Nutritional myodegeneration as a cause of dysphagia in adult horses: three case reports

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ABSTRACT: Three cases of nutritional myodegeneration caused by selenium deficiency in adult horses are described. Difficulty in eating and drinking was a common clinical sign in all horses. Blood biochemistry revealed a marked elevation of muscle enzymes and low glutathione peroxidase activity or low selenium concentration in whole blood in all cases. The treatment with sodium selenite and vitamin E was instituted in all horses. Two of them were euthanized because of continuing muscle injuries, one patient was cured. The post-mortem examination of euthanized horses revealed pale muscles that were distributed with bilateral symmetry on hind and thoracic limbs, diaphragm, tongue, masticatory and intercostal muscles and the myocardium. Histopathology revealed the areas of degeneration and necrosis. Large groups of regenerating fibres and pronounced lymphoplasmocytic reaction among the groups of intact fibres were also present. The clinical outcome of the disease is probably influenced by timely diagnosis and treatment.

Keywords: selenium; α-tocopherol acetate; vitamin E; myopathy; glutathione peroxidase

Dysphagia is difficulty in eating or swallowing feed or water despite having good appetite. The cause of dysphagia in horses may be a painful pathological process in the mouth, obstruction in the oral cavity, pharynx or oesophagus or a neurological problem (Baum et al., 1988a,b). Clinical and neurological examination, endoscopy of the pharynx, oesophagus and guttural pouches as well as haematological and biochemical analysis are usually necessary to establish the cause of dysphagia.

Selenium/vitamin E deficiency, the cause of the white muscle disease in foals (Lofstedt, 1997), was thought to be responsible for the masseter myodegeneration and resulting dysphagia in adult horses in the papers published by Step et al. (1991) and Pearson et al. (2005). Three cases of dysphagia caused by myodegeneration of masticatory muscles that were diagnosed at the Equine Clinic, University of Veterinary and Pharmaceutical Sciences in Brno, are described in this article.

Case histories

Case 1, a three-years-old Czech Warm-blood gelding, was moved to a new stable and introduced into the herd. Difficulties in swallowing water and feed were noticed by the owner five days later. The attending private practitioner found the palpatory sensitive oedema of masticatory muscles. No pathological changes were found during the examination of the oral cavity and flunixin meglumine at the

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dose of 1.1 mg/kg (Finadyne, Shering-Plough, UK) was administered to the horse. The head oedema worsened the next day, therefore the horse was referred to the clinic.

Case 2, a seventeen-years-old Shetland pony mare was introduced into a new herd for a period of one week. After return to the home stable, the inability to chew was observed by the owner. The left-side oedema of the head and a protrusion of the tongue from the mouth developed subsequently. The attending veterinarian presupposed a mandible trauma caused by a kick. Nonsteroidal anti-inflammatory drugs and an enema were administered to the pony. Despite this treatment the general status of the pony worsened during the consecutive three days. The mare was able to swallow only mash and her tongue protruded from the mouth. The brownish colouring of the urine was observed on the third day and the mare was referred to the clinic.

Case 3, a five-years-old Haflinger mare with the history of one-day lethargy and eating difficulties was examined by a private veterinarian. Haematological and biochemical tests were also performed. On the basis of an increased CK and AST activity, a suspicion of myopathy was pronounced. The veterinarian administered sodium selenite at the dose of 0.046 mg/kg and α-tocopherol acetate at the dose of 0.52 mg/kg (Selevit, Biotika a. s., Czech Republic) and referred the mare to the clinic.

Clinical course and outcome

Case 1. The clinical examination revealed an increased rectal temperature (38.7°C) and heart rate (56 bpm). The respiratory rate was normal at admission but the horse was slightly dyspnoeic. The auscultation of the chest was normal, gut sounds decreased bilaterally. The patient could drink but was not able to chew or swallow feed. Muscles of the head were not painful on palpation and the movement of the horse was normal. Endoscopy revealed the oedema of the pharyngeal mucosa that narrowed the pharyngeal lumen; both guttural pouches and oesophagus were normal. Because of persisting dyspnoea, tracheotomy was performed and a trachea tube was inserted to the horse.

Haematological tests did not show any pathological changes, but an increase in the muscle enzyme activity and low glutathione peroxidase activity (GSH-Px) were found in biochemical tests (Table 1). Dark coloration of the urine was noticed. On the basis of clinical findings and blood biochemistry, a diagnosis of nutritional myodegeneration was established. Sodium selenite at the dose of 0.088 mg/kg and α-tocopherol acetate at the dose of 1.0 mg/kg were applied to the horse. Flunixin meglumine was administered daily at the dose of 1.1 mg/kg (Meflosyl 52% inj., Fort Dodge Animal Health Holland, The Netherlands).

The clinical state of the horse worsened slightly during the next five days at the clinic. Heart rate was in the range of 56–64 bpm, respiratory rate 30–36 bpm, and body temperature 38.0–38.9°C. Repeated biochemical tests revealed an ongoing muscle injury. The horse was unable to drink since the third day at the clinic and had to be hydrated using a nasogastric tube. Due to the absence of improvement in the clinical status and sustained elevation of muscle enzymes in blood serum, the horse was euthanized.

Case 2. The body condition of the mare was very good on the day of admission; heart rate was 45 bpm and body temperature 38.2°C. The mare was unable to swallow feed and water. Her tongue protruded from the mouth and the mare was not able to retract it. She resisted any attempts to examine the oral cavity and palpate the masseters. A tracheal collapse was suspected on the basis of the neck palpation. The mare was slightly dehydrated (PCV 0.46 l/l, TPP 92.0 g/l). The blood serum was chylous. An increase in the muscle enzyme activity, low blood glutathione peroxidase activity, low selenium concentration and an increase in serum urea (13.8 mmol/l) were found in biochemical tests

Table 1. Results of biochemical examination in Case 1

Day	CK (U/l)	AST (U/l)	LDH (U/l)	K^+ (mmol/l)	Selenium (μg/l)	GSH-Px (U/l)	Vitamin E (μmol/l)
1	40 584.0	30 990.0	24 366.0	4.2	8.3	below detection value	30.3
4	50 238.0	34 122.0	24 540.0	–	–	–	–
6	69 420.0	–	–	–			61.5

Table 2. Results of biochemical examination in Case 2

Day	CK (U/l)	AST (U/l)	LDH (U/l)	K^+ (mmol/l)	Selenium (μg/l)	GSH-Px (U/l)	Vitamin E (μmol/l)
1	212 220.0	44 430.0	73 560.0	3.2	5.3	541.2	56.5

(Table 2). The radiography of the head, endoscopy of the nasopharynx, oesophagus and guttural pouches did not reveal any pathological changes. The tracheal collapse was confirmed by the endoscopy of the trachea. On the basis of clinical examination and clinical pathology, diagnosis of nutritional myodegeneration complicated by hyperlipaemia and tracheal collapse was established. Considering the prognosis, euthanasia was chosen.

Case 3. The mare was alert on the day of admission and its body condition was very good. Heart rate was 56 bpm and body temperature was 38.6°C. The mare tried to chew hay, but it dropped from the mouth. However, she was able to drink. Increased palpatory sensitivity of masseter muscles was found; the endoscopy of pharynx, oesophagus and guttural pouches was normal. Biochemical results (Table 3) revealed increased muscle enzymes and decreased glutathione peroxidase activity and confirmed the diagnosis of the referring veterinarian – nutritional myodegeneration.

Sodium selenite at the dose of 0.14 mg/kg and α-tocopherol acetate at the dose of 1.56 mg/kg were administered intramuscularly on the second day and at the dose of 0.22 mg/kg and 2.5 mg/kg orally on the fifth day. The mare was unable to swallow since the third day of stay at the clinic. Water with the addition of potassium chloride (50–100 g daily) was administered using a nasogastric tube. The ability to chew and swallow returned on the seventh day of hospitalization. Therefore, the horse was released to the home stable on the tenth day. A biochemical examination performed 30 days after discharge from the clinic was normal and the mare did not have any clinical signs of myopathy during the following two years.

Clinical pathology

Whole blood selenium was measured using the Unicam 939 AA spectrometer and AAS hydride technique.

Whole blood glutathione peroxidase (GSH-Px) was measured by the Paglia and Valentine (1967) method employing the Ransel kit supplied by the Randox Company and the Cobas Mira automatic analyser. The blood serum vitamin E concentration was determined by a fluorometric method using the fluorescence spectrophotometer 204 Perkin-Elmer (Thompson et al., 1971; Bouda et al., 1980). The reference values of blood selenium and GSH-Px in healthy horses were established previously in our laboratory (> 75.0 μg/l, >12 000 U/l) (Ludvikova et al., 2005a) as well as the reference value of serum vitamin E (>6.5 μmol/l) (Ludvikova et al., 2005b).

Post-mortem findings

Gross pathology. Post-mortem examinations of both euthanatized horses (Case 1 and 2) revealed the bilateral and symmetric distribution of pale muscles and streaks in the muscle groups of hind and thoracic limbs, diaphragm, tongue, masticatory and intercostal muscles (Figure 1). Large areas of

Table 3. Results of biochemical examination in Case 3

Day	CK (U/l)	AST (U/l)	LDH (U/l)	K^+ (mmol/l)	GSH-Px (U/l)	Vitamin E (μmol/l)
1	10 038.0	1 909.2	5 994.0	–	2 262.0	32.8
4	5 814.6	2 944.2	–	2.4	–	–
6	1 729.2	2 184.6	–	2.8	–	–
8	402.0	1 593.0	–	2.3	–	–
10	162.0	1 012.8	–	–	3 684.0	–
40	231.0	287.4	–	–	13 590.0	–

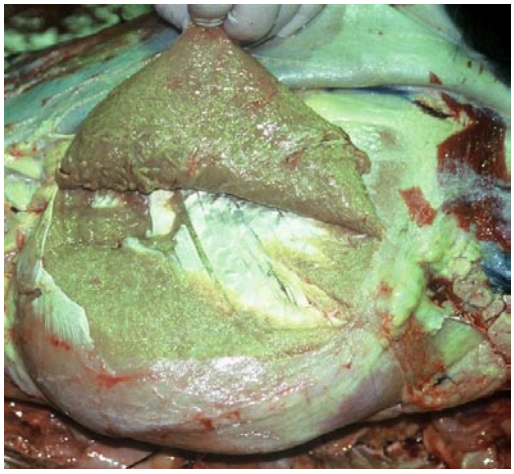

Figure 1. Pale discoloration of masticatory muscles (Case 2)

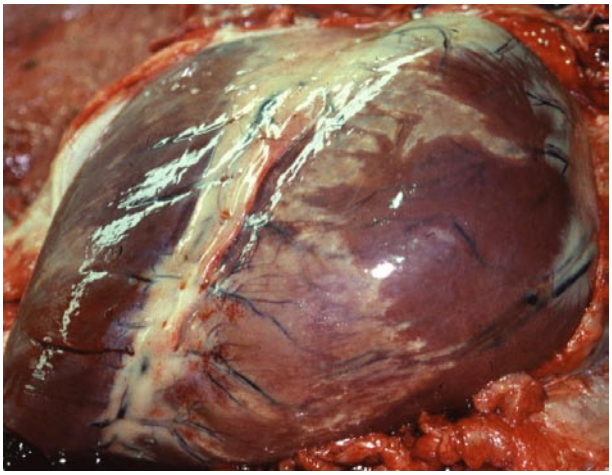

Figure 2. The myocardium with extensive areas of degeneration of both ventricles (Case 2)

the myocardium necroses were observed in both cases (Figure 2). Ulceration of the squamous mucosa along the margo plicatus was found in Case 1. A haematoma in the broad ligament containing four litres of blood, extensive tracheal collapse and enlarged liver with yellow-greyish discoloration were detected in Case 2.

Histopathology. Histopathology of muscle specimens (masticatory muscles, gluteal muscles, myocardium) fixed in formaldehyde was performed. Scattered atrophic fibres and foci of degeneration or necrotic areas with more or less pronounced lymphoplasmocytic reactions (with an admixture of polymorphonuclears) were found among the groups of intact myofibres (Figure 3). Conspicuous was the presence of large groups of regenerating fibres with basophilic cytoplasm (sarcoplasm) showing a positive red fluorescence in slides stained with acridine orange (Figure 4). The extension and severity of lesions were almost the same in all sampled muscles.

DISCUSSION

Masseter degeneration as a cause of dysphagia in adult horses was described in former Czechoslovakia by Kral as early as in 1929. The aetiology of the disease was not known to the author at that time.

Cases with similar symptoms and pathological findings were reported later in the USA (Freestone and Carlson, 1991; Step et al., 1991; Pearson et al., 2005). Selenium and vitamin E deficiency were considered as a causative factor of muscle degeneration

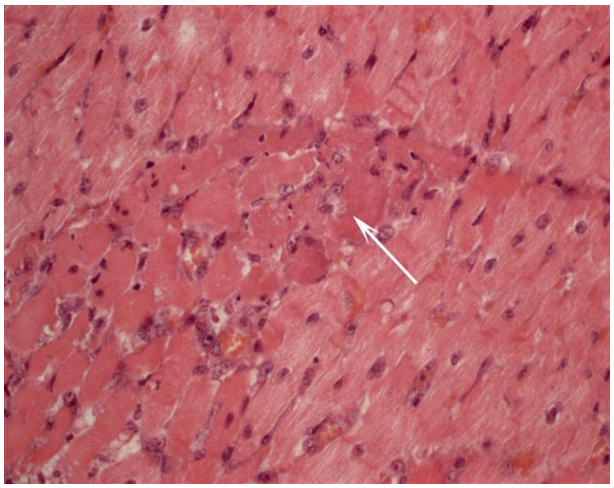

Figure 3. A group of necrotic myofibres (arrow) in skeletal muscle; H&E × 100

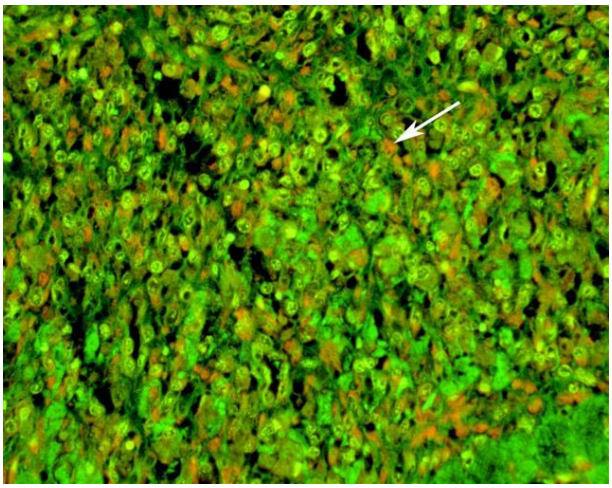

Figure 4. Regenerating myofibres (orange myofibres, arrow); acridine orange × 100)

in all of these cases. Nutritional myodegeneration in foals less than five months old has the same aetiology (Lofstedt, 1997; MacLeay, 2004). The muscles of mastication and deglutition are often affected together with locomotor and cardiac muscles in adult horses. Dysphagia may be the first sign of muscular degeneration noticed by the owner (Freestone and Carlson, 1991; Step et al., 1991). It was also true in all our cases described above.

The concentration of selenium was lower than the reference value in the case report of Step et al. (1991), and lower values of selenium were documented by Pearson et al. (2005) in two out of the eight horses with masseter myodegeneration. In another case from the latter paper, the blood value of selenium was normal but the horse was previously treated with selenium. In five horses the blood selenium concentration was not established. Similarly, lower selenium concentrations in serum were found in two out of the three adult horses with dystrophic myodegeneration in which the concentration of selenium was established (Owen et al., 1977). The selenium concentration was not evaluated in three horses with myonecrosis of the head, neck and diaphragm described by Freestone and Carlson (1991).

Concentrations of blood selenium in Case 1 and 2 in our group of patients were lower in comparison with the reference value for our laboratory. The selenium concentration in Case 3 was not determined. Extrapolating from the level of glutathione peroxidase activity (Ludvikova et al., 2005a), it can be considered low. The supplementation of this horse with selenium/vitamin E led to an increase in the glutathione peroxidase activity to the reference range after 40 days.

Muscular degeneration occurs in regions where the soil is deficient in selenium (Lofstedt, 1997). Our previous study revealed selenium deficiency in 47% of all the animals from a group of 159 horses bred in the Czech Republic (Ludvikova et al., 2005a).

The concentration of vitamin E in horses with dystrophic myodegeneration was evaluated by Owen et al. (1977), Step et al. (1991) and Pearson et al. (2005). Only Step et al. (1991) found a decreased concentration of vitamin E in one horse. The other authors found comparable (Owen et al., 1977) or even higher (Pearson et al., 2005) concentrations of vitamin E than those considered as reference values. Similarly, higher vitamin E concentrations than the reference value for our laboratory were found in all of our cases. The serum of Case 2 was hyperlipaemic and the extremely high concentration of vitamin E could be influenced by turbidity. However, in the other two cases the serum turbidity was not observed. Our results and results of the cited authors indicate that vitamin E deficiency plays a less important role than selenium deficiency in the aetiopathogenesis of nutritional myopathy in the horse.

Stress probably has some role in the pathogenesis of nutritional myodegeneration (Lofstedt, 1997). We can presume that in Case 1 and 2 stress was represented by transport of the horses to new stables and their introduction into a new herd. Case 3 was not stressed and from the clinical signs we can presume that only the masseter and no other muscles were involved. In the cases described in the cited literature, colic is mentioned only once as a possible triggering factor (Owen et al., 1977).

Differential diagnosis of nutritional myodegeneration includes mainly atypical myopathy and equine rhabdomyolysis syndrome (polysaccharide storage myopathy and recurrent exertional rhabdomyolysis) (MacLeay, 2004). Atypical myopathy occurs in horses of all age groups kept on pasture. Clinical signs are very similar to those in nutritional myodegeneration (weakness, stiffness, muscle pain, recumbency, myoglobinuria) (Cassart et al., 2007). However, dysphagia has never been described in atypical myopathy and affected horses can eat and drink well (Brandt, 1997). Dysphagia was present in all of our cases and none of them was kept on pasture. Atypical myopathy occurs very often at more than one horse in a group (Brandt et al., 1997). In all our cases only one horse in the group was affected.

Signs of the onset of equine rhabdomyolysis syndrome appear after a short duration of mostly aerobic exercise (MacLeay, 2004) but none of our cases was in training. An increase in the muscle enzyme activity is a common sign in rhabdomyolysis and it is not possible to use it for differentiation of the cause of rhabdomyolysis (MacLeay, 2004). Low GSH-Px activity and/or selenium concentration is specific to the diagnosis of nutritional myodegeneration (MacLeay, 2004).

The prognosis of nutritional myodegeneration in the adult horse is guarded to unfavourable. In the papers cited above only one from a group of five horses (Owen et al., 1977), none from a group of three horses (Freestone and Carlson, 1991) and none from a group of eight horses (Pearson et al.,

2005) survived. One case of masseter myonecrosis described by Step et al. (1991) also survived. One horse from our group of cases recovered after selenium/vitamin E administration. The horse is still living without any problems after two years. We can conclude that the ability to survive nutritional myodegeneration in adult horses is probably influenced by the early diagnosis and institution of treatment.

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