

WEIGHT LOSS IN HORSES

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Weight loss is a common presenting complaint in horses of all ages. Although the investigation of these horses can be frustrating, a logical approach, based on the mechanisms of weight loss and the most likely differential diagnoses can yield a diagnosis in many cases.

MECHANISMS OF WEIGHT LOSS

1. **Decreased intake**
2. **Increased utilization/ increased losses**
3. **Malabsorption/maldigestion**

I find it useful to try to establish whether I am dealing with a 'healthy' horse (ie no abnormalities identified on PE) or a 'sick' horse, as this can help narrow down the list of possible diagnoses, and thus the appropriate diagnostic tests.

1. Decreased Intake

Healthy Horse

- insufficient feed, especially in stabled horses or on overcrowded pasture
- competition for food in pastured horses
- poor quality/spoiled feed, especially hay, haylage

Systemic disease

- leading to anorexia

Diseases interfering with prehension/mastication and deglutition

- poor dentition
- oral pain - pharyngitis; oral ulceration; synovial pathology - eg temporomandibular joint
- guttural pouch disease - cranial nerve dysfunction. Mycosis, empyema
- oesophageal dysfunction - strictures, partial obstructions, megaesophagus
- chronic grass sickness

2. Increased utilisation/ increased losses

Healthy horse

Nutritional requirements increase with

- increased activity level
- gestation, primarily late gestation
- lactation

Systemic disease

- nutritional requirements may increase with disease
- loss of protein can result in increased catabolism
 - protein-losing enteropathy eg larval cyathostomiasis
 - protein-losing nephropathy (rare)
 - 'third-spacing' of protein - ie loss into a body cavity (pleuritis, peritonitis)
- Chronic pain eg laminitis

3. Malabsorption/Malassimilation

Decreased:

- digestion
- absorption
- assimilation

Dental disease

- poor mastication can result in insufficient break down of especially long stemmed fiber (eg hay) and thus decreased nutrient utilization

Decreased absorption

- infiltration of bowel with inflammatory/neoplastic cells (see below)

Decreased transit time

- diarrhoea

Decreased assimilation

- Liver disease may result in defects in glucose, protein and lipid metabolism

Approach to a horse with weight loss

History

The history should include a general history, as well as asking questions that may help determine which category to place the weight loss in. If the horse is being fed an adequate amount for its activity level/physiologic status, and does not appear to have any problems chewing or swallowing the food, then it is less likely to be a problem with decreased intake. If at all possible, I find it useful to observe the horse being fed, so that I can evaluate the amount being fed, the horse's appetite, and whether there is any competition for the feed, especially in a group feeding situation.

Physical examination

Although in most instances, further diagnostic tests beyond the PE are required, a full and thorough examination is an essential step in identifying the cause of the horse's weight loss. This should include a rebreathing examination, a rectal palpation and a full oral examination. The value of a full oral examination cannot be understated – in a retrospective study of 60 horses with chronic weight loss, dental disease was the second most commonly identified cause, accounting for 12/60 cases. (Parasitism was most common, 18/60)¹

Blood work

A haematology and biochemical screen, as well as evaluation of acute phase proteins may help identify specific organ abnormalities, or may increase the index of suspicion for GIT disease. Increases in hepatic enzyme activity, in particular SDH and GGT, as well as bile acid concentration will point towards liver dysfunction, although will not define the aetiology of the disease. Chronic renal failure is an uncommon cause of weight loss in the horse, and thus if increases in creatinine and urea are identified, pre-renal azotaemia due to hypovolaemia or dehydration must be ruled out. If azotaemia occurs with urine abnormalities (especially dilute urine), then renal disease should be suspected.

Anaemia (mild to moderate: PCV typically 22-30%) is a non-specific finding in many horses with systemic disease, and is most likely due to chronic disease and sequestration of iron.

In my experience, the white cell count is an unreliable indicator of infection in horses. Horses with severe acute GI disease (eg colitis) will typically have a low white cell count, and in some cases, chronic infection will result in a leucocytosis (eg Strangles, *R equi*). A normal white cell count should not be used as evidence of a lack of infection. Globulin concentration, and acute phase proteins, such as SAA and fibrinogen can be useful to identify inflammation or infection. There is evidence to suggest that a low serum iron concentration is a more sensitive indicator of systemic inflammation, especially in the acute phase of the disease. It is important to remember that a high SAA/fibrinogen or a low serum iron is not specific for infection – non-septic inflammation, such as surgery, will cause changes in acute phase proteins, correlating to the severity of the tissue damage.

Faecal analysis

A faecal egg count is routinely done in many horses with weight loss – unfortunately it is rarely rewarding. Despite intestinal parasitism being a relatively common cause of weight loss, because the infections are not patent in cases of larval cyathostomins, FEC are usually negative, although if the laboratory specifically searches for larvae, they may be identified. Remember that horses do not develop strong immunity to cyathostomins, so although parasitism is most commonly seen in younger animals, it should not be ruled out purely based on a horse's age.

Ultrasound – abdominal and thoracic

Ultrasound can be used to assess GI wall thickness (should not be greater than 3mm, apart from the stomach which can be up to 10mm), organ structure, peritoneal fluid characteristics and to identify the presence of masses etc. Despite its utility as a diagnostic tool, ultrasound evaluation of the abdomen is limited by the presence of the gas filled colon, as well as its inability to penetrate further than approximately 30cm. Both transcutaneous and transabdominal ultrasound should be performed.

Abnormalities of the pleural cavity are best identified via ultrasound. Radiographs may be useful if a lesion within the pulmonary parenchyma is suspected, as it will not be identified via ultrasound if covered by aerated lung. If I suspect pulmonary disease, I will typically perform both ultrasound and radiographs. Remember that a normal thoracic auscultation (even a normal rebreathing exam) does not rule out thoracic disease.

Peritoneal fluid analysis

Although I will typically perform peritoneal fluid analysis as part of the work up in cases of weight loss, most of the time the results are normal. Occasionally, a low grade peritonitis (which may be associated with an intra-abdominal abscess) or exfoliative neoplasia is identified.

Gastroscopy

Although gastric ulcers in themselves will typically not cause weight loss, they may cause the horse's appetite to be decreased, leading to weight loss. Gastroscopy can also be used to identify gastric neoplasia (most commonly squamous cell carcinoma) and to obtain duodenal biopsies.

Absorption test

The oral glucose absorption test (also known as the oral glucose tolerance test OGTT) is used to diagnose small intestinal malabsorption. It is a fairly crude indicator of absorptive capacity, and many horses fall into the 'grey zone' of 'partial malabsorption'. The test is traditionally run over 360 minutes, with blood samples taken every 30 minutes for the first 3 hours, and then every hour for the next three. A simplified approach, which utilizes the same amount of glucose, but only a pre-treatment blood sample and one at 120 minutes, has been suggested, which is more practical for field use, and appears to involve minimal loss of diagnostic information.

Intestinal biopsies

Intestinal biopsies can be taken in the standing horse (rectal, duodenal) or via laparotomy/laparoscopy. Full thickness biopsies obtained at surgery will typically provide more useful information than rectal or duodenal, although surgery is both invasive and expensive.

Other diagnostic tests

As indicated by results of work up eg liver biopsy, aspiration/biopsy of masses etc.

A detailed discussion of all causes of weight loss is beyond the scope of this talk, however, two diseases which appear to be more frequently recognized include Inflammatory /infiltrative bowel disease, and Proliferative Enteropathy caused by *Lawsonia Intracellularis*.

Chronic Idiopathic Inflammatory Bowel Disease

Inflammatory Bowel Disease (IBD) refers to a group of conditions of unknown aetiology that result in malabsorption and weight loss in affected horses. The underlying problem in all cases is infiltration of the small intestine (and occasionally large intestine) with inflammatory cells. Several syndromes have been described, based on the type of inflammatory cells involved. The clinical signs typically associated with IBD include: weight loss despite a good appetite; low grade/recurrent abdominal pain; lethargy; and dependent oedema. Dermatitis has also been associated with some forms of IBD.

Diagnosis

Diagnosis of IBD can be challenging. The diagnostic work up will typically include routine blood work, abdominal ultrasonography (both rectal and percutaneous), peritoneal fluid analysis, a glucose absorption test and histopathological evaluation of biopsy samples (rectal and duodenal).

Characteristic findings in horses with IBD include:

Diagnostic test	Result in horses with IBD
Blood work	Hypoproteinaemia, hypoalbuminaemia
Abdominal ultrasound	Thickened SI (+/- LI) wall thickness
Peritoneal Fluid	Normal
Glucose absorption test	Partial/complete malabsorption
Biopsy findings	Infiltration with inflammatory cells

Four types of IBD have been described in the horse, based on the type of inflammatory cells present within the intestine:

Lymphocytic plasmacytic enterocolitis (LPE)

Although LPE is rarely reported in horses, there appears to be no age, sex or breed predilection. Most horses with LPE have a history of weight loss, whilst colic and diarrhoea are less frequently reported. The glucose absorption test is usually abnormal, although rectal biopsies are rarely helpful in diagnosing this condition. LPE is the most common form of IBD diagnosed at the Royal Veterinary College, and duodenal biopsies obtained endoscopically appear to be helpful. In other species, this type of IBD often represents a nonspecific intestinal immune response, and may also be an early stage of intestinal lymphosarcoma. Whether or not this is also the case in the horse is unknown.

Granulomatous enteritis (GE)

This is one of the most commonly reported forms of IBD in horses. The majority of cases reported have been in Standardbreds, some of whom were related, suggesting a possible genetic predisposition. Young horses are more commonly reported. Although GE appears similar to Crohn's disease in humans (linked to persistent infection with *Mycobacterium paratuberculosis*), no such link has been established in horses. Affected horses typically present with signs of progressive and chronic weight loss, and anorexia. Skin lesions, colic and diarrhoea may occur, but are uncommon. The majority of horses with GE have abnormal glucose absorption, and a rectal biopsy may be diagnostic in approximately 50% of cases.

Idiopathic eosinophilic enterocolitis (EC)

Infiltration of the small intestine with eosinophils may occur diffusely, or more commonly, as focal lesions. Although the cause is unknown, it may represent an immune-mediated response to parasites. Circumferential mural bands of infiltrate may cause partial obstruction of the small intestine. Colic is the most frequently reported clinical sign with EC, with weight loss, diarrhoea and dermatitis being uncommonly reported. In contrast to other forms of IBD, hypoalbuminaemia is rarely reported in horses with EC.

Multisystemic eosinophilic epitheliotrophic disease (MEED)

In horses with MEED, eosinophilic infiltration is not restricted to the GIT, but is seen in other organs such as the skin, liver, pancreas and lungs. Weight loss, diarrhoea and dermatitis are commonly reported, whilst colic is rare. Most affected horses are young, and although the cause is not known, an immune-mediated disease and response to parasites has been implicated.

Treatment

Treatment of horses with IBD is classically unrewarding, however treatment with corticosteroids is often attempted. If parasitic involvement is suspected, anthelmintics such as fenbendazole (10mg/kg PO SID 5 days) or moxidectin should be administered. Dexamethasone or prednisolone have been used to treat horses with IBD. If diffuse small intestinal thickening is observed on ultrasound, or the glucose absorption test suggests complete malabsorption, parenteral, as opposed to enteral steroids may be preferable, at least in the initial treatment period, as the bioavailability of orally administered drugs may be low. I will typically start using either dexamethasone 0.1mg or prednisolone 1mg/kg SID. A typical treatment course will usually last for a minimum of 4 weeks, but may need to be longer. I recommend staying at the initial dose for 1 week, then gradually decreasing the dose over the next several weeks. If the clinical signs begin to recur when the horse is weaned off steroids, then I go back to the previous dose rate.

In horses with EC that have circumferential mural bands identified at surgery, resection of the affected areas of small intestine can be curative. Other treatments attempted in horses with IBD include dietary management (high protein, high fat diet) and metronidazole (especially in cases of GE)

Prognosis

In general the prognosis for horses with IBD is considered guarded to poor. However, a proportion of horses do respond to corticosteroids with complete resolution. Horses which continue to lose weight despite parenteral corticosteroids are unlikely to survive.

Proliferative Enteropathy

Proliferative Enteropathy (PE) is a disease of young horses (typically 6-8 months of age) caused by the obligate intracellular bacteria *Lawsonia intracellularis*. The organism is classically associated with disease in pigs, causing growth retardation and diarrhoea in weaned growing pigs. In the last 10-15 years, infection with *L. intracellularis* has

been increasingly reported world wide as a cause of weight loss, protein losing enteropathy and diarrhoea in young horses.

Epidemiology

The disease can occur as isolated cases, or in outbreaks. Initial case series reported a geographical link with pig farms, however the majority of cases have no link to pig operations. Serologic surveys suggest exposure to the organism is more common than overt disease.

The mechanism of transmission in horses is unknown. Studies in pigs show that the organism is passed in the faeces, but is unlikely to survive for longer than 2 weeks in the environment. Clinical cases in horses can occur sporadically, with a long period of time in between cases, suggesting the possibility of an environmental reservoir. A recent epidemiologic study in Brazil confirmed that clinically healthy horses can both shed the organism in faeces, and have evidence of seroconversion. In this study, both young animals and mature horses were tested. The oldest horse that had faecal shedding identified was 16 months of age, although there were several mature horses that were seropositive.

Aetiology

The bacteria infects the small intestinal epithelial cells, in particular the crypt cells. These cells expand and elongate, resulting in the classical appearance of ileal mucosal hyperplasia. The lack of normal crypt cells and therefore, lack of a functional brush border eventually leads to intestinal malabsorption

Diagnosis

1. Clinical signs

The typical presentation in the horse is a weanling, 4–7 mo old, with depression, weight loss (often rapid and profound), subcutaneous oedema, and poor body condition. Diarrhoea is a common but inconsistent finding. Some foals appear to have mild clinical signs, whilst others are severely affected and can die before treatment can be initiated.

2. Biochemistry

The most common clinical biochemical abnormalities are severe hypoproteinaemia and hypoalbuminaemia. Leucocytosis is common, but inconsistent. Elevated serum CK and AST have also been reported, as well as electrolyte abnormalities such as hyponatraemia, hypochloraemia and hypokalaemia.

3. Abdominal ultrasound

Thickening of the small intestine can typically be visualized using abdominal ultrasound, although this may not be a consistent finding.

4. Serology, Faecal PCR

Both serology and faecal PCR can be used in an attempt to confirm the diagnosis. The sensitivity and specificity of each test is not known in the horse. It is recommended to run both tests, as affected foals may only be positive on one test. Positive serology and faecal PCR can be found in clinically normal weanlings and adult horses.

Treatment

Antibiotics which penetrate into cells are required, due to the intracellular location of the organism. Traditionally, either erythromycin alone, or in combination with rifampin have been used, although tetracyclines (oxytetracycline followed by doxycycline) have also been used successfully. Affected foals typically improve rapidly (within several days) following the initiation of treatment. Treatment for several (3-6 weeks) is recommended.

Supportive therapy including IV fluids and colloids may be required in severely ill foals.

Prognosis

Most foals that receive appropriate antibiotic and supportive treatment will survive. A recent study showed that yearling sales price of affected foals was significantly lower (68%) than unaffected foals from the same sire. The long term athletic potential of recovered foals has not been reported, but appears to be normal.

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