Treating equine liver disease

Author: Nicola Menzies-Gow

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Nicola Menzies-Gow provides an update on this condition in horses, summarising recent publications and reports, while looking at signs and imaging modalities.

Summary

Recently reported relevant publications are summarised to highlight what is new in this commonly encountered condition. A review of equine primary liver tumours and a case of hepatic lobe torsion have been reported and intestinal hyperammonaemia should be considered as a differential diagnosis in cases with hepatic encephalopathy. The association between bilateral laryngeal paralysis and hepatic dysfunction is also reviewed. The usefulness of serum protein electrophoresis and alterations in clinicopathological indices of hepatic function in colic patients and septic neonates are evaluated. Finally, an ultrasonographic equine starry sky pattern and the use of technecium-99m labelled PEG-liposomes to potentially detect hepatic pathology are described.

Key words

horse, liver, disease, electrophoresis, bilateral laryngeal paralysis, hepatic dysfunction

EQUINE liver disease was first reported in the literature in 1965 and there have been significant advances in our knowledge since that time. Recently reported relevant publications are summarised, highlighting what is new in this commonly encountered condition.

Diseases
Firstly, a review of the literature relating to equine primary liver tumours revealed they are rare, with only 20 cases documented\(^1\). Hepatoblastoma accounts for 11 of these cases occurring in horses, ranging from late-term aborted foetuses to four years of age, and hepatocellular carcinoma accounts for three cases reported in horses one to two years old. The remaining cases comprised cholangiocellular carcinoma, mixed hamartoma and mesenchymal hamartoma.

Secondly, a case of hepatic lobe torsion in a horse has been reported. The four-year-old Belgian mare presented with a one-week history of fever\(^2\). One episode of colic had occurred at the onset of the clinical signs. The horse improved following treatment with flunixin meglumine, but subsequently deteriorated on several occasions. The mare died before further diagnostic procedures were completed. Postmortem examination revealed torsion of the left medial liver lobe, resulting in diffuse hepatic necrosis and severe peritoneal effusion. Liver lobe torsion is an uncommon condition of uncertain aetiology reported in one other horse. The clinical signs are non-specific and include colic, anorexia, tachycardia, tachypnoea and fever. The difficulty in evaluating the anatomical position of the liver lobes in adult horses, other than by surgery, makes antemortem diagnosis extremely difficult. Serum hepatic enzyme activities, as well as haematology changes, were inconsistent in both reported cases.

However, both had increased serum sorbitol dehydrogenase (SDH) activity and total bilirubin concentration. Torsion of a hepatic lobe, although rare, should be considered as a differential diagnosis in horses presenting with signs of colic, fever of unknown origin, and peritoneal effusion, where the aetiology cannot be readily established, especially if serum biochemical analysis results suggest liver dysfunction. Prompt surgical intervention with resection of the twisted liver lobe is the only treatment option.

Finally, a retrospective review of 36 cases of intestinal hyperammonaemia has been published. This condition should be considered as a differential diagnosis in animals presenting with neurologic signs suggestive of hepatic encephalopathy\(^3\). Clinical pathology reveals hyperammonaemia, but hepatic parameters are normal.

**Clinical Signs**

While the common neurologic signs associated with hepatic encephalopathy are welldocumented, the effects on peripheral nerves are less so. An association between bilateral laryngeal paralysis (BLP) and hepatic dysfunction has been reported. In a review of six ponies and four horses that developed sudden-onset BLP in association with hepatic dysfunction, the clinical, clinicopathological, endoscopic and histopathological findings were documented\(^4\). Nine animals had been referred for the investigation of respiratory distress, and one pony had been referred for weight loss before BLP developed. Nine animals were investigated further – all had clinicopathological evidence of liver disease and histological evidence of liver disease. All had one or more of hepatic encephalopathy (n = 8), hyperammonaemia (n = 6) and endoscopic evidence of BLP (n = 9).
Three of the animals had concurrent pituitary pars intermedia dysfunction. Histopathological examination of the intrinsic laryngeal musculature and recurrent laryngeal nerves \((n = 4)\) and of the region of the nucleus ambiguus \((n = 2)\) did not reveal any abnormalities. Three of the animals were euthanised after they had first been examined, and one improved temporarily before the condition recurred. A temporary tracheostomy was performed in six of the animals, five of which subsequently died or were euthanised; one pony recovered.

**Clinical Pathology**

Previously, beta-gamma bridging \((-\gamma\) bridging\) on serum protein electrophoresis has been suggested as being virtually pathognomonic for hepatic disease. A recent study evaluated all serum protein electrophoretograms from clinical patients generated at the University of Georgia between 1994 and 2008 for the presence of \(-\gamma\) bridging.\(^5\) It found the positive predictive value of \(-\gamma\) bridging for hepatic disease was 32 per cent, whereas the positive predictive value was 36 per cent for infectious disease. It concluded that \(-\gamma\) bridging was not pathognomonic for liver diseases, and it is as frequently found with infectious diseases.

A study evaluated the clinical implications of increased liver enzyme activities in hospitalised neonatal foals.\(^6\) Increased serum gamma glutyltransferase (GGT) and/or SDH activity were common in sick neonatal foals, especially foals with sepsis. Foals with increased liver enzyme activities were more likely to be septic, and septic foals were less likely to survive than foals without sepsis. However, increased liver enzyme activities alone were not a useful negative prognostic indicator.

A recent study aimed to determine changes in blood ammonia, bile acid (BA), bilirubin, triglyceride, and glucose concentrations and liver enzyme activities in perioperative colic patients and the association between these laboratory findings and survival to discharge.\(^7\)

Mildly increased blood ammonia concentrations were present in two out of 32 horses at admission. Postoperative blood ammonia concentrations were normal in all 32 horses, but there were increases in liver enzyme activities as well as in BA, triglyceride, and total bilirubin concentrations.

Horses with markedly increased admission BA concentrations and SDH activities did not survive. BA concentrations and SDH activities decreased postoperatively. There was no association between GGT activity and survival; GGT activity remained increased postoperatively. Blood triglyceride concentration was increased in almost all horses postoperatively; horses that did not survive had higher triglyceride concentrations at 24 to 36 hours postoperatively than horses that survived. Thus, changes in hepatobiliary function are common in colic patients, and the results of this study provide further prognostic indices for colic patients.

**Ultrasonography**
Equine hepatic ultrasonography provides information relating to the size of the liver, changes in the hepatic parenchyma and biliary tracts and biopsy site selection. The normal equine liver parenchyma is homogeneous and of medium echogenicity. The bile ducts are not normally visible, while the branching vasculature can be easily seen.

Hepatic congestion, described as an enlarged liver with decreased echogenicity and prominent vasculature, is a diffuse abnormality recognised ultrasonographically. Changes in parenchymal echogenicity have been noted with hepatic necrosis or fibrosis, hepatic lipidosis, granulomatous liver disease, hepatic amyloidosis and cholangiohepatitis. Changes in the biliary tract, including cholelithiasis and biliary distention, have also been described.

An equine starry sky pattern has been described recently, characterised by the presence of numerous small, hyperechoic foci, some of which cast an acoustic shadow, distributed randomly throughout all visible hepatic parenchyma. Cholelithiasis may produce similar ultrasonographic findings to the starry sky liver, with hyperechoic interfaces that may or may not shadow.

However, choleliths do not have a diffuse distribution throughout the hepatic parenchyma, but are found within the biliary ducts. It would appear this pattern is caused by diffuse hepatic granulomas and is likely incidental and not associated with clinically apparent hepatic disease in most horses. The cause of the granulomas is unknown, but there is some evidence to support chronic parasitic infection.

New imaging modalities for the future

Scintigraphy has long been used to detect bone-related disease and has also been applied to gastrointestinal disease. A recent study evaluated the safety and biodistribution of IV technecium-99m-labelled PEG-liposomes in horses. Liposomes are phospholipid nanoparticles that extravasate at sites of increased vascular permeability and have the potential in equine medicine for diagnostic imaging of infectious, inflammatory and neoplastic lesions. Scintigraphic studies revealed a reproducible pattern of organ distribution, and biodistribution studies revealed the highest concentrations of radiopharmaceutical within the lung, kidney, liver and spleen. Thus the technique may be useful for detecting hepatic pathology. Further studies in diseased animals are required.

References


