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CASO CLINICO: INSUFFICIENZA EPATICA IN UNA FATTRICE CAUSATA DA ASSUNZIONE AL PASCOLO DI PIANTE EPATOTOSSICHE NEL PORTOGALLO MERIDIONALE

CASE REPORT: HEPATIC FAILURE IN A MARE DUE TO HEPATOTOXIC PLANTS PASTURE IN THE SOUTHERN OF PORTUGAL

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Compromise of hepatic function in horses is probably more frequent than commonly described, mainly due to the great regenerative capacity of the liver that precludes clinical sings until 75% of liver parenchyma is affected. Despite exposure to hepatotoxic plants is the most common cause of chronic hepatic failure there are no reports in Portugal concerning this pathology in horses.

Pepa a 4 year old mare, undetermined breed, presented to the Veterinary Teaching Hospital of the University of Évora with signs of intermittent abdominal pain and weight loss for one week. According to the owner, during the last year, 5 horses and 3 cows had died with similar clinical signs at the same pasture. Before death the horses showed anorexia, icterus and neurological deficit. Upon presentation Pepa had a rectal temperature of 39ºC, a heart rate of 62 and a respiratory rate of 24. The mare presented mucosal congestion, oral erosions, slightly reduce gastrointestinal motility and dermatitis in the forelimb fetlock. Rectal palpation was physiological and passage of a nasogastric tube showed no gastric reflux.

Serum biochemistry profile revealed high values for alkaline phosfatase (AP), gamaglutamyl transferase (GGT), aspartate amino transaminase (AST) and total bilirubin. The complete blood count was within physiological limits. After vesical catheterization urinalysis revealed bilirubinuria and hemoglobinuria.

Abdominal ultrasonographic exam showed hepatic hiperecogenicity. Coagulation profile revealed an elevated protrobim time (PTT) so we decided to wait for 3 days until liver biopsy. Meanwhile the mare was kept in stall, without light exposure, feeding only roughage. We started intravenous 5% glucose fluids, supplementation with branched-chain amino acids and extra-label enrofloxacin (5mg/kg BW, sid, IV). During this period Pepa showed intermittent neurological signs with head pressing and compulsive walking.

Ultrasonographic guided liver biopsy was performed in the standing mare sedated with detomidine and butorfanol and under local anaesthesia. Histopathologic exam revealed hepatic megalocytosis, globular cytoplasmic invaginations within the nuclei, hydropic degeneration, intracellular biliar granular accumulation and fibroplasia that lead to the diagnostic of pyrrolizidine alkaloid toxicosis. The treatment was kept as described earlier except the enrofloxacin administration that was discontinued after 10 days of treatment. After one month at the Veterinary Hospital Pepa was send home and it was recommended to be feed with beet pulp and sorghum, kept at stall.

Although the prognosis is usually guarded, especially when there are extensive megalocytosis and fibrosis as in this case, we decided to continue supportative care and, nowadays, 4 months after left our Hospital, Pepa presents no clinical signs and liver probes are within physiological values.

Key words: *pyrrolizidine, alkaloid, hepatoxicosis, biopsy, horse.*

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