Case #45 (569489)

Megan Caudill, DVM

Clinical Pathology Resident
Signalment and History

• 3-month-old male castrated Nubian goat
• Presented for recent onset of anorexia and lethargy
• Housed in fenced yard with a flock of chickens
  • Had been observed ingesting chicken litter
• Sibling had recent onset of pigmenturia, but no other clinical signs
Physical Exam Findings

- Markedly icteric mucous membranes and integument
- Slightly prolonged capillary refill time (~3 seconds)
- Tachycardic (pulse ~180-220)
- Weak, infrequent rumen contractions
## Hematologic and Chemistry Data

<table>
<thead>
<tr>
<th>Test</th>
<th>Result</th>
<th>Reference Interval*</th>
<th>Units</th>
</tr>
</thead>
<tbody>
<tr>
<td>RBC</td>
<td>5.73 (L)</td>
<td>13.6-23.7</td>
<td>x $10^6$/uL</td>
</tr>
<tr>
<td>HGB</td>
<td>4.7 (L)</td>
<td>8.9-13.8</td>
<td>g/dL</td>
</tr>
<tr>
<td>HCT</td>
<td>11.9 (L)</td>
<td>28-44</td>
<td>%</td>
</tr>
<tr>
<td>MCV</td>
<td>20.8</td>
<td>16-22</td>
<td>fL</td>
</tr>
<tr>
<td>MCHC</td>
<td>40.0 (H)</td>
<td>32-34</td>
<td>g/dL</td>
</tr>
<tr>
<td>nRBC</td>
<td>65 (H)</td>
<td>0</td>
<td>/100 WBC</td>
</tr>
</tbody>
</table>

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<thead>
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</tr>
</thead>
<tbody>
<tr>
<td>GGT</td>
<td>215.2 (H)</td>
<td>24-66</td>
<td>U/L</td>
</tr>
<tr>
<td>T. Bilirubin</td>
<td>21.4 (H)</td>
<td>0.1-0.2</td>
<td>mg/dL</td>
</tr>
<tr>
<td>Bile Acids</td>
<td>499.2 (H)</td>
<td>5-69**</td>
<td>umol/L</td>
</tr>
</tbody>
</table>

*Reference intervals obtained from Cornell University Animal Health Diagnostic Center
** Bile Acids reference interval obtained from Marshfield Labs
Asynchronous maturation

nRBC with nuclear fragmentation

Binucleated metarubricyte
Additional Test: Tru-cut Liver Biopsy

Rhodanine stain; left - 40X, right - 100X
Final Diagnosis

Marked hemolytic anemia and secondary blood dyscrasia due to chronic copper toxicosis
Copper Toxicosis

• Most common in sheep -> limited biliary excretion of copper
  • Reported in other species, especially ruminants

• Mechanisms of toxicosis
  • **Dietary excess of copper**
  • Dietary insufficiency of molybdenum
  • Altered biliary excretion of copper
  • Increased hepatocellular copper secondary to pyrrolizidine alkaloids
  • Rare disorders of copper metabolism

• Common dietary sources of copper
  • Improperly formulated rations or mineral supplements*
  • Copper footbaths
  • Fertilizers
  • Fungicides
  • **Swine or poultry manure**

*Permission granted only for viewing on SEVPAC website
Pathogenesis of Copper Toxicosis
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Back to our patient...

• Presumed source of copper – chicken manure on pasture

• Hematologic Abnormalities – likely a combination of pathology and signalment-associated findings
  • “Hyperchromatic” regenerative anemia with Heinz bodies – indicative of hemolysis secondary to oxidative damage
  • Poikilocytosis – very common in young goats and sheep; also anemia in adults
    • Associated with neonatal Hemoglobin C
      • Present in small ruminant neonates longer than other species
      • Adults of these species can revert
  • Dyserythropoiesis – likely due to accelerated erythrocyte production
Acknowledgements

• Many thanks to:
  • Melinda Camus, DVM, DACVP
  • Lisa Kelly, DVM, PhD, DAVCP
  • Elizabeth Howarth, DVM, PhD, DAVCP

References