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Diagnostic Exercise
From the Latin Comparative Pathology Group and the Davis-Thompson Foundation

Fibrinosuppurative valvular endocarditis with myxomatous valvular degeneration in a mare

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Clinical History: A 12-year-old Azteca mare died unexpectedly with no premonitory clinical signs being observed.

Gross Findings: The leaflets of the aortic semilunar valve were expanded by multiple smooth nodular thickenings that blended seamlessly with the adjacent portions of the valve **(Fig 1A, B)**. There was also an irregular focus of tan to red material adhered to the valve **(Fig 1A, B)**.

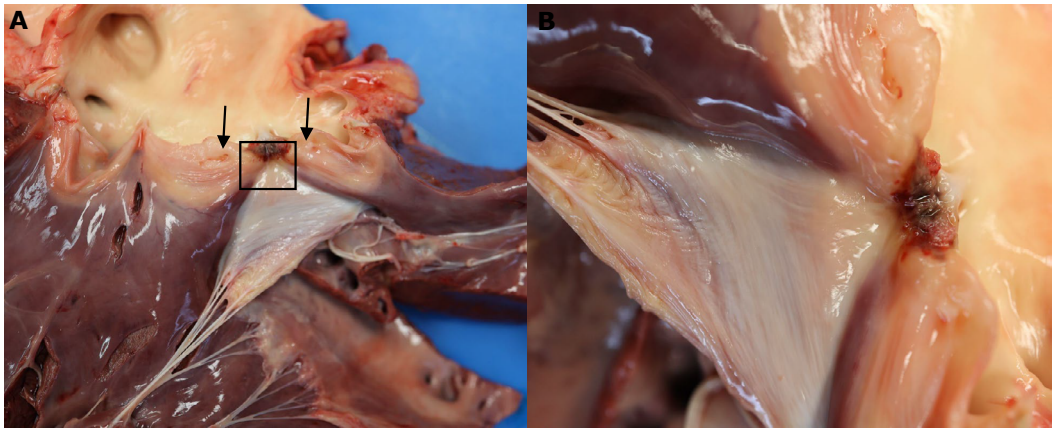

Figure 1 (A). The leaflets of the aortic valve are thickened and firm (arrows). There is a focus of tan to red material adhered to the valve (box). **(B)** Close-up of the aortic valve lesion. The aortic leaflets are thickened, and there is a single irregular focus of dark brown to red material adhered to the valve. Note that this "vegetative" lesion does not extend to the mitral valve leaflet

**The Diagnostic Exercises are an initiative of the Latin Comparative Pathology Group (LCPG), the Latin American subdivision of The Davis-Thompson Foundation (DTF). These exercises are contributed by members and non-members from any country of residence. Consider submitting an exercise! A final document containing this material with answers and a brief discussion will be posted on the DTF website: https://davisthompsonfoundation.org/diagnostic-exercise/*

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Follow-up questions:

- Microscopic description
- Morphologic diagnoses
- Potential causes

ANSWERS:

Microscopic description:

In the examined sections of the aortic semilunar valve, there is expansion of the valve leaflet by pale basophilic myxomatous material **(Fig 2A)** with focal accumulation of fibrin, neutrophils, and reactive fibroblasts lining the surface of the valve, which corresponds to the red vegetative focus observed grossly **(Fig 2B)**. Hemosiderin-laden macrophages, lymphocytes, plasma cells, and erythrocytes are also present within the valve.

Morphologic diagnoses:

1. Heart, aortic semilunar valve:
   - Fibrinosuppurative valvular endocarditis
   - Myxomatous degeneration (consistent with valvular endocardiosis)

Potential causes:

Degenerative valve disease is noted most frequently in older horses. This preexisting valvular dysfunction is a predisposing factor for development of vegetative valvular endocarditis.

Discussion:

Myxomatous valvular degeneration (also called *valvular endocardiosis*) is an important age-related cardiac disease (10). In horses, the aortic valve is the most common site of valvular pathology, typically manifesting as nodular lesions (5). Histologically, the condition is characterized by an accumulation of loose, myxomatous, connective tissue within the valvular matrix (1). Given its slow progression, myxomatous connective tissue is predominantly observed in older horses (3). The disease is thought to be a response to shear forces created as blood flows across the surface of the valve and repeated impact of the leaflets as they close (5).

Valvular degeneration compromises valvular coaptation, resulting in turbulent blood flow within the heart and regurgitation of blood into the previous chamber. When regurgitation is severe, the pressure of the chamber may increase as well, triggering compensatory mechanisms and enlargement of the heart (4, 8). In many horses, signs of aortic valve

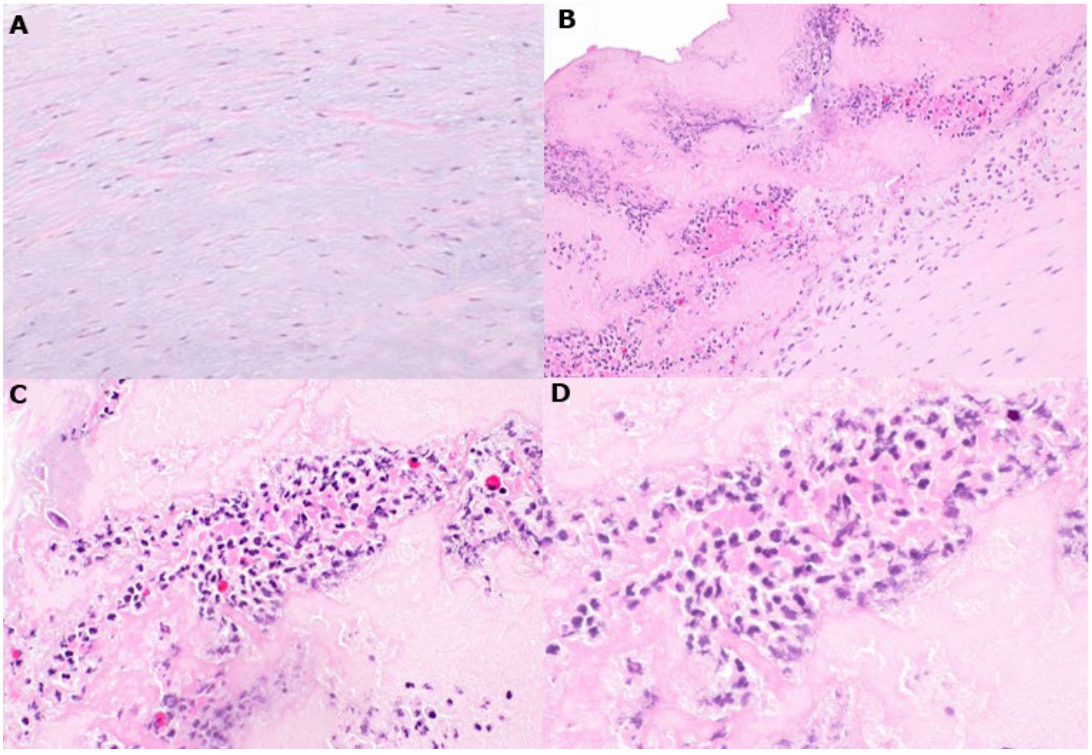

Figure 2 (A). Deposition of basophilic myxomatous material expands the aortic semilunar valve. H&E. **(B)** Focal accumulation of fibrin, neutrophils, and reactive fibroblasts lining the surface of the valve. H&E. **(C, D)** Higher magnification of image B shows the evidence of neutrophilic infiltration in the valve.

degeneration are uncommon because aortic regurgitation is rarely severe enough to cause heart failure (4). However, when heart failure does occur, its manifestations vary depending on the affected valve. For instance, severe mitral valve degeneration can lead to left-sided heart failure, characterized by signs of trouble breathing or coughing. These signs are associated with a backup of pressure in the vessels delivering blood to the left ventricle, which causes fluid to accumulate within the lungs (edema) (4). Conversely, severe tricuspid valve disease can lead to signs of right-side heart failure, with increased pressure in the vessels delivering blood to the right atrium and in the body's veins and capillaries. This can cause fluid accumulation under the skin and in the abdomen (ascites). Additional signs may include jugular distention and exaggerated pulses (4).

Endocarditis is usually the result of bacterial infection (5,10). Infective endocarditis typically involves one of the cardiac valves, although endocarditis of the cavity's wall may occur (3). The valves of the left side of the heart are usually involved (5,1). Grossly, the affected valves have large, adhering, friable, yellow-to-gray masses of fibrin termed "vegetations" (10). Many bacterial species can cause valvular endocarditis. A diverse range of organisms have been implicated in equine infective endocarditis without a single dominant group. S*treptococcus* spp., *Pasteurella* spp./ *Actinobacillus equuli, Pseudomonas* spp., and *Rhodococcus equi* are among the more commonly reported organisms in equine (5,1). Thrombotic involvement of chordae tendineae can occur from extension of valvular vegetations (1).

The development of infectious endocarditis requires previous endocardial injury followed by a period of bacteremia (9). Valvular heart disease can act as a risk factor since the turbulent flow around diseased valves may cause endocardial disruption, making them more susceptible to bacterial colonization (3, 9). Clinical manifestations vary depending on the site and severity of the intracardiac infection, embolization of vegetations, bacteremia, and the development of immune-complex disease. Signs can range from a relatively mild febrile condition to severe systemic signs and heart failure (5). Blood tests, echocardiography and electrocardiography are useful to diagnose this condition (3). Treatment involves high-dose bactericidal antibiotics, ideally based on the result of the blood culture and antibiogram (7). However, broad spectrum antimicrobials should be initiated before blood culture results are available because a delay in antimicrobial therapy may lead to progression of the associated cardiac disease (6). Only early definite diagnosis and early long-term antibiotic therapy can prevent the release of bacteria/emboli and thus manifestation of metastatic disease.

The diagnosis of mitral/aortic valve bacterial endocarditis carries a grave prognosis (6, 2). These horses usually succumb to the infection itself (which is difficult to resolve) or to heart failure secondary to valve incompetence (2). Even when bacteriologic cure is achieved, valvular insufficiency caused by vegetative endocarditis can remain despite reduction of the size and sterilization of the lesion (7). Therefore, endocarditis should not be considered merely a localized infection, but rather, a systemic problem that can cause the death of the animal if it is not diagnosed and treated at a very early stage of the disease.

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