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Pregnancy-associated hyperlipaemia in a mare: A case report

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Abstract

Pregnancy-associated hyperlipaemia is a life-threatening metabolic disorder in mares that develops due to prolonged negative energy balance, particularly during late gestation when energy requirements are markedly increased. This case report describes a 7-year-old pregnant mare presented with anorexia after complete withdrawal of concentrate feeding during the last trimester. Clinical examination revealed depression and anorexia, while laboratory investigations demonstrated grossly lipaemic plasma, severe hypertriglyceridaemia (1664.29 mg/dL), hypercholesterolaemia (370.65 mg/dL), hypoglycaemia, elevated hepatic enzymes, hyperbilirubinaemia, and azotaemia, indicating advanced lipid metabolism disorder with hepatic dysfunction. The mare was treated with oral glucose supplementation, subcutaneous insulin, heparin therapy, and supportive care to correct the negative energy balance and reduce lipid mobilization. Despite intensive treatment, the mare's condition progressively deteriorated, resulting in fetal death followed by maternal death. The poor outcome was attributed to prolonged nutritional deprivation and delayed intervention. This case emphasizes the importance of balanced nutritional management during late pregnancy, early recognition of anorexia, and prompt biochemical evaluation, including serum triglyceride estimation, for timely diagnosis and management of hyperlipaemia to improve maternal and fetal survival in pregnant mares.

Keywords: Pregnant mare, hyperlipaemia, negative energy balance, hypertriglyceridaemia, hepatic dysfunction, late gestation, equine medicine

Introduction

The periparturient period represents a physiologically demanding stage in mare life characterized by increased metabolic and energy requirements associated with late gestation, parturition and onset of lactation. These physiological changes act as significant stressors that trigger complex metabolic adaptations to maintain energy homeostasis. Owing to these metabolic challenges, mares during the periparturient period are more susceptible to developing disorders such as hyperlipidemia, endometritis, subclinical hepatic dysfunction, and reproductive abnormalities, particularly when nutritional intake is inadequate (Arfuso *et al.*, 2023) ^[1]. Hyperlipidemia is characterized by abnormally elevated concentrations of circulating lipids in the bloodstream and commonly develops during periods of negative energy balance and physiological stress. This elevation in blood lipids is initially a normal adaptive mechanism that facilitates the mobilization of energy reserves stored in adipose tissue to meet metabolic demands. However, under certain conditions this compensatory response may become excessive and dysregulated which leads to pathological lipid accumulation in the circulation (McKenzie *et al.*, 2011) ^[6]. In horses, hyperlipidemia is most often characterized by elevated concentrations of triglycerides with increased cholesterol levels. Triglycerides and cholesterol are known as indicators of lipid and energy metabolism. Serum triglycerides concentrations reflect the state of nutrition and hepatic synthesis. As serum triglycerides increase, anorexia often worsens and dyslipidemia becomes self-perpetuating. Hyperlipaemia is used when TG concentrations exceed 5.7 mmol/L (500 mg/dL), the plasma becomes grossly lipaemic and clinical signs of disease are present.

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Conversely, hyperlipidaemia is used when TG concentrations are between 1.0 and 5.7 mmol/L, the plasma remains clear and clinical signs of disease are absent. Severe hypertriglyceridaemia is usually reserved to describe TG concentrations >5.7 mmol/L in large-breed horses that are typically asymptomatic (Dunkel and McKenzie, 2003)^[4]. Although the prognosis is poor in severe cases, clinical signs are often overlooked and diagnosis relies on measurement of triglycerides levels. Treatment is primarily etiological, with the aim of correcting the negative energy balance by enteral or parenteral nutrition (Vikulina *et al.*, 2026)^[2].

Case Report

A pregnant mare of age 7 years was presented to the Veterinary Clinical Complex (VCC), MRCVS, Jhajjar, Haryana, India, with a primary complaint of anorexia. The owner reported that concentrate feeding had been completely discontinued from last trimester because of concern that feeding concentrates might predispose the mare to colic. During this period, the mare was maintained exclusively on roughage with free access to water. No previous medical treatment was reported before presentation.

On presentation, the mare was depressed and anorectic. Rectal temperature was within the normal physiological range. Upon distant examination fetal movements was present, indicating that the fetus was alive at the time of case presentation. No obvious signs of acute abdominal pain were observed during the initial examination.

Blood samples were collected for complete blood count (CBC), liver function analysis, lipid profile estimation and D-dimer assay. Grossly lipaemia was seen in EDTA vial (Fig.1). Hematological evaluation revealed erythrocyte count ($7.17 \times 10^6/\mu\text{L}$), hemoglobin concentration (13.6 g/dL) and hematocrit (38.7%) in normal range and other erythrocyte indices (MCV, MCH and MCHC) remained within the normal reference ranges indicating no blood abnormality. Leukocyte count and differential leukocyte counts were within physiological limits (Table 1), suggesting the absence of marked systemic inflammatory or infectious processes. Platelet count and D-dimer concentration (508 ng/mL) were also within their respective reference intervals, indicating no laboratory evidence of overt disseminated intravascular coagulation. Serum biochemistry demonstrated severe metabolic abnormalities characterized by as well as marked hypertriglyceridemia (1664.29 mg/dL) and hypercholesterolemia (370.65 mg/dL) (Table 2) which confirming profound disturbance of lipid metabolism. Mild hypoglycemia (59.65 mg/dL) supported the presence of prolonged negative energy balance. Hepatic enzymes including AST, ALP and GGT along with total bilirubin were markedly increased indicating significant hepatocellular injury and cholestatic dysfunction secondary to excessive hepatic lipid accumulation. Concurrent azotemia reflected by increased blood urea nitrogen and creatinine concentrations suggested renal hypoperfusion or impaired renal function associated with severe systemic metabolic compromise. Collectively these findings were consistent with advanced hyperlipidemia accompanied by hepatic dysfunction following prolonged nutritional deprivation. Therapy was initiated immediately after diagnosis. Oral glucose (1g/kg body weight given as a 5% solution by stomach tube every hours) supplementation was administered to improve energy availability and reduce further mobilization of body fat reserves. Insulin was

administered subcutaneously (0.1-0.3 IU/kg every 24hrs) to facilitate peripheral glucose utilization and suppress adipose tissue lipolysis. Subcutaneous heparin (40-100IU/kg every hr) was also administered as part of the therapeutic protocol to enhance lipoprotein lipase activity and promote triglyceride clearance from the circulation. Supportive nursing care and close clinical monitoring were continued throughout treatment. Despite therapeutic intervention, the mare clinical condition progressively deteriorated. Subsequent examination confirmed fetal death. The mare also died despite intensive medical management. The unfavorable outcome was considered to be associated with severe metabolic abnormalities resulting from prolonged negative energy balance and advanced hyperlipidemia.



Fig 1: Grossly lipaemic plasma.

Table 1: Hematological profile of the mare diagnosed with hyperlipidemia

Hematology Parameter	Result	Reference Interval
RBC ($\times 10^6/\mu\text{L}$)	7.17	6.6–9.7
Hemoglobin (g/dL)	13.6	11.8–15.9
Hematocrit (%)	38.7	34–46
MCV (fL)	54.0	43–55
MCH (pg)	19.0	15–20
MCHC (g/dL)	35.1	34–37
WBC ($\times 10^3/\mu\text{L}$)	3.70	5.2–10.1
Neutrophils (%)	65	36–79
Lymphocytes (%)	28	18–55
Monocytes (%)	4	0–7
Eosinophils (%)	3	0–16
Basophils (%)	0	0–3
Platelets ($\times 10^3/\mu\text{L}$)	114	94–232
MPV (fL)	6.6	5.3–8.3

Table 2: Biochemical and Coagulation profile of the mare

Biochemistry	Result	Reference Interval
ALT (U/L)	35.12	6–25
AST (U/L)	425.12	199–374
ALP (U/L)	700.42	88–461
GGT (U/L)	116.22	8–33
Total Protein (g/dL)	8.12	5.4–7.5
Albumin (g/dL)	3.85	2.9–3.6
Globulin (g/dL)	4.27	2.3–3.8
A/G Ratio	0.90	0.8–1.5
Blood Urea Nitrogen (mg/dL)	57.71	10–22
Creatinine (mg/dL)	4.25	0.8–1.5
Total Bilirubin (mg/dL)	6.65	0.5–2.1
Direct Bilirubin (mg/dL)	1.33	0.1–0.3
Indirect Bilirubin (mg/dL)	5.32	0.3–2.0
Glucose (mg/dL)	59.65	71–122
Cholesterol (mg/dL)	370.65	68–133
Triglycerides (mg/dL)	1664.29	14–65
Coagulation		
D-dimer (ng/mL)	508	0–1000

Bold values show deviation from normal range.

Discussion

In our study pregnant mare was presented with history of anorexia and mare was on roughage diet which leads to negative energy balance in mare and ultimately leads to hyperlipaemia which is a life-threatening metabolic disorder that develops when prolonged negative energy balance induces excessive mobilization of adipose tissue triglycerides, exceeding the liver capacity for lipid metabolism and resulting in marked hypertriglyceridaemia and hepatic lipidosis as found in earlier studies also. (McKenzie, 2011; Hughes *et al.*, 2004) ^[6, 5]. In our study, complete withdrawal of concentrate feeding in last trimester together with the increased metabolic demands of late pregnancy likely leads to severe negative energy balance. Adequate dietary energy is essential during gestation because fetal growth markedly increases maternal nutrient requirements; prolonged dietary restriction promotes fat mobilization and predisposes mares to metabolic disorders (Vikulina *et al.*, 2026) ^[7]. The grossly lipaemic plasma and serum triglyceride concentration of 1664.29 mg/dL confirmed severe hyperlipaemia which is well established in earlier studies (Dunkel and McKenzie, 2003; Bertin and Fraser, 2020) ^[4, 3].

Marked increases in AST, ALP, GGT and bilirubin indicated hepatocellular injury and cholestasis secondary to hepatic lipid accumulation while concurrent azotaemia suggested systemic metabolic compromise with impaired renal perfusion which align with findings frequently reported in severe equine hyperlipaemia (Watson *et al.*, 1992; Bergero *et al.*, 2008) ^[8, 2]. Treatment aimed at reversing negative energy balance through glucose supplementation insulin administration and heparin therapy was instituted however, successful treatment largely depends on early diagnosis before irreversible hepatic dysfunction develops (McKenzie, 2011) ^[6]. The subsequent fetal and maternal death in the present case is consistent with the poor prognosis associated with advanced hyperlipaemia and emphasizes the critical importance of maintaining adequate nutritional management during late gestation and promptly investigating anorexia with serum triglyceride estimation (Hughes *et al.*, 2004; McKenzie, 2011) ^[5, 6]. Thus to prevent colic and hyperlipaemia in advance pregnant mare a balanced diet with addition of mineral mixture, liver tonic and regular diet monitoring by veterinarian can be recommended.

Clinical Significance

This case highlights the importance of maintaining adequate energy intake in pregnant mares during late gestation. Complete withdrawal of concentrate feeding for an extended period can lead negative energy balance and excessive lipid mobilization, predisposing susceptible mares to hyperlipidemia and its potentially fatal complications. Early recognition of anorexia, prompt biochemical evaluation including serum lipid profiling, and aggressive correction of energy deficits are essential for improving maternal and fetal survival.

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