

February 2022

Laminitis in a standardbred horse, what is hidden behind?

Roberto Rey-Conejo DVM – LABOKLIN

Antonio Meléndez-Lazo DVM, MSc, PhD, DipECVCP, MRCVS – LABOKLIN

SIGNALMENT AND HISTORY

A 16-year-old, neutered male, standardbred horse was presented to the Vet with a 24 hour history of reluctance to move, stiffness and weakness. The owner referred too weight loss over the last few months. The horse had been drinking and urinating more than usual according to the yard staff (the stable bed appeared completely soaked all mornings during the last week). The horse was stabled during the night in wood shavings and on a small paddock with grass during the day.

The horse was adopted 2 years ago, previous clinical and travel history was unknown.



Fig. 1: Note the abnormal standing position

Photo credits: Roberto Rey Conejo DVM

PHYSICAL EXAMINATION:

On general physical examination, the horse presented a body condition score of 4/9. At rest, the horse was standing with the forelegs out in front of the usual position with the hind legs tucked underneath the body (fig. 1) and was shifting weight from one foot to the other. Both front feet were warm to the touch and there were strong bounding pulses in the digital arteries which run down the sides of the fetlock. Heart rate was 52 bpm (30-40) and respiratory rate 12 bpm (8-16) with a rectal temperature of 38.2°C (37.5-38.5). Mucous membranes colour was pink and capillary refill time less than 2 seconds with correct pulse. Intestinal motility was present in abdominal auscultation.

Growth rings around the hoof wall were visible, which generally indicates that it has suffered from laminitis in the past (fig. 2).



Fig. 2: Growth rings on one of the front feet

Photo credits: Roberto Rey Conejo DVM

INVESTIGATION

Haematology and biochemistry

Complete blood count was done as part of routine diagnostic workup and results are listed in table 1. Serum biochemistry was performed, and the results are listed in table 2.

Table 1: CBC results

Parameter	Results	Reference Interval	Units
PCV	29	30 – 50	%
Red blood cells	5.6	6.0 – 12.0	$\times 10^6/\mu\text{L}$
Haemoglobin	11.5	11 – 17	g/dL
MCV	46	37 – 55	fL
MCH	17	13 – 19	pg
MCHC	35	31 – 36	g/dL
White blood cells	8.6	5.0 – 10	$\times 10^3/\mu\text{L}$
Segmented neutrophils	7.5	3.0 – 7.0	$\times 10^3/\mu\text{L}$
Band neutrophils	0	0 – 0.6	$\times 10^3/\mu\text{L}$
Lymphocytes	1.1	1.5 – 0.4	$\times 10^3/\mu\text{L}$
Monocyte	0	0 – 0.4	$\times 10^3/\mu\text{L}$
Eosinophils	0.1	0 – 0.3	$\times 10^3/\mu\text{L}$
Basophils	0	0 – 0.15	$\times 10^3/\mu\text{L}$
Platelets	211	90 – 300	$\times 10^3/\mu\text{L}$

Table 2: Serum biochemistry results

Parameter	Results	Reference Interval	Units
Albumin	39	25 – 24	g/L
Creatinine	1.2	71 – 159	$\mu\text{mol/L}$
AP	501	< 450	U/L
BUN	14	3.3 – 6.7	mmol/L
GGT	47	< 25	U/L
AST	394	140 – 400	U/L
GLUCOSE	6.2	3 – 5	mmol/L
CK	283	90 – 300	U/L
Total Bilirubin	2.9	8.6 – 59.9	$\mu\text{mol/L}$
TP	59	55 – 75	g/L
LDH	350	< 400	U/L
Globuline	20	< 51	g/L

What is your interpretation of the haematology?

Mild normocytic normochromic anaemia likely reflects anaemia of chronic inflammation. Mild neutrophilia and lymphopenia probably reflect a response to endogenous or exogenous steroids (stress leukogram).

Is the biochemistry giving you any hints in order to lead your diagnosis?

Mildly increased of GGT and FA is presumably due to mild hepatic damage. High glucose concentration might reflect ongoing endocrine problem or just be an artefact due to previous food intake.

Which are your differential diagnoses for the findings in the physical exam and haematology and biochemistry?

Equine metabolic syndrome (EMS) – Although this horse is not overweight, adiposity is not

inextricably linked with insulin dysregulation, and it is possible for equids to have EMS in association with a lean phenotype.

Pituitary pars intermedia dysfunction (PPID) - Changes in hair coat, such as failure to shed fully and curly hair once called hypertrichosis may occur but not all horses present this feature.

Diabetes mellitus – Rare in horses but some cases described.

What further test/tests could you do in order to confirm your diagnosis?

X-rays, urinalysis, insulin and ACTH.

X-rays

The x-rays confirmed the rotation and sinking of the pedal bone (ongoing laminitis).

Urinalysis

A sample of urine was obtained and urinalysis was performed (table 3).

Urinalysis results were normal and diabetes mellitus was dismissed.

Table 3: Urinalysis results

Specific gravity	1.025	1025 – 1060
pH	7.5	7.0 – 8.4
Glucose	Negative	Negative
Ketones	Negative	Negative
Bilirubin	Negative	Negative
Proteins	Trace	Negative
Blood	Negative	Negative
Leukocytes	Negative	Negative
Sediment	Normal	Normal

Parameter	Results	Reference intervals	Units
ACTH	115	< 30 negative 30 – 50 Questionable > 50 positive	pg/mL
Insulin	37	< 20	µU/mL

For the ACTH test, the sample was taken in mid-February, so reference intervals were adjusted due to seasonal variation. ACTH levels were elevated and, therefore, diagnosis of PPID was made.

High levels of insulin were detected. Insulin dysregulation or insulin resistance plays a central role in equine metabolic syndrome and hyperinsulinemia is probably the most important pathophysiologic feature of insulin resistance in horses. However, resting hyperinsulinemia can occur to a greater extent than explained by equine metabolic syndrome.

Decreased hepatic clearance of insulin might also contribute to hyperinsulinemia. More than 70% of the insulin secreted by pancreatic beta cells is normally cleared from the portal blood by the liver in horses.

Laminitis associated with insulin dysregulation can arise in association with glucocorticoid administration and pituitary pars intermedia dysfunction (PPID). So, in this case, the insulin dysregulation is most likely caused by the high ACTH levels and therefore for the PPID.

DIAGNOSIS

Pituitary pars intermedia dysfunction, with ongoing laminitis due to insulin resistance.

TREATMENT AND FOLLOW UP

Pergolide is the drug of choice for treatment of PPID. Initial dosage of 2 µg/Kg (1mg/500kg) was started.

Laminitis was discussed with the farrier and special pads were made for the animal.

The horse was also put on a diet. Insulin sensitivity can be improved by avoiding feeds high in starch and sugar. The horse was fed hay soaked for 30 minutes in cold water every 12 hours to lower the sugar content prior to feeding. Pasture access was limited to one to two hours a day in addition to elimination of all treats such as sugar cubes.

Initial response to the treatment was assessed during the first 30 days of treatment. The horse attitude and activity increased (despite being in box rest) and there was also an improvement in PU/PD. Laminitis progressed well and biochemistry and hormones levels were checked again in order to do a follow up.

All parameters, included insulin, were within normal limits. The owner was advised to keep the dosage constant and make an appointment with the vet every 6 months with one extra appointment happening on fall season to ensure the treatment is adequate during this period.

SUMMARY

Equine PPID's disease is one of the most common diseases of horses greater than 14 years of age. The clinical signs are associated with abnormally elevated ACTH levels in the blood. This syndrome is caused by a pathological enlargement of the middle lobe of the pituitary gland (pars intermedia) which results in over production of hormones. The enlargement of the pituitary gland is often referred to as a pituitary adenoma.

The most common symptoms found in PPID are hirsutism (not always present - as in this case), muscle loss, laminitis, PD/PU, lethargy, redistribution of fat and prone to infections.

Horses with PPID may also present signs of insulin resistance. Insulin resistance is described as a failure of tissues to respond appropriately to insulin. Insulin resistance can contribute to weight loss (mainly muscle), PD/PU and increased susceptibility to disease. A recent study (Baskerville et al 2018) included the strong association between hyperinsulinemia and laminitis. It demonstrated that insulin activate lamellar epithelial cells resulting in a proliferative effect that might be the cause of laminitis. Further studies need to be performed.

In conclusion, when a horse presents clinical signs of laminitis, weight loss and PD/PU but no hirsutism or fat condition are present, PPID along with insulin resistance should be considered as a possible diagnosis.

References:

- Durham, A.E.; Frank, N. ECEIM consensus statement on equine metabolic syndrome. *J. Vet. Intern. Med.* 2019, 33, 335–349
- Baskerville CL, Chockalingham S, Harris PA, Bailey SR. The effect of insulin on equine lamellar basal epithelial cells mediated by the insulin-like growth factor-1 receptor. *PeerJ.* 2018;6:e5945
- McFarlane D. Equine pituitary pars intermedia dysfunction. *Vet Clin North Am Equine Pract.* 2011;27(1):93–113
- Durham et al. Pituitary pars intermedia dysfunction and pituitary gland hyperplasia. *J. Am. Vet. Med. Ass.* 231, 417–426.