EquineReview

Introduction: Peritonitis in horses, equine bleeding and the pathogenesis of gamma glutamyl-transferase activity in Thoroughbred horses are discussed in this month's selection of three recent papers for equine practitioners.

https://doi.org/10.12968/ukve.2021.5.2.82

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Short and long-term survival in horses with peritonitis

In a retrospective study in Vet Surg (2021;50:323–335. https://doi.org/10.1111/vsu.13564) the authors aimed to identify etiology and other findings in horses with peritonitis and report factors associated with survival. They hypothesised there would be significant differences in clinicopathological variables between survivors and non-survivors. Essential inclusion criterion was peritoneal fluid with a white blood cell concentration of >25,000 cells/μL. Veterinary medical records over a 10-year period were examined. Horses presenting with gastrointestinal rupture, peritonitis after abdominal surgery or castration were excluded. The inciting cause, when known, was recorded; cases were considered idiopathic when no aetiology was determined. Long-term follow up was by telephone. A total of 72 horses matched the inclusion criteria. Colic was the most common complaint (47%), followed by fever, inappetence and trauma. Transabdominal ultrasound was performed in 96% of horses. Increased peritoneal fluid was present in 11/38 of horses that underwent ultrasound where other abnormalities were found. Actinobacillius equuli and Corynebacterium pseudotuberculosis were the most common bacteria cultured (24% and 20%, respectively). A definitive diagnosis was made in 61% of cases; the most common cause was infectious agents, followed by trauma, gastrointestinal thickening and eosinophillic peritonitis. Of the surviving horses, 83% survived short-term and 88% were alive at one-year follow-up. Of the horses with a definitive diagnosis, 25% were euthanised compared to 4% of idiopathic cases. Positive peritoneal fluid culture, surgery, increased lactate and high packed cell volume were associated with death.

Genetics of equine bleeding disorders

Dahlgren et al (*Eq Vet J.* 2021; 53:30–37. https://doi.org/10.1111/evj.13290) reviewed the above topic. A basic large animal coagulation panel includes platelet count, activated partial thromboplastin time, prothrombin and fibrinogen. Fibrin degradation products and D-dimers can also be measured, although these are generally less informative for diagnosing genetic bleeding disorders. Factor assays can also be performed. Haemophilia A is diagnosed by measuring factor VIII activity. Von Willebrand factor (vWF) levels and function can be measured. The total plasma vWF quantifies vWF using an ELISA, which is then measured against a standard curve. Ristocetin cofactor is used to measure vWF activity. Thromboelastography evaluates viscoelastic changes in whole blood to evaluate platelet function. Platelet function can be tested in vivo using template bleeding time. Inherited equine disorders affecting the coagulation cascade include haemophilia A, which is caused by mutations in the F8 gene (X linked recessive) that cause e VIII deficiency. This leads to recurrent bleeding, and haematomas have been reported. Prekallikrein deficiency is a rare blood disorder in horses. Inherited disorders affecting platelet function include vWF disease, Glanzmann's thrombasthenia and atypical equine thrombasthenia. The above diseases are reported to cause epistaxis and potentially could contribute to the heritability of exercise-induced pulmonary haemorrhage.

Pathogenesis of high serum gamma-glutamyl transferase activity in Thoroughbreds

High serum γ-glutamyl-transferase (GGT) activity is thought to be a marker of maladaptation to training and possibly poor performance in racehorses. The cause has not yet been established. Horses do not typically have increases in other liver specific enzymes. Mann et al (Eq. Vet. J 2021, https://doi.org/10.1111/evj.13435) aimed to investigate possible metabolic and infectious causes for the high GGT syndrome, using a pilot case control study and nested case control study. In the pilot case control study, 7 case samples and 7 control samples were used. Metabolomics testing was performed to explore the metabolic background and possible differences between cases and controls. In a nested case control study, 27 case control pairs (n=124) were submitted for metabolomic analysis of 214 metabolites. All samples were tested for the presence of equine hepacivirus or equine parvovirus-hepatitis by end-point reverse transcriptase polymerase chain reaction (RT-PCR) and PCR, respectively. Six metabolites were found to have increased normalised abundance compared with controls: L-glutamate, L-hydroxyglutamate semialdehyde, eicosenoic acid, palmitoleate, docosahexaenoic acid and taurolithcholate. Similar numbers of controls and cases tested positive for viruses. Mild increases in hepatocellular injury and cholestatic markers was present in cases versus controls, which suggested a degree of liver disease in a subset of cases. The authors concluded that high GGT syndrome is likely a combination of oxidative stress and cholestasis. EQ