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Case reports

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Vestibular syndrome.
A 13 day old filly was admitted with a history of suspected trauma. On admission the foal was recumbent and blood was noted to be coming from the right ear with a marked turning of whole head to neck to the right and a head tilt to the right. Nystagmus and abnormal positioning of both eyes was present. The filly was quite bright and aware of her surroundings and would make unsuccessful attempts to stand. Hematology was normal.

Radiographs of the head revealed differences between the petrous temporal bones and although a distinct fracture line could not be identified a fracture or multiple fractures was suspected. Initial treatment consisted of broad spectrum antibiotics, NSAID’s, corticosteroids, fluid therapy and supportive care.

The filly deteriorated neurologically over the following two days and on the third day an ultrasound examination of the atlanto-occipital space was performed which revealed a large blood clot. The filly at this point was unaware of her surroundings, had bilateral dilated pupils and was unable to stand. Given the deterioration in her condition, the uncertain and likely poor prognosis and costs of ongoing indefinite therapy, it was decided to euthanize the filly.

Necropsy examination demonstrated multiple fractures of the right petrous temporal bone, blood clots with the cranium and proximal spinal canal.

Vestibular syndrome 2.
This 3 month old colt with a history of suspected trauma and head tilt to right was examined at the farm. On examination the foal was able to stand and follow the mare, was bright and alert, nursing but could stumble and fall and would circle to the affected side if agitated or blindfolded. Bilateral nystagmus was present and the right eye was in an abnormal position. The foal was treated with broad spectrum antibiotics, anti-inflammatories, corticiosteroids, fluid therapy and supportive care.

The foal was re-examined 3 weeks later and showed improvement but still had a marked head tilt. He made a full recovery over the following 6 weeks.

Tetanus
This colt foal was admitted at 10 days of age with neurologic signs and signs of sepsis. On presentation the foal had a very stiff and stilted gait with a raised tailhead. The foal was very sensitive to touch and auditory stimuli, both pupils were dilated and unresponsive, there was a fixed flaring of the nostrils more marked initially on the right, tapping on the forehead would induce prolapsed of the third eyelids and palpation of the hindlimbs would initiate tense muscular contractions. Also, there was marked swelling and effusion of the right carpus and a marked leucocytosis (18.6 K/µL with a predominant neutrophilia). An initial diagnosis of septicemia with tetanus was made.

Treatment consisted of broad spectrum antibiotics, NSAID’s, omeprazole, tetanus antitoxin,
muscle relaxants and supportive care (including maintenance in a darkened stall with ear plugs to minimize external stimuli).

The third day it was noted that the foal had poor GI motility likely related to the sepsis, enteral feeding was stopped and TPN was started. On day 9 the foal was offered some milk in a bucket. The foal was willing and able to nurse at this time but the extension of the neck required to place the foal in a nursing position would induce tetanic spasms so we opted for bucket feeding which could be performed without overly stressing the foal.

The antitoxin was discontinued on day 10. On day 11 the foal now had sufficient muscle relaxation to be able to nurse and the muscle relaxants were discontinued. The foal was now going outside for short periods.

**Mediastinal Abscess**
This 4 month old filly was admitted with a history of chronic (1 month) accumulation of thoracic fluid that had previously cultured positive for R.equi. The fluid had been drained with a teat cannula on three occasions, but sequential thoracic ultrasounds revealed progressive fluid accumulation.

On admission the foal was noted to be dull but was responsive, she was in poor body condition despite a reported good diet. She had a marked leucocytosis (21.00K/uL) and hyperfibrinogenemia (1168 mg/dL). Ultrasonographic examination of the thorax revealed a large amount of free thoracic fluid to approx 8cm above the level of the shoulder. The fluid was anechoic with no evidence of fibrin accumulation. There was consolidation of the ventral lung margins. A caudal displacement of the heart with rotation to the right was also noted during the ultrasound examination. Further examination revealed a mass occupying much of the cranial mediastium with adhesions to many of the vessels in the area. With a prior positive culture for R.qui an abscess was suspected. A sample of the mass was taken under standing sedation which yielded a caseous material consistent with an abscess.

Given the dimensions of the abscess (estimated to be at least 13cm in diameter), its relationship to vascular structures and difficulty in accessing the site the foal was given a poor prognosis. It was felt that debulking the abscess would give the foal the best chance of survival but such a surgery would be technically difficult given the location. A drain was placed in the abscess cavity following surgery and lavages were performed a number of times of daily.

Serial thoracic ultrasounds demonstrated a decrease in the volume of thoracic fluid. The drain was removed after 4 days when it was no longer productive. The foal remained in the hospital for a total of 3 weeks after which it was discharged to continue antibiotic therapy at home. At follow-up 4 months later the foal was reported to be doing well and growing normally.

**Hypoplasia of colon and cecum**
A 5 day old foal was admitted with a history of colic. The referring vet had noted that the foal had a short episode of diarrhea 2 days earlier and on the current day had a small amount of gastric reflux on passage of a nasogastric tube.
On admission the foal had a HR of 92BPM, a respiratory rate of 28BPM, a rectal temperature of 39.4C with congestion of the mucous membranes. Notable hematology results were: HCT 37.8% and WCC 20K/ul. Abdominal ultrasound revealed marked distension of the small intestine (between 3-4cm) with fluid contents, marked distension of the stomach (2L of reflux was obtained immediately and 450ml more over the following 40mins). There was no motility evident and no signs of colic. Visualisation of the large bowel was not possible but this was considered to be related to the extensive and marked distension of the small intestine.

An initial diagnosis of enteritis/enterocolitis was made. The foal was placed on broad spectrum antibiotics and fluid therapy with prokinetics. Repeat abdominal ultrasound examination later that evening revealed further distension of the small intestine. The foal had minimal reflux and no signs of colic, but no motility was present. Through the night and following morning the foal continued was unresponsive to prokinetics. At this point it was thought that the foal may have a primary motility disorder. On the afternoon of the second day the foal started to become moderately painful and another abdominal ultrasound was performed and on this occasion a small intestinal intussusceptions was noted. This intussusceptions was likely produced by the administration of prokinetics. The foal was taken to surgery where a hypoplasia of the colon and cecum was diagnosed. An intussusception of the ileum was present as diagnosed by ultrasound. The foal was euthanized.
CASE 2a and 2b

History/Physical Exam: 1 day old foal presented with a history of increased respiratory rate of 80 BPM with an abdominal lift. The foal was born as a dystocia. The foal is still nursing but immediately lies down after nursing. The remainder of the vital parameters are of normal limits. The mucus membrane color was pink with a normal capillary refill time. Formed manure has been noted.

Complete Blood Cell Count/Fibrinogen and Serum Biochemistry are unremarkable

Differentials:
- Meconium Impaction
- Aspiration/Bacterial Pneumonia
- Enterocolitis
- Fractured Ribs
- Diaphragmatic Hernia

Ultrasound Exam:
- Case 2a: Fractured Ribs
- Case 2b: Diaphragmatic Hernia with Fractured Ribs

Treatment Plan:
- 1) Stabilization of the Rib Fractured
- 2) Repair the diaphragmatic hernia

DISCUSSION:
Signs that should direct an examiner’s attention to the possibility of costal fractures include groaning or grunting in the foal, plaques of subcutaneous edema overlying the ribs or along the ventrum of the thorax, especially behind the elbows, and flinching when the rib area is palpated. Audible or palpable crepituation or a clicking sensation when the hand is gently pressed over an affected area is common. Nearly all the fractured ribs in the foals studied were broken at or within several centimeters of the costochondral junction. The distal rib fragment tends to displace axially and is usually the instrument of myocardial injury when it occurs. Subcutaneous emphysema is occasionally present, and though not recorded frequently in association with rib fractures in the records examined in this study, the distinctive presence of subcutaneous emphysema, particularly over the sides of the thorax or in the axillary area, should alert the examiner to the possibility of a broken rib. Wetting of the hair with alcohol or water significantly increases the visibility of chest wall indentation and plaques of edema overlying injured ribs. In some cases, it is necessary to clip the hair in order to obtain the best images, but in our hands, alcohol alone as a wetting agent is often sufficient. Though fractured ribs are attended by significant pain in humans, after several days of hospitalization many foals will demonstrate normal vigor and play activity in the stall, even when multiple broken ribs are present.
For this reason, analgesic administration should be judicious in affected foals, as any movement or jostling of the thorax can cause rib fragments to lacerate internal vascular structures or the heart, resulting in sudden death. Not surprisingly, myocardial laceration or puncture were unequivocally fatal events in this study. This complication occurred equally among fractures involving both the left and right rib arcades. Sudden death is not an uncommon feature of broken ribs in foals, and clients are warned frankly of this unfortunate possibility when foals are discharged from the hospital. Recommendations for two to three weeks of confinement to a small space, with sedation if necessary, are typically included in the treatment orders for discharged foals.

Flail chest occurs when several consecutive ribs are fractured, leading to an incompetent segment of chest wall. The foal’s respiratory efforts are hampered by the failure of the affected rib arcade to lift and participate in the process of creating negative pleural pressure for inspiration. During expiration, the failure of the affected chest segment to collapse as a unit likewise impedes the normal development of positive airway pressure and timely exhalation. When a flail chest is present, the involved segment of chest wall will sink inwards during the inspiratory excursions of the abdomen and diaphragm.

Foals presenting to the Hagyard Equine Medical Institute’s NICU with rib fractures and concomitant pulmonary injury are generally managed by encouraging lateral recumbency with the affected side down. When pulmonary contusion is significant, positioning the foal with the undamaged lung on top minimizes ventilatory embarrassment. Foals which struggle excessively or are active enough when ambulatory to warrant fear of displacement of a fracture fragment may be sedated to encourage immobility and rest. These generalizations are subject to modification in foals with pneumonia associated with sepsis, in which sternal positioning is required for optimal gas exchange and overall well-being. Rib fractures can lacerate intercostal blood vessels, the internal thoracic artery, may bleed from the fractured bone ends, or tear intercostal muscles. Contusion and extravasation of blood in the thoracic wall was a finding in several foals in this study, and the pulmonary artery was lacerated in one case. The resultant hemothorax may compromise the foal by leading to cardiovascular collapse from intracavitary volume loss and shock, and by the tamponade effect on the lungs of fluid and positive pressure in the pleural space. When thoracic auscultation or ultrasound demonstrated the presence of hemothorax, the pleural blood was not removed if the foal did not experience significant dyspnea, to allow for autotransfusion and the ameliorating effect of positive pleural pressure on continued bleeding. When respiratory compromise was judged to be present as a result of hemothorax, thoracentesis was performed to remove some or all of the blood in the pleural space. In the three cases in this survey when the foals presented with hemorrhagic shock secondary to blood loss into the pleural space, blood transfusion along with the judicious removal of free blood successfully stabilized these foals. Two of the three foals treated with blood transfusions and thoracentesis survived to discharge. Foals were also treated with broad-spectrum antibiotics, intravenous fluid support, and the attendant treatments and instrumentation typical in foals suffering the effects of birth asphyxia and/or sepsis, since most of the affected foals had these conditions as the primary presenting complaint.
The feasibility of surgical treatment of fractured ribs has been pursued in a number of foals at this practice (Hagyard Equine Medical Institute) since 1999, when the injuries involve ribs which are in proximity to the heart and when the foal’s value dictates the procedure to be feasible. The results of a survey of foals managed by the application of dynamic compression plates to fractured ribs are currently being assembled for profiling in a future presentation (Robert Hunt and F.T. Bain, personal communication).

The long-term monitoring of foals successfully discharged from the hospital and returned to farm care is done with serial ultrasound imaging. The formation of a hematoma or thrombus at the broken bone ends is typical of a rib fracture injury in the acute stages. Serial visits to image the site at the farm document the evolution of this finding to bony callus formation, and eventual smoothing and remodeling of the callus with time. Four to six weeks are considered to be necessary for stabilization of the thoracic wall following rib fractures. Deformation of the lung surface and thickening of the visceral pleura may be semi-permanent to permanent findings in the long-term assessment of these foals. Some chronic fractures may be incidental findings when the foal is being examined at an older age. For example, previous fracture sites and deformation of the visceral pleura by the bony callus are an occasional finding in older foals being imaged for rhodococcal pneumonia or other thoracic disease.

Though previous studies have documented fractured ribs in foals to be benign and unassociated with significant complications, in the population of neonatal foals hospitalized at this referral center, the injury is a significant cause of morbidity and mortality. Foals should be examined for this injury in post-partum assessments, and foals born in complicated or difficult deliveries should be especially scrutinized. In this study colts were more commonly affected than fillies, a finding which may be related to size and resultant birth trauma. A higher number of involved ribs was associated with increased risk of death in our foals, a finding which corroborates findings in similar studies of this injury in children. Though not documented by this study, the human propensity to assist in the birth of foals by applying traction to the forelegs of foals during the delivery process may also play a role.
CASE: Hypotension and Kidney Compromise in 3 day old foal

HISTORY: The thoroughbred filly was born uneventfully 3 days before presentation. The foal developed diarrhea 6 hours before presentation. The referring veterinarian administered Ceftiofur and Metronidazole before arrival.

PHYSICAL EXAM:
On arrival the filly was ambulatory, weak and lethargic. The heart rate was 100 BPM, Respiratory rate of 30 BPM with a temperature of 102.5 F. The foal was not nursing. The abdomen was “tucked up” with ice cold distal limbs. Bilateral enophthalmus were noted. The mucus membrane color was injected with a 4 sec capillary refill time.

INITIAL BLOOD WORK:

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
<th>Normal Range</th>
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<tbody>
<tr>
<td>WBC</td>
<td>7600/μl</td>
<td>Neutrophils 77%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Bands 8%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Lymphocytes 15%</td>
</tr>
<tr>
<td>Fibrinogen</td>
<td>300 mg/dl</td>
<td>(133-140 mg/dl)</td>
</tr>
<tr>
<td>Neutrophils</td>
<td>77%</td>
<td>(2.5-5.0 mg/dl)</td>
</tr>
<tr>
<td>Bands</td>
<td>8%</td>
<td>(25-32 mg/dl)</td>
</tr>
<tr>
<td>Lymphocytes</td>
<td>15%</td>
<td>(11-26 mg/dl)</td>
</tr>
<tr>
<td>HCT</td>
<td>45.9%</td>
<td>(0.8-1.8 mg/dl)</td>
</tr>
<tr>
<td>IgG</td>
<td>800 mg/dl</td>
<td>(11-26 mg/dl)</td>
</tr>
<tr>
<td>Na</td>
<td>119 mEq/L</td>
<td>(133-140 mEq/L)</td>
</tr>
<tr>
<td>K</td>
<td>8.6 mEq/L</td>
<td>(25-32 mEq/L)</td>
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<tr>
<td>CO2</td>
<td>12 mEq/L</td>
<td>(11-26 mg/dl)</td>
</tr>
<tr>
<td>BUN</td>
<td>52 mg/dl</td>
<td>(25-360 U/L)</td>
</tr>
<tr>
<td>Creatinine</td>
<td>7.9 mg/dl</td>
<td>(11-26 mg/dl)</td>
</tr>
</tbody>
</table>

ULTRASOUND ABDOMEN:
- Abdomen: Large Colon Fluid Distention
  - Right Kidney was small as was the bladder size
  - No free fluid noted

PROBLEM LIST:
1. Hypotensive Shock
2. Colitis
3. Azotemia

INITIAL PLAN:
- Resuscitative colloids Hetastarch 10/ml/kg bolus
- Fluid Therapy (4ml/kg/hr) utilizing Normosol R with Dextrose to make the fluids a 5% dextrose solution with 75 meq Sodium bicarbonate per liter of fluids administered
Once hydrated then started on Diuretics

- Mannitol (1 gram/kg IV TID)
- Furosemide 0.25 mg/kg IV TID

- Antimicrobials to aid in decreasing the chances of Bacterial Translocation
  - Ceftiofur 4mg/kg IV TID
- Metronidazole was discontinued after the Clostridium perfringens and difficile ELISA results were negative
- Rotavirus POSITIVE on ELISA

Re-evaluation of Blood Work (12 hours after arrival)

- Electrolytes
  - Na 125 mEq/L (133-140 mEq/L)
  - K 7.6 mEq/L (2.5-5.0 mEq/L)
  - CO2 8 mEq/L (25-32 mEq/L)
  - Creatinine 8.4 md/dl (0.8-1.8 mg/dl)

Revised Plan:

- Animal still has not started to urinate and is still febrile 103.5F
- Increased mannitol to QID and Furosemide to 1mg/kg IV QID
- Increase Sodium Bicarbonate to 100 meq per liter of fluid given

Re-evaluation of Blood Work (24 hours after arrival)

- WBC = 8500/μl
- Fibrinogen 300 mg/dl
- Neutrophils 81%
- Bands 5%
- Lymphocytes 14%
- HCT = 38.10%
- Total Protein 4.8 g/dL (Albumin 2.5 g/dL)
- Na 127 mEq/L (133-140 mEq/L)
- K 6.3 mEq/L (2.5-5.0 mEq/L)
- CO2 14 mEq/L (25-32 mEq/L)
- BUN 64 mg/dl (11-26 mg/dl)
- Creatinine 8.6 mg/dl (0.8-1.8 mg/dl)
- ALK Phosphorus 979 U/l (25-360 U/L)
- CK 696 U/L (67-377 U/L)

Interpretation after 24 hours of Hospitalization:

- Poor perfusion (Hypotension) characterized with worsening of the azotemia and increased CK levels
- Animal still spiking low grade fevers
  - Will wait another 24 hours for the antimicrobial to reach steady state before deciding to change the antimicrobial therapy
- Now has Diarrhea. Starting to nurse more aggressively
PLAN:

- Maintain current crystalloid therapy as 5% dextrose with 100 meq of Sodium Bicarbonate per liter of fluids
- Add Vasopressors Dobutamine and Dopamine CRI Infusion
  - 3 μg/kg/min
  - Add 200 μg of Dopamine and 250 μg of Dobutamine to 500ml Saline and administer at 0.45 ml/kg/hr equals approximately 3 μg/kg/min

48 hours of Hospitalization

WBC = 6200/μl  Fibrinogen 300 mg/dl
   Neutrophils 89%
   Bands 5%
   Lymphocytes 11%

HCT= 36.10%

Total Protein 4.6 g/dL (Albumin 2.5 g/dL)

Na 131 mEq/L (133-140 mEq/L)
K 4.1 mEq/L (2.5-5.0 mEq/L)
CO2 16 mEq/L (25-32 mEq/L)

BUN 63 mg/dl (11-26 mg/dl)
Creatinine 6.8 mg/dl (0.8-1.8 mg/dl)

ALK Phosphorus 926 U/l (25-360 U/L)
CK 128 U/L (67-377 U/L)

Calcium is at 8.9 mg/dl (11.6-13.2 mg/dL)

Interpretation at 48 hours of Hospitalization

- Animal now has profuse watery diarrhea
- Maintain current crystalloid and vasopressor therapy
- Add Calcium Gluconate at 50 meq/L added to only 3 of the liters

Animal was hospitalized for 6 days and made a full recovery from its diarrhea

- Rotavirus was detected in the feces
CASE 3: Ivermectin Toxicity

HISTORY

A 4-week-old Thoroughbred Foal presented to Hagyard Equine Medical Institute for acute blindness, ataxia, and depression following an overdose of an over-the-counter ivermectin-based de-worming medication. With symptomatic and supportive care, the foal recovered fully and regained its vision.

- Enhances Inhibitory Neurotransmission
  - Glutamate-gated Chloride ion channels
    - Binds Selectively with high affinity in invertebrates (Nerves and Muscle cells)
      - Increase permeability to chloride ions \(\rightarrow\) Hyperpolarization
  - May also interact with other ligand-gated chloride channels
    - For EXAMPLE GABA

Clinical Signs associated with ivermectin Toxicity Include:

- Ataxic
- Depression,
- Forelimb and hind limb ataxia,
- Drooping of the superior and inferior lips
- Muscle fasciculations.
- Bilateral mydriasis, decreased pupillary light reflexes, and absent menace reflexes were evident