Hepatobiliary Diseases in the Horse

A PowerPage Presented By

The incidence of hepatobiliary disease in horses is relatively low. However, when it does occur, profound clinical signs may be observed because of the multiple functions that the liver serves. This PowerPage reviews the more common diseases affecting the liver including Acute Serum Hepatitis (Theiler's Disease), Chronic Active Hepatitis, and Pyrrolizidine Alkaloid Toxicosis.

Key Points

- The prognosis for hepatobiliary disease is variable, depending on the type. Generally acute serum hepatitis has a guarded to poor prognosis whereas other forms of hepatobiliary disease have a guarded to fair prognosis
- Acute serum hepatitis is the **most common cause of acute hepatitis and hepatic failure in horses**
- Acute serum hepatitis may be **associated with administration of tetanus antitoxin**
- Etiology of chronic active hepatitis is unknown
- Hepatocytes may appear normal in chronic active hepatitis
- PA toxicity definitive diagnosis based on observation of **fibrosis, megalocytosis and bile duct proliferation**

Acute Serum Hepatitis (Theiler's Disease, Serum Hepatitis)

- Idiopathic acute hepatic disease; unidentified virus or plant toxin are possible causes
- **Most common cause of acute hepatitis and hepatic failure in horses**
- Primarily affects adult horses:
  - Some cases associated with administration of **tetanus antitoxin** or other equine biological 4-10 weeks prior to onset of hepatitis
  - Pathophysiology of disease unknown
- Clinical Signs: Acute depression, anorexia, severe **icterus, photosensitization, hepatoencephalopathy**, pica
- Diagnosis: Microscopic changes include widespread hepatic necrosis and inflammatory cell infiltrate with mononuclear cells and neutrophils in portal areas noted along with proliferation of bile ductules
- Treatment: Non-specific supportive therapy; fluid therapy, dextrose supplementation, anti-inflammatory therapy, nutritional support, control of hepatoencephalopathy
- Prognosis and Necropsy Findings: Favorable if there are no severe signs of hepatoencephalopathy; poor with signs of bleeding or hepatic encephalopathy. Necropsy may demonstrate severe decrease in liver size and severe icterus (Figure 1)

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Figure 1: Necropsy of horse with serum hepatitis (left) demonstrating icterus of the tissues; closer image (right) of the liver that is firm and discolored

Chronic Active Hepatitis

- **Chronic** inflammatory response of liver to unknown stimulus. Potential causes include plant or chemical toxins, environmental chemicals, ascending bacterial infections from biliary tract, or immune mediated disease
- Clinical Signs: Similar to other causes of liver disease but may be more **chronic** in nature. This would include progressive weight loss, intermittent fever, icterus, inappetance and photosensitization
- Diagnosis: **Varying degrees of fibrosis** in the portal areas as well as cellular infiltrate and biliary hyperplasia. **Hepatocytes may appear normal**
- Treatment**:** Corticosteroids, supportive care, fluid therapy, antibiotics if bacterial cholangiohepatitis present
- Prognosis**:** Fair to good if only early hepatic changes with mild fibrosis and response to steroids. Poor if chronic hepatic changes and/or hepatic failure

Pyrrolizidine Alkaloid Toxicity

- **Chronic** progressive intoxication resulting from the **consumption of plants** containing pyrrolizidine alkaloids (PA).
  - More common plants include: *Senecio sp, Crotalaria sp* and *Heliotropium sp*.
- Pathophysiology
  - Toxin absorbed by GI and transported to liver where it is metabolized by hepatocytes to pyrroles
  - Pyrroles cross-link DNA causing an anti-mitotic effect
  - Hepatocytes cannot divide and form **megalocytes** (large cells) as cytoplasm expands without nuclear division
  - Cells subsequently die and are replaced by connective and fibrous tissue
- Clinical Signs: Non-specific signs of liver disease include weight loss, **icterus, and photosensitization**
- Diagnosis**:** History of consumption of PA-containing plants. Definitive diagnosis based on observation of **fibrosis, megalocytosis and bile duct proliferation (hyperplasia)** on microscopy
- Treatment**:** Remove PA-containing plants and provide supportive care
- Prognosis**:** Dependent on degree of hepatic change

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