



## HYPERLIPAEMIA

### **EPIDEMIOLOGY**

(Risk Factors)

- Donkeys
- Small pony breeds - Shetlands, Welsh Mountain, Miniature ponies
- Uncommon in horse breeds, but has been reported.
- Females, more than stallions/geldings (only partly due to pregnancy and lactation)
- Obesity
- “Stress” – transport, weather, disease.
- Age – increased risk with age – age related decline in insulin sensitivity, Unusual < 18 months.
- Underlying primary disease
- G.I. disease: parasites, enteritis, colitis, impaction, dysphagia, choke, lymphosarcoma etc.
- Hyperadrenocorticism
- Laminitis
- Metritis
- Hepatopathy
- Peritonitis

### **PATHOPHYSIOLOGY**

Energy is stored as triglyceride in adipose tissue, this is broken down when stressed/undernutrition, by hormone sensitive lipase [HSL] to Free Fatty Acids [FFA]. FFA taken up by liver oxidised for energy or used to make glucose and ketone bodies. Excess FFA re-esterified to triglyceride [TG]. Insulin should shut down HSL. If hyperlipaemic there is insulin insensitivity, so FFA released in excess of livers' capacity for oxidation, gluconeogenesis and ketogenesis. Hence increasing amounts of triglyceride released into circulation. (There is limited ketogenesis in equines compared to the bovine/ovine).

The enzyme responsible for clearing TG from the circulation is Lipoprotein lipase [LPL]. Its activity is possibly inhibited in the azotaemic, endotoxic animal, although studies show it is working at an increased rate in hyperlipaemic animals.

Liver becomes filled with lipid – hepatic failure.

Fatty infiltration of other tissues – multi-organ failure.

Factors that increase activity of HSL

- **ACTH**
- **Glucocorticoids**
- **Catecholamines**
- **Glucagon**
- **Growth Hormone**
- **Thyroid Hormone**
- **Obese animals have a higher resting TG level and are less sensitive to insulin.**
- **Pregnancy and lactation reduce insulin sensitivity.**

**Clinical Signs** – Dull, depressed, anorexic, pyrexia, tachycardia, tachypnoea, gut stasis – dry mucous covered faeces, ataxia, oedema, congested mucous membranes, halitosis, hepatic encephalopathy, “sham” eating and drinking.

## **Diagnosis**

CS and plasma triglyceride concentrations – visual assessment of plasma helpful.

\*Take blood samples early in clinical examination – early diagnosis – instigate treatment on first visit\*

Assess biochemistry - liver and kidney function

- PCV and white cells
- glucose, electrolytes
- acid base balance

## **Treatment**

1. Primary & Concurrent Disease
  - esp. consider parasitism in all cases.
  - ? abort mare – risks
  - wean foal early
2. Fluid Therapy
  - maintain/restore circulatory volume.
  - correct electrolytes/acid base.
3. Symptomatic Therapy
  - NSAIDS, analgesics, anti-ulcer
  - Multivitamins, anabolics
  - plasma transfusions, antibiotics
4. Nutritional Support
  - maintain positive energy balance
5. Normalise lipid metabolism
  - insulin and glucose - reduce triglyceride mobilisation
  - heparin – increase triglyceride clearance.

## **Need to consider**

- Degree of severity
  - TG concentration
  - hepatic/renal function
  - TG < 20 – better prognosis
  - TG > 20 – worse prognosis
- Response to treatment
  - TG declining or rising
- Gut stasis present or gut active
  - do not use oral feeds if ileus
- Any voluntary food intake?
  - encourage with best grazing, hand feeding, treats, sandwiches?
- Economics
  - Cost rises dramatically with IV fluids, consider prognosis and underlying disease especially in older animals – dental problems, neoplasia.

## **Treatment details**

- NSAIDS
  - use low dose flunixin meglumine in cases with hepatic, renal impairment
  - 0.25mg/kg B – TID
- Multivitamins
  - e.g. Haemo 15 IV/IM 4ml , Equiton orally.
- Anabolics
  - appetite stimulant e.g. Laurabolin – nandrolene laurate 5ml IM
  - hepatic encephalopathy – Neomycin 50-100mg/kg & Lactulose 150-200ml QID ( to limit ammonia production and absorption)

- |                  |                                                                                                                                                                                                                                            |
|------------------|--------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Insulin          | - 0.1 – 0.3iu/kg bw<br>- 30 – 80iu protamine zinc insulin IM SID – BID only with glucose, but ?efficacy due to insulin insensitivity.                                                                                                      |
| Heparin          | - 100 – 200iu/kg BW BID I/V theoretically increases activity of lipoprotein lipase and increases clearance of TG from circulation, but LPL is working maximally therefore ?efficacy, also risk of haemorrhage especially if liver failing. |
| Anti-ulcer drugs | - Ranitidine (“Zantac”) 3x300mg tablets BID (? I/V injection for more rapid effect)<br>- Sucralfate (“Antepsin”) 10ml BID/TID ( ? efficacy for ulcers of the non glandular mucosa)<br>- Omeprazole (“Gastrogard”) ? cost and availability  |

## **Fluids and Nutritional Support**

### **Mild cases/preventative if inappetant – TG up to 6mmol/l**

- (a) 100g glucose, 5g sodium bicarbonate, oral multivitamins (e.g. Equiton, Visorbin), twice daily as a drench.
- (b) 30 – 80iu insulin BID
- (c) Can be given as a drench (50ml syringe) or nasogastric tube. (13mm pony size tube)

### **Moderate cases – TG up to 10mmol/l**

- (a) 1g/kg BW 5% glucose, i.e. 1litre/50kg by drench or nasogastric tube QID. Do not use if ileus.
- (b) 30 – 80iu insulin with glucose.

N.B Lectade has 50g dextrose per sachet.

### **Severe cases – complete anorexia**

- (a) Fluid therapy – maintenance approx 60ml/kg per day, + assess % dehydration.

Supply as intravenous polyionic fluids with lactate or pyruvate as precursors of bicarbonate.  
(Care with bicarbonate use to reverse acidosis unless acid base measurement available)

Body will reverse much of acidosis as hydration and renal function improve.

- (b) If ileus present need IV glucose as 5% Dextrose 1 – 2ml/kg/hour.\*

Overdosing - diuresis, hypoglycaemia, dehydration.

\*Not always possible to run IV drips continuously over a day. At the Donkey Sanctuary we use 2.5 – 3 litres twice or three times daily over approx. 45-60 minutes.

- (c) If gut function present – (auscultate and rectal examination). It is preferable to use a more complete food than just oral glucose. e.g. commercial enteral feeds – Clinifeed, slurry of pony nuts in solution, Complan, Ready Brek.

Calculate daily ration and give a small amount as often as possible, no more than **3l** volume for donkey, **5l** for a small pony. N.B. It may be possible to use a large bore 50ml syringe and for owners to do this at home.

## **Two methods (both approximate)**

1. DE (Daily digestible energy) – DE in Mcal/day (= 0.975 + 0.021 x BW in kg) x “stress” factor 1.2 –2.

Feed 75% on day one increasing to 100% on day four.

2. Energy requirements MJ/day = 487 x W(kg)<sup>.75</sup>

e.g. 150kg donkey needs 21 MJ/day

100g Complan provides 1.8MJ

1155g “ “ 21MJ = 3.5litre Complan

Probably practical to do this calculation for one feed used and adjust as necessary.

\* Always contraindicated to use glucocorticoids.

\* Keep companions together

\* Assess likely “stress” of hospitalising patient, may prefer to treat at home.

\* Owner attitude vital – if they are willing to hand feed, oral drench and administer oral medicines – prognosis much better. Always try and encourage and maintain voluntary feed intake.

\* Discuss finances early in disease

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