



# CYTOLOGICAL AND PATHOLOGICAL FINDINGS IN A HORSE WITH HEMOCHROMATOSIS DUE TO CHRONIC IRON OVERLOAD



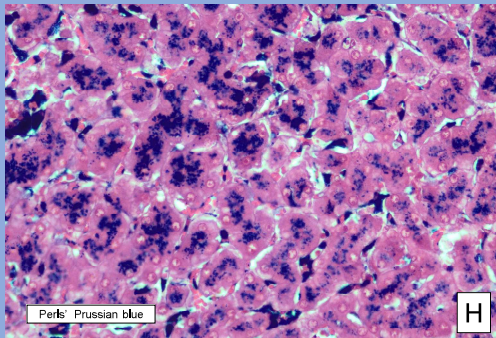
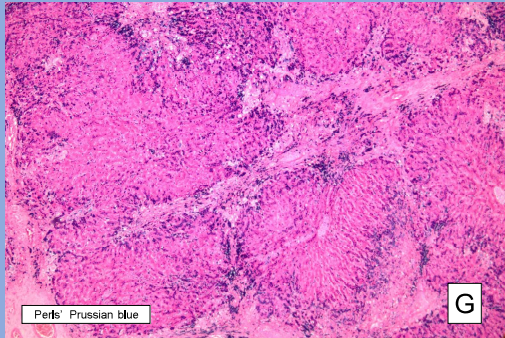
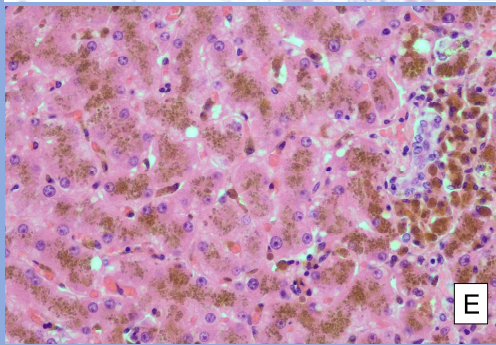
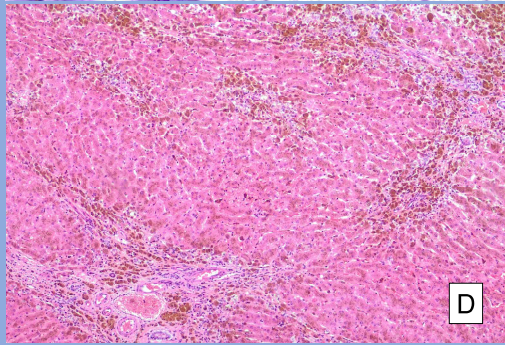
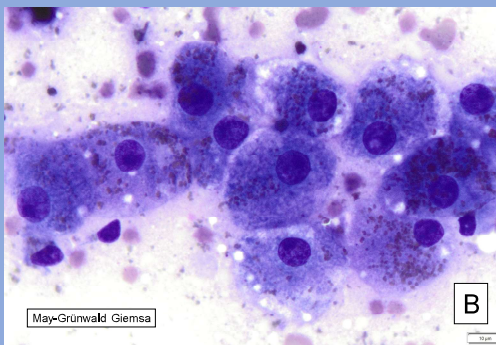
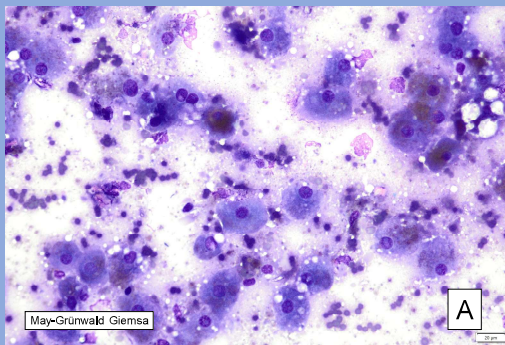
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Hemosiderosis of the liver has been rarely described in horses. Most reported cases are acute and related to iron toxicosis due to feed supplements or repeated blood transfusions. Chronic iron overload (mostly due to contamination of water sources) has been recently reported in equids and is characterized by a slow development of the disease, subtle clinical signs and a common fatal outcome. A 19-years old male Andalusian horse was referred to the Veterinary Teaching Hospital of Cordoba due to chronic weight loss, apathy and jaundice. The animal lived in a stable with other equids, but no clinical signs were noted in the rest. All animals had free access to water and forage. The patient had not received any previous blood transfusion.

| Parameter            | Ranges    | Result     |
|----------------------|-----------|------------|
| Bilirubin (mg/dL)    | < 3,5     | <b>5,2</b> |
| AST (IU/L)           | 140 – 410 | <b>463</b> |
| AP (IU/L)            | 62 – 228  | <b>335</b> |
| γGT (IU/L)           | < 44      | <b>257</b> |
| LDH (IU/L)           | < 400     | <b>623</b> |
| GDH (IU/L)           | < 12      | <b>51</b>  |
| Bile acids (μmol/L)  | < 12,8    | 10,7       |
| Ammonia (μmol/L)     | < 30      | <b>70</b>  |
| Fe (μmol/L)          | 18 – 54   | <b>133</b> |
| Ferritin (ng/mL)     | 65 – 300  | <b>799</b> |
| TIBC (μmol/L)        | 54 – 90   | <b>126</b> |
| Prothrombin time (s) | 9 – 11    | <b>15</b>  |
| APTT (s)             | 30 – 55   | 43         |

There were no changes in hematology. Serum biochemistry was markedly altered (Table). An abdominal ultrasound revealed severe hepatic atrophy with rounded liver margins, loss of normal echogenicity and multifocal hyperechoic foci (C). An ultrasound-guided fine-needle aspiration cytology was performed, showing common presence of a golden granular cytoplasmic pigment in hepatocytes and macrophages, marked anisocytosis and mild anisokaryosis in hepatocytes, marked fibrosis and moderate macrophagic inflammation (A-B). A presumptive diagnosis of liver hemosiderosis and secondary hepatopathy was established. The animal had to be euthanized due to a rapid deterioration and a necropsy was performed. The liver appeared small, with rounded borders and a coarse appearance (F), being markedly firm and fibrous. Histology revealed a severe bridging and portal fibrosis, with severe periportal hepatocellular degeneration (D) and marked hepatocellular and histiocytic siderosis (E). Perls' Prussian blue staining demonstrated that the pigment observed was iron (G-H). Since this patient had not received any previous transfusion, a chronic iron nutritional overload causing hepatopathy and hemochromatosis was finally diagnosed. Until recently, equine hemochromatosis was thought to be either of primary origin (hereditary) or secondary to severe hepatic damage. A recent study, where 21 equids were exposed to high dietary iron intake (due to excess iron content in drinking water) for prolonged periods, demonstrated that hemochromatosis can precede hepatic damage in these species. Both the evolution, clinicopathological changes and pathological findings are similar in our case to those referred in that study. Although owners rejected more testing (or testing other animals), it is noteworthy that the horse in this case was in an area where heavy metal are commonly found in water sources (Río Tinto, Huelva, Spain) and the drinking water came from a well near the mentioned river.



### REFERENCES:

- Theelen, M.J. et al. (2019) Chronic iron overload causing haemochromatosis and hepatopathy in 21 horses and one donkey. *Equine Vet J* 51(3):304-309. .
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- Pearson, E.G. et al (1994) Hepatic cirrhosis and hemochromatosis in three horses. *J Am Vet Med Ass* 204, 1053-1056.