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Equine Polysaccharide Storage Myopathy in an American Paint Horse

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Abstract : A 4-year-old, American Paint horse gelding with recurrent episodes of exertional rhabdomyolysis was diagnosed with equine polysaccharide storage myopathy (EPSSM). The common clinical signs were muscle weakness, loss of muscle mass, exercise intolerance, difficulty holding up limbs for farrier, sensitivity of grooming, cramping with reluctance to move and the inability to rise. Through histological examination of skeletal muscle specimens, this case was confirmed of EPSSM.

Key words **:** horse, equine polysaccharide myopathy (EPSSM).

Introduction

Equine polysaccharide storage myopathy (EPSSM) is characterized by abnormal accumulation of glycogen and glycogen-related polysaccharide in skeletal muscle fibers(2,3,5,6-8,10, 11,13). The purpose of this study reported here was to evaluate muscle samples of a horse and determine the incidence of EPSSM.

Case

A 4-year-old, American Paint horse gelding was presented for necropsy with clinical histories of recurrent episodes of muscle stiffness, muscle fasciculations, exercise intolerance, weakness, reluctance to move, abnormal gait, and recumbency for a year. Because of the poor prognosis, the horse was euthanized and submitted to the Veterinary Medical Diagnostic Laboratory at University of Missouri-Columbia.

At necropsy, no significant gross abnormalities were noted. Multiple sections of vital organs and skeletal muscles from both the left and right semimembranosus muscles as well as skeletal muscle from the fore limbs and distal aspect of the rear limbs were prepared for histopathology.

Microscopically, the semimembranosus muscle had occasional myofibers that were smaller in size when compared to the surrounding fibers and slightly more eosinophilic with rounded or angular outline. Some of the hypereosinophilic myofibers contain either numerous irregular, basophilic "lakes" against confluent aggregates of dense eosinophilic material (Fig 1). Those basophilic lakes were strongly positive with periodic acid-Schiff (PAS) staining after amylase-treatment (Fig 2). Skeletal muscles from left and right front proximal

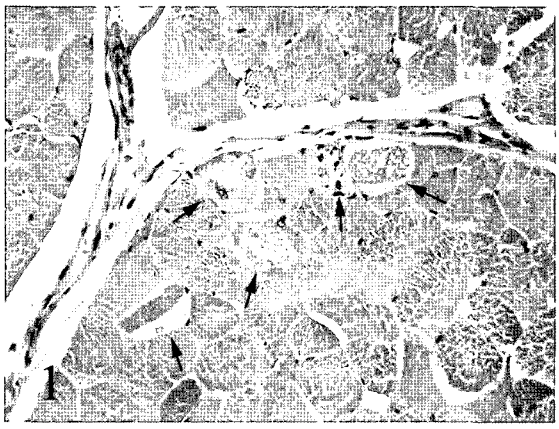

Fig 1. Semimembranousus muscle. Note the irregular, basophilic "lakes" against the hypereosinophilic myofibers (arrow). Hematoxylin and eosin. 400×.

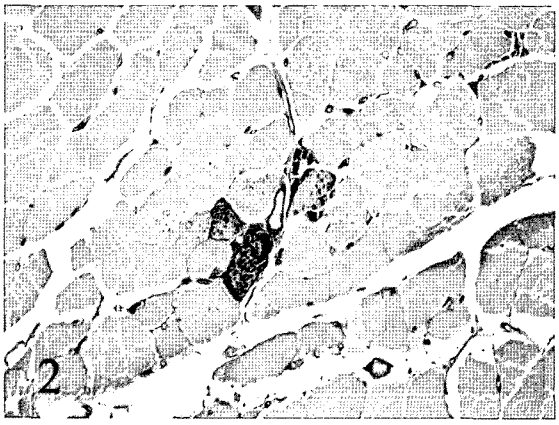

Fig 2. Semimembranousus muscle. Note the amylase-resistent, PAS (periodic acid-Schiff reagent) positive inclusions in the degenerate muscle fibers. PAS. 400×.

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and distal limbs and left and right distal limbs exhibited similar lesions seen in the semimembranosus muscle but milder in severity. No significant changes were noted in the lung, liver, kidney, spleen, heart, stomach, intestines, and brain.

Discussion

Pedigree analysis of Quarter horses with EPSSM has suggested autosomal recessive inheritance. It is more likely that this disorder in the draft horse breeds is inherited as a dominant trait, perhaps with incomplete penetrance.

EPSSM has been associated with a variety of manifestations of neuromuscular dysfunction, including exertional rhabdomyolysis, postanesthetic myopathy, sudden onset of weakness and recumbency, generalized weakness and muscle atrophy (1), and abnormal hind limb gait, including shivers (9). Serum activities of creatine kinase (CK) and aspartate aminotransferase (AST) are often slightly to markedly increased in draft horse-related breeds with EPSSM (9).

Therefore, biopsy evaluation of muscle samples stained for glycogen should be considered for draft horse-related animals with histories of increased serum levels of CK or AST, exertional or postanesthetic myopathy, or sudden death.

The excessive accumulation of glycogen within the skeletal muscle tissue has been found to be a result of increased insulin sensitivity in some affected breeds. This increased insulin sensitivity results in rapid clearance of glucose from the bloodstream following a carbohydrate meal followed by deposition of the glucose in the form of glycogen within muscle tissue. The defect leading to the accumulation of abnormal polysaccharide has yet to be reported (14).

Based on the abnormal storage of glycogen and complex carbohydrate and the positive response to a diet high in fat and low in soluble carbohydrates (12), this disease is presumed to be a carbohydrate metabolism disorder affecting skeletal muscle. Horses with polysaccharide storage myopathy fed a high-fat and low-carbobydrate diet should have normal serum muscle enzyme activities by four months after the dietary change (12). Rich pasture has not been reported as a trigger factor for episodes of rhabdomyolysis, but some pastures can be high in carbohydrate and low in roughage, and pasture analysis may be warranted as part of dietary management (16). However, horse owners should be advised to ensure that horses have regular daily exercise and to maximize their period at pastures and recommended that horses suffering recurrent bouts of rhabdomyolysis should be evaluated by means of a muscle biopsy.

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American Paint Horse에서의 다당류저장성근질환

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요 약 : 노책성 횡문근융해증의 재발을 보이는 거세된 4세령 Paint horse가 다당류저장성근질환으로 진단되었다. 말이 폐사하기전 보인 임상증상으로는 근약화, 근손실, 운동불내성, 장제시 사지를 들어올리는데 어려움이 있었고, grooming 시 민감한 반응, 이동 혹은 기립시 장애였다. 골격근 샘플의 조직검사를 통해서 말의 다당류저장성근질환으로 확진되었다.

주요어 : 말, 다당류보관근질환