History

An 8-hour-old 50.5-kg (111-lb) female American Quarter Horse foal was evaluated by its primary veterinarian because of colic of 7 hours’ duration. Initially, the foal was reluctant to nurse and was tube fed colostrum. Shortly thereafter, the foal began to roll on the ground and appeared to be in pain. The veterinarian subsequently used a point-of-care semiquantitative enzyme immunoassay to measure serum IgG concentration, which was 800 mg/dL. A single dose of butorphanol tartrate (0.06 mg/kg [0.03 mg/lb], route of administration unknown) was administered, along with multiple enemas. Some meconium was voided, but the clinical signs did not abate. The foal was referred to the Virginia-Maryland Regional College of Veterinary Medicine large animal teaching hospital.

Clinical and Clinicopathologic Findings

At the initial evaluation at the hospital, the foal was quiet, alert, and responsive. It could stand when supported but buckled in the hind limbs and had general signs of colic. The foal was estimated to be 6% dehydrated and had injected mucous membranes and a small amount of loose stool around the anus. Rectal temperature, pulse rate, and respiration rate as well as the remainder of the physical examination findings were considered normal.

Results of an initial CBC indicated that the foal’s total neutrophil concentration was within the reference interval for a neonatal foal (neutrophil count, 7,198 neutrophils/µL; reference interval, 1,340 to 9,600 neutrophils/µL) with a mild left shift (band neutrophil count, 164 band neutrophils/µL; reference interval, 0 to 150 band neutrophils/µL) and mild lymphopenia (654 lymphocytes/µL; reference interval, 700 to 2,100 lymphocytes/µL). These findings were interpreted as inflammation with stress. Results of additional blood tests, including serum biochemical analysis and assessments of plasma fibrinogen and serum lactate concentrations, were unremarkable on the basis of published, age-matched reference intervals. Serum IgG concentration was confirmed to be ≥ 800 mg/dL with a point-of-care semiquantitative enzyme immunoassay (the same type used by the referring veterinarian).

The next day, abdominal ultrasonography of the 2-day-old foal revealed a small amount of free anechoic abdominal fluid, which was sampled via abdominocentesis. The fluid was yellowish-white and opaque (Figure 1) with a total protein concentration of 4.5 g/dL (determined by refractometry), erythrocyte concentration of 27,000 RBCs/µL, and total nucleated cell concentration of 32,400 cells/µL.

Formulate differential diagnoses from the history, clinical findings, and Figure 1—then turn the page →
Cytologic Evaluation Findings

Direct, sediment, and cytocentrifuged preparations of the abdominal fluid sample were examined; all preparations were moderately cellular with well-preserved nucleated cells. The nucleated cell population consisted of 82% nondegenerate neutrophils, 16% macrophages, 2% lymphocytes, and occasional reactive mesothelial cells; the cells were present on a pale eosinophilic background with sparse erythrocytes and pyknotic cells. The macrophages frequently contained a moderate amount of basophilic cytoplasm and frequent colorless, punctate, variably sized intracytoplasmic vacuoles (Figure 2). The lymphocytes were primarily small lymphocytes, with few medium- and large-sized lymphocytes and rare reactive lymphocytes. No infectious agents or neoplastic cells were observed.

Because the foal had a history of tube feeding (colostrum by the referring veterinarian as well as dam’s milk by hospital staff), iatrogenic gastric rupture and leakage of colostrum or milk into the abdominal cavity was considered. A direct preparation of dam’s milk was made and stained with a modified Wright stain. On cytologic evaluation of the direct milk preparation, aggregates of a flocculent, lightly basophilic material (milk) were observed; such aggregates were not apparent in the foal’s abdominal fluid sample. These findings excluded iatrogenic gastric rupture as a diagnosis.

Additional Laboratory Findings

The triglyceride concentration in the abdominal fluid sample was 1,936 mg/dL. Subsequently, a concentrated smear of the fluid sample was stained with oil red O stain, and the vacuoles within the macrophages stained intensely red, suggesting the presence of lipid (Figure 3). Positive and internal negative controls for oil red O (positive control, mayonnaise; negative controls, neutrophils and lymphocytes) stained appropriately.

Cytologic Diagnosis and Case Summary

Cytologic diagnosis and case summary: chylous ascites with secondary neutrophilic inflammation in a foal.

Comments

For the foal of the present report, initial differential diagnoses based on the gross appearance of the abdominal fluid sample and its total protein and cell concentrations included chylous effusion, gastric rupture with leakage of milk or colostrum into the abdominal cavity, and exudate. The finding of numerous macrophages with clear, punctate intracytoplasmic vacuoles was suggestive of chylous effusion, which was confirmed by the high fluid triglyceride concentration and oil red O staining of the
abdominal fluid. Lipid-laden, oil red O-stain–positive macrophages in abdominal fluid have been seen after nasogastric mineral oil administration to an equid with a rectal tear, but the foal of this report had no history of mineral oil administration. Moreover, mineral oil does not appear grossly chylous (white and opaque) as did the abdominal fluid sample obtained from the foal. The numerous nondegenerate neutrophils without any visible infectious agents detected in preparations of the abdominal fluid sample likely represented sterile peritonitis secondary to the irritating and inflammatory effects of an effusion within the peritoneum (particularly chyle). Peripheral lymphopenia may have been secondary to stress or the loss of lymphocyte-rich chyle into the peritoneal cavity. However, lymphocytes were a minor component of the foal’s effusion.

Chylous abdominal effusions are rare in horses and of 6 previous reports, only 2 describe the condition in neonatal foals. Chylous effusions develop when lipid-rich fluid leaks from a lymphatic vessel or vessels into the peritoneal, pericardial, or thoracic cavity. Mechanisms that can lead to leakage of chyle include increased hydrostatic pressure within lacteals (most often due to impaired venous or lymphatic drainage) or increased lacteal vessel wall permeability. Diseases reported to result in chylous ascites in horses include colonic torsion, obstruction of lymph flow owing to intra-abdominal lymphadenopathy, lymphatic tears induced by intra-abdominal adhesions, primary or secondary intestinal lymphangiectasia, and congenital aplasia or hypoplasia of lymphatics.

Although chylous ascites has been reported for only a few foals, the condition is better described in the human medical literature. In a 1985 review of 41 cases of pediatric chylous ascites, the predisposing disorders were lymphatic abnormalities (54%), lymphatic obstruction (27%), and trauma (19%). In a more recent review, the most common cause of atrumatic chylous ascites among 61 pediatric patients was primary lymphangiectasia (83.6% of cases); the remaining 10 cases were attributed to Costello syndrome (3.3%), familial visceral myopathy (3.3%), intestinal malrotation (3.3%), ascariasis (1.6%), sarcoma (1.6%), and unknown causes (3.3%).

In the case described in the present report, the history and physical examination findings were not compatible with increased resting venous pressure (ie, due to cardiac insufficiency) or intestinal torsion. Abdominal imaging did not reveal evidence of lymphadenopathy or a compressive mass that could obstruct lymphatic vessels, and intra-abdominal adhesions were considered unlikely in a neonatal foal. Therefore, the initial differential diagnoses for the chylous ascites in the foal of this report included intestinal lymphangiectasia (either congenital or acquired secondary to increased intralacetal hydrostatic pressure as a result of dystocia or meconium impaction), rupture of a major lymphatic vessel during parturition, and congenital aplasia or hypoplasia.

The foal’s clinical signs of colic and chylous ascites resolved with hospitalization and supportive care, including IV fluid therapy with isotonic crystalloids, administration of a plasma transfusion (1 L, IV, once), initial antimicrobial treatment with cefotiofur sodium (2.2 mg/kg [1 mg/lb], IV, q 12 h) that was later changed to penicillin G potassium (22,000 U/kg [10,000 U/lb], IV q 6 h) and amikacin (25 mg/kg [11.4 mg/lb], IV, q 24 h), NSAID treatment with flunixin meglumine (0.5 mg/kg [0.23 mg/lb], IV, q 12 h), and administration of the gastroprotectant proton-pump inhibitor omeprazole (4 mg/kg [1.8 mg/lb], PO, q 24 h). The foal was discharged to the owner without complications after 7 days of hospitalization and was subsequently lost to follow-up.

Exploratory laparotomy was not performed in this case, and a definitive etiogenesis for the colic episode and chylous ascites was not identified. However, intestinal lymphangiectasia was the likely diagnosis, given the foal’s initial stable condition, mild signs of abdominal pain, and rapid clinical improvement with medical treatment. Dystocia or meconium impaction likely compressed the intestinal lymphatic vessels, resulting in increased intralacetal pressure and subsequent lymph leakage into the abdominal cavity. Other causes of neonatal chyloperitonitis, such as congenital malformations, malignancy, rupture of a major lacteal vessel, and intestinal torsion or volvulus, were deemed unlikely, considering that these conditions typically require surgery to alleviate the ascites and have a high mortality rate (even following surgical intervention).

References