Foal Medicine II: Uncommon Problems of Neonatal Foals

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Abstract

Awareness of uncommon conditions is useful when evaluating the neonatal foal. Because the initial presentation of a sick foal is often nonspecific, mental preparation of differential diagnoses, including uncommon conditions, will aid the clinician in arriving at the correct diagnosis. An overview of several uncommon conditions with common presentations is provided.

Primary presenting problem: ADR/Floppy Foal

Less common (relative to hypoglycemia, neonatal maladjustment syndrome and sepsis discussed in Part I of this lecture) causes of lethargy, inappetence, weakness and recumbency in the neonatal foal include neonatal isoerythrolysis (NI), uroabdomen, botulism, congenital cardiac anomaly, hyponatremia, nutritional muscular dystrophy (white muscle disease), Tyzzer's Disease, and glycogen branching enzyme deficiency (GBED) in Quarter Horses. This list is not all inclusive but represents several differentials to consider. The three more common of these uncommon conditions are described.

Neonatal isoerythrolysis

Neonatal isoerythrolysis is characterized by tachycardia, tachypnea, lethargy and pale or icteric mucous membranes. Rarely, severe neurological signs are observed, consistent with kernicterus. Affected foals are generally 1-4 days of age, but occasionally older at presentation. Dams of NI

foals are often, but not always, multiparous. Donkey and mule foals appear to be at increased risk for development of NI. Variable degrees of anemia develop with NI, with most clinical cases showing 20% or lower packed cell volume (PCV). The rate of onset and severity of clinical signs is determined by the quantity and activity of absorbed alloantibodies from the dam's milk . Diagnosis of NI is supported by saline agglutination crossmatch of the foal's red blood cells and mare's serum. Coomb's test may yield false negatives. Because some equine alloantibodies act only as hemolysins, agglutination tests may be falsely negative.

Treatment: The need for transfusion of oxygen-carrying red blood cells is determined by clinical signs related to signs of hypoxia (tachycardia, tachypnea, weakness), rather than a specific PCV. Initial blood transfusion from a healthy gelding blood donor is generally safe without crossmatching. However, if subsequent transfusions are required, major and minor cross matching is recommended. One to two liters of whole blood transfused from a donor to a 50kg foal is commonly performed. The formula for calculating blood volume for transfusion more specifically is:

$$\text{Transfusion volume} = \frac{\text{Desired PCV} - (\text{Recipient PCV} \bullet 0.08 \bullet \text{Body weight in kg})}{\text{Donor PCV}}$$

An NI foal should be muzzled to prevent nursing from the dam until 24 hours of age (time of gut closure), if diagnosed prior to that time. Nutritional support for the foal during that period can be provided with milk replacer.

Uroabdomen (ruptured bladder)

Foals with uroabdomen may present with generalized lethargy, inappetence, abdominal distension, frequent posturing to urinate, scrotal edema, colic, or any combination of these signs. Rarely, dyspnea or respiratory distress develops with extreme abdominal distension. Colts may be overrepresented owning to their narrow pelvic anatomy, but this is not confirmed. Rupture of any part of the urinary tract may lead to uroabdomen, most often in the first 1-6 days of life in the newborn foal. The dorsal wall of the bladder is reportedly the commonest site of rupture, with other sites on the bladder, urachal, urethral and ureteral defects also reported. Periparturient traumatic injury to the urinary tract, congenital defect, and septic foci rupture are some of the proposed and confirmed causes of uroabdomen. The pathophysiology is incompletely understood in many cases. Typical laboratory findings include hyperkalemia, hyponatremia and hypochloremia. Foals receiving intravenous fluids may not display these abnormalities. Sonographic exam reveals hypoechoic fluid accumulation in the abdomen and possibly direct visualization of the rupture. Definitive diagnosis is made when the peritoneal fluid: serum creatinine ratio is 2:1 or greater.[2]

Treatment: Surgical correction is nearly always required to repair a rupture of defect of the urinary tract. Medical management utilizing a Foley catheter has been described. Correction, or at least improvement of serum electrolytes is critical prior to anesthesia. Hyperkalemia in excess of 5.5 can be life-threatening. Intravenous fluid therapy aimed at reducing potassium concentration in the blood includes administration of 0.9% or 0.45% saline with 5% dextrose. Peritoneal fluid drainage is indicated if dyspnea secondary to abdominal distension is present. Rushing to surgery without first resolving dangerous electrolyte imbalances carries a high risk of anesthetic death.

Botulism

Flaccid paralysis, tremors (shaker foals), recumbency, pupil dilation, dysphagia, respiratory distress and acute death are signs attributed to botulism in foals. In contrast to adult botulism cases which involve pre-formed toxin, foals develop toxicoinfectious botulism. The causative organism, Clostridium botulinum, can survive and proliferate in the foal's GI tract, creating necrotic foci in the neonatal liver. In this manner, the foal is constantly exposed to newly formed toxin.

Treatment: Antimicrobial therapy (penicillin, metronidazole, or oxytetracycline), combined with toxin neutralization with botulinum antitoxin are mainstays of therapy. Intensive supportive care and mechanical ventilation may be required in severely affected foals. Prevention (although not 100% effective) is recommended in endemic areas (northeastern and mid-atlantic regions of the US) by vaccinating pregnant mares.[3]

Primary presenting problem: lame/abnormal gait foal

Uncommon causes of lameness, or more likely, abnormal ambulation in the neonatal foal include rib fractures, occipitoatlantoaxial malformation, and cerebellar abiotrophy.

Rib Fractures

Rib fractures are a somewhat common fracture type among neonatal foals. Attendant clinical signs may include stiffness when walking, increased recumbency, or non-specific lethargy. Palpation of the ribs is a recommended step in the physical exam of a neonate. Risk of myocardial puncture, hemothorax or pneumothorax is high if rib fractures displace. Diagnosis is made by palpation, radiography and/or ultrasonography. The latter has been found a more sensitive tool than radiography.[4]

Treatment: Surgical repair of fractured ribs may be indicated if overlying the heart or displacement has occurred. Otherwise, avoidance or direct pressure on the thorax and over exertion is recommended. On some farms, screening for rib fractures is routine in all newborn foal examinations.

Occipitoatlantoaxial malformation

Occipitoatlantoaxial malformation (OAAM) is a developmental defect of the atlas (C1) axis (C2), and occipital bone. This malformation results in spinal cord compression, manifesting as abnormal head carriage and an ataxic gait in affected animals. Ataxia may progress with age from mild incoordination and weakness of the limbs to the inability to stand. Depending on the severity of the disorder affected foals may be stillborn, develop neurological deficits in the neonatal period, or as a young adult.

Diagnosis of the malformed atlas and axis is confirmed with radiographs. OAAM is believed to be inherited as an autosomal recessive defect in Arabian horses, but different mutations appear to be involved. The condition has also been reported in Miniature Horses, Quarter Horses, Morgans, Standardbreds and Friesians. The author has diagnosed this condition in a Thoroughbred. Researchers believe there may be multiple forms of OAAM, some involving multiple organ involvement.[5,6]

Treatment: no treatment exists. Genetic testing for one of the mutations involved in OAAM is commercially available, but a negative test does not exclude the condition as alternate genetic mutations likely exist.

Cerebellar abiotrophy

Cerebellar abiotrophy (CA) is another inherited neurological condition primarily seen in the Arabian breed. Autosomal recessive inheritance has been confirmed in Arabian crossbreeds as well as purebreds. Progressive neuronal degeneration of the cerebellum results in intention tremors, a stiff, stilted gait, and ataxia in foals. While most commonly observed in weanling aged foals, the author has diagnosed CA in multiple Arabian neonates. When the collection of clinical signs described are observed in a foal with Arabian pedigree, DNA testing is warranted to confirm the diagnosis.

Treatment: no treatment exists. Arabian breeding programs are encouraged to use the genetic test for screening purposes to avoid continuation of this genetic defect.

Primary presenting problem: colicky foal

The list of uncommon causes of colic in the neonatal foal is likely endless. From medical colic of unknown origin to mesenteric root torsion, neonatal foals, like adult horses, develop colic of every kind. Two uncommon but definitively diagnosable causes of colic clinicians may encounter include intestinal aganglionosis (lethal white foal syndrome) and intestinal atresia.

Lethal White Foal Syndrome (LWFS)

Currently termed "lethal white overo syndrome", this autosomal recessive genetic disease seen primarily in Paint horses occurs specifically in the overo phenotype horse. Affected foals born to frame overo carriers of the LWFS gene are all white in color, with blue eyes and pink skin, as well as aganglionic segments of the distal intestinal tract (frequently ileum and colon). A defect

of the endothelin-B receptor results in abnormal development of melanocytes and intestinal ganglia.[7] Affected foals appear normal at birth, stand and suckle, but become colicky within hours due to functional intestinal obstruction. Meconium is retained in the colon and barium contrast study of the distal GI tract may show marked narrowing of the intestinal lumen of the affected segment.

Treatment: none. Genetic testing of all overo phenotype breeding stock is recommended to avoid producing a foal with this condition.

Atresia coli

Atresia coli is a congenital abnormality with sporadic appearance and unknown heritability or underlying cause. In this condition, a portion of the distal GI tract (most often distal large colon or proximal small colon) is absent or not patent. Clinical signs of colic with abdominal distension develop within the first 24 hours of life in affected foals. Distinguishing meconium impaction from atresia coli can be difficult, however, it is imperative as the prognosis for the former is very good and for the latter, grave. Digital rectal exam and enema should help distinguish meconium impaction from atresia coli. The presence of any amount of meconium will exclude atresia coli, while absence of meconium does not confirm it. Barium enemas can be used to aid in the diagnosis, however the results may be difficult to interpret with absolute certainty. The same difficulty exists with proctoscopy due to straining and intestinal motility, however this technique has been used to confirm atresia coli. Definitive diagnosis may require exploratory celiotomy.

Treatment: depending on segment and length of colon affected, surgical repair by anastomosis of the two ends may be attempted.[8]

Summary

Familiarity with uncommon causes of common presentations allows the clinician to extend the differential diagnosis list when initial differentials are excluded.

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