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REVIEW

Equine hyperlipaemia: a review

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FFA	Free fatty acid/s
GIT	Gastrointestinal tract
HSL	Hormone-sensitive lipase
IV	Intravenous
LPL	Lipoprotein lipase
PPID	Pituitary pars intermedia dysfunction
TGC	Triglyceride/s
TPN	Total parenteral nutrition
VLDL	Very low density lipoprotein/s

Disturbances of lipid metabolism that result in the accumulation of TGC in the blood are common in equine species.[1-9] Hyperlipidaemia is a physiological response in hypophagic[9] and exercising horses[10] that is often an incidental finding on serum biochemical analysis.[9] The condition is corrected when feed intake improves[9] or exercise ceases.[10] Hyperlipaemia however, is a pathophysiological response to prolonged negative energy balance associated with gross lipaemia, marked hypertriglyceridaemia, fat infiltration of body tissues and organ dysfunction.[2,3,5] Given the high mortality associated with hyperlipaemia, it is of considerable concern to veterinarians dealing with susceptible equine populations.[3,4] This review describes the current understanding of the epidemiology, pathogenesis, clinical findings, diagnosis, pathology, treatment and prevention of equine hyperlipaemia.

Epidemiology

Various risk factors for the development of hyperlipaemia have been identified (Table1). Many reports confirm that hyperlipaemia predominantly affects pony breeds,[3-6,11] with Shetland ponies especially prone.[1,3,5,6,11] The increased prevalence in Shetland ponies is likely to represent a true breed predisposition, and a familial basis has been proposed.[4] The disease is also well described in donkeys[2,12] and miniature breeds.[7,8,13] Hyperlipaemia is uncommon in light horse and draft horse breeds.[2,9,14]

In ponies hyperlipaemia usually affects mature animals, occuring only rarely in animals younger than 18 months-of-age (Table 1).[1,5,6] It has been suggested that the prevalence of the disease in ponies may increase with age,[4] although this may simply reflect the increased reproductive activity and incidence of obesity in mature animals. In contrast, age does not appear to be a risk factor in miniature breeds.[7,8,15] In one study of nine hyperlipaemic miniature horses and donkeys, six were aged 6 months or less, while the oldest was 30 years.[7] The difference in age susceptibility between pony and miniature breeds may be influenced by differences in predisposing factors, discussed below.

The disease is most common in mares of all breeds, accounting for between 74 and 100% of affected horses (Table 1).[1,5-8] It has been reported infrequently in stallions and geldings.[1,6-8] Reproductive activity appears to increase the susceptibility of pony mares to hyperlipaemia, with late pregnancy and early lactation being predisposing factors,[1,4-6] leading to a seasonal pattern of incidence.[1,4] The prevalence of hyperlipaemia in reproductively inactive pony mares is similar to that of stallions and geldings combined.[5,6] While hyperlipidaemia and hyperlipaemia are also more common in females of miniature breeds, reproductive activity does not appear to be a strong predisposing factor, with 65 to 71% of affected females reproductively inactive.[7,8] Potential reasons for a predominance of females include a sex-related predisposition to disturbances in lipid metabolism or underlying

primary diseases.[8] Alternatively it may reflect the sex ratio of miniature breed populations. In contrast to pony breeds, no seasonal incidence of hyperlipaemia is evident.[8]

In ponies and donkeys hyperlipaemia is usually a primary disease process, and stress and obesity appear to be particularly important predisposing factors.[5,6,12] An underlying disease process is present in only approximately one third of hyperlipaemic ponies.[1,5] In contrast, hyperlipaemia in miniature breeds is reported to be secondary to an underlying disease process in 83 to 100% of cases (Table 1),[7,8] and in horses is almost invariably induced by concurrent disease, with azotaemia or PPID.[2,9,14] Secondary hyperlipaemia may occur as a result of any disease that results in negative energy balance.[1,5,7-9,13,14] Food deprivation, either accidental, intentional, or relative to the increased metabolic demands of pregnancy or lactation, is a common predisposing factor in primary and secondary hyperlipaemia.[1,6-8]

Table 1. Epidemiological factors in five case studies of equine hyperlipaemia arranged by year of publication (adapted from Watson and Love [3]).

	1978[1]	1985[6]	1992[5]	1994[7]	1995[8]
Number of cases	15	30	18	9	23
Breed					
Shetland pony	11	27	11	—	—
Other pony breeds	4	3	7	—	—
Miniature horses	—	—	—	5	23
Miniature donkeys	—	—	—	—	4
Age	2-15 years	4-9 years	18 months-20 years	2 days-30 years	2 months-15 years
Sex					
Stallions	0	3	0	0	5
Geldings	0	0	4	2	1
Mares	15	27	14	7	17
- pregnant	3	4	9	2	5
- lactating	9	22	2	0	1
- reproductively inactive	3	1	3	5	14
Predisposing factors					
Primary disease process	4	NA	6	9	19
Obesity	9	24	12	1	5
Stress	NA	15	1	NA	NA
Food restriction	2	15	1	NA	4
Disease duration (days)	6 - 10	7 (average)	3 - 24	3 - 12	1 - 7
Mortality (%)	80	57	67	22	50

NA = information not available

Pathogenesis

Normal triglyceride metabolism

The physiological metabolism of adipose tissue in equidae has been well described.[2-5] Briefly, TGC in adipose tissue continually undergo lipolysis due to the action of HSL, producing glycerol and FFA.[5] Lipolysis is balanced by the esterification of FFA with glycerol to reform TGC in adipocytes, so a net release of FFA generally does not occur[3] (Figure 1). Hormone-sensitive lipase is under sensitive regulation (Figure 1), and the release of FFA into the systemic circulation can be rapidly adjusted to meet the ever changing energy needs of the body.[3,4] During fasting the activity of HSL is increased, resulting in the net release of FFA into the circulation.[3] In addition, serum insulin concentrations are often reduced during fasting, resulting in reduced esterification in adipose tissue and further promoting the release of FFA.[16] The majority of FFA are transported to the liver where they are either oxidised completely via β-oxidation to provide energy, partially oxidised for ketone production, or re-esterified to form TGC.[3,4,9] The production of ketone bodies is limited because the pathway for their synthesis is poorly developed in the horse.[2,10] Triglycerides produced by the liver are released into the circulation in the form of VLDL.[2-4] In the peripheral tissues FFA are released from VLDL under the action of LPL, and are used as an energy source.[3,8] Once this metabolic response to fasting has met the body's energy requirements, the activity of HSL is normally suppressed by insulin and no further FFA are released from adipose tissue.[3]

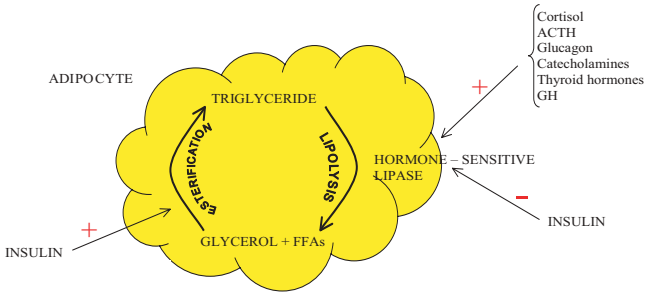

Figure 1. Normal triglyceride metabolism in equine adipocytes. - = inhibition, + = stimulation, ACTH = adrenocorticotrophic hormone, GH = growth hormone, FFAs = free fatty acids. (Adapted from Watson and Love [3]).

Triglyceride metabolism in hyperlipaemia

Hyperlipaemia is initiated by negative energy balance, the result of a variety of potential causes discussed above, with profound increases in the activity of HSL (Figure 2). Marked lipolysis results, and FFA are released into the circulation at a rate that exceeds the liver's ability to process them via oxidative pathways.[3] Most FFA are re-esterified to form TGC with a subsequent increase in the rate of TGC enriched VLDL production and release from the liver.[2,3,5] Hyperlipaemia results when the release of TGC from the liver overwhelms clearance mechanisms in the peripheral tissues.[8] Consequently hyperlipaemia is associated with marked elevations in plasma VLDL[17] and TGC[1-4,7-9,17] concentrations.

In pony breeds tissue insensitivity to insulin (insulin resistance) is considered to be an important predisposing factor for the development of hyperlipaemia, particularly in obese animals.[4,5,16,18] Because insulin is an important regulator of HSL activity, insulin resistance may result in an inability to regulate adipose tissue lipolysis appropriately.[4,18] Similarly in donkeys, insulin resistance may contribute to the pathogenesis of hyperlipaemia, with positive correlations between plasma insulin and TGC concentrations, and plasma insulin concentration and body weight.[19]

The mechanism of tissue insensitivity to insulin is incompletely understood. Potential causes include a decrease in the concentration or affinity of insulin receptors, or a post receptor defect resulting in alterations of intracellular signal transduction.[4,20] Changes in hormone homeostasis during pregnancy (in particular progesterone) and lactation,[4,21-23] and increased plasma concentrations of cortisol and ACTH in PPID[20] or stress[4] may exacerbate tissue insulin resistance through antagonism of the action of insulin on HSL.[3,4,21] Further, the pancreatic secretion of insulin is diminished with increasing gestational age, which may be a physiological mechanism to maintain adequate blood glucose concentrations, ensuring the nutritional requirements of the foetus are met.[21] Other factors postulated to predispose to a reduction in tissue sensitivity to insulin include fasting,[24] inactivity[25] and increasing age.[4]

Additional factors that may contribute to the development of hyperlipaemia include the effects of endotoxaemia and azotaemia (Figure 2). Endotoxin can rapidly induce changes in lipid metabolism, with stimulation of increased hepatic synthesis and secretion of TGC laden VLDL.[26] High blood endotoxin concentrations may also inhibit clearance of TGC by LPL in peripheral tissues.[26] Azotaemia is thought to be an essential factor in the development of hyperlipaemia in horses by interfering with the action of LPL.[2]

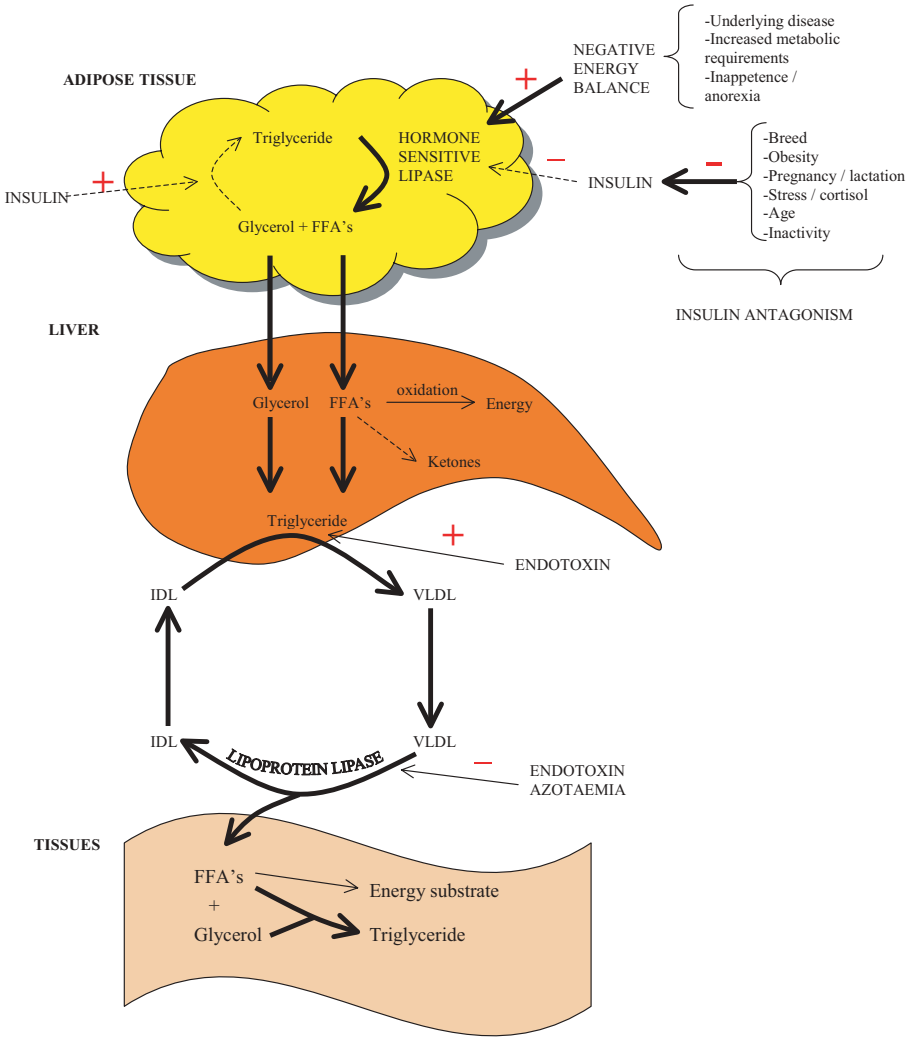

Figure 2. The pathogenesis of equine hyperlipaemia: reduced metabolic pathways are indicated with dashed arrows, while increased metabolic pathways are indicated with bold arrows. - = inhibition, + = stimulation, FFAs = free fatty acids, VLDL = very low density lipoprotein, IDL = intermediate density lipoprotein.

It has been suggested previously that a principal mechanism for the development of hyperlipaemia is a reduced clearance of VLDL from the circulation, resulting in hypertriglyceridaemia.[16] However, the activity of LPL is not reduced in hyperlipaemia;[17,27] rather, its activity is reported to be increased two-fold in response to increased substrate (VLDL) concentrations.[17] It can therefore be concluded that overproduction, rather than reduced clearance of TGC laden VLDL, is responsible for the development of hyperlipaemia.[17,27]

Clinical findings

The clinical signs of hyperlipaemia are well described, and their usual progression is outlined in Table 2. Clinical signs are non-specific and generally result from hepatic and/or renal dysfunction due to lipidosis. Signs may be preceded or complicated by those of an underlying disease process.[3,7,8] Hyperlipaemia is rapidly progressive, with the interval between the onset of clinical signs and death usually 1 to 10 days,[1,4,5,7,8,15] although periods of up to 3 weeks have been reported.[5]

Many animals exhibit signs of mild abdominal pain that are likely to be the result of stretching of the liver capsule secondary to TGC accumulation,[3] or primary disorders of the GIT.[7,8,15]

Table 2. Progression of clinical signs in equine hyperlipaemia.[1,3-9,11,12,14,15]

Stage of disease	Clinical signs
Initial	Signs of depression and lethargy Inappetence Adipsia Weakness Reduced gastrointestinal motility and faecal output Mucous coated inspissated faeces
Mid/progressive	Reluctance to move Muscle fasciculations Intermittent abdominal pain Diarrhoea Central nervous system dysfunction; ataxia, sham drinking, dysphagia, head-pressing, circling
Late/terminal	Recumbency Convulsions, champing, nystagmus, mania Abortion

Diarrhoea observed in hyperlipaemic animals may reflect disturbance of GIT function secondary to decreased feed uptake,[9] or the effect of an underlying disease process including GIT para-

sitism,[1,7,9] and enterocolitis.[7,8,14,15] Icterus is common and is likely to result from a combination of hepatic dysfunction and inappetence.[9] Ventral subcutaneous oedema may occur due to hypoalbuminaemia,[7] fat embolism[1] or vascular thrombosis.[1,9]

Hyperlipaemia is associated with a high mortality rate, particularly in ponies (Table 1) and horses, with the condition reported to be fatal in 57 to 80%[1,5,6] and 80%[9] of cases respectively. Miniature breeds appear to have a better prognosis, with mortality rates of 22 to 50% reported.[7,8] Survival in miniature breeds is thought to be dependant on the ability to treat the underlying primary disease successfully.[7] Consideration must also be given to differences in treatment type and intensity as being responsible for the reduced mortality rates in miniature breeds compared to ponies. Nutritional support provided to miniature horses and donkeys in a recent study[7] was frequently more aggressive than that given to ponies of earlier studies,[1,5,6] and may have contributed to the survival rate of 78%.[7]

Diagnosis

A diagnosis of hyperlipaemia cannot be made from clinical signs alone due to their non-specific nature. Used in combination with history and signalment, a high index of suspicion can be raised and a preliminary diagnosis can be made, particularly if the plasma/serum is opalescent (Figure 3).[4] Diagnostic confirmation is best achieved by quantification of serum TGC concentrations.[3,4] In healthy horses and non-pregnant ponies TGC concentrations are usually less than 1.00 mmol/L,[16,28] while in ponies during the last trimester of pregnancy concentrations have been reported to reach 2.83 mmol/L.[23] In healthy donkeys concentrations of up to 2.94 mmol/L have been reported,[19,29] with a positive correlation between body condition and serum TGC concentration.[29]

Mild elevations of serum TGC concentrations (up to 5.6 mmol/L) are often subclinical, detected only by laboratory means, and are not associated with lactescent plasma or fatty infiltration of the liver.[2,9] Such elevations are termed hyperlipidaemia, and are usually indicative of insufficient feed intake[9,16] and the requirement for nutritional support.[2,9] The condition rapidly corrects when feed intake, and therefore energy balance, improves.[2] In contrast, hyperlipaemia involves elevation of serum TGC concentrations to values greater than 5.6 mmol/L, opalescent plasma, hepatic lipidosis and severe clinical signs.[5,9] The degree of hypertriglyceridaemia present is variable: in one study mean TGC concentrations were 25.4 mmol/L,[5] however values in excess of 75 mmol/L have been reported.[1,5] Plasma concentrations of FFA, glycerol, cholesterol and phospholipids may also be elevated in hyperlipaemia,[2,17] however, such increases are small and insufficient for diagnosis.[2,3,17] Ketonaemia and ketonuria are not features of hyperlipaemia.[1,2]

Haematological changes are non-specific and may include haemoconcentration,[11] a stress leukogram,[14] or changes associated with underlying disease including neutrophilia,[11,12] neutropenia,[11] left shift,[11,12] and hyperfibrinogenaemia.[12] Serum biochemical evaluation is useful in determining the presence of organ dysfunction, and the metabolic status of hyperlipaemic patients (Table 3). Hepatic and liver dysfunction is common: in a study of 18 hyperlipaemic ponies, there was evidence of renal or hepatic dysfunction in all cases, with 15 having co-existing hepatic and renal pathology.[5] Hepatic dysfunction is reflected by increases in serum activities of enzymes of hepatocellular and biliary origin, and an elevated serum bilirubin concentration.[2,5,7,11-14] Elevations in

Figure 3. Blood sample from a pony with hyperlipaemia showing marked opalescence of the plasma.

Table 3. Serum biochemical variables, with expected changes, for diagnosis and monitoring of equine hyperlipaemia.

Indicator	Variable	Expected change
Metabolism	Triglycerides	↑
	Glucose	↓/N/↑
	Electrolytes	↓/N/↑
Acid-base status	pH	↓
	HCO_3	↓
	PCO_2	↓
Hepatic function	Iditol dehydrogenase	↑
	Aspartate transferase	↑
	Alkaline phosphatase	↑
	γ-glutamyl transferase	↑
	Total bilirubin	↑
	Total bile acids	↑
Renal function	Urea	↑
	Creatinine	↑
	Electrolytes	↓/N/↑

HCO_3 = Bicarbonate
PCO_2 = Partial pressure of CO_2 in blood
↑ = Increased, N = Normal range, ↓ = Decreased

serum total bile acid concentrations would also be expected in cases of hyperlipaemia with hepatic dysfunction, however their use in this disease has been reported rarely.[7] Serum bile acid concentrations are a sensitive indicator of liver function, and are technically easier and more readily measured than serum ammonia concentration measurements or exogenous dye clearance tests.[30] Their inclusion in a serum biochemical profile of hyperlipaemic animals would therefore seem justified. Liver biopsy is seldom indicated given the aetiology of liver dysfunction (lipidosis) usually being apparent on serum biochemical analysis, and the possible existence of a coagulopathy.[7] While prolonged clotting times have been reported in hyperlipaemia,[3,7] clinical coagulopathies are rare, possibly due to the acute nature of the disease and there being insufficient time for decreased hepatic production of coagulation factors to become apparent.[30] Hyperlipaemic animals are frequently azotaemic, which is generally assumed to be an indication of renal dysfunction,[5] often supported by post-mortem findings of severe renal lipid infiltration.[1] However in some cases the increase in serum creatinine and urea concentrations may be prerenal in nature and resolve with

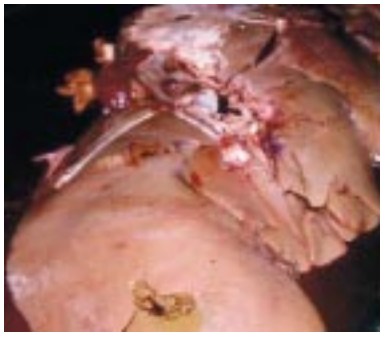

Figure 4. Necropsy specimen of the liver from a pony with hyperlipaemia. The liver has a characteristic pale, swollen and friable appearance.

fluid therapy.[7]

Hypoglycaemia is common in hyperlipaemic ponies and donkeys[1,2,12] despite the likely presence of insulin resistance, which would be expected to induce hyperglycaemia. Hypoglycaemia may be a reflection of depleted hepatic glycogen stores, and impaired hepatic gluconeogenesis and glycolysis due to lipidosis and increased plasma glucagon concentrations.[30] Thus hypoglycaemia and tissue insensitivity to insulin may co-exist in hyperlipaemic animals. Hyperglycaemia may occur in hyperlipaemic patients secondary to PPID.[2,7,9]

Metabolic acidosis is common, associated with marked base deficits,[3,15] and decreased arterial partial pressures of carbon dioxide may be found due to respiratory compensation.[15]

Interpretation of serum biochemical data may be complicated by interference of biochemical assays from lipids present in the blood. Lipaemia may impair end-point spectrophotometric determinations, resulting in false elevations in glucose, calcium, phosphorus, and bilirubin, and false reductions in total protein and albumin concentrations.[31] Lipaemic serum should be cleared by either ultracentrifugation or chemically by the use of polyethylene glycol 6000 prior to analysis.[31]

Controversy exists as to the prognostic value of serum biochemical values in hyperlipaemic animals. While some authors have documented an association between serum TGC concentrations and prognosis,[8] other studies have found disease outcome is unrelated to serum TGC concentrations or other serum biochemical measurements.[5,7] Favourable changes in successive serum biochemical profiles in hyperlipaemic patients have been associated with clinical improvement, and daily monitoring is recommended.[5] A major determinant of survival in hyperlipaemic patients appears to be the nature and response to therapy of any underlying disease.[7,8] Successful management of concurrent disease may contribute to the higher survival rates reported in miniature breeds, in contrast to the lower survival rates in ponies with primary hyperlipaemia.

Necropsy findings

Gross and histopathological findings in hyperlipaemia are well described and are principally associated with widespread deposition of lipids (mainly TGC) in body tissues.[1-3,11] The liver (Figure 4) and kidneys are most severely affected with lipid infiltration characterised grossly by pallor, swelling and development of a greasy texture.[1,2,14] Histopathological examination confirms the presence and severity of tissue lipidosis. Other lesions occasionally reported include lipid infiltration of skeletal muscle, adrenal cortex and the myocardium,[1] pancreatitis,[8] and a variety of vascular lesions including focal haemorrhages, pulmonary oedema, left ventricular myocardial infarction, renal infarction, and venous thrombosis.[1] In cases of secondary hyperlipaemia, changes associated with underlying disease may also be found.[2,15]

Treatment

Several authors have described the treatment of hyperlipaemia on the basis of nutritional support, identification and treatment of any underlying disease, correction of fluid, electrolyte and acid-base abnormalities, reduction of adipose tissue lipolysis and enhanced clearance of plasma lipids.[3,4,9]

Nutritional support is considered the most important factor in the treatment of hyperlipaemia.[3,7] Nutritional support reverses negative energy balance, increases blood glucose concentrations, promotes release of endogenous insulin and inhibits mobilisation of peripheral adipose tissue.[7] Varying intensities of nutritional support have been described, from the provision of highly palatable foodstuffs (including freshly cut grass, leafy hay, rolled grains, sweetfeeds or pasture),[3,4] enteral feeding[3-5,12] and parenteral nutrition.[7] Enteric nutrition has been attempted with a variety of preparations including glucose and electrolyte solutions,[3-5,12] commercially available enteric feeding preparations[7,12,13] and gruel made from lucerne, dried grass or pelleted feeds.[3,4,8,14] Oral glucose preparations have been advocated as a useful energy source, although they are unlikely to provide daily caloric requirements. In contrast, commercial enteric formulations have a higher energy density and provision of energy requirements can be achieved more easily.[7,13] Consequently such preparations have been recommended in the treatment of hyperlipaemia,[7,12,13] and their use may have contributed to the improved survival rate in a recent study[7] compared to earlier studies in which oral glucose solutions were used.[1,5,6] Patients should be observed after each feeding for signs of abdominal distension and pain, bruxism or evidence of gastric reflux through the nasogastric tube.[7]

Parenteral nutrition in the form of a constant IV infusion of 5% dextrose has been described, particularly in patients with GIT dysfunction.[3,8] While hypoglycaemia may be corrected,[7] nutritional requirements of the patient will not be met, and repeated monitoring of blood glucose and electrolyte concentrations is recommended.[3] Total parenteral nutrition has seldom been documented in the treatment of hyperlipaemia, although its use has been reported in a foal that did not tolerate enteric feeding due to ileus.[7] TPN is expensive, requires particular care in maintenance, and is associated with a variety of potential complications including metabolic disturbances (hyperglycaemia, hyperosmolarity, electrolyte imbalances, worsening hyperlipaemia and azotaemia), thrombophlebitis, venous thrombosis and sepsis.[32] If GIT function is present, enteric feeding is therefore preferred over TPN.[7]

The virtue of identifying and treating any underlying disease has been long recognised[2] and, as discussed previously, appears to be a prognostic determinant, particularly in miniature breeds.[7,8] Gastrointestinal parasitism was found to be a common predisposing disease in several early reports,[1,6] a finding corroborated by a recent study in which 33% of hyperlipaemic animals had evidence of a GIT nematode infection.[7] A previous suggestion that anthelmintics should be included in the treatment of hyperlipaemia[3] appears justified.

Correction of any fluid, electrolyte and acid-base derangements and support of organ function can be provided through the use of appropriate polyionic crystalloid solutions administered IV.[33] Supplementation of IV fluids with 5% glucose is useful in patients with hypoglycaemia.[7] Plasma is indicated for the treatment of hypoproteinaemia/hypoalbuminaemia when present.[7] If severe metabolic acidosis exists (bicarbonate concentration less

than 12 mmol/L, base deficit greater than 10 mmol/L), IV sodium bicarbonate is required,[15,33] with the deficit calculated using the following equation:

Bicarbonate (mmol/L) = 0.4 x body weight (kg) x base deficit[33]

Half of the calculated deficit should be given over 4 hours, after which the acid-base status should be re-assessed, with continued bicarbonate administration provided if necessary.[34]

The use of exogenous insulin and heparin has long been advocated in hyperlipaemic patients in an attempt to reduce lipolysis (through inhibition of HSL) and enhance peripheral tissue clearance of TGC (through increased LPL activity) respectively.[1,4,8,9,14] The aim of such treatments is normalisation of plasma lipid concentrations, however, the rationale for their use has subsequently been questioned, given the frequent presence of peripheral insensitivity to insulin and maximal activity of LPL in hyperlipaemic patients.[3] A recent study[7] in which neither insulin nor heparin was used reported a survival rate of 78%, further supporting the claim that both agents are clinically ineffective in the treatment of hyperlipaemia.[3] Further research is required for the identification of pharmacological agents that effectively reduce the mobilisation of adipose tissue TGC in equidae.

Prevention

Guidelines for the prevention of hyperlipaemia have been based on identification of susceptible animals and risk factors, and implementation of appropriate management strategies. Prevention should focus on those risk factors that are modifiable - energy balance, body condition, and stress[3-5,9] - through provision of adequate nutrition,[3,4,9] exercise[25] and avoidance of stressful situations such as transport, changes in management and GIT parasitism.[3,4] Risk factors such as breed and pregnancy/lactation cannot be altered, however they indicate a high risk status and the requirement for close monitoring and preventative measures.[3,4] Periodic blood sampling of high risk animals can be used to monitor for opalescent serum or elevated plasma TGC concentrations prior to the onset of clinical signs.[1,3,4,7] Such measures can allow the detection of impending hyperlipidaemia/hyperlipaemia and correction of negative energy balance, with a much improved prognosis.[1,4]

Recent studies have examined the effect of feeding a fat supplemented diet on lipid metabolism in ponies[35] and horses.[36] Both studies found a significant reduction in plasma TGC concentrations associated with a 50%[35] to 79%[36] increase in LPL activity. While these findings suggest that fat feeding improves plasma TGC clearance, further studies are needed to determine the effect of fat feeding in the prevention and treatment of hyperlipaemia.[35] If fat supplemented diets improve tissue utilisation of FFA, it is possible that such diets may reduce the risk of development of hyperlipaemia in susceptible animals. Possible limitations of dietary fat supplementation include glucose intolerance[35] and insulin resistance, and an inability to address the principal pathological process in hyperlipaemia, namely uncontrolled lipolysis.

Conclusion

The epidemiology, clinical signs, diagnosis and pathology of hyperlipaemia are well established, and much of the pathophysiology determined, permitting identification of susceptible animals and the effect of various risk factors on uncontrolled lipolysis. However, hyperlipaemia is still a disease associated with high morbidity and mortality despite various reported treatment modalities. Previously advocated pharmacological therapy (exogenous insulin, heparin) is ineffectual in altering disease outcome, and most treatment success is currently achieved through appropriate nutritional support, treatment of underlying disease and correction of fluid, electrolyte and acid-base derangements. Identification and use of pharmacological agents that effectively inhibit HSL with a reduction in the mobilisation of TGC in adipose tissue would be expected to improve treatment success significantly and reduce mortality in hyperlipaemic patients. Further research is required also to evaluate the efficacy of fat-supplemented diets in the prevention of hyperlipaemia, particularly in susceptible Equidae populations.

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BOOK REVIEW

***Treatment of behaviour problems in dogs and cats**, 2nd edn, Askew HR, Blackwell Publishing Asia, Carlton South,2003, 391 pages. Price $191.40. ISBN 1 4051 0620 4.*

This book is written by an experimental psychologist, not a veterinarian, so the emphasis and the classification of various problems is different from most other currently available behavioural medicine textbooks. It is divided into three sections. The first section (six chapters) deals with pet behaviour counselling, how pets relate to the family (human animal bond), how to conduct a consultation and how to increase compliance. It explains misconceptions and ethical issues involved in dealing with animals with behaviour problems. The content and presentation of this section are very good.

The second section deals with the treatment of canine behavioural problems. The main emphasis is on various types of aggression (six chapters) with one chapter each on fear, separation anxiety and elimination problems. The final chapter in this section is labelled miscellaneous behavioural problems and covers everything from uncontrollability during walks to stereotypic behaviours.

There is also a chapter on drug therapy co-authored by a veterinarian. Most of the information here appears to have been taken from the text books written by American behaviourists. Although indications and dose rates include cats the discussion concentrates on dogs. The chapter is critical of the use of drugs in the treatment of behavioural problems in dogs, contrasting with the section on cats, in which drugs are commonly recommended. The comments on experimental methods and the interpretation of the results of drug trials are well founded.

The third and shortest section of the book is the treatment of feline behavioural problems. There is one chapter dealing with urine marking, another dealing with inappropriate urination and defaecation, and another dealing with fear and aggression. The last chapter on cats covers a miscellaneous and unrelated group of behaviours, including unfriendliness to owners, running away from home and stereotypic behaviour. The book concludes with a chapter covering the future of the field.

Behavioural problems are dealt with by outlining the causal factors, followed by a description of possible treatments. Although most of the causal factors are well described, the recommended treatments tend to be somewhat contradictory. Although many of the recommendations for treatment include positive behaviour modification methods, there is still a focus on punitive techniques. These can be dangerous, especially when dealing with aggressive animals, and are not recommended by veterinary behaviourists. In addition, some of the scenarios given in each chapter as examples for modifying behaviour may actually reward inappropriate behaviours. Interestingly, throughout the book the author mentions that punishment is not effective in modifying behaviour unless alternative behaviours have been taught.

The use of head collars on dogs, which is now an important aspect of behaviour modification, is hardly mentioned. The author also fails to distinguish adequately between behavioural problems and training problems.

Although many psychotropic medications are mentioned in the chapter on drug therapy, with appropriate precautions and indications, some of the recommendations for the treatment of behavioural problems are outdated (for example the use of progestins). Progestins are now not recommended by veterinary behaviourists because of their significant side effects and the availability of safer and much more effective drugs. The use of phenothiazines, (for example acepromazine) and anticonvulsants, (for example phenobarbital) in behavioural problems is no longer recommended because they treat non-specific signs rather than the underlying causes.

Despite the fact that the field of behavioural medicine is growing rapidly, with many new publications and updates on behavioural theory and practice, only 14 of the 25 chapters have any references dated after 1995 (the first edition was released in 1996). A considerable volume of new information could have been included and have enhanced the overall value of the book.

In summary the first section on counselling is excellent and provides a great background on the subject. The other two sections and treatment options, while they have some useful information, are not always consistent with current veterinary behavioural practices, especially in terms of therapeutic and behaviour modification recommendations.

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