Clinical Snapshot

A 2-hour-old Thoroughbred colt presented to the clinic with a history of seizures since birth. The mare had placentitis and was treated with antimicrobials, NSAIDs, alfenogest, and pentoxifylline during the last few weeks of gestation. The foal was full-term, weighed 90.2 lb (41 kg), and was in poor body condition (2 of 9). There were no signs of prematurity. On initial clinical examination, the neonate had abnormal mentation, a heart rate of 150 beats/min, a respiratory rate of 56 breaths/min, and a rectal temperature of 98.1°F (36.7°C). The mucous membranes were pale and injected. There were no palpable peripheral pulses, and the extremities were cold. The indirect blood pressure was 88/56 mm Hg, the arterial oxygen saturation was 81%, and the $P_{co_2}$ was 44 mm Hg. The complete blood count and serum chemistry profile revealed leukopenia (2800 leukocytes/µL), hypoglycemia (54 mg/dL), and a low serum IgG concentration (<100 mg/dL). The foal exhibited seizure activity (i.e., severe extensor rigidity that was more pronounced in the forelimbs, opisthotonus, nystagmus) and could not sit in a sternal position or rise. The foal continued to exhibit seizures despite aggressive medical therapy and was euthanized due to progression of the seizures. A necropsy was performed.

1. What is your differential diagnosis based on the foal’s history and the clinical findings?
2. How would you have treated this neonate?
3. How would you have proceeded if the seizure activity had continued despite treatment and the owner had wanted to proceed?
4. Based on your diagnosis, what central nervous system abnormalities would you expect on the postmortem examination?

See page 62 for answers and explanations.
Clinical Snapshot

Answers and Explanations Case Presentation #2

SEE PAGE 60 FOR CASE PRESENTATION.

1. Differential diagnosis: septicemia; hypoxic ischemic encephalopathy; neonatal encephalopathy; or perinatal asphyxia; metabolic abnormalities; central nervous system trauma; developmental anomalies; and toxicosis.

2. The foal was treated with the following:
   - Intravenous bolus of Normosol R (40 mL/kg)
   - Intravenous polyionic crystalloids with 2.5% dextrose (4 mL/kg/hr)
   - Hyperimmune plasma (20 mL/kg IV)
   - Antioxidants: dimethyl sulfoxide (0.8 g/kg IV) and allopurinol (45 mg/kg per nasogastric tube)
   - Vitamins: ascorbic acid (5 g) and thiamine (500 mg) IV (added to crystalloids)
   - Antimicrobials: amikacin (25 mg/kg IV q24h) and potassium penicillin (20,000 U/kg IV q6h)
   - Anticonvulsants: magnesium sulfate (0.025 g/kg/hr); diazepam (0.1 mg/kg twice in the first 2 hours); and midazolam (0.04 mg/kg/hr via constant-rate infusion), which was increased to 0.08 mg/kg/hr after 2 hours and 0.12 mg/kg/hr after another 2 hours
   - Intranasal oxygen insufflation (6 L/min)

3. If seizure activity had continued despite treatment, reasonable options would have included skull radiography, ultrasonography of the cisterna magna, cerebrospinal fluid collection, computed tomography, magnetic resonance imaging, and electroencephalography.

4. In this case, postmortem examination revealed severe cerebellar hypoplasia. While this is a rare finding in this type of presentation, antemortem diagnosis could have been made using magnetic resonance imaging.