Muscle Lesions Dynamic and Severity in Acute Rhabdomyolysis in Horses

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Abstract. There were examined two mixed breed mares that had acute rhabdomyolysis. Necropsy revealed spread areas of acute muscular dystrophy, hypostatic lungs congestion and hyperemia, and myoglobinuria due to nephrosis. In histological samples muscle fibers were hypertrophied and showed sarcoplasm homogenization. Hypertrophied myofibers were necrotized and hyalinized. The majority of myofibers that have degenerative and necrotic lesions with hyaline deposits inside (Zenker hyaline necrosis) were fragmented. Despite of the presence of necrotic lesions local cell reaction degree is quite discreet indicating a fast evolution. Rhabdomyolysis affected only some myofibers not all muscle, which is in conformity with bibliographic dates. In areas with extended myofibers necrosis circulatory disturbances are intense, being accompanied by pronounced interstitial edema. Despite of that the capillaries maintained their structural integrity.

Key words: horse, rhabdomyolysis, muscle lesion, necrosis

INTRODUCTION

Acute rhabdomyolysis (that is known as "Monday morning disease") it is clinically characterized by fast appearance and evolution, movement disturbances, affected muscle hypertrophy (especially buttock muscles), sweating, and other signs of pain and discomfort. The signs could appear during or after exercises, and rarely being encountered in exhaustive exercise. In severely affected horses, even minimal exercise, such as walking out of a stall can cause clinical signs (Art et al., 2000; Touzot-Jourde, 2000; McGavin and Zachary, 2007).

Regarding the disease causes are incriminated several factors but the most important being high grain feeding and lack of regular exercises. Muscle lactic acidosis, vitamin E and/or selenium deficiency, hypothyroidism and electrolyte abnormalities are some other situations that are incriminated to be involved into acute rhabdomyolysis pathogenesis (Harris, 1998; Valberg et al., 1999; Beech, 2000).

Recent studies have found that mentioned factors aren’t obligatory primary causes in this disease. It is still thought that E vitamin or selenium deficiency can exacerbate signs of rhabdomyolysis but doesn’t represent a primary cause for it. Horses (especially pure breed horses) with light myopathy forms have an increased quantity of polysaccharides (Valberg et al., 1997; Sprayberry et al., 1998; Bloom and Valentine, 1999), but there are some cases where some thoroughbreds had an abnormal calcium homeostasis in skeletal muscle fibers (Beech et al., 1993; Hodgson, 1993). Because muscle necrosis doesn’t induce clinically pain and regional muscle swelling it seems that in disease pathogenesis could be involved some other etiological factors, such as oxidative injuries of muscle fibers membrane (Cătoi, 2003; McGavin and Zachary, 2007).
MATERIAL AND METHODS

The biologic material for this material had been represented by two mares of 6 respectively 7 years. The both of them are mixed breed. In both cases were noticed poor forms of acute rhabdomyolysis, and mares died in 2 and respectively 4 days from disease debut.

Necropsy revealed spread areas of acute muscular dystrophy, hypostatic lungs congestion and hyperemia, and myoglobinuria nephrosis. There were harvested samples for histology, respectively several thin slides of 5 mm thickness from the buttock muscles, such as: gluteus muscle, semitendinosus and semimembranosus. The muscle pieces were fixed in Stieve mixture for 24 hours, and embedded into paraffin and sectioned about 6 microns. The histology slides were stained using Goldner trichrome technique.

Using microscopically exam was observed muscle lesions extinction, character and gravity in acute rhabdomyolysis, which appeared after exercise.

RESULTS AND DISCUSSIONS

In affected muscles were observed lesions that had quite different extinction and gravity from one muscle fascicle to the other ones. The most discrete signs are represented by hypertrophy of some fibers comparatively to some others, which seems to be atrophied. Other changes which indicate an active lesion process but with a reduced intensity are disappearing of striate muscle pattern represented by transversal bands. In areas with advanced lesions could be noticed that myofibers aren’t very well individualized because they have homogenous structure (Fig.1). Also, there are some vascular permeability disturbances materialized by discreet interfibrillar edema (Fig.2). In fact are some changes that indicate rhabdomyolysis debut.

Microscopic exam reveal muscle fibers hypertrophy and sarcoplasm homogenization and transversal bands disappearing. In areas with hypertrophied myofibers could be observed muscle necrosis and their hyalinization (Fig.3). The majority of myofibers that have degenerative and necrotic lesions with hyaline deposits inside (Zenker hyaline necrosis) are fragmented (Fig.4). In this manner are formed hyaline blocks associated with local hyperemia. In some muscle areas are encountered myofiber fibrils lysis together with granular and/or vacuolar and hyaline dystrophy. In these regions vascular permeability changes are more pronounced and accompanied by different degrees of interstitial edema. Following to brutal dystrophic processes results granule-hyaline complexes that could have a small size in some myofibers or could be much larger in some other fibers.
Fig. 1. Incipient necrosis of striated muscular cells with sarcoplasm homogenization (Goldner’s Trichrome stain x200).

Fig. 2. Intercellular edema (Goldner’s Trichrome stain x200).

Fig. 3. Focal myofiber necrosis and hyalinization (Goldner’s Trichrome stain x400).

Fig. 4. Necrosis with fragmentation of muscular cell (Goldner’s Trichrome stain x200).
The last situation is due to hyaline material condensation forming in this manner massive blocks that are going to enlarge affected fibers and interstitial space (Fig. 5). Due to myofibers hyaline degeneration and enlargement, hyaline blocks compress mechanically neighboring muscle cells, which also appear to have dystrophy and degenerative processes (Fig. 6). Degenerative processes are indicated by the lost of striate pattern in this myofibers, and active myofiber fibrils lysis. Hyaline dystrophy is going to expand into myofibers, forming some hyaline blocks which interrupt myofibers continuity.

![Fig. 5. Massive block of hyalin (Goldner’s Trichrome stain x400).](image)

![Fig. 6. Advanced coagulation necrosis of muscular cells (Goldner’s Trichrome stain x200).](image)

Between necrotic myofibers are met muscle cells with normal feature indicating that rhabdomyolysis affect only some myofibers not all muscle. In conformity with bibliographic dates in the first stage are affected type II muscle fibers (Valberg et al., 1993; Snow and Valberg, 1994; McGavin and Zachary, 2007). That’s why the extinction of Zenker hyaline necrosis it is the consequence of disease evolution stage. Despite of the presence of necrotic lesions local cell reaction degree is quite discreet indicating a fast evolution. Macrophage infiltrate was obvious only in a few muscle regions with degenerative and necrotic myofibers lesions.

In some areas muscle dystrophy is very advanced and comprises muscle cells fascicles with the majority of myofibers affected. In respective areas circulatory disturbances are intense, being accompanied by pronounced interstitial edema. Despite of that the capillaries maintain their structural integrity, even in areas with cellular and interstitial alterations. Inside vessels could be noticed normal red blood cells or micro thrombus due to intravascular coagulation. All described aspect indicates that muscle lesions are much more intense than vascular ones. Also, more than a half from muscle cells has obvious signs of degeneration and necrosis, and only a few vessels present micro thrombus. This is the situation in the moment of muscle tissue harvesting in acute rhabdomyolysis in horses.
Described aspects, respectively skeletal myofibers necrotic lesions in buttock muscle (coagulation necrosis type with subsequent hyalinization), reduced local cellular reaction (discreet or absent macrophage infiltrate), and necrotized myofibers fragmentation forming hyaline blocks (rhabdomyolysis) indicate acute rhabdomyolysis.

CONCLUSIONS

There were examined 2 mixed breed mares that had acute rhabdomyolysis, both of them died in 2 and respectively 4 days from disease debut.

Necropsy revealed spread areas of acute muscular dystrophy, hypostatic lungs congestion and hyperemia, and myoglobinuria nephrosis.

In histological samples muscle fibers were hypertrophied and showed sarcoplasm homogenization, and a miss of transversal bands. Hypertrophied myofibers were necrotized and hyalinized. The majority of myofibers that have degenerative and necrotic lesions with hyaline deposits inside (Zenker hyaline necrosis) were fragmented. Despite of the presence of necrotic lesions local cell reaction degree is quite discreet indicating a fast evolution.

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REFERENCES

