Case Report

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

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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 provoked pleuropneumonia.

Key words: pleuropneumonia, postoperative, septic thrombophlebitis, horse

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 unusual 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 examination 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 fibrinous 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].

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...