asystole (up to 24 seconds in duration). Mean ventricular heart rate for the duration of the continuous telemetry recording (approximately 4 hours) was 15.4 ± 8.9 beats/min. The horse experienced 2 collapse episodes during hospitalization. The first collapse (Video S1) occurred during transport to a padded stall and appeared to be initiated upon exiting the barn. Heart rate was 16 beats/min immediately before the collapse, and no change was noted on the ECG recording before this collapse episode. After collapse, the mare developed an accelerated idiojunctional/idioventricular rhythm and intermittent periods of atrial flutter/fibrillation with a junctional escape rhythm before ultimately returning to 3rd-degree AVB (Fig 2). The second collapse was preceded by a 16.5 second period of ventricular asystole; the horse became tachypneic and subsequently collapsed.

Atropine[d] (0.015 mg/kg IV) was administered to evaluate whether decreased parasympathetic tone would increase AV conduction. No change in rhythm was noted after administration of atropine. Dexamethasone[c] (0.1 mg/kg IV) was administered to treat potential myocardial inflammation. An abbreviated echocardiogram performed from the right parasternal window included a 4-chamber view, right and left outflow tract views, and short axis of the left ventricle. The entire heart could be visualized on a 30 cm screen. A standard 4-chamber view disclosed subjectively enlarged atria and right ventricle for a Quarter Horse, with the right ventricle almost as large as the left ventricle. The left atrial diameter was subjectively mildly enlarged at 12.9 cm, and mild pericardial effusion was present. Short-axis view of the left ventricle from the right identified a thin myocardium, with an interventricular septum thickness in diastole of 2.3 cm (reference range, 2.8 ± 0.2 cm) and left free wall thickness in diastole of 1.6 cm (reference range, 2.5 ± 0.3 cm).[11] The aortic root measured 7.4 cm at the sinus of Valsalva and the pulmonary artery measured 8.3 cm. Color flow Doppler identified a small to medium jet of diastolic tricuspid regurgitation. A complete echocardiogram could not be performed because the patient's rapid deterioration posed a danger to personnel safety. Emergency pacemaker implantation was recommended, but euthanasia was elected by the owner. Necropsy was performed.

Gross examination disclosed a moderate degree of right ventricular and left atrial dilatation, a firm brown liver, and cyathostomin endoparasitism of the ventral colon. On histology, diffuse hepatic centrilobular degeneration, manifested as cytoplasmic vesiculation, cell atrophy, and sinusoidal congestion, was observed. Eosinophilic myocarditis with a lymphocytic component was seen in the right ventricle, left ventricle, and interventricular septum. Within the right ventricular myocardium, several mural bundles of Purkinje fibers were surrounded by a low density eosinophilic and lymphocytic infiltrate (Fig 3). Unfortunately, AV nodal tissue was not acquired in the tissue sections taken. Dilated lymphatic vessels were observed in the left ventricular myocardium. Immunohistochemical staining for *Sarcocystis neurona* failed to identify protozoa. *Borrelia burgdorferi* real-time PCR on formalin-fixed, paraffin-embedded cardiac tissue was negative. *Bartonella henselae* PCR[e] on formalin-fixed cardiac tissue was negative. Eosinophilic cellular infiltrates were not seen in other tissues examined histologically.

A modified McMaster's fecal floatation identified 800 strongyle-type ova per gram (in January). Liquid chromatography–mass spectrometry (LC-MS)[f] for monensin on stomach contents and frozen liver tissue failed to identify detectable monensin concentrations. Serologic *Borrelia burgdorferi* multiplex testing[g] was negative for outer surface protein (OSP) A antibodies (median fluorescent intensity [MFI] = 115), negative for OSPC antibodies (MFI = 73), and equivocal for OSPF antibodies

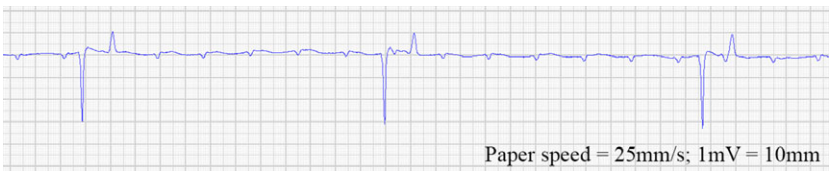

Fig 1. Modified base-apex ECG tracing obtained from an 11-year-old Quarter Horse mare evaluated for collapse. Third-degree AV block with a junctional (high ventricular) escape rhythm can be seen. Note the variable PR intervals, narrow QRS complexes (122 ms), atrial rate of 80 beats/min, and ventricular rate of ~20 beats/min. Negative P waves are likely associated with electrode placement, but may also represent ectopic activity or altered conduction due to atrial myocardial disease or stretch.

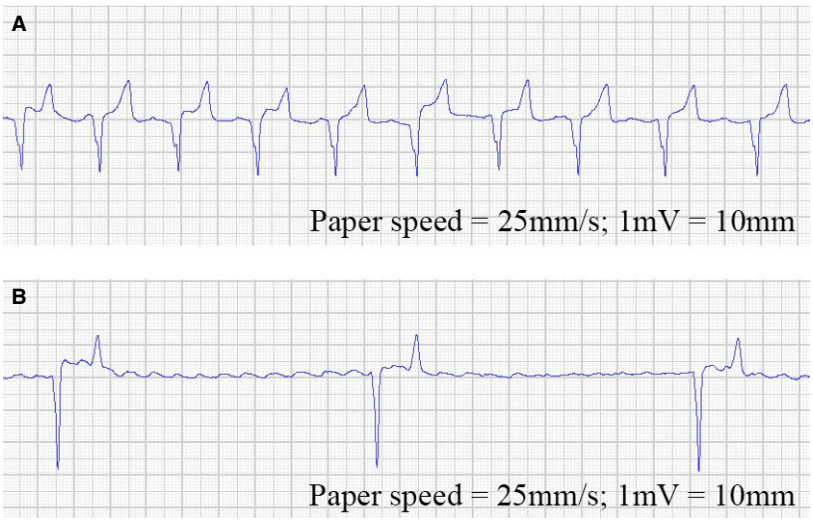

Fig 2. Modified base-apex ECG tracing obtained immediately after episode of collapse. Complete AV block is seen. (**A**). An accelerated escape rhythm (idiojunctional/idioventricular) is evident (QRS width = 120–132 ms; ventricular rate of 70 beats/min). P waves are indistinct. (**B**). The rhythm briefly changes to atrial flutter/fibrillation with a narrow escape rhythm (QRS width = 116 ms; ventricular rate of 18 beats/min) before returning to the rhythm seen in Figure 1.

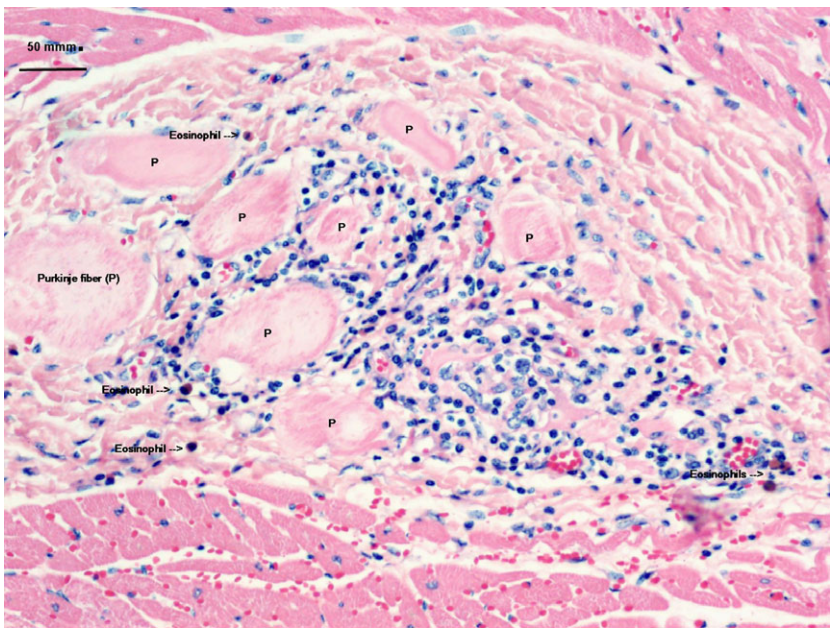

Fig 3. Hematoxylin–eosin stain of the right ventricular myocardium (20×). An infiltrate of lymphocytes and a few eosinophils are focused on a bundle of Purkinje fibers deep within the right ventricular myocardium.

(MFI = 935). Serologic combined SAG 2, 4/3 ELISA[h] for *Sarcocystis neurona* identified a weak titer of 1:250. Serologic ELISA[g] for *Neospora hughesi* was negative with a titer <1:500. Serologic Western blot for *Toxoplasma gondii* was negative.

Discussion

This horse was presented for collapse at rest and was subsequently diagnosed with 3rd-degree AVB, an uncommon arrhythmia in horses. Histology disclosed eosinophilic myocarditis, a rare form of myocarditis that has been described in humans but not previously in horses. A definitive etiologic agent or inciting allergen could not be identified, highlighting the difficulty in determining a specific cause of myocarditis in horses.

A number of differential diagnoses should be considered when presented with a horse that collapses at rest, although cardiovascular and neurological causes, along with disorders of sleep, are most common.[12] Collapse often is classified as either syncopal or non-syncopal. Syncopal collapse previously has been reported in

association with complete AVB and sick sinus syndrome in horses.[12–14] Other bradyarrhythmias such as advanced 2nd-degree AVB, sinus arrest or marked sinus bradycardia and rapid tachyarrhythmias also should be considered. Although intermittent cardiac arrhythmias can pose a diagnostic challenge, persistent arrhythmias usually are readily apparent on initial diagnostic evaluation, as was the case in this horse.

Additional clinical signs can accompany collapse in horses and might help guide differential diagnosis. This horse had evidence of systemic hypoperfusion secondary to bradyarrhythmia, which likely resulted in poor cardiac output and syncopal episodes. The bilateral mydriasis likely was due to cerebral ischemia after systemic hypoperfusion. Similarly, ocular ischemic syndrome is a condition in humans resulting in bilateral mydriasis caused by severe arterial hypoperfusion of the eye arising from cardiovascular occlusion or stroke.[15] In addition, the azotemia seen in this horse may be explained by systemic hypoperfusion.

Our horse exhibited 3rd-degree AVB with escape complexes of 2 morphologies, both of which were narrow and therefore likely to have originated either in junctional or high ventricular regions. The short period of accelerated idiojunctional/idioventricular rhythm was composed of narrow QRS complexes (Fig 2A), suggesting this rhythm also was a junctional rhythm. In addition, the horse developed a period of atrial fibrillation/flutter (Fig 2B), which was likely secondary to atrial inflammation and dilatation associated with myocarditis. Atrial tachycardia and complete AVB can be confused, but may be distinguished by the observation of a fast atrial rate and normal ventricular rate in atrial tachycardia, contrasted with a mildly increased atrial rate, slow ventricular rate, and AV dissociation in complete AVB. Diastolic AV valve regurgitation is observed commonly during AV block; our horse exhibited diastolic tricuspid regurgitation during atrial relaxation. The relative pulmonary artery enlargement in this horse was assumed to be secondary to bradycardia-induced forward failure.

Myocarditis is found as a cause of AV block in 6% of affected humans,[7] and the complete AV block seen in our horse likely was secondary to eosinophilic myocarditis, although histological sections of the AV node and His Bundle were not obtained. Eosinophilic myocarditis is a rare form of cardiac disease in humans and is most commonly caused by hypersensitivity or allergic reactions.[8] The disease is often fatal if left untreated, and unfortunately, the disease is usually not recognized clinically and is only discovered at necropsy.[16] A large number of medications have been associated with development of eosinophilic myocarditis in people, ranging from antimicrobials such as chloramphenicol and tetracyclines to anti-inflammatory drugs such as phenylbutazone.[8] Eosinophilic myocarditis also has been associated with protozoal infections (e.g, *Trypanosoma, Toxoplasma, Trichinella*).[17] However, the cause of eosinophilic myocarditis in humans is often unknown.[16] Our horse had no known exposure to medications associated with development of eosinophilic myocarditis in humans, and serologic testing for protozoal infections did not elucidate a cause. The clinical relevance of the parasite burden noted in this horse is unclear. Parasite migration theoretically may result in development of the disease, but there was no evidence of parasite migration. Clinical signs of eosinophilic myocarditis in people range from vague, mild, and self-limiting, to acute syncope and death. Humans with eosinophilic myocarditis may have marked peripheral eosinophilia.[8] However, eosinophilia may be absent in the early stages of the disease and a subset of patients never develop eosinophilia during the course of disease.[8] Similarly, our horse did not exhibit peripheral eosinophilia or evidence of other eosinophilic disease. Additionally, administration of corticosteroids before admission may have affected the peripheral eosinophil count.

Histopathologic examination of myocardium in people with eosinophilic myocarditis shows a mixed inflammatory infiltrate with varying numbers of eosinophils; eosinophils may be few and focal or there may be widespread infiltration by eosinophils.[16] Similarly, our horse had a mixture of eosinophils and lymphocytes within the myocardial tissue sampled. Given the combination of lymphocytic and eosinophilic myocardial inflammation seen in this horse, it is possible the clinical syndrome was driven by an immune response similar to eosinophilic myocarditis described in humans. Eosinophilic myocarditis in humans also has been a feature of hypereosinophilic syndromes.[8] Hypereosinophilic disease in horses has been described as part of multisystemic eosinophilic epitheliotropic disease syndrome (MEEDS) or in conjunction with eosinophilic enteritis, eosinophilic pneumonia, or dermatologic disease. Cardiac involvement in humans with eosinophilic disease is common and multisystemic eosinophilic disease in dogs has been described to affect the heart,[18] but eosinophilic disease affecting the heart has not been described previously in horses. The lymphocytic component of our horse's disease likely is associated with the mixed inflammatory infiltrate seen in eosinophilic myocarditis cases in humans. However, lymphocytic infiltrates are seen most commonly in myocarditis in humans, and are commonly associated with viral infections or autoimmune responses.[19]

Primary inflammatory myocarditis is rare in horses, and an eosinophilic component has not been described previously.[10] *Streptococcus equi* has been reported as a cause of interstitial myocarditis in a foal.[20] *Bartonella henselae* has become an important recognized cause of myocarditis and endocarditis in dogs, cats, and other domestic species.[21] Myocarditis caused by *Borrelia burgdorferi* has been reported in both humans and dogs.[22,23] Other reported causes of myocarditis in veterinary species include *Toxoplasma gondii* in a llama,[24] *Neospora caninum* in a dog,[25] and *Sarcocystis neurona* in sea otters and raccoons.[26] An eosinophilic component to these diseases has not been described.

Antemortem diagnosis of myocarditis remains difficult in horses. Diagnosis often is presumptive and based on history, physical examination findings, echocardiography, ECG, and cardiac troponin I concentrations.

Cardiac troponin I may be increased in the acute stage of myocarditis, but may return rapidly to normal concentrations.[27] In addition, cTnI concentrations may remain low if blood is sampled shortly after the insult, before reaching its peak. In 1 study, horses with monensin-induced myocardial damage did not have increased cTnI concentrations until 24 hours or later after myocardial insult.[28] Therefore, normal concentrations do not rule out myocardial injury.[29] Our horse had a relatively mild increase in cardiac troponin I concentration, but only a single measurement was obtained early in the clinically apparent disease course, and this concentration may have increased with time. Echocardiography in humans with eosinophilic myocarditis may be normal or may show severe left ventricular systolic dysfunction, with endomyocardial scarring, valvular changes, and thrombus formation seen in the final stage of disease.[8] Cardiac magnetic resonance imaging and computed tomography also have been described in the diagnosis of eosinophilic myocarditis in humans.[8] The chamber enlargements are suggestive of dilated cardiomyopathy or may be secondary to chronic volume overload. In myocarditis in humans, acute dilated cardiomyopathy is 1 of the most dramatic presentations of acute myocarditis, with a link between clinical myocarditis and acute dilated cardiomyopathy shown in studies evaluating endomyocardial biopsies.[30,31] The gold standard for the diagnosis of myocarditis in humans is based on endomyocardial biopsy,[32] a procedure that has been performed in dogs and recently has been reported in horses.[33,34] However, right-sided biopsy as described in the equine veterinary literature may miss localized or multifocal disease.

Corticosteroid treatment for myocarditis was initiated in our horse, but the definitive treatment of complete AVB requires implantation of a cardiac pacemaker, as reported previously.[13] In an emergency situation, placement of a temporary pacing catheter into the right ventricular apex via the jugular vein can be performed in the standing horse. The temporary catheter then can be used to maintain adequate heart rate during sedation and implantation of a permanent rate-adaptive pacemaker.[14,35] Cost and potential complications of permanent pacemaker insertion and maintenance may dissuade horse owners from this option, especially with uncertain safety implications regarding future use as a riding horse. In humans, rate-adaptive pacing has resulted in improved exercise tolerance independent of heart rate changes.[36] A recent study in humans found that chronic ventricular pacing did not affect the physical capacity of pediatric and young patients, but did have a negative impact in older patients.[37] However, no studies in horses have evaluated the impact of pacemaker implantation on athletic performance. When pacemaker implantation is not an option, corticosteroid treatment is thought to be beneficial to treat inflammatory changes in the AV node and His Bundle.[13] In hindsight, the diagnosis of eosinophilic myocarditis in this horse would have prompted corticosteroid or other immunosuppressive therapy and potentially complicated successful maintenance of a permanent pacemaker because of an increased risk of infection. Anticholinergics, such as atropine or glycopyrrolate, can be used in an attempt to improve AV conduction by inhibiting parasympathetic input.[6] Administration of atropine in our horse resulted in no improvement in conduction, confirming that excessive parasympathetic stimulation was not the cause of the slow conduction. Dopamine and isoproterenol also have been used to treat AV block, but these drugs may result in tachyarrhythmias.[1] Both dopamine and isoproterenol act on β1-adrenergic receptors as positive inotropes to increase myocardial contractility and thus cardiac output.[38]

Footnotes

[a] Cardiac troponin I, Stratus CS, Dade Behring Inc., Deerfield, IL
[b] Televet 100 Version 6.0.0, Engel Engineering Service GmbH, Heusenstam, Germany
[d] Atropine Sulfate, 15 mg/mL, Neogen Corporation, Lansing, MI
[c] Dexamethasone Solution, 2 mg/mL, Agri Laboratories, Ltd., St. Joseph, MO
[e] Bartonella henselae PCR, Galaxy Diagnostics, Research Triangle Park, NC
[f] Liquid chromatography–mass spectrometry, Pennsylvania Animal Diagnostic Laboratory System, Kennett Square, PA
[g] Lyme Multiplex, Animal Health Diagnostic Center, Cornell University, Ithaca, NY
[h] Equine Diagnostic Solutions, LLC, Lexington, KY

Conflict of Interest Declaration: Authors declare no conflict of interest.

Off-label Antimicrobial Declaration: Authors declare no off-label use of antimicrobials.

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Supporting Information

Additional Supporting Information may be found online in the supporting information tab for this article:

Video S1. Supplemental video obtained from an 11-year-old Quarter Horse mare evaluated for collapse.