Septicemia

Presentation and Diagnosis:
Foals with septicemia typically present at ≤ 1 week of age (or occasionally at older ages) for general weakness/lethargy or for specific signs such as colic, diarrhea, or lameness. Key findings on physical examination that suggest septicemia include fever, hyperemic or injected mucous membranes, petechial hemorrhages on the pinnae or mucous membranes, pitting edema in the distal limbs, uveitis, or evidence of localized infection such as pneumonia, diarrhea, joint effusion or physeal enlargement, lameness, umbilical infection or patent urachus. Foals with septic shock may have altered mentation and appear comatose; return of normal mentation after stabilization and fluid resuscitation helps rule out neonatal encephalopathy as a cause of the altered mentation. Evidence of placentitis in the dam (early udder development, prepartum vaginal discharge, premature parturition, placental abnormalities, or post-partum metritis) should also raise suspicion of in utero sepsis in the foal; such foals should be treated with antimicrobials even if they don't look sick. Common clinicopathologic abnormalities in septic foals include leukopenia or leukocytosis, increased band neutrophils, hyperfibrinogenemia, low serum IgG (especially persistently low), and hypo- or hyperglycemia. Additional diagnostic evaluation in any foal with suspected sepsis should include aerobic and anaerobic blood cultures, and may include thoracic/abdominal imaging, fecal diagnostics, umbilical ultrasound, radiographs of affected joints/physes, synoviocentesis, or cerebrospinal fluid analysis.

Management:
Antimicrobial Therapy: The most important part of management of septicemia in neonatal foals is early and aggressive antimicrobial therapy. If sepsis is suspected based on evidence of placentitis in the mare, or clinical signs or leukogram abnormalities in the foal, broad spectrum antimicrobial therapy should be initiated immediately without waiting for blood culture results. Parenteral administration is preferable to oral administration, and appropriate initial antimicrobial choices include:

1) a combination of penicillin G and an aminoglycoside
   a. potassium or sodium penicillin – 22,000-44,000 IU/kg IV q 6 hours; or procaine penicillin 300,000 – 500,000 IU/kg IM q 12 hours
   b. amikacin 25 mg/kg IV or IM q 24 hours or gentamicin 6.6 mg/kg IV or IM q 24 hours (may have a greater potential for nephrotoxicity than amikacin in foals);
2) a 3rd generation cephalosporin
   a. ceftiofur 5 mg/kg IV or IM q 12 hours
   b. may be combined with penicillin as above to increase gram positive coverage.
Antimicrobial therapy should be guided by blood culture results and the foal’s clinical response, and should be continued for 7-10 days in foals at risk for sepsis (dystocia, cesearean section, placentitis) or foals with suspected sepsis but no localizing signs of infection. In foals with localizing signs of infection or positive blood cultures, parenteral antimicrobial therapy should be continued until the leukogram is within normal limits, and then followed by oral antimicrobial therapy for an additional 10-14 days. Options for oral antimicrobials include:

1) a potentiated sulfonamide  
   a. trimethoprim-sulfamethoxazole 20-30 mg/kg PO q 12 hours  
   b. may be combined with rifampin (5-10 mg/kg PO q 12 hours) to increase tissue penetration  
2) a 3rd generation cephalosporin  
   a. cefpodoxime (10 mg/kg PO q 8-12 hours)  
3) a macrolide in combination with rifampin as above  
   a. erythromycin 25-30 mg/kg PO q 6-12 hours  
   b. or azithromycin 10 mg/kg PO q 24 hours for 5 days, then q 48 hours  
   c. or clarithromycin 7.5mg/kg PO q 12 hours  

In general, bacteriostatic agents should be avoided if possible in foals with suspected septicemia.

**Fluid Therapy:** Neonatal foals have a higher maintenance fluid requirement (80-100 ml/kg/day) than adult horses, but milk consumption and any plasma administered should be included in calculation of a daily fluid plan to avoid overhydration. Foals also have difficulty handling an excessive sodium load, so 0.45% sodium chloride/2.5% dextrose is usually a suitable maintenance isotonic fluid choice. Administration of 2.5-20% dextrose solutions may be necessary for correction of hypoglycaemia, depending on the severity. Rapid or large volume administration of dextrose in water, even if isotonic, can result in significant electrolyte derangements and should be avoided. Small to moderate amounts of dextrose may be added to isotonic balanced polyionic crystalloids, but if large-amount or long-duration dextrose supplementation is necessary, parenteral nutrition should be considered. Foals that cannot nurse or be fed enterally should be maintained on parenteral nutrition or IV fluids supplemented with dextrose at 4-8 mg/kg/min, as neonates have very limited glucose reserves. Hypovolaemic or hypoxic foals are often hyperlactataemic; fluids containing lactate such as lactated Ringers solution should be avoided in these cases. Foals with partial or complete failure of transfer of passive immunity should receive equine plasma, at 20-40 ml/kg IV. Due to ongoing immunoglobulin consumption during sepsis, repeat plasma transfusions may be required to fully resolve hypogammaglobulinaemia.

**Nutritional Support:** Foals that are willing and able to nurse and have no signs of colic should be allowed to nurse the mare freely. Orphan foals can be fed mare’s milk or equine milk replacer via a bottle or pan every 2-4 hours. Weak or recumbent foals that are unable to nurse adequately should be fed via an indwelling nasogastric tube. Small bore tubes that will allow the foal to nurse around them once stronger can be maintained for long periods of time (weeks). In general, neonatal foals should receive 20-25% of their body weight in milk per day divided into ~12 feedings to grow
adequately. However, some clinicians elect to rest the GI tract in foals with severe FPT, leukopenia, diarrhea, or suspected sepsis for 12-24 hours, as milk can be an excellent medium for pathogenic bacterial growth in a poorly defended GI tract. Small volume feedings (2-5% of body weight in milk/day) are then initiated after plasma transfusion and broad spectrum antimicrobial therapy is initiated. If these small volume feedings are tolerated, the foal is then gradually increased to a full milk ration over the next several days. Foals that have not been nursing for any other reason should also be gradually reintroduced to feeding in this manner. Parenteral nutrition should be considered in any foal that cannot tolerate enteral feeding for > 24-48 hours.

**Other Therapy:** Some clinicians routinely administer acid suppressants (proton pump inhibitors and/or H2-blockers) and/or gastroprotectants (e.g. sucralfate) to all sick neonatal foals. However, studies have not documented convincing evidence for acid-induced gastric ulceration or shown benefits for gastroprotectant therapy in sick neonatal foals, and an acidic gastric pH is an important intestinal defense mechanism. Thus, use of acid suppressants and gastroprotectants should be limited to foals with documented gastroduodenal ulcer disease (see below), foals on prolonged non-steroidal anti-inflammatory therapy, foals with severe diarrhea, colic, or those that are not nursing for prolonged periods. General supportive care should include maintenance of recumbent foals on clean, dry bedding to minimize the development of decubital ulcers. Recumbent foals should be maintained in sternal recumbency as much as possible to improve ventilation and minimize atelectasis. In addition, the foal should be assisted to stand and then returned to the opposite recumbency every 2 hours. Corneal lubrication should be applied several times a day to recumbent or obtunded foals to prevent corneal ulceration.

**Neonatal Encephalopathy (NE)**

**Presentation and Diagnosis:**
Signs of NE in foals can range from a bright, alert foal that has difficulty nursing to recumbency, coma, and seizures. Foals with septic shock or bacterial meningitis can present with similarly altered mentation or neurologic deficits, so blood culture should be performed in all foals with neurologic signs, and cerebrospinal fluid analysis considered in foals with deficits that persist after fluid resuscitation. A diagnosis of NE can be made when sepsis and bacterial meningitis are ruled out based on clinical signs or the above diagnostic testing. Advanced CNS imaging (CT or MRI) may be performed to rule out congenital structural CNS anomalies. Some foals with NE have extensive ischemic damage, CNS hemorrhage or necrosis evident on MRI, but gross abnormalities are typically not identified with MRI in most cases.

**Management:**
The most important component of the management of NE involves supportive care for the foal while allowing the CNS time to heal and recover. NE often occurs in conjunction with septicemia, failure of transfer of passive immunity, or dystocia-related complications such as fractured ribs, so concurrent management of these conditions is
also important. Finally, some clinicians advocate the use of specific therapeutic modalities to decrease CNS inflammation and edema, though given the lack of definitive understanding of the specific pathogenesis of NE in foals, use of such agents is controversial.

In general, supportive care for foals with NE is very similar to management of sepsis, with antimicrobial prophylaxis, fluid and nutritional support, and general nursing care. Specific anti-inflammatory therapy may include NSAIDS (flunixin meglumine 0.5-1.1 mg/kg IV q 24 hours) or dimethyl sulfoxide (1.0 g/kg diluted in IV fluids (as a <10% solution) q 12-24 hours) as a free radical scavenger. Mannitol (0.25-1.0 g/kg IV q 6-12 hours) may be used to decrease cerebral edema, but due to its diuretic effects requires careful monitoring of hydration status and electrolyte balance.

Other therapy that may be beneficial in some cases includes:
- Thiamine – 10 mg/kg in IV fluids q 12-4 hours to support neuronal metabolism
- Vitamin C – 0.5 – 1.0 g/kg PO q 24 hours as an anti-oxidant
- Vitamin E – 10-20 IU/kg PO q 24 hours as an anti-oxidant
- Caffeine – 10 mg/kg PO loading dose, then 2.5-3 mg/kg q 24 hours as a general CNS stimulant in somnolent foals with irregular respiratory patterns or periods of apnea
- Magnesium sulfate – 20 mg/kg in IV fluids q 24-48 hours as a mild relaxing agent in hyperesthetic/hyperreactive foals

**Neonatal Isoerythrolysis (NI)**

**Presentation and Diagnosis:**
Foals with NI usually present at 24-72 hours of age (or occasionally older) and are most often from multiparous mares (but not always!). Donkey and mule foals appear to be at increased risk for development of NI. Owners typically report non-specific signs of weakness, increased recumbency, or lethargy, and may also note discolored urine, icterus, or tachypnea. The presence of tachycardia, tachypnea, and pale or icteric mucus membranes in a neonatal foal warrants suspicion of NI. Rarely foals with severe NI develop profound neurologic deficits and seizures due to kernicterus, which is a very poor prognostic indicator. Variable degrees of anemia may be seen in NI, and in acute hemolysis the degree of anemia does not always correlate with the severity of the foal's signs. A diagnosis of NI is supported by autoagglutination of the foal's blood in an EDTA tube or a positive Coomb's test.

**Management:** Most foals with NI require provision of oxygen carrying capacity, which is most easily provided with a whole blood transfusion. The need for transfusion should be based on clinical signs related to tissue hypoxia (tachycardia, tachypnea, weakness, hyperlactatemia, and venous oxygen pressure < 30 mmHg) rather than a specific hematocrit. Washed red blood cells from the dam are suitable for transfusion to foals with NI, but adequate washing can be time consuming and technically challenging in a field setting. An initial transfusion from a healthy gelding donor can be usually be
performed safely without cross-matching, but major and minor cross-matching is recommended before subsequent transfusions if possible. Most foals require 1-2 liters of whole blood in an initial transfusion, but an appropriate transfusion volume can be calculated with the following formula:

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\text{Desired PCV} - (\text{Recipient PCV} \cdot 0.08 \cdot \text{Body weight in kg}) = \frac{\text{Liters of whole blood}}{\text{Donor PCV}} \text{ needed}
\]

The foal should be prevented from nursing from the mare until about 72 hours of age, and the mare milked out frequently and the colostrum/milk discarded until that time. Nutritional support for the foal during that period can be provided with milk replacer as described above. Additional fluid therapy to maintain normovolemia and support perfusion or administration of dextrose or parenteral nutrition may be necessary in some foals that are severely compromised. Foals with NI are at increased of septicemia, so blood cultures should be submitted and foals should be covered with broad spectrum antimicrobials and monitored carefully.

**Uroperitoneum**

**Presentation and Diagnosis:**
Rupture of the urinary bladder may occur from trauma associated with parturition, but it can also result from mural ischemia or other unknown factors during the neonatal period; thus, foals may present with uroabdomen anytime during the first few weeks of life. Owners may observe the foal straining to urinate or note an abnormal urine stream, but more often the foal presents for inappetance, lethargy or colic. Gross abdominal distension is almost always present and can be severe, and extensive ventral edema may be noted if the urachus is ruptured. The classic clinicopathologic abnormalities associated with uroperitoneum include azotemia, hyponatremia, and hyperkalemia, but these can be variable, especially if the foal is receiving intravenous fluids. A large amount of hypoechoic free peritoneal fluid is seen with trans-abdominal ultrasonography, and can be confirmed as urine by documentation of a creatinine concentration > 2 times the plasma creatinine concentration. Occasionally a defect in the bladder wall can be identified ultrasonographically, but failure to do this does not exclude a diagnosis of uroperitoneum.

**Management:**
Management of uroperitoneum in almost all cases requires surgical correction of the mural defect in the bladder. However, many foals with uroperitoneum present with profound fluid and electrolyte derangements and require stabilization prior to surgery. Intravenous fluid therapy with 0.9% sodium chloride with 2.5-5% dextrose is acceptable to correct the most typical electrolyte derangements in most cases. However, 0.45% sodium chloride + 2.5% dextrose may be indicated in foals with severe and prolonged hyponatremia and severe hyperkalemia, to avoid increasing blood sodium concentrations too rapidly. If possible, the urine should be slowly drained from the peritoneal cavity to decrease abdominal distension and improve azotemia and
electrolyte derangements, but concurrent fluid administration is imperative to prevent severe hypovolemia. In most cases, a teat cannula can be used to sufficiently drain the abdomen, though herniation of some omentum when the teat cannula is removed is common. If this happens, cutting the herniated omentum at the level of the skin with sterile scissors and placement an antiseptic wrap over the site usually prevents further complications. Broad spectrum antimicrobial therapy should be provided to all foals with uroperitoneum, and the foal evaluated closely for concurrent septicemia. If surgical correction of the bladder rupture is delayed to stabilize the foal, an indwelling urinary catheter should be placed to keep the bladder decompressed. Occasionally, in foals with very small bladder defects, maintenance of bladder decompression with an indwelling urinary catheter for several days may permit adequate resolution without surgery.

**Meconium Impaction**

**Presentation and Diagnosis:**
Foals with meconium impaction usually present with signs of colic during the first 24-48 hours of life. Tenesmus and tail flagging are typically observed, and moderate to severe gross abdominal distension usually develops fairly rapidly. Digital rectal examination may reveal firm fecal material in distal impactions, but the absence of meconium on rectal examination does not exclude the possibility of a more proximal impaction. Consistent clinicopathologic abnormalities are not usually present, but foals with meconium impaction frequently have concurrent failure of transfer of passive immunity and thus are also at increased risk for septicemia. Meconium is typically visualized within the small colon and rectum on abdominal radiography, and associated proximal intestinal gas distension may be quite severe. Meconium can also be visualized within the distal colon with trans-abdominal ultrasonography, appearing as highly echogenic material within the intestinal lumen. Abdominocentesis and peritoneal fluid analysis may be performed to rule out other causes of abdominal distension in foals (uroabdomen, small intestinal strangulation), but are not necessary in all cases.

**Management:**
Most foals with meconium impaction can be managed medically with laxative therapy and supportive care, but surgical resolution is occasionally necessary. Initial medical management should include prevention of nursing by muzzling the foal and intravenous polyionic fluids at 80-100 ml/kg/day to maintain hydration. A higher fluid rate may be needed in foals that present with dehydration from prolonged colic and decreased nursing. Glucose support should be provided with 2.5-5% dextrose in IV fluids, and the foal monitored closely for hypoglycemia and electrolyte derangements (hypernatremia and hypokalemia are most common). Laxative therapy should include 200-400 ml mineral oil (for an average 50 kg foal) every 12-24 hours. An enema of 500 ml warm soapy water (a few drops of dish detergent in 500 ml water) administered via gravity flow via a red rubber catheter inserted no further than ~4-6 inches into the rectum may be helpful for foals with distal impactions. If the impaction is extensive on radiographs or fails to resolve with the above therapy, a retention enema with 4% acetylcysteine may be helpful. To prepare, add 40 ml 20% Mucomyst™ to 160 ml water, place a lubricated
foley catheter (30 Fr with a 30ml balloon) ~3 inches into the rectum and inflate the balloon gently, slowly infuse 120-180 ml of acetylcysteine solution into the rectum via gravity flow, occlude the catheter tip for 15-45 min, then deflate the balloon and remove the catheter. Enema fluid should never be pumped or administered by force to minimize the risk of rectal or small colonic rupture, and no more than 2-3 soapy or acetylcysteine enemas should be administered as severe rectal mucosal edema can occur. Analgesic therapy with flunixin meglumine (0.5 - 1.1 mg/kg IV q 12-24 hours) and sedation with diazepam (0.05-0.2 mg/kg IV) and/or butorphanol (0.01-0.04 mg/kg IV or IM) may be used to provide pain relief during medical therapy. Occasionally a constant rate infusion of lidocaine (loading dose of 1.3 mg/kg IV, then 0.05 mg/kg/min IV CRI) can be used for additional analgesia in foals that remain colicky despite the above therapy, but the foal should be monitored very closely for signs of lidocaine toxicity (ataxia, altered mentation, seizures). In foals that remain intractably colicky or fail to pass meconium despite aggressive therapy, exploratory laparotomy is indicated.

References are available from the author.