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Editorials

Liver disease: contributions to diagnostic and prognostic aids

Liver disease in horses has been studied progressively as an organ responsible for a myriad of clinical presentations. Equine hepatic insufficiency was characterised for clinicians (Tennant *et al*. 1973) into categories that included megalocytic hepatopathy, acute hepatic necrosis, chronic hepatitis and cholangitis biliary cirrhosis. These diseases identified plant pyrrolizidine alkaloid toxicity, serum hepatitis (Theiler's disease), idiopathic hepatitis, and biliary cirrhosis associated with cholangitis or choleliths, respectively. These entities have received further definition with plant toxicities that include grasses, clovers and moulds (aflatoxicosis: Cysewski *et al*. 1982); acute bacterial diseases (*Clostridium novyi*, Gay *et al.* 1980; *Bacillus/Clostridium piliformis*, Swerczek *et al.* 1973), iatrogenic iron toxicity (Divers *et al.* 1983); chronic active hepatitis (Carlson 1989); parasitic disease (hydatidosis, Cranley 1982); congenital-genetic compromises, including portal caval shunts, hyperammoniaemia, hyperbilirubinaemia and hyperlipidaemia (Gay *et al.* 1979); neoplasia and cholangitis, cholelithiasis and choledocholithiasis (Schneider 1997; Divers 2002; Pearson 2002; Peek and Divers 2000).

The liver is an organ that seems to be endowed with remarkable compensatory and regenerative ability. Unfortunately, compensatory abilities can delay the clinical presence of liver disease until the proverbial threat of 'too late' may usher in reality, with the onset of more profound clinical signs. While even severe cases of hepatic coma may recover, many will not, due to advanced liver changes that are beyond the regenerative capabilities of the organ. Clinical decompensation can occur when a significant percentage (60–75%) of the liver is no longer functional, either as an acute disorder or when chronic insults to the liver have surpassed the abilities of cellular regeneration. The clinician is challenged in defining those individuals at high risk prior to the onset of liver failure, or the prognosis for patients that are treatable vs. end stage.

Our profession has diagnostic advantages in current veterinary practice to aid in the determination of liver disease. Ultrasonography (Rantanen and Byars 1998) enables us to visualise the organ to determine size and make a subjective assessment of texture, displacement, presence of abscessation, choleliths within dilated canaliculi, and nodular disruptions of capsular and parenchymal tissue more characteristic of neoplasia. The biopsy procedure is no longer a 'blind shot' at the 12–13th right intercostal space; ultrasound allows for a strategic biopsy site with the least patient risk followed by the additional support of microbiology and histopathology for the biopsy sample. Beyond routine chemistry analysis, specialised tests are increasingly more available. Laboratory-available testing of specific enzymes, such as sorbitol dehydrogenase (SDH), fractionated bilirubin, bile salts, blood ammonia concentration, coagulation and functional testing, although not routine, are available for patients with the need for added diagnostic aids prior to biopsy.

The liver is an organ with the reputation for having the 'work ethic' to overcome numerous insults, yet when clinical signs do become apparent, can we determine the extent of damage reliably enough to justify the expense of treatment in a potentially treatable organ failure? When clinical signs are apparent, what tests are appropriate to guide therapy and provide for the most accurate prognosis? What level of routine laboratory tests signal a true insult to the liver to flag further, less routine tests? If liver disease is present, but not clinically apparent in performance horses, do they then perform at less than their maximal capabilities? Are lower levels of enzyme elevations a reflection of true liver disease or merely an adaptation to stresses? What risk factors and clinical signs can alert the practitioner to a suspected liver patient or group of patients? The liver is a complicated and clinically 'fickle'organ with consequences of clinical compromise that initiate a myriad of justifiable questions.

Knowledge of liver disorders has been extended by papers presented in this issue of *EVJ* (pp 534, 542, 549 and 554). Durham *et al.* (p 534) categorised liver biopsies and gave a numerical score that provided a statistical evaluation as a means for objective assessment of survivors and nonsurvivors in mature horses with liver disease. In another report by Durham *et al.* (p 542), a retrospective study was carried out of noninvasive diagnostic criteria in 116 horses with liver disease to determine the validity of prognosis. The statistical analysis and assessments supported noninvasive diagnostic aids as being indicated but of limited value. The severity of clinical signs provided the most useful prognostic assessment.

Case subject details were reviewed by Smith *et al.* (p 549), where the statistical analysis of age, gender and breed was applied for prognostic guidelines to aid in probable decision-making regarding hepatic disease. The

work of Durham *et al.* was then extended to assess the noninvasive diagnostic approach to liver disease against the invasive biopsy (p 554). The reliability of clinical, ultrasound and single or combined clinicopathological laboratory tests for positive or negative predictive values for the presence of liver disease did not compare favourably with biopsy findings. The biopsy retained its gold standard reputation for diagnostic and prognostic information. These papers and their predecessors provide for the practitioner's need to know which individual is most at risk, how to proceed with patient evaluation, and if treatments known to be effective in liver disorders can be justified economically or humanely.

Liver disease in horses remains a difficult clinical challenge in equine practice even while important tools have been provided by the current papers presented in *EVJ*. As clinicians become more knowledgeable, we can alter our mindsets the better to comprehend that liver failure cannot definitively be called failure in an organ capable of regenerative healing. As importantly, we can more objectively apply the aids to determine the difference.

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