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Clinical Writing Assessment 1

Complete Failure of Transfer of Passive Immunity, Sepsis, Omphalophlebitis, and Equine Neonatal Maladjustment Syndrome in a Brabant Belgian Neonate

Case Summary: A 3-day-old Brabant Belgian filly was presented for a 12-hour history of recumbency. Equine neonatal sepsis was diagnosed based on the sepsis score and a positive blood culture, and a serum [IgG] of 0 mg/dL on presentation confirmed the diagnosis of complete failure of transfer of passive immunity. Ultrasonography of the urogenital structures was consistent with an umbilical vein abscess. The filly's clinical and behavioral signs were consistent with equine neonatal maladjustment syndrome. The filly responded well to therapy (which included supportive care, antimicrobials, plasma transfusions, and fluid therapy) and was reported to remain well one month and three months after discharge.

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Signalment: 3-day-old Brabant Belgian filly

History:
The filly presented for "straining" to urinate and/or defecate of 24 hours' duration and recumbency of 12 hours' duration. Prior to referral, the filly was not evaluated by a veterinarian, and no medications were administered by the owners.

The dam was an apparently healthy multiparous 17-year-old Brabant Belgian mare with historically normal foalings. The mare was reported to be properly vaccinated prior to foaling. However, vaccine records were not available at the time of presentation. The owner did not report evidence of premature udder development or lactation in the dam. The filly was reported to be to term, but the exact gestational age was unknown. The foaling occurred in a stall and was observed by the owner and reported to be normal with no evidence of dystocia. The filly stood within 1-2 hours of foaling and nursed within 2-3 hours, but the exact times and amount of colostrum consumed were unknown by the owner. The mare's colostral quality was also unknown. The mare was reported to pass her placenta within a normal time period, but the exact time was not known. The placenta was reported to be normal by the owner, but it was not evaluated by a veterinarian. The owner was unsure if the filly passed meconium. The filly was reported to be active and was often seen by the mare's udder for the first 36-48 hours of life. The filly's umbilicus was not disinfected on the farm. The amount of milk produced by the mare and consumed by the foal was unknown during this time period.

Physical Examination:
At presentation, the filly was recumbent and unable to rise on the trailer with a dull, unresponsive mentation. She was carried off the trailer and placed on a foal bed for the remainder of the examination. She was estimated to weigh 80 kg. The filly was tachycardic with a heart rate of 152 beats/min. Her respiratory rate was 32 breaths/min and her rectal temperature was mildly increased at the high end of the neonatal reference range at 102.5^{o}F.[1] The filly's ribs were palpably within normal limits bilaterally with no evidence of fracture. The filly's mucous membranes were pale pink and tacky with no petechiation and a capillary refill time of 2-3 seconds. Her jugular refill time was delayed bilaterally, and she had a prolonged skin tent time. The filly's distal extremities were warm to the touch, but her peripheral pulses were weak. The filly's sclerae were mildly injected bilaterally, and both eyes were sunken. The filly was estimated to be approximately 8% dehydrated. No murmurs or arrhythmias were heard on cardiac auscultation, and normal bronchovesicular sounds were heard on thoracic auscultation. Quiet but present borborygmi were heard on gastrointestinal auscultation in all quadrants. The filly's joints were palpably within normal limits with no evidence of effusion, heat, or pain. The external umbilical remnant was dry but was palpably mildly thickened. There was a small ('1 finger') umbilical hernia palpated in the abdominal wall. No urination or defecation was observed during the physical examination. A truncated neurologic assessment was performed revealing normal pupillary light reflexes, both direct and consensual. There was an absent menace response bilaterally. The filly was unresponsive to stimuli and had an absent suckle response. A full neurologic examination was not performed in order to quickly initiate treatment. A complete blood count (CBC),

serum chemistry, serum amyloid A (SAA), PCV/TP, plasma [lactate], blood [glucose], serum [IgG], and blood culture were performed on presentation (Appendix I). Thoracic, abdominal, and urogenital ultrasound was performed on presentation (Appendix III). A sepsis score was created incorporating the physical examination and initial bloodwork data (Appendix IV).

Problem List and Differential Diagnoses:
The problems identified on the filly's initial physical examination included recumbency, obtunded mentation, absent menace, injected sclerae, absent suckle reflex, mild fever, a thickened umbilical remnant, umbilical hernia, dehydration with hypoperfusion, and tachycardia.

Recumbency in a neonate can be caused by trauma, angular limb deformity, weakness, electrolyte derangements, sepsis, botulism, nutritional myodegeneration, or neurologic disease (neonatal encephalopathy, hydrocephalus, meningitis, trauma). The filly's mentation was also obtunded, which can be seen as a result of neurologic disease, hypoglycemia, sepsis, acidemia, and electrolyte disturbances. A truncated neurologic examination revealed an absent menace response, but this is a learned response that is incompletely developed for the first two weeks of life.[2] The filly's sclerae were mildly injected, which is observed after trauma and with sepsis. The filly lacked a suckle reflex on presentation. The suckle reflex requires an intact motor supply to the tongue and sufficient cerebral function. Loss of the suckle reflex is often the first sign of neurologic dysfunction, often neonatal encephalopathy.[2] Other causes of an absent/reduced suckle reflex include prematurity/dysmaturity, weakness, or nutritional myodegeneration

The filly's umbilical remnant was palpably mildly thickened on physical examination. Differentials for abnormalities of the external umbilical remnant include infection, hematoma, hernia, or a patent urachus. A small ('one finger' in diameter) umbilical hernia was palpated in the body wall cranial to the umbilicus. Umbilical hernias are often congenital and can resolve on their own in 3-5 months. There was no evidence of visceral incarceration within the hernia, and it was considered clinically insignificant. Ultrasonography of the umbilical vein was consistent with omphalophlebitis (Appendix III). The umbilicus is a key portal of entry of bacteria causing sepsis in neonates, therefore increasing the suspicion of sepsis in this filly.[3]

The filly was clinically dehydrated, with evidence of hypoperfusion as indicated by tachycardia, decreased peripheral pulses, prolonged jugular fill and skin tent, sunken eyes, pale pink and tacky mucous membranes with a prolonged capillary refill time, and abnormal mentation. A state of shock occurs when the cardiovascular system is unable to supply a sufficient amount of oxygen to the cells resulting in a shift to anaerobic metabolism. This was suspected due to her clinical signs and hyperlactatemia (Appendix I). Additionally, hyperlactatemia can result from sepsis-induced tissue hypoxia from alterations in cellular metabolism. Hypovolemic shock develops from a decrease in circulating blood volume, which is caused by external fluid loss, hemorrhage, or third spacing. Distributive shock results from abnormal circulation in the microvasculature despite normal cardiac output, with the most common form being septic shock. The filly

did not have evidence of isotonic fluid loss and was hypernatremic, making hypovolemic shock less likely. Sepsis was suspected in the filly and it was thought that marked dehydration and suspected septic shock were responsible for these clinical findings.

The filly had a mildly elevated body temperature at presentation (102.5°F), which later progressed to more marked pyrexia of 103.8°F after initial resuscitation. The causes of elevated body temperature include fever and hyperthermia. Fever results from an increase in the hypothalamic set point, whereas hyperthermia is an increase in body temperature without an increase in this set point. The most common cause of fever is infectious in origin (bacterial, viral, fungal) but can also be associated with immune-mediated disorders (paraneoplastic syndrome or IMHA). Hyperthermia can result from increased environmental temperature, overexertion, or certain medications (macrolide antimicrobials). The filly's elevated body temperature was more consistent with a fever, rather than hyperthermia given other clinical signs indicative of infection such as evidence of omphalophlebitis and suspect sepsis.

The filly was tachycardic on presentation. Tachycardia can be caused by pain (colic, trauma), electrolyte derangements, sepsis, dehydration, and hypovolemia. The filly had a history of straining, but an abdominal ultrasound did not reveal any abnormalities in the gastrointestinal tract such as meconium retention or intussusception (Appendix III). The filly was clinically dehydrated and suspected to have sepsis; therefore, these were thought to be the cause of the filly's tachycardia.

A thoracic ultrasound was performed, which showed pleural thickening indicating a disruption in the normal pleural surface due to accumulation of exudate or cellular debris (Appendix III). Potential causes for these findings include mild bronchopneumonia or dependent atelectasis. The filly was recumbent on the farm, trailer ride, and during the examination. Therefore, the likely cause of the mild pleural thickening was due to atelectasis from recumbency. Ultrasonography of the ribs was within normal limits, confirming the absence of rib fractures, as ultrasonography is more sensitive than palpation and radiography in their diagnosis.[4]

There were multiple problems identified on the filly's initial bloodwork. The initial PCV, blood lactate, and blood glucose concentrations showed erythrocytosis, hyperlactatemia, and hyperglycemia. The CBC showed erythrocytosis and leukopenia characterized by neutropenia and lymphopenia. The serum chemistry identified elevations in [ALP] and [SDH], decreased [CK] hyperbilirubinemia, hypertriglyceridemia, hypernatremia, hyperchloremia, hypocalcemia, hyperphosphatemia, hypomagnesemia, hyperosmolality, elevated anion gap, and hypoproteinemia characterized by hypoglobulinemia. The SAA was elevated, and the serum [IgG] was consistent with complete failure of transfer of passive immunity. All reference ranges in the Appendices are for adult horses, which is how the laboratory data were provided.

The normal foal spun PCV and hematocrit (Hct) should fall into the lower portion of the adult reference range.[5] The filly's PCV and Hct values were at the top end of the adult reference range and therefore should be considered abnormally high. Erythrocytosis can

be relative or absolute. Relative erythrocytosis can be caused by hemoconcentration or splenic contraction. Dehydration, resulting in a loss of plasma volume, is the most common form of hemoconcentration and results from diarrhea, sweating, water deprivation, or diuresis. Absolute erythrocytosis is less common and can result from primary erythrocytosis or secondary erythrocytosis due to chronic pulmonary disease or right-to-left cardiovascular shunting. In this patient, hemoconcentration due to dehydration from lack of free water is the most likely explanation to the elevated PCV and Hct. The MCH was very mildly elevated and not thought to be clinically relevant.

The filly was markedly leukopenic, characterized by marked neutropenia and lymphopenia. Neutropenia in neonates can be caused by sepsis, prematurity, *in utero* infection with equine herpesvirus-1 during late gestation or neonatal alloimmune neutropenia.[5] The neonatal lymphocyte count is lower than the adult count at approximately 1.4 $x10^9$/L and increases to adult values around 3 months of age.[5] However, the filly remained lymphopenic with this adjusted reference value. Lymphopenia is observed in foals with an immunodeficiency or sepsis. Additionally, foals in intensive care units have been shown to have persistent lymphopenia associated with stress.[5] The filly also had an elevated SAA; an acute phase protein produced by the liver that increases with inflammation. The filly's SAA was markedly elevated, and in foals with SAA values greater than 100 mg/L, there is a high likelihood for infection.[5]

The filly was hyperlactatemic and hyperglycemic using a stall side lactate meter and glucometer, respectively. The equine neonatal lactate is elevated at birth but decreases to adult levels in 24 hours.[5] Hyperlactatemia results from tissue hypoxia and poor perfusion associated with dehydration and hypovolemia, impaired clearance associated with hepatic disease, increased protein catabolism and muscle activity, and/or from increased metabolism and the activation of inflammatory mediators in sepsis/SIRS. In neonates, blood glucose reaches its lowest value two hours after birth. Subsequently, gluconeogenesis begins and the blood glucose increases over the first 24-48 hours and can be higher than adult values. Other causes of hyperglycemia in foals include glucose administration, catecholamine release, neonatal encephalopathy, sepsis, and SIRS, which can induce insulin dysregulation or alter gluconeogenesis.[5] The filly had an elevated anion gap on the general chemistry. The anion gap is the difference between the unmeasured cognate anions of Na^+ and K^+ and the unmeasured cognate cations of Cl^- and HCO_3^-. A high anion gap metabolic acidosis is caused by an increase in unmeasured anions. These unmeasured anions may include lactate, glycols, ketoacids, salicylates, methanol, and urea. A blood pH was not acquired on presentation, but the elevated anion gap in conjunction with hyperlactatemia is consistent with a high gap metabolic acidosis.

The filly was moderately hypernatremic on presentation. Hypernatremia can result from water deprivation, administration of sodium-containing fluids, free water loss (diarrhea, fever, tachypnea), and diuretic administration. Additionally, foals fed milk replacer that does not contain an adequate amount of water often experience hypernatremia. In this foal, the most likely cause of hypernatremia was a combination of water deprivation (as it was unable to stand and nurse) and free water loss due to fever. The filly was also mildly hyperchloremic. Hyperchloremia can be caused by retention of chloride due to renal

dysfunction, increased chloride intake, acid-base disturbances, and decreased extracellular fluid volume. The corrected chloride was calculated and was within normal limits (Appendix V). Therefore, the hyperchloremia was thought to be due to decreased water intake from lack of nursing. The filly's elevated osmolality could be caused by an increase in osmotically active particles (sodium, potassium, glucose, urea nitrogen), decreased water intake, or loss of free water. The filly's hyperosmolality was likely due to decreased water intake from lack of nursing, and free water loss from the filly's fever.

The filly was mildly hypocalcemic and hypomagnesemic based on total calcium/magnesium values in the chemistry panel. In foals, both ionized and total calcium have been reported to be lower than adult horses.[6] Other causes of hypocalcemia in horses include colic, acute diarrhea, sepsis, hypoparathyroidism, and cantharadin toxicosis. It is most likely that this filly's hypocalcemia was due to a combination of normal foal variation and sepsis. Additionally, there is an association of hypomagnesemia with hypocalcemia, as they are often both seen in horses suffering from cantharadin toxicosis, endotoxemia, enterocolitis, or ileus.[7] Hypomagnesemia can also be induced due to a dietary deficiency of magnesium. In this filly, the most likely cause of the hypocalcemia and hypomagnesemia was sepsis.

The filly had an elevated serum [ALP] when using the adult reference range. However, ALP is elevated in neonates due to increased osteoblastic activity.[5] Similarly, the serum [phosphorus] was outside of the adult reference range, but it is normal for neonates to have elevated phosphorus due to bone metabolism.[5] The filly had a serum [CK] below the adult reference range. However, newborn foals often have low CK levels, and this was not thought to be clinically significant.[5]

The filly's serum [SDH] was mildly elevated. Sorbitol dehydrogenase is a marker of hepatocellular injury and can be elevated with primary liver disease, inflammatory gastrointestinal disease, sepsis, and SIRS. In this filly, the SDH was only mildly elevated and not considered clinically significant. The filly had mild hypertriglyceridemia. It can be normal for foals <2 weeks old to have elevated serum cholesterol and triglyceride concentrations compared to adults.[5] However, hypertriglyceridemia in foals can also be seen in active liver disease, sepsis, SIRS, and neonatal encephalopathy causing alterations in lipid metabolism. The filly had a mild hyperbilirubinemia due to elevated unconjugated bilirubin. During the first week of life, neonatal hyperbilirubinemia is commonly seen associated with elevated unconjugated bilirubin.[5] This is the result of the breakdown of neonatal red blood cells and most likely the cause of hyperbilirubinemia in this case. Other causes of hyperbilirubinemia include hemolysis, decreased hepatic function seen in patients with sepsis or SIRS, or primary liver disease such as infectious hepatitis (Tyzzer's disease) or congenital disorders. However, with only a slightly elevated serum [SDH] and normal [AST] and [GGT], a primary liver disease was less likely the cause of the mild hyperbilirubinemia.

The filly was hypoproteinemic characterized by marked hypoglobulinemia, likely due to a deficiency in IgG. This was supported by a serum [IgG] of 0 mg/dL, thus characterizing the filly as having complete failure of transfer of passive immunity. This can be due to

maternal causes such as premature lactation (placentitis, premature placental separation, twins), poor colostrum quantity or quality (maiden or older mare), or failure to lactate (fescue toxicosis).[8] Foal causes of inappropriate serum IgG concentrations include failure to ingest colostrum (weakness, muscular deformity) or failure to absorb colostrum (prematurity, necrotizing enterocolitis).[8] Progressively decreasing IgG concentrations can be observed in septic foals due to catabolism of immunoglobulins. The filly was reported to be born at term and to stand and nurse within the first 3 hours after foaling. The dam was multiparous and was not observed to have premature lactation. Therefore, the filly's low [IgG] was suspected to be due to poor colostral quality or quantity.

The filly urinated after the initial work up. A free-catch urine sample was collected and evaluated (Appendix I). The filly's USG was considered isosthenuric. A normal equine neonate should have hyposthenuric urine due to the large amount of milk a normal neonate ingests.[5] A USG in the isosthenuric range could indicate renal dysfunction often seen with SIRS.

Case Management:

The patient was carried from the trailer and placed on a foal bed. After an initial examination was performed, the left jugular vein was clipped and aseptically prepared for intravenous catheter placement. A 14-gauge Mila™ over-the-wire catheter was aseptically placed and sutured into the left jugular vein. This was a non-reactive polyurethane catheter that was chosen for long-term use and to decrease the risk of thrombophlebitis in critically ill neonates. A catheter wrap was placed using sterile gauze and Elastikon™ to keep the catheter site clean and dry. Blood was drawn from the catheter for a CBC, serum chemistry, SAA, lactate, blood glucose, and blood culture (Appendix I). On physical examination, the filly was estimated to be approximately 8% dehydrated. She was administered 2 L of lactated Ringer's solution intravenously throughout the initial work-up to begin correcting her dehydration. Lactated Ringer's solution was chosen for resuscitation as it is a balanced isotonic replacement crystalloid. Dextrose was not added to the fluids, as a stall-side blood glucose concentration was within normal limits. After the initial examination and fluid boluses were complete, the filly had become more responsive and made attempts to stand, indicating a positive response to initial fluid therapy. This positive response to fluid therapy, without the use of vasopressors, made hypovolemic or septic shock less likely. A maintenance fluid rate of 125 ml/hour was calculated (Appendix V) based on the filly's estimated weight of 80 kg. Throughout Day 1, the fluid rate was increased to 175 ml/hour due to persistent tachycardia and hyperlactatemia, suggesting decreased perfusion. A blood pressure was not obtained but would have aided the evaluation of perfusion. Sodium chloride (0.45%) with 2.5% dextrose supplemented with calcium gluconate (10 ml/L) and potassium chloride (20 mEq/L) was used as maintenance isotonic fluids.

The filly's working diagnosis was sepsis, presumptively due to complete failure of transfer of passive immunity leading to omphalophlebitis. This diagnosis was supported by the filly's elevated sepsis score (Appendix IV) and later confirmed by a positive blood culture (*Actinobacillus spp., Escherichia coli*). A sepsis score of >12 predicts sepsis 93% of the time.[9] Additionally, the filly's clinical signs of tachycardia, fever, decreased suckle

reflex, recumbency, weakness, and decreased capillary refill time could all be attributed to sepsis. Sepsis is the leading cause of death in equine neonates and results from a systemic response to an infection (usually bacterial). The umbilicus was suspected to be the most likely portal of entry for bacterial sepsis in this case, however the gastrointestinal or respiratory tracts are more common sites of bacterial entry causing sepsis.[10] The biggest risk factor for sepsis in this case was complete failure of transfer of passive immunity. However, other risk factors for sepsis include dystocia, illness in the dam, gestational age, and environmental conditions. In this case, the foaling was reported to be uneventful, the mare was reportedly healthy, and the filly was born at term, making these risk factors less likely. Some of the most common bacterial isolates from foals diagnosed with sepsis includes *E. coli*, *Enterococcus* spp., *Streptococcus* spp., *Actinobacillus* spp., and *Staphylococcus* spp.[11]

The bacterial identification and susceptibility results (Appendix II) were not available initially, and therefore the filly was given broad-spectrum antimicrobials. She was started on potassium penicillin for primary Gram-positive coverage (22,000 IU/kg IV q6h), amikacin for primary Gram-negative coverage (25 mg/kg IV q24h), and metronidazole for primary anaerobic coverage for the umbilical vein abscess (10 mg/kg PO q12h).[12] The risk of antimicrobial-induced colitis was low due to the patient's age and lack of established hindgut fermentation. Potassium penicillin is a bactericidal β-lactam antibiotic that can cause signs of colic and agitation if given too quickly, and therefore the medication was diluted in sterile saline (Appendix VI) and given over 5 minutes. Amikacin is a bactericidal aminoglycoside that can be nephrotoxic due to acute tubular necrosis. The filly's renal values were assessed every 24-48 hours to ensure there was no evidence of azotemia while on amikacin (Appendix I). The filly's renal values were within normal limits on presentation and remained normal throughout hospitalization. Metronidazole is a bactericidal nitroimidazole antibiotic with side effects of anorexia, ataxia, or depression at high doses.

On Day, 1 the filly was administered 2L of hyperimmune equine plasma for immune support due to an [IgG] of 0 mg/dL. Due to the filly's size (80 kg) and [IgG], it was estimated that she would require approximately 4L of plasma to achieve an IgG of 800 mg/dL, which is considered adequate transfer of passive immunity. Intravenous plasma was chosen because the filly was >24 hours old and colostral antibodies would not be adequately absorbed through the gastrointestinal tract. The plasma transfusion was performed over 2 hours, and the filly's heart rate, respiratory rate, and temperature were monitored closely. The filly did not have any adverse reactions to plasma administration (fever, tachycardia, tachypnea, colic, or anaphylaxis). The filly was scheduled to receive two additional liters of hyperimmune plasma on Day 2, so as to not administer an excessive fluid load on Day 1. Following the second transfusion, the filly's [IgG] would be rechecked to determine if an adequate [IgG] had been achieved.

A 14 Fr x 50-inch Mila™ feeding tube was passed through the right naris into the stomach and sutured into place. This was performed to supplement the filly with mare's milk, as she was unable to stand and nurse on her own. Complications of feeding tubes include irritation/trauma to the larynx/pharynx, milk aspiration, and over-feeding. In

order to prevent milk aspiration, placement of the feeding tube was confirmed by palpation and endoscopy to be intra-esophageal. Additionally, the filly was placed in sternal recumbency or stood while administering milk through the feeding tube and remained in that position for 15-30 minutes after feeding. In order to prevent over-feeding, the filly was initially only given 2% of her body weight (150 ml) of mare's milk every 2 hours. This was increased to 200 ml every 2 hours overnight, as she tolerated the feedings well throughout the day as indicated by lack of colic signs and normal blood glucose values. The filly was checked for reflux prior to feeding, and the feeding was performed using a gravity flow enteral feeding bag.

The filly received sucralfate (10 mg/kg PO q6h) throughout hospitalization for prophylactic treatment of gastric ulcers. Sucralfate is a mucosal adherent but also inhibits pepsin and buffers hydrogen ions. Gastric ulcers in equine neonates are thought to result from ischemic damage to the gastrointestinal tract from shock, sepsis, or trauma.[13] Additionally, gastrointestinal health was promoted by administering small, frequent feedings of mare's milk (q2h).

The filly had visual assessments (attitude, urination, defecation, activity, position) performed hourly and physical examinations performed q4h (Day 1-discharge). The filly's recumbency was switched q2h to reduce the formation of bedsores and atelectasis of the recumbent lung. The filly's umbilical remnant was dipped in a 0.5% chlorhexidine solution (q8h Day 1-Day 3) to prevent further infection of the umbilical structures. The filly's blood glucose (q4h Day1-discharge) and lactate (q8h Day 1-2, q24h Day 3-4) concentrations were evaluated stall-side to ensure the filly did not become hypo- or hyperglycemic and to ensure adequate perfusion, respectively. The filly's PCV/TP (Day 1 q4h, Day2-8 q8h) were monitored to assess perfusion and make appropriate adjustments to the fluid therapy plan. The filly's urine was collected at least once daily for the first 4 days to measure the USG to assess hydration status.

Throughout Day 1, the filly became much more alert. Four hours after presentation, the filly made attempts to stand, and eight hours after presentation, the filly stood with assistance. However, the filly was unable to form a coordinated suckle and showed no interest in the dam. The filly's blood [lactate] increased over the first 4 hours of hospitalization and remained elevated for the first 36 hour before normalizing. This was thought to be a result of sepsis. Eight hours after admission, the filly's temperature increased to 103.8 ^{o}F, and she was tachycardic and tachypneic. She was administered a single dose of ketoprofen (1.1 mg/kg IV). Ketoprofen is a non-selective COX inhibitor with side effects including gastric ulceration and renal injury. The filly was already receiving sucralfate for gastric ulcer prevention, and her renal values were within normal limits at presentation and would be monitored closely with the use of amikacin. The filly's pyrexia improved with a single dose of ketoprofen. She did not receive additional NSAIDs throughout hospitalization.

Day 2: A repeat CBC was performed, revealing a significant increase in the filly's white blood cell count (Appendix I). There was evidence of band neutrophils, supporting a response to infection. A NOVA Metabolic panel was obtained that indicated an increase

in creatinine. Because the filly was receiving a nephrotoxic medication (amikacin), the IV fluid rate was increased to 175 ml/hr to enhance renal perfusion. The filly's [Na] remained elevated but improved from the previous day. Additionally, the filly's [Mg] was just slightly below the reference range. A repeat NOVA Metabolic panel was performed in the evening, which revealed a decrease in creatinine, indicating improved renal perfusion. The filly's [lactate] normalized by the end of the day. The filly remained borderline pyrexic (102.5 ^{o}F), but intervention with an NSAID was not required. The filly's USG was hypersthenuric, indicating appropriate renal concentrating abilities. The filly's daily milk intake was increased to approximately 5% (350 ml q2h) then 7% (450 ml q2h) of her body weight. Because the filly was still receiving much less than the normal 20-25% bodyweight in milk, it was not surprising that her urine was hypersthenuric. The filly became mildly hyperglycemic throughout the day but never required intervention. This was thought to be the result of sepsis and/or stress. The filly received an additional 2 L of hyperimmune plasma IV. Again, no adverse events were noted during the plasma transfusion. Throughout the day, the filly became progressively stronger and more alert and was able to stand without assistance. She developed a strong suckle reflex but showed no affinity for the udder even when placed directly underneath the mare's flank.

Day 3: The filly's [IgG] on recheck was only 755 mg/dl. An additional liter of plasma was administered IV without complications (total of 5 L). This was not unexpected, as critically-ill foals often do not show the expected increase in [IgG] based on grams of IgG/kg body weight as seen in healthy foals.[7] Additionally, septic foals catabolize IgG, leading to persistent hypoglobulinemia. A repeat NOVA Metabolic panel was performed revealing a normal creatinine, mild hyperglycemia, hypernatremia, mild hyperchloremia, mild hypocalcemia, and hypomagnesemia. No changes were made to the fluid therapy plan. Urine was obtained, and a USG showed mild hypersthenuria. The filly's daily milk intake was increased to ~10% (650 ml q2h) then 15% (800 ml q2h) of her body weight, and she showed no evidence of colic or diarrhea. The filly continued to stand and walk unassisted and had a strong suckle reflex. However, due to her continued lack of affinity for the mare despite being physically strong enough to stand and walk unassisted, the filly was diagnosed with neonatal maladjustment syndrome. Foals diagnosed with this condition can appear normal at birth and not show clinical signs for up to 24-48 hours. This condition is most commonly associated with peripartum events (dystocia, premature placental separation) causing hypoxia. Other causes associated with the placenta include placentitis, fescue toxicity, and placental edema. However, this filly did not have any of these risk factors, and therefore her condition was most likely associated with neonatal events such as sepsis.

Day 4: A repeat CBC revealed a leukocytosis characterized by a mature neutrophilia with a left shift. Leukocytosis occurs in the neonate as a result of infection, SIRS, or stress. The filly's omphalophlebitis was most likely the cause of the leukocytosis. A NOVA Metabolic Panel was performed, revealing a normal creatinine and an improved (lower) sodium concentration. The filly was still mildly hyperchloremic and hypomagnesemic. A repeat [IgG] showed that adequate transfer of passive immunity was achieved, with an [IgG] of 919 mg/dl. A urine sample was collected and the USG was hyposthenuric, which

is normal in a neonatal foal receiving an adequate amount of milk (Appendix I). The filly's milk intake was increased to ~20% of her body weight (1 L q2h). However, the mare's lactation level could not support this, so the filly received a 50:50 mix of mare's milk:milk replacer. If mixed improperly, milk replacer can cause hypernatremia. Care was taken to ensure the milk replacer was mixed appropriately. The filly's IV fluid rate was decreased to 150 ml/hr and then to 125 ml/hr to begin weaning her off IV fluids. The antimicrobial susceptibility report was completed for the *Actinobacillus* spp. isolate and showed susceptibility to the current antimicrobial therapy. The filly retained a strong suckle reflex, but her affinity for the dam had not improved.

On Day 5, the filly's CRI of IV fluids was discontinued, and she was transitioned to 500 ml boluses q6h of 0.45% NaCl + 2.5% dextrose. This was decreased to 300 ml boluses on Day 8, and all IV fluids were discontinued on Day 9. The filly appeared systemically healthy at this time, with no evidence of fever, tachycardia, or tachypnea. She remained in the hospital to receive nutritional support via her nasogastric feeding tube, as she continued to show no affinity for the dam. The filly was positioned at the dam every 2 hours to assist nursing; however, she was unable to find the teat. On Day 8, the filly latched onto the teat and nursed and swallowed successfully. The nasogastric tube feedings were subsequently discontinued, and she was closely monitored every hour to ensure she was adequately nursing. A repeat umbilical ultrasound was performed and revealed improvement of the umbilical vein abscess (Appendix III). On Day 9, the filly was started on chloramphenicol (50 mg/kg PO q6h), and potassium penicillin, amikacin, and metronidazole were discontinued. Chloramphenicol is a bacteriostatic acetamide antibiotic with a broad spectrum of coverage, including anaerobes. This medication was chosen as it has good penetration into abscesses and can be given orally at home. The filly's owners were educated on the complication of aplastic anemia in human patients and warned to wear gloves when handling this medication. The filly continued to nurse well over the next 24 hours, and her IV catheter was removed. The filly was discharged on Day 10 with instructions to administer chloramphenicol for the next three weeks. It was recommended that the filly be evaluated by the rDVM in 3-5 days after discharge to evaluate a CBC and SAA and to have a repeat umbilical vein ultrasound performed 7-10 days after discharge, as well as before discontinuing antibiotics.

Follow Up:
The rDVM called one week after discharge and reported that the filly was energetic and nursing well. The rDVM reported that a re-check ultrasound of the umbilical vein abscess showed subjective improvement. A repeat CBC was also performed at that visit, and the white blood cell count was reported to be within normal limits. The filly returned to the hospital 3 months after discharge for a routine umbilical hernia repair. The surgery was uneventful, and the filly was discharged 24 hours after surgery.

Self Reflection:
This case had multiple weaknesses in the history, as there were many gaps in information surrounding the foaling. Information that would have been useful to this case, but was unknown by the owner, included the exact gestational age to assess if the filly was premature/dysmature, the mare's colostrum quantity and quality, and the appearance and

[redacted]

weight of the placenta to rule in/out placentitis. Additionally, improvements could have been made during the initial work-up and subsequent monitoring. The filly was suspected to be dehydrated and in septic shock based on clinical and laboratory findings. It would have been beneficial to obtain a blood pressure at the time of presentation and after fluid resuscitation to objectively evaluate the patient's perfusion status. An arterial blood gas could have been performed at admission to provide information on oxygenation, as well as the patient's pH. The filly was placed on a nephrotoxic drug (amikacin), and therefore renal values were monitored every 24-48 hours while on this medication, and a USG was performed when urine was collected. The filly's plasma amikacin concentration could have been determined and a urinalysis performed for evaluation of casts indicating renal injury. Lastly, an antimicrobial susceptibility was not performed for the *E. coli* isolated from the blood culture due to a miscommunication with laboratory personnel. This information would have been beneficial to ensure that the antimicrobial plan was appropriate.

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Appendix I – Bloodwork

Hematology Report # 1

Date/Hospital Day –	**Normal Reference Ranges**	5/6/19 Day 1	5/7/19 Day 2	5/9/19 Day 4
HCT%	29-48	47	-	-
RBC x 10^6/ul	5.7-10.4	10.3	-	-
MCV fl	41-54	46	-	-
MCH pg	34.5-37.1	**33.1**	-	-
MCHC %	N/A	N/A	-	-
RDW %	16.0-20.0	16.5	-	-
MPV fl	4.9-7.9	6.9	-	-
Hemoglobin g/dl	10.1-17.5	15.7	-	-
Reticulocytes/ul	N/A	N/A	-	-
RBC Morphology	Pathologist's comments	No comment	-	-
Nucleated Cells/ul	-	-	-	-
Nucleated RBC's/ul	-	-	-	-
White Blood Cells/ul	4,100-9,700	**600**	7,500	**14,900**
Metamyelocytes/ul	FIND	0.0	0.0	0.0
Band Neutrophils/ul	0.0-100	0.0	**200**	**600**
Seg. Neutrophils/ul	2,400-7,000	**100**	5,900	**11,900**
Lymphocytes/ul	1,000-4,900	**400**	1,000	2,100
Monocytes/ul	100-500	100	500	100
Eosinophils/ul	0.0-400	0.0	0.0	100
Basophils/ul	0.0-200	0.0	0.0	0.0
Reactive Lymphocytes	Present/not present	Present	Present	Present
Leukocyte Morphology	Pathologist's comments	No comment	Anisocytosis Poikilocytosis	No comment
Platelets/ul	102-213	241	-	-
Sed. Rate mm/hr		-	-	-
Coomb's (Direct/Indirect)		-	-	-
LE prep		-	-	-
Antinuclear antibody		-	-	-
Antiplatelet antibody		-	-	-
Fibrinogen mg/dl		-	-	-
OSPT sec.		-	-	-
APTT sec.		-	-	-
Fibrin degradation products ug/ml		-	-	-

*If your laboratory units differ from those listed above, or if units are not noted, insert correct laboratory units. Include normal ranges of your laboratory in the column provided. Please add any additional parameters measured as required.

Biochemistry Report #1

Date/Hospital Day --		**Normal Reference Ranges**	5/6/19 Day 1	5/7/19 Day 2	5/7/19 Day 2
Parameter	**Units**				
Creatinine	mg/dl	0.8-1.8	1.0	1.4	1.1
ALP	IU/L	57-220	**1647**	-	-
AST	IU/L	179-396	171	-	-
LDH	IU/L	-	-	-	-
SDH	IU/L	0-5	**6.7**	-	-
GLDH	IU/L	-	-	-	-
GGT	IU/L	6-28	23	-	-
CK	IU/L	166-593	**95**	-	-
BUN	mg/dl	9-23	20	19	16
Total bilirubin	mg/dl	0.5-2.5	**5.53**	-	-
Direct bilirubin	mg/dl	0.1-0.3	0.20	-	-
Amylase		-	-	-	-
Lipase		-	-	-	-
Urea	mg/dl	-	-	-	-
Triglycerides	mg/dl	14-64	**144**	-	-
Glucose	mg/dl	83-114	**117**	-	-
Na + (NOVA)	mmol/L	134-139	**-**	150	140.4
Na	mEq/L	133-144	**152**	-	-
K+ (NOVA)	mmol/L	2.2-4.0	-	3.22	-
K	mEq/L	3.0-5.4	3.30	-	-
Cl- (NOVA)	mmol/L	104-111	**-**	112.0	-
Cl	mEq/L	95-104	**107.2**	-	-
Ca++ (NOVA)	mg/dl	5.5-6.5	**-**	4.83	4.22
Ca	mg/dl	11.2-13.4	**9.2**	-	-
P	mg/dl	1.4-5.0	**5.1**	-	-
Mg	mg/dl	1.29-2.21	**1.2**	-	-
Mg++ (NOVA)	mg/dl	1.11-1.60	**-**	**1.06**	**0.82**
Osmolality	mOsm/kg	268-289	**305**	-	-
Total protein	g/dl	5.3-7.2	**4.0**	-	-
Albumin	g/dl	2.6-3.7	3.2	-	-
Globulin	g/dl	2.1-4.3	**0.8**	-	--
Cholesterol	mg/dl	N/A	-	-	-
Uric acid	mg/dl	N/A	-	-	-
Ammonia	ug/dl	<60	-	-	-
Bile Acids		8-12	-	-	-

Cortisol			-	-	-
pH		7.38-7.42	-	7.402	7.431
PO_2	mm Hg	-	-	-	-
PCO_2	mm Hg	-	-	-	-
HCO_3	mmol/L	24-33	28	-	-
Total CO_2	mEq/L	-	-	-	-
Base Excess	mEq/L	-	-	-	-
Anion Gap	mEq/L	11-19	**20**	-	-

*If your laboratory units differ from those listed above, or if units are not noted, insert correct laboratory units. Include normal ranges of your laboratory in the column provided. Insert any other laboratory parameters measured as required.

Biochemistry Report #2

Date/Hospital Day --		**Normal Reference Ranges**	5/8/19 Day 3	5/9/19 Day 4	
Parameter	**Units**				
Creatinine	mg/dl	0.8-1.8	0.9	0.8	
ALP	IU/L	57-220	**-**	-	
AST	IU/L	179-396	-	-	
LDH	IU/L	N/A	-	-	
SDH	IU/L	0-5	**-**	-	
GLDH	IU/L	N/A	--	-	
GGT	IU/L	6-28	-	-	
CK	IU/L	166-593	**-**	-	
BUN	mg/dl	9-23	21	12	
Total bilirubin	mg/dl	0.5-2.5	**-**	-	
Direct bilirubin	mg/dl	0.1-0.3	-	-	
Amylase		N/A	-	-	
Lipase		N/A	-	-	
Urea	mg/dl	N/A	-	-	
Triglycerides	mg/dl	14-64	**-**	-	
Glucose	mg/dl	83-114	**145**	-	
Na + (NOVA)	mmol/L	134-139	**151**	**142.8**	
Na	mEq/L	133-144	**-**	-	
K+ (NOVA)	mmol/L	2.2-4.0	2.76	3.65	
K	mEq/L	3.0-5.4	-	-	
Cl- (NOVA)	mmol/L	104-111	**112.7**	**113.4**	
Cl	mEq/L	95-104	**-**	-	
Ca++ (NOVA)	mg/dl	5.5-6.5	**4.59**	5.22	
Ca	mg/dl	11.2-13.4	**-**	-	
P	mg/dl	1.4-5.0	**-**	-	
Mg	mg/dl	1.29-2.21	**-**	-	
Mg++ (NOVA)	mg/dl	1.11-1.60	**1.06**	**1.02**	

Osmolality	mOsm/kg	268-289	-	-	
Total protein	g/dl	5.3-7.2	-	-	
Albumin	g/dl	2.6-3.7	-	-	
Globulin	g/dl	2.1-4.3	-	-	
Cholesterol	mg/dl	-	-	-	
Uric acid	mg/dl	-	-	-	
Ammonia	ug/dl	<60	-	-	
Bile Acids		8-12	-	-	
Cortisol		-	-	-	
pH		7.38-7.42	7.42	7.41	
PO_2	mm Hg	-	-	-	
PCO_2	mm Hg	-	-	-	
HCO_3	mmol/L	24-33	32	-	
Total CO_2	mEq/L	-	-	-	
Base Excess	mEq/L	-	-	-	
Anion Gap	mEq/L	11-19	-	-	

Immunoglob – IgG/TIA Foal

	Normal Value	**Day 1**	**Day 3**	**Day 4**
IgG (mg/dL)	> 800	0	755	919

Stall Side PCV/TP/Lactate/Blood Glucose Daily Monitoring Report # 1

	Unit	**Day 1**	**Day 2**	**Day 3**	**Day 4**	**Day 5**
PCV (28-43)	%	**Q4h:** 44, 39, 44, 39, 37, 39	**Q8h:** 35, 33, 31	**Q8h:** 31, 33, 31	**Q8h:** 32, 33, 33	**Q8h:** 29, 30, 33
TP (5-7.5)	g/dL	**Q4h:** 5.0, 5.2, 5.6, 5.2, 5.6, 5.9	**Q8h:** 6.0, 6.4, 6.2	**Q8h:** 6.0, 6.2, 5.6	**Q8h:** 5.4, 5.2, 5.6	**Q8h:** 5.9, 5.8, 5.6
Lactate (<2)	mmol/L	**Q8h: 2.9, 4.8, 3.8**	**Q4h: 4.3, 3.8, 2.9**, 1.7	**Q24h:** 1.2	**Q24h:** 1.0	
Blood Glucose (80-120)	mg/dL	**Q4h:** 117, 108, **77**, **70**, 83, 90	**Q4h:** 101, 150, 117, **174**, **180**, 113	**Q4h: 163, 163, 133, 140, 138, 166**	**Q4h: 121,** 109, **168,** 111, **164, 154**	**Q4h: 136,** 105, **141**, 119, 120, 53

Stall Side PCV/TP/Blood Glucose Daily Monitoring Report #2

	Unit	Day 6	Day 7	Day 8
PCV (28-43)	%	**Q8h:** 31, 26, 33	**Q12h:** 32, 33	**Q8h:** 33, 32, 33
TP (5-7.5)	g/dL	**Q8h:** 5.4, 5.0, 5.6	**Q12h:** 5.8, 5.6	**Q8h:** 5.6, 5.6, 5.6
Blood Glucose (80-120)	mg/dL	**Q4h:** 117, **152**, 83, 119, 95, 82	**Q4h:** 116, **140,** 118, 98, 93	**Q8h:** 107, 110, 91

Urinalysis Report #3

Date/Hospital Day --	**Normal Reference Ranges**	Day 1	Day 2	Day 3	Day 4
Source(eg cath/void/cysto)	-	Void	Void	Void	Void
Color	-	Light yellow	Light yellow	Light yellow	Light yellow
Appearance	-	Clear	Clear	Clear	Clear
Specific gravity		1.012	1.016 1.028	1.016	1.016 1.008
pH		-	-	-	-
Protein		-	-	-	-
Glucose		-	-	-	-
Acetone		-	-	-	-
Bilirubin		-	-	-	-
Blood		-	-	-	-
Urobilinogen		-	-	-	-
Casts		-	-	-	-
Hyaline/LPF		-	-	-	-
Granular/LPF		-	-	-	-
Other		-	-	-	-
Leucocytes/HPF		-	-	-	-
Epithelial Cells/HPF		-	-	-	-
Erythrocytes/HPF		-	-	-	-
Crystals		-	-	-	-
Bacteria		-	-	-	-
Other		-	-	-	-
Osmolality mOsm/kg		-	-	-	-

* Include normal ranges of your laboratory in the column provided. Please add any additional parameters measured as appropriate.

Appendix II: Microbiology

Blood Culture: Positive for *Actinobacillus sp.* and *E. coli*

Antimicrobial Susceptibility Report # 1 – *Actinobacillus spp.*

Drug		
Amikacin	<=4	S
Ampicillin	<=0.25	NI
Azithromycin	<=0.25	NI
Cefazolin	<=4	S
Ceftazidime	<=1	S
Ceftiofur	<=0.25	NI
Chloramphenicol	<=4	S
Clarithromycin	2	NI
Doxycycline	<=2	S
Enrofloxacin	<=0.25	NI
Erythromycin	2	I
Gentamicin	2	S
Imipenem	<=1	S
Oxacillin	<=0.25	NI
Penicillin	0.5	NI
Rifampin	<=1	S
Tetracycline	<=2	S
Trimethoprim/Sulfamethoxazole	<=0.5	S

Salmonella Biosecurity Report # 1

Hospital Day	Day 1	Day 2	Day 3
Salmonella Fecal Culture	Negative	Negative	Negative

Appendix III – Ultrasound Examinations

Thoracic Ultrasonography (Day 1): The examination was performed with a 6.5 MHz micro-convex transducer. There were mild "comet tails" observed bilaterally, cranioventrally extending from ICS 7-5 indicating pleural roughening at these areas. The filly was recumbent during the examination and trailer ride to the hospital. As such, these ultrasonography findings were thought to be due to atelectasis of the dependent lung from lying down or mild bronchopneumonia. There were no other abnormalities identified on the pleural surface. There was no evidence of free pleural fluid within the pleural cavity to indicate the presence of pleural effusion.

Abdominal Ultrasound (Day 1): The examination was performed using a 6.5 MHz micro-convex transducer. There was no evidence of free fluid in the abdomen to indicate the presence of a peritioneal effusion. The stomach extended to the ninth intercostal space on the left and was filled with gas as evidenced by reverberation artifact. Portions of the small intestines were seen in the inguinal region, bilaterally and ventrally. The small intestinal loops observed were motile and non-distended, with no evidence of strangulation or intussusception. The foal was observed to strain on the farm, but ultrasonography of the transverse/descending colons did not reveal any structures consistent with meconium.

Urogenital Ultrasound (Day 1): The examination was performed using a 7.5 MHz linear transducer. The urinary bladder was moderately distended with an intact wall. The umbilical vein measured 0.83 cm in diameter at the base of the umbilical remnant and consistently increased in diameter moving cranially. At the level of the xiphoid, the umbilical vein was seen adjacent to the liver and distended to 2.5 cm in diameter with a hyperechoic center. The subcutaneous tissue surrounding the umbilical vein appeared thickened and edematous. The umbilical arteries were followed from the umbilicus to the bladder, and both measured < 1.0 cm in diameter. The urachus appeared within normal limits.
Interpretation: Umbilical vein abscess/ omphalophlebitis

Re-check Urogenital Ultrasound (Day 8): A recheck examination of the umbilical vein was performed using a 7.5 MHz linear transducer. The umbilical vein was followed from the umbilical remnant to the liver and measured 1.8 cm diameter at the level of the xiphoid next to the liver. The center of the umbilical vein was less hyperechoic than the previous examination, and the surrounding tissues appeared less thickened. The umbilical arteries and urachus were within normal limits.
Interpretation: Resolving umbilical vein abscess/ omphalophlebitis

Appendix IV: Foal Sepsis Score Sheet

Foal Sepsis Score	**4**	**3**	**2**	**1**	**0**	**This Case**
CBC						
Neutrophil Count		**<2.0x10^9/L**	2.0-4.0 or >12.0	8.0-12.0	Normal	3
Band Neutrophil Count		>0.2 x10^9/L	0.05-0.20		**<0.05**	0
Doehle bodies, toxic, granulation, or vacuolization in neutrophils	Marked	Moderate	Slight		**None**	0
Fibrinogen			>600	410-600	<400	--
Other Lab Data						
Hypoglycemia (mg/dL)			<49	49079	**>79**	0
IgG (mg/dL)	**<200**	200-400	400-800		>800	4
Arterial Oxygen		<40 Torr	40-50	51-70	>70	--
Metabolic Acidosis				**Yes**	No	1
Clinical Exam						
Petechiation, scleral injection not secondary to eye disease or trauma		Marked	Moderate	**Mild**		1
Fever (^{o}F)			**>102**	<100	Normal	2
Hypotonia, coma, depression, convulsions			**Marked**	Mild	Normal	2
Anterior uveitis, diarrhea, resp. distress, swollen joints, open wounds		**Yes** *****Counting umbilical vein abscess			No	3
Historical Data						
Placentitis, vulvar discharge prior to delivery, dystocia, long transport of mare, mare sick, foal incduced		Yes			**No**	0
Prematurity		<300 days	300-310	311-330	**>330**	0
Total Points						**16**

- A score of 12 or higher correctly predicts sepsis 93% of the time. A score of 11 or less predicts non-sepsis correctly 88% of the time.

Appendix V: Calculations

Calculation 1: Corrected Chloride Day 1

Corrected chloride = Measured chloride x (Normal sodium/Measured Sodium) :
107.2 x (135/152) = **95.2 mEq/L**

Calculation 2: Fluid Calculations (Holiday-Segar formula)

Maintenance Fluid Rate:
100 ml/kg/day first 10 kg
50 ml/kg/day second 10 kg
15 ml/kg /day for the remaining body weight

Est weight: 80 kg
10 kg x 100 = 1,000 ml = 1 L
10 kg x 50 = 500 ml = 1.5 L
60 kg x 25 = 1,500 ml = 1.5 L
Total per day = 3 L = 125 ml/hour

Dehydration:
80 kg x 8% dehydration = 6.4 L

Actual Fluids Administered at presentation:
2 L bolus of LRS + 2 L hyperimmune plasma + CRI at 150 ml/hr

Appendix VI: Medication Dilutions

Potassium Penicillin:
Bottle diluted to a concentration of 500,000 IU/mL
Volume of antibiotic administered: 4 mL (2 Million units)
Dilution: 4 mL potassium penicillin diluted in 20 mL sterile saline