Journal of Veterinary Internal Medicine
Open Access

Standard Article

J Vet Intern Med 2017;31:894–900

Factors Associated with Survival in 97 Horses with Septic Pleuropneumonia

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Background: Septic pleuropneumonia is a common cause of morbidity and mortality in horses, but there is limited data available regarding factors associated with survival.

Hypothesis/Objectives: To identify factors predictive of survival in horses with septic pleuropneumonia.

Animals: A total of 97 horses with septic pleuropneumonia at 2 referral institutions.

Methods: A retrospective study was performed. A diagnosis of septic pleuropneumonia was based on the presence of sepsis, pleural effusion, and positive bacterial culture from tracheal aspiration (TA) or pleural fluid (PF).

Results: Thirty-one percent of horses had a recent history of travel. Clinical signs included lethargy (78%), tachycardia (75%), tachypnea (60%), fever (43%), prolonged capillary refill time (22%), and ventral edema (14%). The most common clinicopathologic abnormality was hyperfibrinogenemia (79%). Increased serum creatinine concentration at presentation was negatively associated with survival (OR, 5.13; CI, 1.88–14.01; $P = .001$) and return to work (OR, 6.46; CI, 1.10–37.92; $P = .034$). Eighty-four TA and 67 PF samples were submitted for culture, 98 and 84% of which were positive, respectively. The most common isolate was *Streptococcus equi* subsp *zooepidemicus*. Tracheal aspirates were more sensitive than PF for bacterial growth, but some organisms isolated from PF were not isolated from TA. Thoracotomy was positively associated with survival (OR, 0.13; CI, 0.01–0.83; $P = .028$).

Conclusions and Clinical Importance: Increased serum creatinine concentration is a negative prognostic indicator and is likely a reflection of dehydration. Submission of TA and PF is recommended. Thoracotomy should be considered as a treatment for pleuropneumonia.

Key words: Creatinine; Horse; Pleuropneumonia; Thoracotomy.

Pleuropneumonia is not uncommon in horses. Risk factors for development of pleuropneumonia include long-distance transportation, prolonged head elevation, exercise, general anesthesia, and viral respiratory infections that lead to decreased mucociliary clearance and immune suppression.[1–4] Clinical signs of pleuropneumonia include lethargy, tachycardia, tachypnea, pleurodynia, fever, nasal discharge, and anorexia.[1] Many horses with pleuropneumonia present to tertiary care facilities with sepsis, as a possible sequelae to severe bacterial infection is systemic inflammatory response syndrome (SIRS).[5] Following a diagnosis of septic pleuropneumonia, aggressive treatment is necessary to increase the likelihood of complete resolution and return to function.

Abbreviations:

CBC	complete blood count
CRT	capillary refill time
NSAID	nonsteroidal anti-inflammatory drug
PF	pleural fluid
SIRS	systemic inflammatory response syndrome
TA	tracheal aspirate

A diagnosis of pleuropneumonia is achieved through diagnostic imaging, cytological evaluation and microbial culture. Transthoracic ultrasound can be useful in identifying the optimal site for thoracocentesis and can provide valuable information regarding the severity of disease. Location and volume of pleural fluid (PF), recurrence of fluid following thoracentesis, identification of fibrin deposition and pleural adhesions, and pneumothorax can be assessed.[6,7] Samples for cytological evaluation and microbial culture may be obtained by thoracocentesis and/or collection of a tracheal aspirate (TA).[8,9] The most common bacterium isolated from horses with pleuropneumonia is *Streptococcus equi* subsp *zooepidemicus*.[8,10] Other commonly cultured aerobes include other *Streptococcus* sp., *Pasteurella* sp., *Escherichia coli, Klebsiella pneumoniae, Enterobacter* sp., and *Actinobacillus* sp.[8,11] Anaerobic isolates, including *Bacteroides* sp., *Clostridium* sp., *Fusobacterium* sp., and *Peptostreptococcus* sp, are less commonly isolated and have been associated with a poorer prognosis in some studies.[12,13]

Medical treatment of septic pleuropneumonia consists of antimicrobial and anti-inflammatory therapy, pleural fluid drainage, and ancillary care that might include

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This work was performed at Purdue University in West Lafayette, IN, and Hagyard Equine Medical Institute in Lexington, KY. No sources of funding were used for this project. Results were presented at the 2015 American College of Veterinary Internal Medicine Forum, Indianapolis, Indiana

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Submitted July 19, 2016; Revised November 3, 2016; Accepted January 19, 2017.

DOI: 10.1111/jvim.14679

intravenous fluid therapy, oxygen supplementation, and nutritional support. Failure of medical management to achieve complete resolution is often due to persistence of fibrinous adhesions and accumulation of pleural fluid and necrotic lung tissue.[14] Surgical intervention might be indicated in these cases and includes thoracotomy and possible rib resection to facilitate sufficient drainage of infected material.[11,15]

Reported survival rates for horses diagnosed with pleuropneumonia range from 43.3% to 96%.[4,9,13,14,16,17] With respect to return to previous work, 1 study reported that 61% of racehorses treated for pleuropneumonia went on to race at least once, with 56% of those horses winning at least 1 race.[16] A second study showed that 52% of Thoroughbred racehorses returned to racing after treatment of pulmonary abscessation.[18] Complications of pleuropneumonia include IV catheter-associated thrombophlebitis, pleural abscessation, antimicrobial-associated diarrhea, and laminitis.[16,17]

Currently, there is limited data regarding factors associated with survival in horses with pleuropneumonia, particularly among septic cases. Therefore, the objectives of this study were to describe the signalment, historical complaint, physical examination findings, clinicopathological values, diagnostic procedures, treatment, complications, and outcome in horses with septic pleuropneumonia referred to tertiary care facilities, and to identify variables associated with survival. We hypothesized that the presence of anaerobes in TA or PF samples and development of laminitis would be significantly associated with survival.

Materials and Methods

Data Collection

Medical records from the Purdue University College of Veterinary Medicine (PU-CVM) and Hagyard Equine Medical Institute (HEMI) were reviewed over a 13-year period (January 2001 to December 2014) to identify horses diagnosed with septic pleuropneumonia. Inclusion criteria included the presence of unilateral or bilateral pleural effusion as determined with transthoracic ultrasonography, in addition to a diagnosis of sepsis. Sepsis was defined as the presence of SIRS and a positive bacterial culture from a TA or PF.[19] The diagnosis of SIRS was based on the presence of 2 or more abnormalities including hyperthermia (>101.5°F) or hypothermia (<97°F), tachycardia (>45 beats/min), tachypnea (>24 breaths/min), leukopenia (WBC < 6,000/μL), leukocytosis (WBC > 12,000/μL), or >10% band neutrophils.[19]

Data collected included signalment, pertinent history, physical examination findings and routine blood work results upon presentation, diagnostic tests performed, bacterial organisms cultured (from TA and PF), the presence or absence of laminitis, in-hospital treatment, and outcome. Only 1 TA and/or PF sample was collected per horse at admission. A successful outcome was defined as survival to hospital discharge. Return to previous work was also recorded for applicable cases.

Data Analysis

Horses were grouped by outcome (survived to hospital discharge or euthanized/died prior to discharge). A Shapiro-Wilk test was used to test for normality, and differences in nonparametric continuous data were assessed by the Wilcoxon rank-sum test. Categorical data were compared with a chi-square or Fisher's exact test. The association of clinical signs, clinicopathologic factors, and treatments with outcome was analyzed initially with logistic regression in a univariate fashion. Variables screened in univariate and retained with $P < .15$ were assessed via multivariate analysis using forward stepwise logistic regression, and statistical significance was set at $P < .05$. Statistical analysis was performed using commercially available statistical software.[a]

Results

Signalment/History

Ninety-seven horses met the inclusion criteria: 40 from PU-CVM and 57 from HEMI. Horses ranged from 2 months to 29 years of age with a median age of 2.5 years. Forty-six horses were female and 48 were male (26 geldings and 22 stallions). Gender was not indicated in the medical records of 3 horses. Breeds included Thoroughbreds (n = 58), Standardbreds (n = 17), Quarter Horses (n = 9), Arabians (n = 3), and 1 each of 9 other breeds. Breed was not indicated in the medical record of 1 horse.

Thirty-one percent of horses (30/97) had a history of recent travel (within 6 weeks of presentation); the median duration of travel was 14 hours and occurred a median of 14 days prior to presentation (range: 1 to 45 days). The most common historical complaints included fever (81 horses; 84%), lethargy (76 horses; 78%), inappetance (75 horses; 77%), increased respiratory effort (64 horses; 66%), nasal discharge (60 horses; 63%), exercise intolerance (33 horses; 36%), weight loss (18 horses; 19%), and colic (13 horses; 13%). Prior to presentation, 79 horses (83%) received a nonsteroidal anti-inflammatory drug (NSAID), 67 (71%) received an antimicrobial drug, and 8 (8%) received corticosteroids. Prior medications were not associated with survival ($P > .168$).

Clinical Signs

The most common clinical signs identified during initial presentation included lethargy (76 horses; 78%), tachycardia (73 horses; 75%), tachypnea (58 horses; 60%), fever (42 horses; 43%), prolonged capillary refill time (CRT, 19 horses; 22%), and ventral edema (13 horses; 14%). One horse was presented with lameness at the walk and was diagnosed with laminitis. The median duration of clinical signs prior to presentation was 6 days (range: 12 hours to 90 days). Eight horses (8%) were diagnosed with laminitis, either on presentation (1) or during hospitalization (7).

Clinicopathologic Values

A complete blood count (CBC), plasma fibrinogen concentration, and serum biochemical profile were available for 91 horses. Hyperfibrinogenemia was identified in 79% of cases. The most common CBC abnormalities included band neutrophilia (60%), mature neutrophilia (53%), neutropenia (22%), and increased

hematocrit (18%). The most common serum biochemical abnormalities included hypoalbuminemia (70%), hyponatremia (62%), hyperbilirubinemia (56%), hyperglycemia (43%), hyperglobulinemia (41%), hypochloremia (40%), and elevated serum creatinine concentration (14%) Blood lactate concentration on admission was available for 14 horses, 6 (43%) of which had hyperlactatemia (>2.0 mmol/L).

Culture Results

Eighty-four TA samples and 68 PF samples were submitted for culture, 98% and 84% of which were positive, respectively. TA samples and PF samples were submitted in 55 cases, with both yielding positive results in 39 (71%). Of these, the same organisms were isolated from both TA samples and PF samples in 20% of submissions. The most common aerobic organisms cultured from TAs were *Streptococcus equi* subsp *zooepidemicus* (84%), *Actinobacillus* sp. (33%), *E. coli* (28%), *Staphylococcus* sp. (15%), *Klebsiella* sp. (13%), and *Pseudomonas* sp. (10%). The most common aerobic organisms cultured from PF were *Streptococcus equi* subsp *zooepidemicus* (67%), *Actinobacillus* sp. (16%), *E. coli* (11%), and *Staphylococcus* sp. (7%). Twenty-one of 84 TA samples (25%) were submitted for anaerobic culture, 8 of which (38%) were positive. Thirty-four of 67 PF samples (51%) were submitted for anaerobic culture, 10 of which (29%) were positive. The presence of anaerobes was not associated with survival ($P = .664$).

Treatment

Ninety-four of 97 horses were treated. Of the untreated horses, 2 were euthanized and 1 died upon presentation. All treated horses received antimicrobial treatment during hospitalization. A combination of penicillin and gentamicin were used in 77 horses (81%). Other antimicrobials used were metronidazole (65 horses; 68%) and enrofloxacin (10 horses; 11%). In 30 horses (32%), the antimicrobials were switched in-hospital due to poor response to initial treatment. Ninety-three horses (99%) received NSAID therapy, which was reported as flunixin meglumine in all cases. Intravenous isotonic crystalloid fluids were administered to 70 horses (74%). Other treatments included omeprazole (67 horses; 72%), pentoxifylline (18 horses; 19%), heparin (14 horses; 15%), and intranasal oxygen (13 horses; 14%).

Of the 94 horses that were treated, digital hypothermia of all 4 feet was used immediately upon presentation in 1 horse that was presented with laminitis in all 4 feet. Front-limb prophylactic digital hypothermia was used in 23 (24% of treated horses), 4 (17%) of which developed laminitis (3 with front feet affected and 1 with all 4 feet affected). Digital hypothermia was performed continuously with 5-L IV fluid bags filled with crushed ice q 1 to 2 hours to the level of the pastern. Of the 94 horses that were treated, 12 (13%) were placed on foot pads, 2 (17%) of which developed laminitis; it was unclear in these 2 cases whether foot pads were used prophylactically or as a treatment for laminitis. One horse with no digital treatment developed laminitis in both front feet.

Thoracocentesis was performed in 71 of 94 horses (75.5%) within 24 hours of admission, 60% of which were bilateral and 33% of which were unilateral. Of the 42 horses with the volume of PF recorded, the median volume from thoracocentesis from the right and left hemithoraces was 6 (range 0–96) and 6 (range 0–30) liters, respectively. Fluid volume was not associated with survival ($P > .117$). In 5 cases (7%), the site of thoracocentesis was not recorded. At least 1 indwelling thoracostomy tube was left in place to facilitate pleural drainage for at least 24 hours in 61 cases (87%).

Eleven horses had a thoracotomy performed, which was positively associated with survival to discharge ($P = .030$) whether thoracotomy was unilateral (8 cases) or bilateral (2 cases). The site of thoracotomy was not recorded in 1 horse. Indications for thoracotomy included visualization of a well-defined abscess on transthoracic ultrasound or failure of thoracostomy tube drainage. Pneumothorax was not observed in any horse. All 11 horses that had a thoracotomy survived to hospital discharge; 2 were euthanized within 2 months of hospital discharge. The median duration of hospitalization from initial presentation to thoracotomy was 9 days (range: 1–40 days), and the median time from the first clinical sign to thoracotomy was 21 days (range: 13–120 days). A rib resection was performed in 8 horses, all of which survived to hospital discharge. Two horses were alive several months after discharge, 1 had been euthanized, and 5 were lost to follow-up.

Outcome

Sixty-five horses (67%) survived to discharge, and the median duration of hospitalization was 6 days (range: 0–107 days). Nonsurvivors included the 3 horses that were not treated. Of the 8 horses diagnosed with laminitis, 5 (62.5%) survived to discharge There was no association with laminitis and survival ($P = .78$). Thrombophlebitis was diagnosed in 8 horses (8%), and antimicrobial-associated diarrhea was diagnosed in 1 horse (1%); these events were not associated with survival ($P > .62$). Variables associated with increased or decreased odds of death from univariate analysis are presented in Table 1. In multivariate analysis of presentation and treatment variables, only serum creatinine concentration and thoracotomy were significantly associated with survival/death. Serum creatinine concentration was associated with increased odds of death (OR, 5.13; 95% CI, 1.88–14.01; $P = .001$) whereas thoracotomy decreased odds of death (OR, 0.13; 95% CI, 0.01–0.83; $P = .028$). For every 1.0 mg/dL increase in serum creatinine concentration, the risk of death increased by 5-fold. Of the 53 horses for whom follow-up data were available, 4 (8%) were euthanized due to unresolved pleuropneumonia within 2 months of discharge. Thirty-five horses (66%) returned to previous work. Of the 35 horses that returned to work, 23

Table 1. Variables for serum biochemical analysis, complete blood count, and treatment modalities that were significantly ($P < .05$) associated, in univariate analysis, with death in horses with septic pleuropneumonia. Horses were categorized as survivors (discharged alive, n = 65) or nonsurvivors (euthanized or died, n = 32).

Variable[a]	Odds ratio (95% CI) for risk of death	*P*-value
Blood urea nitrogen (mg/dL)	1.06 (0.02–1.16)	.007
Creatinine (mg/dL)	5.13 (1.88–14.01)	.001
Sodium (mmol/L)	0.89 (0.81–0.98)	.016
Chloride (mmol/L)	0.84 (0.76–0.92)	<.001
Total bilirubin (mg/dL)	1.67 (1.26–2.22)	<.001
Hematocrit (volume %)	1.10 (1.04–1.15)	.001
Hemoglobin (g/dL)	1.30 (1.11–1.52)	.001
Red blood cells (M/μL)	1.43 (1.16–1.77)	.001
Band neuts (K/μL)	3.30 (1.23–8.89)	.018
Thoracotomy	0.13 (0.01–0.83)	.028
IV fluids	4.60 (1.26–16.87)	.021
O_2 therapy	4.29 (1.27–14.53)	.019

[a]Other evaluated variables were as follows: glucose, phosphorus, calcium, potassium, anion gap, total CO2, total protein, albumin, globulin, AST, GGT, creatine kinase, lactate, WBC, segmented neutrophils, lymphocytes, monocytes, eosinophils, fibrinogen, NSAID administration, bronchodilator, heparin, pentoxifylline, gentamicin, amikacin, ceftiofur, enrofloxacin, oxytetracycline.

returned to racing (1 Standardbred, 22 Thoroughbreds), 5 returned to showing (3 Quarter Horses, 1 Thoroughbred cross and 1 Saddlebred), 1 Thoroughbred racehorse became a show horse, 1 Quarter Horse show horse became a trail/pleasure horse, 1 Thoroughbred broodmare resumed breeding, and information was unclear in 4 cases. Racehorses that became broodmares (3) following recovery from pleuropneumonia were considered not to have returned to work. Increased serum creatinine concentration at presentation was negatively associated with return to work (OR, 6.46; CI, 1.10–37.92; $P = .034$).

A comparison of CBC, plasma fibrinogen concentration, and serum biochemical analysis data between survivors and nonsurvivors is presented in Table 2 (CBC, plasma fibrinogen concentration) and Table 3 (serum biochemical analysis).

Discussion

The main findings of the study reported here were that increased serum creatinine concentration on presentation was negatively associated with survival and return to work, and in a few cases, thoracotomy was positively associated with survival. Decreased fluid intake and increased fluid loss into the pleural cavity likely led to a prerenal reduction in glomerular filtration rate in the horses described in this study. An association between prerenal azotemia and a poor outcome has been previously identified in horses, and may have been a reflection of hypovolemia.[20] Acute kidney injury secondary to ischemia could not be ruled out, especially given the absence of hyperlactatemia in the majority of cases with an available blood lactate concentration on admission. Our results indicate that judicious administration of NSAIDs and nephrotoxic antimicrobials such as aminoglycosides and tetracyclines might be important in horses with pleuropneumonia given the potential for prerenal or renal azotemia. In this study, however, nonsurvival in horses with increased creatinine concentration was likely a reflection of disease severity rather than a result of renal failure. Facilitation of pleural drainage by thoracotomy may be an effective therapy that allows removal of purulent debris, fibrin, and necrotic tissue when a thoracostomy tube has failed.[21,22] Previous studies have demonstrated that patient selection is critical for a successful outcome and that a thoracotomy should only be performed in horses with large, persistent pockets of thick fibrin and debris.[15] It is likely that financial commitment from owners played a role in patient selection, but this information was lacking in the medical records, highlighting a limitation of the retrospective nature of the study. The most common complication associated with thoracotomy is

Table 2. Complete blood count and plasma fibrinogen concentration data from horses with septic pleuropneumonia. Horses were categorized as survivors (discharged alive, n = 65) or nonsurvivors (euthanized or died, n = 32). The number of horses for which data are available is indicated for survivors and nonsurvivors (N), and data are expressed as median and range.

	Survivors		Nonsurvivors	
Variable	N	Median (range)	N	Median (range)
Hematocrit (volume %)	64	36.25 (24.7–63)	31	45.8 (27.6–67.3)
Hemoglobin (g/dL)	63	12.7 (8.8–20.8)	30	15.35 (9.5–24.2)
Red blood cells (M/μL)	64	8.36 (5.45–13.9)	30	10.35 (6.35–15.9)
White blood cells (K/μL)	64	12.2 (1.63–33.1)	30	10.7 (2.33–48.7)
Segmented neuts (K/μL)	57	8.6 (0.16–27.1)	28	5.485 (0–44.8)
Band neuts (K/μL)	57	0.11 (0–1.45)	27	0.28 (0–1.93)
Lymphocytes (K/μL)	56	1.9 (0.2–5.2)	28	2.325 (0–7.2)
Monocytes (K/μL)	56	0.285 (0–2.09)	28	0.35 (0–1.7)
Eosinophils (K/μL)	56	0 (0–0.2)	28	0 (0–0)
Plasma fibrinogen concentration (mg/dL)	60	535 (200–1,400)	29	453 (100–1,200)

Table 3. Serum biochemical analysis data from horses with septic pleuropneumonia. Horses were categorized as survivors (discharged alive, n = 65) or nonsurvivors (euthanized or died, n = 32). The number of horses for which data are available is indicated for survivors and nonsurvivors (N), and data are expressed as median and range.

	Survivors		Nonsurvivors	
Variable	N	Median (range)	N	Median (range)
Glucose (mg/dL)	59	114 (31–253)	29	124 (70–187)
Blood urea nitrogen (mg/dL)	58	13 (0–65)	29	21 (0–54)
Creatinine (mg/dL)	61	1.2 (0.7–4.6)	30	1.55 (1.1–4.9)
Phosphorus (mg/dL)	58	3.75 (1.1–11.1)	29	5 (1.4–9.3)
Calcium (mg/dL)	59	10.8 (7.5–12)	29	10.6 (6.2–11.8)
Sodium (mmol/L)	61	131 (118–141)	30	129.5 (117–137)
Potassium (mmol/L)	61	3.7 (2.6–6)	30	3.75 (2.4–5.6)
Chloride (mmol/L)	61	98 (84–106)	30	92.5 (81–102)
Anion gap (mmol/L)	58	10 (2–18.9)	29	12 (4–26.6)
Total CO_2 (mmol/L)	60	25 (14–30)	30	25 (16–33)
Total protein (g/dL)	61	6.5 (3–12.3)	31	6.3 (4.8–10.5)
Albumin (g/dL)	59	2.7 (1.4–3.7)	29	2.6 (2–3.6)
Globulin (g/dL)	59	3.9 (1.5–10.1)	29	3.6 (2.1–6.7)
AST (IU/L)	59	179 (74–741)	29	243 (100–1,117)
Creatine kinase (IU/L)	57	81 (21–1,524)	29	78 (31–720)
GGT (IU/L)	59	19 (5–110)	29	19 (8–59)
Total bilirubin (mg/dL)	59	2.5 (0.4–6.6)	29	4.6 (0.8–14)
Lactate concentration (mmol/L)	8	1.55 (0.9–3)	6	2.35 (0.8–3.3)

pneumothorax; however, a thoracic drain can be placed near the thoracotomy site to ensure that the incomplete mediastinum is sealed by the fibrinous material prior to performing the thoracotomy.[21,23] Timing of thoracotomy has been considered an important factor in reducing the likelihood of pneumothorax, as fibrinous adhesions and mature abscess formation that prevent room air from entering the pleural space are typically present in the chronic phase of disease.[9] In the current study, timing of thoracotomy did not affect the outcome and no horses developed pneumothorax. This might have been due to the use of transthoracic ultrasound to determine the character of pleural fluid, the presence of fibrinous adhesions, and the presence of mature abscesses.[24,25] All horses that underwent thoracotomy in this study survived to hospital discharge, which is similar to a previous study that reported a survival rate of 88%.[15]

As the predominant breed presented to HEMI is Thoroughbreds, this likely explains the overrepresentation of Thoroughbreds in this study. Clinicopathologic abnormalities were consistent with previous reports of pleuropneumonia in horses.[9,11] Hyperfibrinogenemia was the most common abnormality identified in this study and in a study of 43 horses with pleuropneumonia.[9] Fibrinogen is an acute-phase protein that increases in response to inflammatory stimuli. Neutropenia, band neutrophilia, and mature neutrophilia reflect various stages of inflammation and neutrophil demand. Increased hematocrit was likely a reflection of dehydration or splenic contraction. Hypoalbuminemia, hyponatremia, and hypochloremia likely reflected loss into the pleural fluid, while hyperbilirubinemia was likely due to anorexia. Hyperglycemia commonly occurs secondary to stress, and hyperglobulinemia was likely due to antigenic stimulation.

The organisms cultured in this study were consistent with those of previous studies.[8,9,12,13] Colonization of the lower respiratory tract with normal oropharyngeal flora can occur when various risk factors lead to suppression of the immune system and/or impairment of mucociliary bacterial clearance. Opportunistic bacteria can also gain access to the lungs when pulmonary defense mechanisms are compromised or when an overwhelming bacterial load is present. We did not find an association with the presence of anaerobic bacteria and a higher incidence of nonsurvival, which is consistent with a previous study.[9] However, only approximately 50% of the samples in the current study were submitted for anaerobic culture, as HEMI does not routinely submit samples for anaerobic culture. Therefore, there was insufficient data to determine whether the cases in which anaerobes were isolated had a worse prognosis.

We found that TA was more sensitive than PF in yielding positive bacterial growth, which is possibly due to a dilution effect in PF. This is consistent with a previous report that found that 100% of TA were positive for bacterial growth while only 57% of PF samples were positive for growth.[8] Of the 39 horses from which both TA and PF samples were positive for bacterial growth in this study, the exact same isolates were identified in only 20% of cases. Moreover, isolates from 11 PF samples were not identified in corresponding TA samples. This might be due to growth of bacteria within newly formed pleural abscesses. Taken together, our data suggest that both TA and PF samples should be submitted for bacterial culture because PF does not always yield bacterial growth, and because isolates cultured from airways might differ from those cultured from PF. Anaerobic submissions were limited in this study, but TAs were slightly more sensitive than PF in yielding anaerobic growth (38% versus 29%).

Antibiotics did not vary significantly between institutions. Penicillin and gentamicin in combination were the most common antimicrobials used throughout the study period, indicating that significant antimicrobial resistance has not developed. Given that some reports show a poorer prognosis, if anaerobic bacteria are involved in the pathophysiology of pleuropneumonia, metronidazole was commonly administered, even in cases where anaerobic culture of TA or PF was not performed.[12,13] Flunixin meglumine was used in almost all cases to address SIRS and to prevent fulminant laminitis. No therapy was associated with outcome.

Only 8 horses (8%) in this study developed laminitis during hospitalization. There was no association with laminitis and survival or return to work. Of the 8 horses with laminitis, 63% survived. Although this is similar to the overall survival rate, there were too few cases to draw conclusions regarding the risk of death due to laminitis. Similarly, there were not enough horses with laminitis to allow recommendation of prophylactic digital hypothermia or the use of foot pads. However, since laminitis developed in only a relatively small percentage of horses treated with prophylactic digital hypothermia, it is likely that this treatment contributed to the overall low incidence of laminitis in this study. Rapid recognition of disease, treatment by the referring veterinarian, and prompt referral were also factors that likely led to a low incidence of laminitis.

There are significant limitations to this retrospective study. Details such as duration of thoracostomy tube placement and exact timing of TA and PF sampling were often missing in the medical records. Some clinicopathological values such as blood lactate concentration and blood gas analyses are often performed but not transferred to medical records, which make it difficult to interpret the relatively few cases with such available data. Also, it was difficult to ascertain whether foot pads were placed prophylactically or as a treatment for laminitis, and the exact timing of ice boot filling was not standardized across cases or institutions. Long-term outcome was difficult to assess due to horses that were lost to follow-up. Finally, it is difficult to capture financial considerations of owners using a retrospective approach.

In conclusion, severity of dehydration as reflected by increased serum creatinine concentration was predictive of nonsurvival in horses with septic pleuropneumonia. To increase sensitivity of microbial culture, submission of both TA and PF is recommended. Thoracotomy should be considered as a treatment for pleuropneumonia in cases that are nonresponsive to conventional therapy, as this procedure was associated with a positive outcome.

Footnote

[a] SAS 9.2; SAS Inc, Cary, NC

Acknowledgment

Conflict of Interest Declaration: George E. Moore serves as consulting editor for Experimental Design and Statistics for the Journal of Veterinary Internal Medicine. He was not involved in review of this manuscript.

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

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