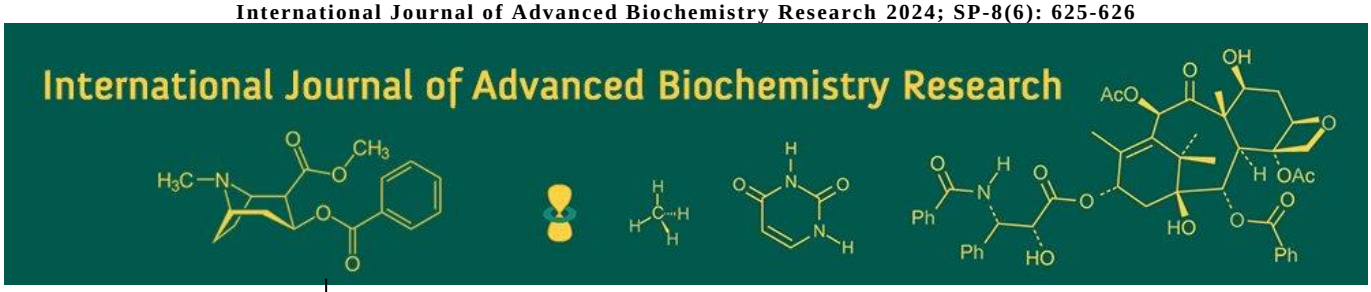

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Hepatic encephalopathy in Marwari mare

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Abstract
Hepatic encephalopathy (HE) is a serious neurological complication associated with liver dysfunction, characterized by cognitive impairment, altered consciousness, and neuromuscular disturbances. This case study investigates the presentation, diagnostic challenges, and therapeutic approaches in a Marwari mare diagnosed with HE. The mare presented with acute onset of behavioral changes, lethargy, and disorientation, prompting clinical evaluation and diagnostic tests revealing hepatic dysfunction. Treatment included dietary management, lactulose administration, and supportive care aimed at ammonia reduction and hepatic function restoration. Despite initial challenges, the mare responded positively to therapy, highlighting the importance of early recognition and targeted intervention in managing HE in equine patients, particularly those of unique breed backgrounds like the Marwari. Further research is warranted to refine diagnostic criteria and therapeutic strategies tailored to breed-specific susceptibilities.

Keywords: Hepatic encephalopathy, Marwari mare, diagnostic

Introduction
Liver disease is a relatively common cause of illness in the mature horse and is most commonly recognised by identification of icterus, weight loss, and behavioural changes (Pearson, 1999) [5]. Liver is frequently exposed to bacterial and chemical toxins due to which hepatic diseases are more prevalent. Hepatic encephalopathy (HE) is a commonly reported reversible neurocognitive ailment occurring in animals with acute or chronic liver disorders. As a result of hepatic insufficiency, HE is distinguished by inappropriate behaviour, poor motor function, and changes in mental status. Horses with HE frequently exhibits despondency, circling, head pressing, ataxia, futile wandering, and prolonged yawning, which are followed by recumbency and coma (Johns *et al* 2007) [3]. On clinical examination, animals can show cortical vision impairment along with mydriasis, poor response towards the touch on inner nares and hypotonia of lower lips (Divers, 2015) [1]. The pathophysiology of this condition is very complex. Hence, the occurrence of the disease condition is mainly determined by presence of hyperammonaemia along with additional markers of impaired liver function, which are all based on the animal's history, physical examination, clinical signs, laboratory results and diagnostic imaging data (Mair and Divers 2017) [4]. The line of treatment in case of HE is mainly based on reduction of cerebral oedema, decreasing the level of intestinal origin neurotoxins mainly ammonia, maintaining electrolyte, glucose and acid base balance along with adequate blood supply (oxygenation) to brain and other vital organs (Divers, 2015) [1].

Materials and Methods
A five and half year old, Marwari mare was presented with the history of depressed mentation, ataxia, compulsive walking, head pressing, protrusion of tongue, normal feed and water intake, straining while urination, hunched back, episodes of respiratory distress since last 10 days. The animal was previously suspected for bacterial encephalitis and was treated by trimethoprim + sulfamethoxazole and ranitidine orally for 11 days with no response to treatment.

Diagnosis

Diagnosis of hepatic encephalopathy was made on the basis of history and clinical signs. There was regular deworming history. On the day of presentation, the rectal temperature was 100.2°F, heart rate was 52 beats per minute, normal pink mucus membrane and normal capillary refill time i.e. 2seconds. On auscultation there was mild crackle sound showing some degree of pneumonic changes. Lymph nodes were normal. There was mild respiratory distress with respiration rate 48 per minute. Blood sample was collected in EDTA vial and serum vial for various haemato-biochemical analysis. The following table is depicting hematobiochemical changes during day on presentation, day 3 and day 10. There was increased bilirubin concentration, ALKP, LDH, CK and GGT concentration in all the blood samples collected on day 0, 3 and day 10.

Parameter	Day 0	Day 3	Day 10	Reference value
Hb (g%)	12.5	10.3	10.2	11.0–19.0
TLC (10^3 per µl)	8.6	7.6	7.0	5.40–14.30
TEC (10^6 per µl)	9.97	6.69	6.95	6.8–12.9
PCV (%)	46.70	39.1	39.6	32–53
Platelets (per µl)	398000	380000	180000	100,000–600,000
Neutrophils (10^9 per µl)	7.224	5.776	4.06	2.3–8.5
Lymphocyte	1.204	1.824	2.8	1.5–7.7
Eosinophils	0.172	0	0.14	0–1.0
Total Bilirubin (mg/dl)	2.5	2.9	2.4	0.9-2.04
SGOT (IU/L)	250	286	273	220–600
ALKP (IU/L)	721	679	647	140–400
LDH (IU/L)	580	650	470	160–410
SGPT (IU/L)	17	180	216	220–600
BUN (mg/dl)	16.7	18	16	10–24
Creatinine (mg/dl)	1.36	1.8	1.2	0.9–1.9
Glucose (mg/dl)	102	116	135	75–115
GGT (IU/L)	96	53	46	4–44
Creatine Kinase (IU/L)	453	562	143	145–380

Treatment and Discussion

The mare was treated with 100 ml of lactulose orally four times a day for 10 days along with inj. Ceftriaxone tazobactum @ 15 mg per kg IM, inj flunixin @ 1.1 mg per kg IM, inj. NSS 10lts and probiotic latifur 10g daily. The animal showed considerable improvement on day 3 with increased feed and water intake. There was still some incoordination and animal showed somnolence and irresistible forward pressing, but overall, there was improvement in condition. There was some respiratory distress along with depression. On day 10 the animal was completely normal and there was complete recovery from signs of encephalopathy. Respiratory stridor due to bilateral laryngeal paralysis is a rare complication of HE (Pearson, 1999, Dixon *et al*., 2001) [6, 2]. HE occurs as a common consequence of hepatic disease in the horse. Management considerations should include treatment of the underlying liver disease, and specific therapies to decrease ammonia production and absorption. The early diagnosis and treatment of hepatic encephalopathy can result in good prognosis and response to treatment.

Conclusion

In conclusion, the diagnosis of hepatic encephalopathy in the mare was based on clinical signs and supported by hematobiochemical changes observed over the course of treatment. Initial symptoms of respiratory distress and neurological impairment gradually resolved with a therapeutic regimen including lactulose, antibiotics, anti-inflammatory agents, intravenous fluids, and probiotics. The prompt intervention led to significant improvement by day 3 and complete recovery by day 10, highlighting the efficacy of early diagnosis and comprehensive management. Continued vigilance for complications such as respiratory distress underscores the importance of ongoing care in cases of hepatic encephalopathy in horses.

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