J. Vet. Sci. (2004), **5**(1), 75–77

JOURNAL OF Veterinary Science

Case Report

A hematogenic pleuropneumonia caused by postoperative septic thrombophlebitis in a Thoroughbred gelding

Seung-ho Ryu, Joon-gyu Kim, Ung-bok Bak, Chang-woo Lee[1,*] and Yonghoon Lyon Lee[2]

Equine Hospital, Korea Racing Association, Kwachon 427-070, Korea
[1]Department of Clinical Pathology, College of Veterinary Medicine, Seoul National University, Seoul 151-742, Korea
[2]Department of Anesthesia, Pain Management and Perioperative Medicine, Boren Veterinary Medical Teaching Hospital and College of Veterinary Medicine, Oklahoma State University, Stillwater, OK 74074, USA

A 7-year-old Thoroughbred gelding was admitted to Equine Hospital, Korea Racing Association for evaluation and treatment of colic. Based on the size and duration of the large colonic and cecal impaction, a routine ventral midline celiotomy and large colon enterotomy were performed to relieve the impaction. Six days following surgery the gelding exhibited signs of lethargy, fever, inappetence and diarrhea. Eleven days following surgery, the jugular veins showed a marked thrombophlebitis. On the sixteenth day of hospitalization the gelding died suddenly. Upon physical examination, the horse was febrile, tachycardic and tachypnoeic. Thoracic excursion appeared to be increased; however, no abnormal lung sounds were detected. No cough or nasal discharge was present. Hematology revealed neutrophilic leukocytosis. Serum biochemistry was normal but plasma fibrinogen increased. In necropsy, fibrinopurulent fluid was present in the thoracic cavity. There were firm adhesions between visceral pleura and thoracic wall. White, mixed and red thrombi were formed in both jugular veins from the insertion point of IV catheter. Histopathological examination showed fibrinopurulent inflammation and vascular thrombosis in the lung. The pleura showed edematous thickening and severe congestion. The clinicopathological and pathological findings suggest that septic thrombi associated with septic thrombophlebitis metastasized into the pulmonary circulation and were entrapped in the pulmonary parenchyma and provoked pleuropneumonia.

Key words: pleuropneumonia, postoperative, septic thrombophlebitis, horse

*Corresponding author
Phone: 82-2-880-1273; Fax: 82-2-880-8662
E-mail: anilover@snu.ac.kr

Pleuropneumonia is a clinically important equine disease, predisposed by a number of identifiable factors. The majority of acute pleuropneumonia occurred in Thoroughbreds (89%). Among pleuropneumonic horses, 61% were in race training at the onset of illness, 31% had been recently transported a long distance and 11% had evidence of exercise induced pulmonary hemorrhage [2].

Viral respiratory tract disease or exposure to horses with respiratory tract disease were determined to be risk factors for the development of pleuropneumonia [1].

Acute disease is associated with the isolation of facultatively anaerobic organisms, especially *beta-haemolytic Streptococcus spp*., *Pasteurella/Actinobacillus spp*., *Bacteroides oralis* and *Bacteroides melaninogenicus* [10,11,15]. Putrid odor was associated with the pleural fluid and/or breath in 62% of the horses from which anaerobes were isolated. In these horses, the survival rate was significantly less than for horses from which odoriferous specimens were not isolated [15].

Horses with chronic pleuropneumonia had a history of lethargy and inappetence for longer than two weeks. *Actinobacillus equuli* was isolated, either alone or in combination with other bacteria, from thoracic fluid [2].

Primary infection was 24%, whereas 76% was secondary to another disease process (inhalation of food or saliva, thoracic trauma, generalized infection, airway disease, neoplasia or thromboembolism). Of the horses with primary pulmonary infections, 91% appeared to be associated with a previous episode of stress; this took the form of long distance travel in 73% [7]. Epidemiological studies suggest that other factors including the immune state of the equine population influence the distribution and severity of respiratory disease [8]. Most reports of pleuropneumonia are bronchogenic.

The chronic nature and cost of ongoing treatment and limitations on choice of antimicrobial agents warrant a poor prognosis for survival and a poorer prognosis for return to athletic endeavour [10].

The clinical features and progression of an unusal hematogenic pleuropneumonia induced by postoperative septic thrombophlebitis in a Thoroughbred gelding are described here.

Case history: A 7-year-old Thoroughbred gelding was admitted to Equine Hospital, Korea Racing Association for evaluation and treatment of colic which had lasted 3 days duration. Rectal palpation identified an impaction of the large colon and cecum. Based on the size and duration of the impaction, abdominal surgery was recommended. A routine ventral midline celiotomy and large colon enterotomy were performed to relieve the impaction. No other lesions were noted on thorough exploration of the remainder of the intestinal tract and abdomen. Routine postoperative therapy was instituted.

Six days following surgery the gelding exhibited signs of lethargy, fever, inappetence and diarrhea. Eleven days following surgery, the jugular veins showed a marked thrombophlebitis. On the sixteenth day of hospitalization the gelding developed a sudden death.

Clinical examination: During the course of the treatment, the horse was often febrile (39.6°C), tachycardic (72 beats/min) and tachypnoeic (52 breaths/min). Thoracic excursion appeared to be increased. Lung sounds were quieter than normal in all lung fields considering the character of rapid and deep breathing; however, no abnormal sounds were detected. No cough or nasal discharge was present.

Clinical pathology: Hematology revealed neutrophilic leukocytosis. Preoperative number of neutrophil was 2,800/μl but increased to 28300/μl on Day 7 following surgery. Serum biochemistry was normal but plasma fibrinogen increased from preoperative 400mg/dl to 800/dl on Day 7 following surgery.

Pathological findings: A complete gross and histological examination was performed. There was fibrinopurulent fluid in the thoracic cavity. There were firm adhesions between visceral pleura and thoracic wall (Fig. 1). The lungs were firmer and dark red. The trachea was clean. White, mixed and red thrombi were formed in both jugular veins around the insertion point of IV catheter (Fig. 2). Both kidneys were enlarged in size (25 cm × 15 cm). The remainder of the gross necropsy was unremarkable.

Histopathological examination showed fibrinopurulent inflammation and vascular thrombosis in the lung. Mucus and purulent debris in bronchioles and bronchi were not seen. The pleura showed fibrionous thickening (Fig. 3). Congestion of alveolar walls, inflammatory cell accumulation (bronchiolar lymphadenopathy) (Fig. 4) and septic thrombi in both jugular veins were seen.

Although pleuropneumonia can occur spontaneously, it is often associated with a stressful event such as transportation, recent illness from viral disease or recumbency under general anesthesia [14]. Catheter related damage to the intima, chemical damage by irritating medications (hyperosmotic solutions, phenylbutazone and guaifenesin), reduced host resistance and bacterial infection attributable to the underlying illness were thought to be possible causes of the thrombophlebitis [9,3,5,4].

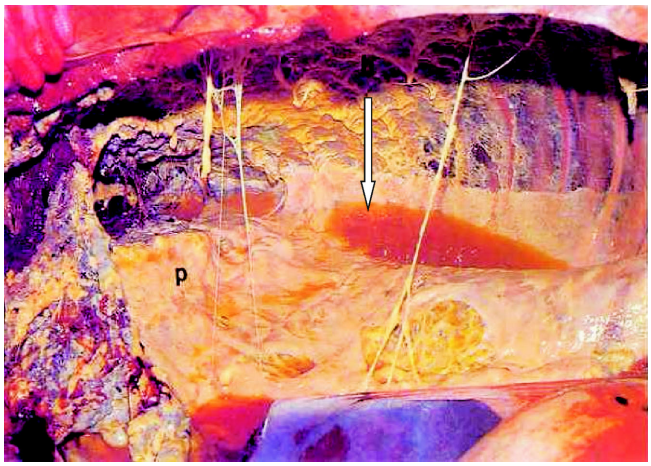

Fig. 1. Fibrinopurulent fluid (h) in the thoracic cavity and firm adhesions between visceral pleura (p) and thoracic wall are shown.

Fig. 2. White, mixed and red thrombi were formed in both jugular veins around the insertion point of IV catheter.

Because of the lack of evidence for a bacterial contamination, the cause of pleuropneumonia in this case remains unclear. However, clinical signs of the current case including swelling, vascular occlusion, fever, pain and clinicopathological changes including neutrophilic leukocytosis and elevation in plasma fibrinogen concentration strongly suggest a bacterial contamination.

It was suggested that leukocytes play a primary role in the initiation of vein thrombosis [12]. This is consistent with the findings in the gelding of this report having white, mixed and red thrombi in both jugular veins around the insertion point of the IV catheter.

The clinicopathological and pathological findings suggest that septic thrombi associated with septic thrombophlebitis metastasized into the pulmonary circulation, were entrapped

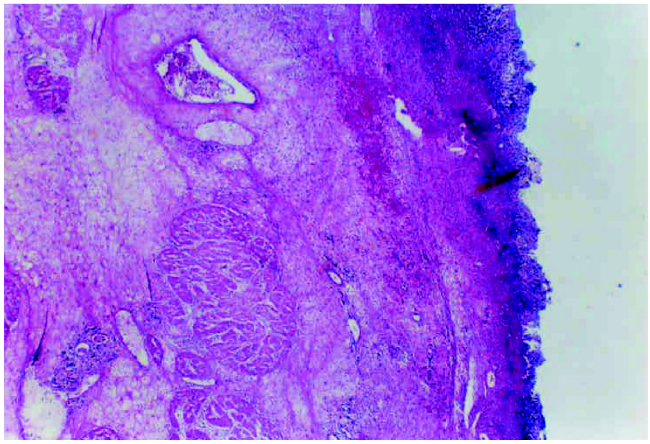

Fig. 3. Fibrinous thickening of pleura is shown.

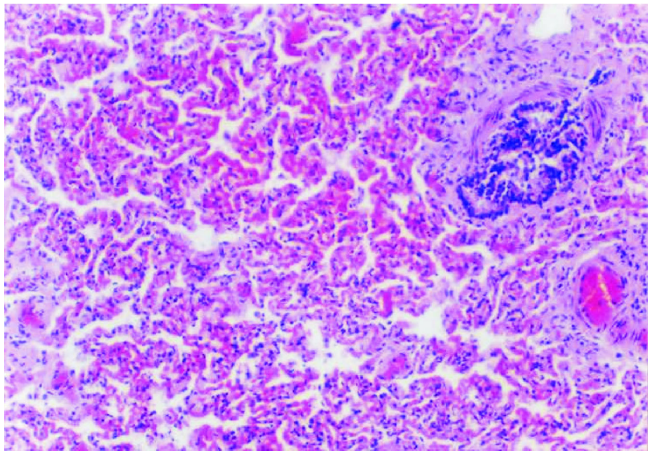

Fig. 4. Congestion of alveolar walls and inflammatory cells accumulation (bronchiolar lymphadenopathy) are shown.

in the pulmonary parenchyma and provoked acute pleuropneumonia. Attention should be paid to thrombophlebitis in placing the catheter and technique of catheter maintenance during long term treatment. Heparin significantly reduced the incidence of thrombosis, the washout of catheter with heparin being more effective than systemic heparin [6]. The duration of catheterization can be increased to 14 days or longer with minimal complications by using catheters made of materials (especially silastic) that are less stiff or rigid [13]. The current case highlights once septic thrombophlebitis develops, monitoring and prevention of respiratory diseases should be carried out to minimize an untoward clinical outcome.

References

1. **Austin SM, Foreman JH, Hungerford LL.** Case-control study of risk factors for development of pleuropneumonia in horses. J Am Vet Med Assoc 1995, **207**, 325-328.
2. **Collins MB, Hodgson DR, Hutchins DR.** Pleural effusion associated with acute and chronic pleuropneumonia and pleuritis secondary to thoracic wounds in horses: 43 cases (1982-1992). J Am Vet Med Assoc 1994, **205**, 1753-1758.
3. **Dickson LR, Badcoe LM, Burbidge H, Kannegieter NJ.** Jugular thrombophlebitis resulting from an anaesthetic induction technique in the horse. Equine Vet J 1990, **22**, 177-179.
4. **Herschl MA, Trim CM, Mahaffey EA.** Effects of 5% and 10% guaifenesin infusion on equine vascular endothelium. Vet Surg 1992, **21**, 494-497.
5. **Mackay RJ, French TW, Nguyen HT, Mayhew IG.** Effects of large doses of phenylbutazone administration to horses. Am J vet Res 1983, **44**, 774-780.
6. **Maffei FH, Rollo HA, Fabris VE.** Prevention of experimental venous thrombosis induced by contrast medium in the rat. Acta Radiol 1980, **21**, 249-252.
7. **Mair TS, Lane JG.** Pneumonia, lung abscesses and pleuritis in adult horses: a review of 51 cases. Equine Vet J 1989, **21**, 175-180.
8. **Powell DG.** Equine infectious respiratory disease. Vet Rec 1975, **96**, 30-34.
9. **Pusterla N, Braun U.** Ultrasonographic evaluation of the jugular vein of cows with catheter-related thrombophlebitis. Vet Rec 1995, **137**, 431-434.
10. **Raidal SL.** Equine pleuropneumonia. Br Vet J 1995, **151**, 233-262.
11. **Raidal SL, Love DN, Bailey GD.** Inflammation and increased numbers of bacteria in the lower respiratory tract of horses within 6 to 12 hours of confinement with the head elevated. Aust Vet J 1995, **72**, 45-50.
12. **Schaub RG, simmons CA, Koets MH, Romano PJ 2d., Stewart GJ.** Early events in the formation of a venous thrombus following local trauma and stasis. Lab Invest 1984, **51**, 218-224.
13. **Spurlock SL, Spurlock GH, Parker G, Ward MV.** Long-term jugular vein catheterization in horses. J Am Vet Med Assoc 1990, **196**, 425-430.
14. **Sweeney CR.** Pleuropneumonia. In: Large Animal Internal Medicine. p 516. C, V, Mosby Company, St. Louis, 1990
15. **Sweeney CR, Divers TJ, Benson CE.** Anaerobic bacteria in 21 horses with pleuropneumonia. J Am Vet Med Assoc 1985, **187**, 721-724.