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Microbiologic and pathologic findings in an epidemic of equine pericarditis

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Abstract. During the spring and summer of 2001 and in association with the mare reproductive loss syndrome, 22 terminal and 12 clinical cases of equine pericarditis were diagnosed in central Kentucky. *Actinobacillus* species were the principal isolates from 8 of 10 nontreated, terminally affected and 3 of 10 clinically affected horses. *Enterococcus faecalis* and *Streptococcus zooepidemicus* were cultured from the remaining 2 nontreated terminal cases. No viruses were isolated in tissue culture. Nucleic acid of equine herpesvirus–2 was detected in pericardial and tracheal wash fluids of 3 and 1 individuals, respectively. Microscopic alterations in sections of heart and parietal pericardium were consistent with chronic fibrinous bacterial pericarditis. This report confirms a significant role of *Actinobacillus* species in equine pericarditis and describes an epidemic of this infrequently observed syndrome in the horse.

Introduction

Pericarditis is an uncommon condition in the horse. Confirmed etiologies include bacterial infections,[1,6,11,20,21,32] trauma,[2,5,30] neoplasia,[7] and pericarditis secondary to inflammatory processes in adjacent lung, pleura, or lymph node.[1,2,4–7,11,20,21,30,32] Viral pericarditis has not been documented in the horse but is presumed to occur on the basis of rising antibody titers in some affected individuals, historical information suggesting previous viral respiratory tract infection, and inability to isolate bacteria from pericardial fluid samples.[10,25] The cause in the majority of cases is undetermined.[11,13,14,23,32]

Between May 7, 2001, and September 14, 2001, 22 horses with terminal pericarditis and serum, a combination of pericardial, pleural, or tracheal wash fluids from 12 horses with sonographic evidence of pericarditis were submitted to the Livestock Disease Diagnostic Center at the University of Kentucky, Lexington, Kentucky, for diagnostic testing. These cases of pericarditis occurred concurrently with components of the mare reproductive loss syndrome (MRLS) (early- and late-term equine abortions and stillbirths). The findings of case–control epidemiologic investigations of MRLS and this outbreak of fibrinous pericarditis have suggested that certain pasture- and management-related factors and environmental conditions increased the risk of MRLS-associated early- and late-term abortions and pericarditis.[9,10,12,26] The greatest risk factor for the development of fibrinous pericarditis was exposure of horses to the eastern tent caterpillar (ETC).[26] This report describes the microbiologic and pathologic features of an epidemic of fibrinous pericarditis in horses, which occurred in central Kentucky in the spring and summer of 2001 and concurrently with MRLS.

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Materials and methods

Records at the Livestock Disease Diagnostic Center were searched for all cases of terminal equine pericarditis and for submissions of pericardial and pleural fluids from horses with clinical pericarditis that were received between January 1 and December 31 of 2001. Only those individuals with terminal pericarditis and no evidence of pneumonia or inflammatory processes in the pleura or mediastinum were selected. Accession forms accompanying pericardial, pleural, and tracheal wash fluid samples from horses with clinical pericarditis were reviewed for historical data and clinical diagnoses, and only horses with sonographically diagnosed pericarditis, without clinical evidence of pneumonia, were included. One clinical case of constrictive pericarditis was excluded because of the lack historical information and limited diagnostic testing.

Horses for necropsy were examined for gross tissue lesions. Samples of lung, liver, spleen, kidney, small and large intestine, and bronchial and mesenteric lymph nodes were routinely collected for bacterial culture and virus isolation. Myocardium, pericardial fluid, or pleural fluid, in various combinations, were also collected from all affected horses.

For the isolation of aerobic bacteria from tissues, blood agar plates (BAP) and eosin–methylene blue agar plates (EMB) were inoculated. Pericardial fluids (1 ml) or swabs of pericardial fluid were also placed in 6.0 ml of tryptic soya broth, incubated at 37 C for 18–24 hr, and then subcultured to BAP and EMB. All plates were incubated at 37 C for 18–

24 hr in an 8% CO_2 atmosphere and examined for bacteria. Plates were incubated for an additional 24–72 hr at 37 C in an aerobic incubator and reexamined. Significant bacteria were identified using standard bacteriological procedures and tested for antibiotic susceptibility by the Bauer–Kirby disk diffusion procedure.[8]

Culture of *Mycoplasma* species was attempted on selected terminal (2) and clinical cases (13) by inoculating agar plates[a] and broth[b] with pericardial fluid and incubating this media in 8% CO_2 at 37 C. The broth was incubated until growth was evident or for 5 days and then subcultured to agar plates.[a] Using a stereomicroscope, all plates were examined daily for 7 days for the presence of colonies characteristic of *Mycoplasma.*

For virus isolation, pericardial and tracheal wash fluids from 15 horses (terminal and clinical cases) were inoculated onto 4 cell lines: RK-13KY, VERO, equine dermal, and equine kidney. Cultures were incubated at 37 C for 60 min and maintenance medium was added. Maintenance medium consisted of minimal essential media with 5% bovine calf serum or fetal calf serum, penicillin, streptomycin, gentamicin, and fungizone. Cultures were incubated at 37 C and observed daily for cytopathic effect. After 7–10 days incubation, the cell cultures were frozen, thawed, and inoculated onto fresh cell cultures. Cultures were checked daily and reported as no virus isolated if no cytopathic effect was observed after 7–10 days of incubation. In addition, pleural or pericardial fluids, or both, were tested for type A influenza virus using a commercially available kit[c] and according to manufacturer's instructions.

Fluids and sera submitted were tested by virus neutralization test against equine herpesvirus–1 (EHV-1). Specimens were diluted in 2-fold dilution series in 96-well plates with an EHV-1–positive control serum. One hundred to 300 tissue culture infectious dose 50% ($TCID_{50}$) of EHV-1 virus was added to the diluted sera and incubated at 37 C for 1 hr. Equine dermal cells were added and the plates incubated for 3 days. The plates were then checked for herpesvirus cytopathic effect and the antibody titer determined using the highest dilution of serum or fluid with no cytopathic effect as the endpoint. The hemagglutination inhibition test for antibodies was performed using A/Eq/Prague/56 and A/Eq/Ky/94 equine influenza viruses. Sera were first treated with kaolin and absorbed with chicken red blood cells. Sera were diluted in a 2-fold dilution series and tested against 4 hemagglutination (HA) units of the equine influenza viruses. Antibody titers were reported as the highest dilution of serum with complete inhibition of hemagglutination. Pericardial fluid from 3 horses and tracheal wash fluid from 1 horse also were tested for the presence of nucleic acid of EHV-1, EHV-2, EHV-4 by nested polymerase chain assay using nucleotide sequences from the glycoprotein B gene and methods described previously.[22,31]

Samples of lung, heart, liver, kidney, spleen, and small and large intestine were collected from the majority of horses for microscopic examination. Parietal pericardium was collected in 15 cases. Tissues were immersed in 10% neutral-buffered formalin, processed for sectioning, embedded in paraffin, sectioned at 5 µm, and stained with Harris hematoxylin and eosin.

Results

Horses with pericarditis came from 22 different farms located in 8 counties in central Kentucky and 1 county in eastern Kentucky. Thirty cases occurred in May and June, 1 in July, 2 in August, and 1 in September. The last 4 cases submitted (Jul–Sep) were terminally affected individuals that had been treated unsuccessfully for 2–3 months. Breed, age, and sex data were available for 23 individuals. Sixteen of the affected horses were Thoroughbreds, 3 were Quarter Horses, 1 was a Rocky Mountain Spotted Horse, and 1 was a Hanovarian. Two horses were of unknown or mixed breeding. The average age of affected individuals was 6.4 years, with a range extending from 3 weeks to 30 years. Fifteen animals were female and 8 male. Two terminally affected mares were pregnant with normal-appearing fetuses.

Clinical signs and findings included pericarditis (17), pericardial, pleural, and peritoneal effusions (5); sudden death (4); fever (2); abdominal pain, which in 1 case was intermittent (2); respiratory distress/pneumonia (2); weight loss (1); failure to gain weight in a younger individual (1); tachycardia (1); and tachypnea. Twelve of the terminally affected horses had received treatment for pericarditis before submission. Six of these individuals developed recurrent effusions or constrictive pericarditis (or both). Two other horses developed severe colitis.

Bacteria were isolated from samples of pericardial fluid or myocardium (or both) and were considered significant in 13 of 32 cases (40%). No bacterial growth occurred in 15 cases. In 4 cases, the isolates were considered contaminants. Pericardial fluid samples from 2 of the clinical cases were not submitted for aerobic culture. *Actinobacillus* species were the only isolates from samples of pericardial fluid or heart (or both) of 10 horses. Actinobacilli were recovered from multiple tissues in 4 of these individuals. An *Actinobacillus* species and *Escherichia coli* were isolated from the heart of 1 horse. *Streptococcus zooepidemicus* and *Enterococcus faecalis* were each isolated from a single animal. All horses with terminal pericarditis from which no bacteria were isolated had been treated with antibiotics before submission. No *Mycoplasma* species were cultured from pericardial fluid of 15 horses.

Only 3 of the *Actinobacillus* isolates demonstrated resistance to the tested antibiotics. One isolate was resistant to sulfa drugs, ampicillin, and carbenicillin; 1 to ampicillin and tricarcillin; and 1 to ampicillin. Moderate susceptibility of isolates was documented for amikacyin (6), kanamycin (5), gentamicin (4), and tribrissen (2).

No viruses were isolated in tissue culture from sam-

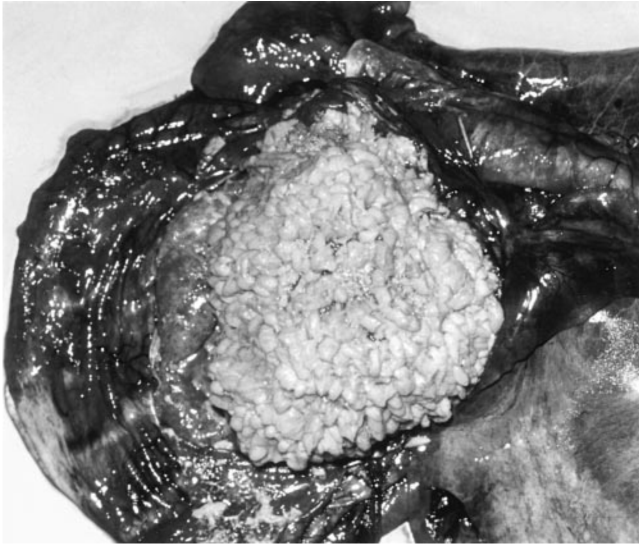

Figure 1. Fibrinous pericarditis in a horse. Notice the villonodular deposition of fibrin.

ples submitted from 15 individuals. Viral antigen was not detected in tissues by immunofluorescent methods. Equine herpesvirus–2 nucleic acid was detected by polymerase chain reaction assay in pericardial (3) and tracheal (1) fluids from 4 horses. Antibody against equine influenza–1 and EHV-1 was detected in 1 and 4 individuals, respectively.

Peritoneal and pleural effusions were either unavailable for visual examination or observations were not recorded in 14 of 22 horses with terminal pericarditis. Effusions of 7 horses were serous to serosanguinous, clear, and often contained strands and sheets of fibrin. In 1 individual, pleural and peritoneal fluid was red tinged.

In 16 individuals, the pericardial sac was oval to round, opaque, mottled to diffusely red, and fluid filled. The fluid was clear, yellow, and contained strands of fibrin. The fluid volume recorded in 9 cases ranged from 0.1 to 4.0 liters. In most cases, the parietal pericardium was thickened, congested, edematous, and was either partially or completely covered by a sheet of fibrin 0.5–1.5 cm thick. An easily detachable layer of fibrin 1.0–3.0 cm thick covered the epicardium (Fig. 1). Characteristically, fibrin deposition resulted in the formation of villous-like appendages that measured 1.0–2.0 cm in length. In cases of constrictive pericarditis, the parietal pericardium was 1.0–3.0 mm in thickness, white, dense, fibrous, and multifocally or diffusely adhered to the epicardium by loose, delicate, or dense fibrous connective tissue. The lumen of the pericardial sac was either obliterated or partitioned into multiple interconnecting fibrin-filled chambers by adhesions between the parietal and visceral pericardium. The epicardial surface was thickened by fibrous tissue. Hearts were reported as enlarged and dilated in 2 horses.

Lungs of affected individuals were normal except for areas of pressure atelectasis that were typically located lateral and adjacent to a dilated pericardial sac. Pulmonary edema was reported in 2 individuals. The liver in 6 horses was enlarged, dark red, and congested. The capsules were taut and occasionally dimpled, and the parenchyma was firmer than normal when incised. One horse had an abscessed retropharyngeal lymph node. Subcutaneous edema occurred in 2 animals.

Microscopic alterations in sections of heart, with minor exceptions, were similar, varying primarily on the age of the reactive/reparative response and intensity of cellular infiltration. The epicardial surface was covered by sequential layers of edematous granulation tissue and fibrin. The outer fibrin layer was of variable thickness, fenestrated, and infiltrated with neutrophils and lesser numbers of macrophages, lymphocytes, and plasma cells. Cellular infiltrates were unevenly distributed and occasionally formed dense aggregates. A transitional zone occurred at the point where the fibrin layer merged with the leading edge of granulation tissue. In this area, capillary buds, fibroblasts, and macrophages had infiltrated the fibrin sheet. The layer of granulation tissue varied in maturity from loose and edematous to dense and fibrous and covered an ulcerated epicardium (Fig. 2). Cellular infiltrates included lymphocytes, plasma cells, macrophages, and low numbers of neutrophils. The subepicardium was mildly thickened by the accumulation of fibroblasts and mononuclear inflammatory cells and by the deposition of collagen (Fig. 3).

Myocardial changes were minimal to mild and multifocal. Lesions included edema and separation of cardiac myocytes in the superficial myocardium; fibroplasia or fibrosis of the interstitium (or both) of the superficial myocardium, especially along branches of arterioles; infiltration of the myocardial interstitium with low numbers of lymphocytes, plasma cells, and neutrophils; and scattered foci of myocardial mineralization and necrosis. Mild, focal lymphocytic vasculitis was an uncommon lesion seen in the myocardium of 2 horses. With the exception of vasculitis, alterations were similar in the parietal pericardium.

Discussion

The 34 cases of primary equine pericarditis occurred in central Kentucky from May through September of 2001 and represent a substantial increase in the incidence of this syndrome when compared with the previous year's prevalence of 4 cases/year. The total number of horses affected with pericarditis during this pe-

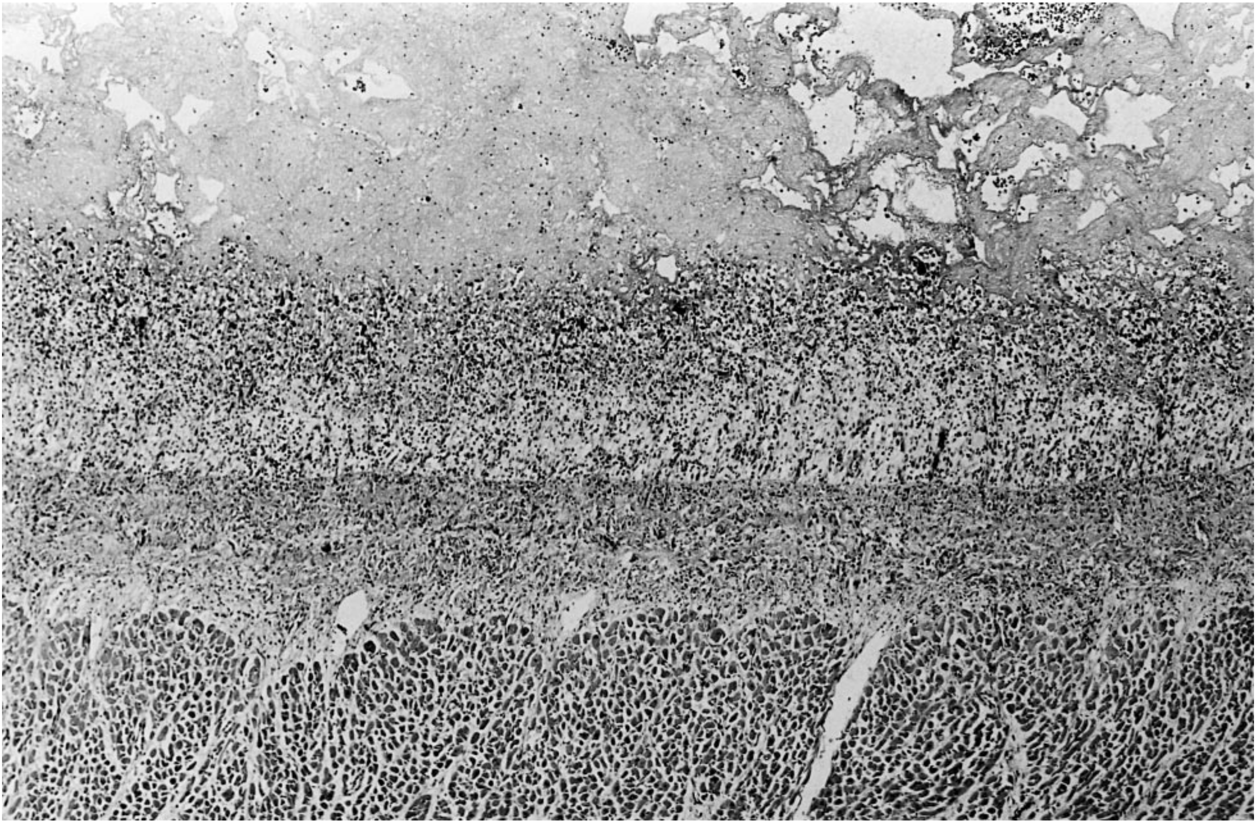

Figure 2 Photomicrograph of a section of heart from a horse with pericarditis. The epicardial surface is covered by successive layers of granulation tissue and fibrin.

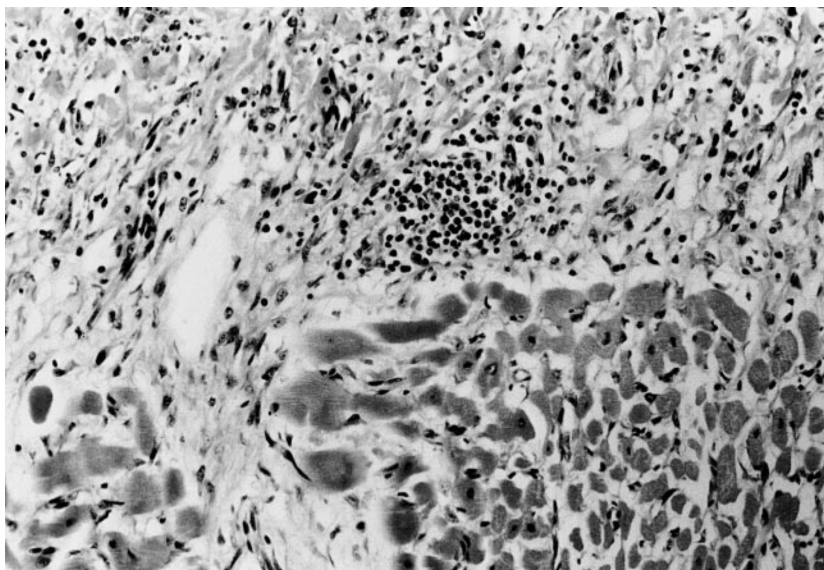

Figure 3. Focal accumulation of lymphocytes in the subepicardium. Notice the extension of fibrous tissue along perivascular channels and the lack of histologic alterations in the adjacent myocardium.

riod and thus the magnitude of this outbreak is unknown. Horses with subclinical pericarditis or animals that responded to antimicrobial therapy in the field and from which no samples were collected for analysis would not have been reported.

The distribution of cases according to time of submission (terminal and clinical cases) and geographic location (terminal cases) and a case–control epidemiologic study[26] indicate a point source epidemic and a causative role for an environmental factor that was present for a short, focal period of time. Exposure of affected horses to the ETC was identified in bivariate and multivariate logistic regression analyses as the greatest risk factor for the development of pericarditis.[26] These findings suggest that an agent or toxin associated with the ETC had a primary role in the pathogenesis of the condition. That spring, there had been a massive eruption of the ETC that coincided with the cases of pericarditis. Interestingly, the pericarditis syndrome was temporally associated with 2 other syndromes in horses with similar epidemiologic features: the MRLS, which included early- and late-term abortions and birth of premature, weak, often septic, foals[9,10,12] and an endophthalmitis syndrome. Neither a direct association nor a mechanism by which caterpillar-associated toxins or agents contributed to the pathogenesis of pericarditis in these horses has been established.

The majority of horses with pericarditis were Thoroughbreds and female. This reflects the breed and sex distribution of horses within central Kentucky and is not an indication of breed or sex susceptibility. The age distribution of affected individuals was similar to that in other reports of equine pericarditis. Eleven of 23 animals, however, were less than or equal to 2 years of age, suggesting young horses were more likely to develop pericarditis than older individuals. This observation is in agreement with the findings of an epidemiologic investigation of this outbreak, which suggested younger horses were at greater risk to develop pericarditis than older individuals.[26]

Bacterial pericarditis was diagnosed in all the terminal nontreated and 36% of the clinical cases on the basis of positive culture results. The bacterial isolation rate for terminal and clinical cases, excluding horses treated with antibiotics before sample collection, was 62% (13 of 21). In contrast, of the 63 cases of pericarditis identified in the literature since 1938, only 24 animals (or 38% of the affected individuals) were diagnosed with bacterial pericarditis.[1,2,4–7,11,13,14,16,20,21,25,27,30,32] In those reports, diagnoses were based on the isolation of bacteria from pericardial fluid[1,6,11,21]; cytologic evaluation of pericardial fluid and detection of acute inflammation with degenerate neutrophils or the presence of gram-positive or gram-negative bacteria, or both[1,21,32]; and clinical findings suggestive of sepsis.[21,25] The contribution of bacteria to the pathogenesis of pericarditis in many of these cases however was difficult to assess and perhaps understated. This was because affected horses received antibiotics before collection of samples for bacterial culture or historical information regarding antibiotic therapy was either unknown or not indicated.

There is evidence to suggest that the cases of terminal pericarditis from which no microorganisms were isolated also were bacterial in origin. The gross and microscopic alterations in the parietal pericardium and heart of affected individuals were similar in all cases and could be consistent with a bacterial etiology. *Actinobacillus* species isolated in this series of cases were susceptible to a broad range of antibiotics, which have the potential to attain therapeutic levels in the pericardium[28] and may have effectively eliminated infection before necropsy. This assumption is supported by 2 individuals from which actinobacilli were isolated from pericardial and pleural fluids before treatment and were culture negative at necropsy 4 and 5 days later, respectively.

The microorganisms isolated in these and previous cases of equine pericarditis are commensal bacteria of mucosal surfaces in the horse that sporadically produce secondary or opportunistic infections.[8] The isolation of *Actinobacillus* species from the majority of individuals in this study and from an earlier study[6] suggests that this microorganism is pericardiotrophic in the horse. Authors of previous studies have suggested that bacteria have an important but secondary role in the pathogenesis of pericarditis, with bacterial infection being secondary to virus infection or immunosuppression (or both).[25,32] A similar function is attributed to the bacteria isolated in these cases. It is unlikely that primary infections with commensal bacteria could have resulted in the abrupt onset and disappearance and broad geographic distribution of pericarditis cases. Perhaps, impairment of either the innate or acquired immune systems by an agent/toxin associated with the ETC contributed to a temporary breakdown of mucosal defense mechanisms.

The inability to isolate virus from samples submitted from terminal and clinical cases of pericarditis was neither an unexpected finding nor does it exclude previous virus infection as a predisposing factor in bacterial pericarditis in the horse because the inflammatory process in the heart and pericardium had progressed to the chronic stage. It is also likely that at the time of diagnosis the clinical cases were subacute to chronic in duration. Successful isolation of virus from chronic inflammatory processes is more difficult.

The significance of detecting EHV-2 nucleic acid in pericardial and pleural fluid samples from 4 horses is unknown. Equine herpesvirus–2 is a member of the

gammaherpesvirinae subfamily of herpesviruses[29] and is widely distributed in horse populations, as determined by serologic surveys and virus isolation studies.[17,18,24] The virus, either as a primary agent or predisposing factor, has been associated with upper respiratory tract infections and pneumonia in the horse.[3,15,19] The detection of EHV-2 nucleic acid in these horses most likely reflects the broad distribution and biology of the virus in horse populations. Further investigation, however, is needed to determine the significance of EHV-2 virus infection in equine pericarditis.

Microscopic alterations in the pericardium were of severe chronic exudative inflammation and relatively nonspecific from an etiological perspective. Lesions in the subepicardial myocardium were considered an extension of the severe inflammatory reaction occurring on the surface of the epicardium. There were no lesions in either the myocardium or epicardium indicative of either active or previous viral myopericarditis. If previous or concurrent viral infection was a factor in these cases, the agent is likely immunosuppressive or pericardiotrophic (or both), producing minimal morphologic alterations at the cellular level and either not detected by standard diagnostic methods or by currently available molecular tests for equine viral pathogens.

The results of this study suggest that bacterial infection of the equine pericardium occurs more frequently than previously noted and that the breakdown of mucosal barriers may be an important factor in the pathogenesis of this condition. A contributory role of viral infection in the pathogenesis of equine pericarditis was not identified using current diagnostic methods. This indicates that either more sensitive assays are needed for the detection of viral antigen or nucleic acid in cases of equine pericarditis or that previous or current viral infection is not a significant contributing factor in affected horses. To the author's knowledge, this is the first description of an epidemic of equine pericarditis in North America.

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Sources and manufacturers

a. PPLO Agar with inhibitors, Remel, Lenexa, KS.
b. PPLO broth, Remel, Lenexa, KS.
c. Directigen Flu A, Becton Dickinson and Company, Sparks, MD.

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