Case 8

Peritoneal fluid from a foal

Contributors

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Cytological Description

Macroscopically, the peritoneal fluid was yellow and cloudy. It was classified as an exudate based on the elevated total nucleated cell count and total protein concentration. The sample contained a high number of inflammatory cells and few red blood cells. A slight pink background containing numerous variably-sized clear vacuoles was also observed (figure 1). The inflammatory cell population was composed of slightly to moderately degenerate neutrophils (68%), vacuolated macrophages (30%) and small lymphocytes (2%). Occasionally, large rods and pleomorphic cocci were observed within neutrophils and rarely within macrophages (figures 2 and 3). Well demarcated and variably-sized clear vacuoles were also observed in the cytoplasm of numerous neutrophils and macrophages (figure 2). These vacuoles look similar to those observed in the background and were suspected to contain lipids.

Diagnosis

The results of the cytology were consistent with severe septic peritonitis. A gastric or intestinal perforation was suspected based on the possibility of the presence of lipid of milk

origin within the effusion. Presence of a pleomorphic bacterial population was also in favor of this hypothesis.

An Oil Red O stain was performed and resulted in a red-orange coloration of both the intracytoplasmic and extracellular vacuoles (Figures 4 and 5), demonstrating their lipid nature.

Clinical outcome

The suspicion of gastric perforation was confirmed by gastroscopy and the foal was euthanized due to the poor prognosis. Necropsy revealed 2 liters of a turbid abdominal fluid containing fibrin and milk clots. Two perforated ulcers, respectively of 1.5cm and 0.5 cm in diameter were found, located along the small curvature of the stomach, at the junction between glandular and non glandular mucosae. Histopathologic examinations of skeletal muscles were compatible with the diagnostic of nutritional myopathy due to selenium deficiency.

It was concluded that clear vacuoles observed on cytology were secondary to the presence of milk within the abdomen.

Discussion

Gastric ulcers of the non-glandular mucosa are frequent in foals less than one month of age and are most commonly not associated with clinical signs. [1,2] Clinically active ulcers in neonatal foals affecting the gastric mucosa are believed to be secondary to an underlying problem. [2] In these cases, the only clinical signs present may be depression or partial anorexia until perforation occurs.

The most specific and sensitive method for diagnosing gastric ulcers is visualization by endoscopic examination [1] as it has been done in this case. However cytology of peritoneal fluid may help to confirm perforation by revealing a septic peritonitis. In this particular case, the presence of an abundant quantity of vacuoles containing lipids that was

compatible with milk was another argument in favor of gastric perforation. To our knowledge, cytology of peritonitis with presence of milk in a foal has never been described before.

The Oil Red O staining is a simple, inexpensive, and useful stain for demonstrating the presence of lipids in cytologic samples. In the dog, it can be used to assist the diagnosis of liposarcoma by confirming that cytoplasmic vacuoles have lipid content. [3] Our case indicates that Oil Red O stain can be used when the presence of milk is suspected within the abdomen in a newborn or young animal with peritonitis due to intestinal perforation.

Acidic gastric pH, which appears to be one of the causative agents of gastric ulcers in adult horses, does not seem to play a major role in critically neonatal foals, in which gastric ulcers are more probably linked to mucosal ischemia and reperfusion injury. [1] Thus, prophylactic treatment of critically ill neonates for gastric ulcers (such as mucosal protectant, histamine type2 receptor antagnosist and proton-pump inhibitors), which has been standard therapy for years, is no longer recommended. A report revealed that incidence of gastric ulcers in critically ill neonatal foals was similar between years when prophylactic treatment was standard of care and years when it was no longer in use. [4]

In our case, the foal also presented a nutritional myodegeneration due to selenium deficiency. This disease is not reported as a specific causative agent of gastric ulcer in foals. However, dehydration and decubitus may have favor gastric ulcer formation by decreasing mucosal blood flow. Myopathy due to selenium deficiency is quite a frequent problem affecting foals and calves in Quebec (Canada) because of its low levels in the soil. This myopathy in its skeletal form frequently has a slow onset characterized by muscular weakness or stiffness. [5,6] Dysphagia or respiratory distress can also be observed. Mechanisms by which selenium deficiency leads to myodegeneration are not fully understood. However selenium is an essential component of several glutathione peroxidases involved in the protection against free radicals. Hence, selenium deficiency is supposed to enhance oxidative damages on muscular cells. [5] Diagnosis is based on clinical

signs, epidemiological evidence of poor-selenium diets, and elevated CK, AST and LDH activities. In acute presentation, hyperkaliemia, hyperphosphatemia, hyponatremia, and hypochloremia may also be observed with foals. [5] Those changes were observed in our case. Considering electrolytic disorders and the mild azotemia, a bladder rupture was also initially considered. However it was considered less likely based on a clear visualization of the bladder by ultrasounds examination as well as the absence of abdominal fluid accumulation. The absence of bladder rupture was confirmed by a lower creatinine concentration in the fluid compared to the serum.

Determination of selenium status by laboratory analysis of tissue biopsies, especially liver biopsies, and whole blood may help to confirm the diagnosis. [5] Treatment is based on selenium and vitamin E administration and supportive therapy including fluids perfusion and antibiotics. Prevention consists on oral supplementation of pregnant mares with selenium. In Quebec, pregnant mares are usually supplemented but it has not been done in our case, as the pregnancy was unexpected in this animal.

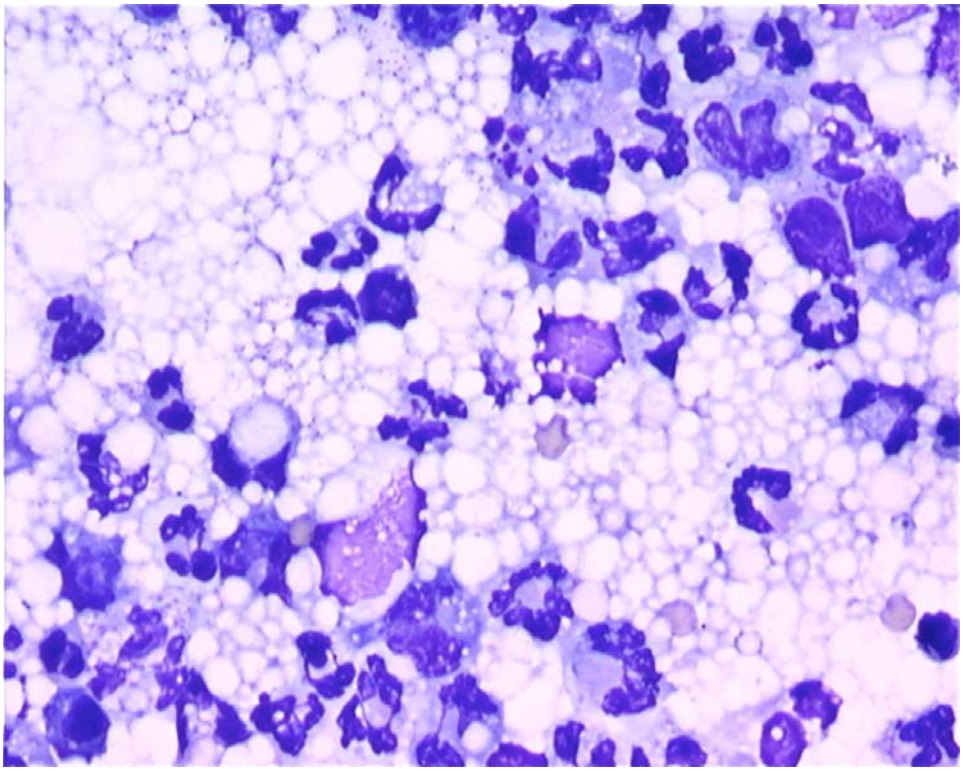

Figure 1: Peritoneal fluid (500x, Wright-Giemsa)

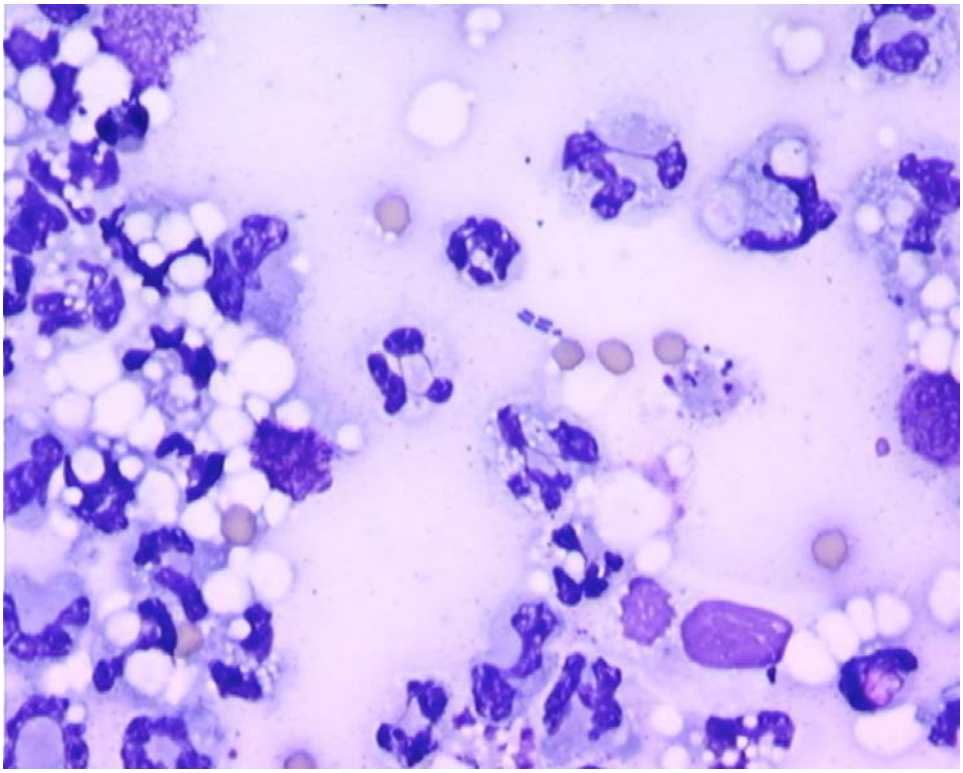

Figure 2: Peritoneal fluid (500x, Wright-Giemsa)

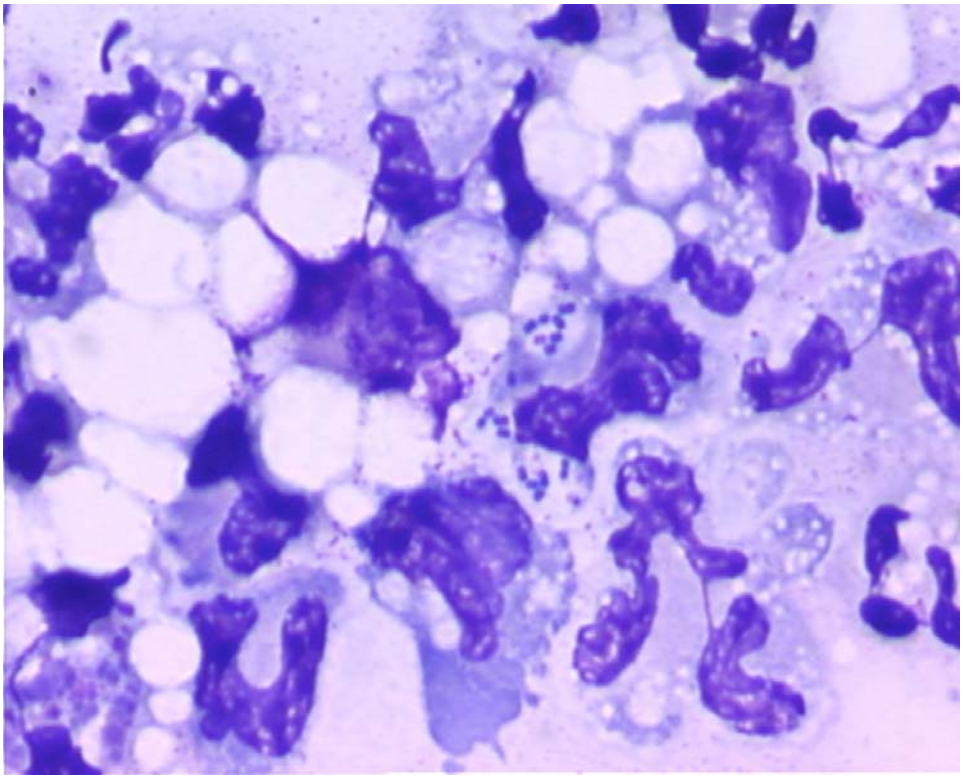

Figure 3: Peritoneal fluid (1000x, Wright-Giemsa).

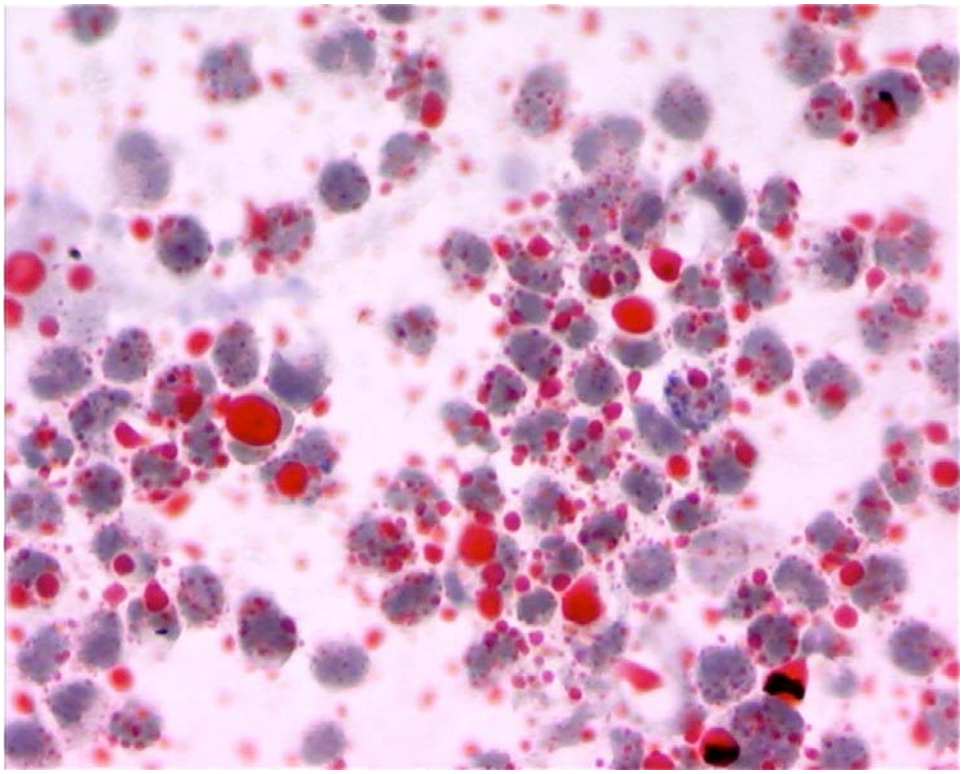

Figure 4: Peritoneal fluid (500x, Oil Red O).

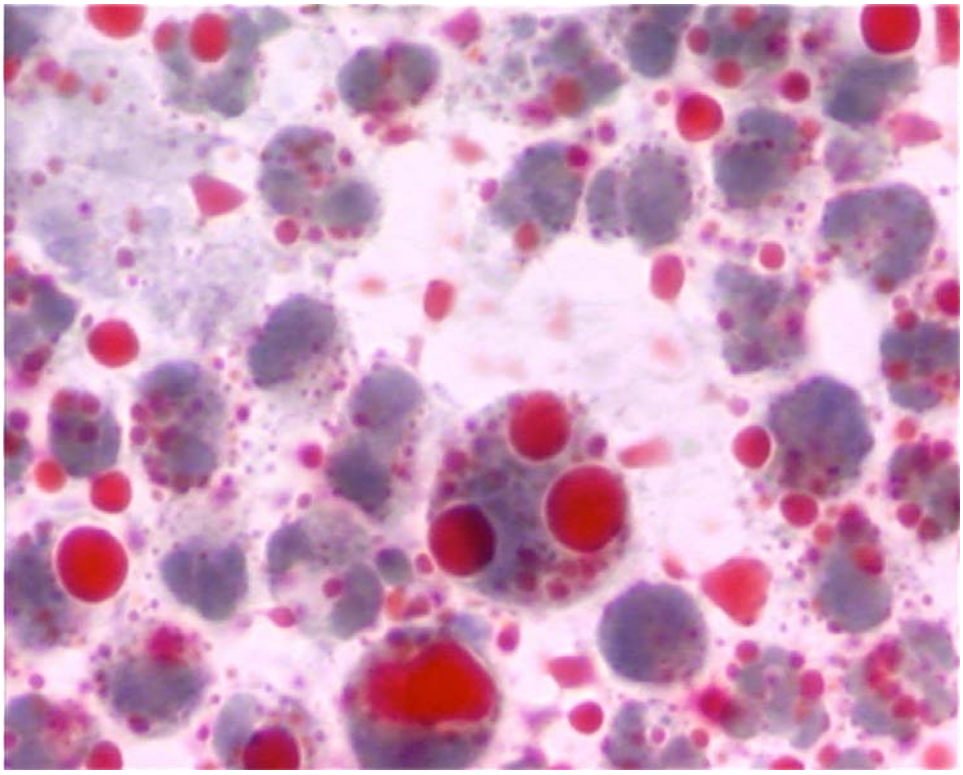

Figure 5: Peritoneal fluid (1000x, Oil Red O). Extracellular and intracytoplasmic vacuoles stain positive.

References:

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