

An Overview of Equine Azoturia

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Introduction

Exertional myopathy in horses is a syndrome of muscle fatigue, pain, or cramping associated with exercise. It is multifactorial myopathy affecting mainly draft horses and less frequently the race horses. The disease is a metabolic muscular disorder of horses characterized clinically by stiffness in gait, lameness, swelling and hardening of massive muscles (Ememe and Edeh 2022). Biochemically, the presence of myoglobin pigments in urine (myoglobinuria).

Incidence, occurrence and predisposing factor:

1. The disease affects mostly draft (draught) horses but race horses are sporadically affected.
2. The disease occurs during exercise after a period of at least 2 days of complete rest on a full working ration

Why Does the Illness Occur?

The causes and triggers of azoturia are not well known, and they vary slightly amongst animals. A draught horse that has been rested over the weekend and is fed to capacity frequently returns to work and becomes ill again.

1. Overconsumption of Carbohydrate: The primary reason is that the draught horse is fed its entire working ration while it is resting on the weekends, and when it returns to work a few days later, it has an illness attack. During rest, muscle glycogen builds up. When consumed during activity, it generates excessive lactic acid at a rate faster than blood vessels can remove it, which causes lactic acid accumulation. This causes local tissue damage (myopathy) and constriction of the blood vessels, resulting in decreased blood flow to the tissues and further reduction in lactic acid removal. Accumulation of sarcolactic acid in muscles produces swelling and hardening of muscle (hard board-like).
2. Local Hypoxia

3. Thiamine Deficiency
4. Vitamin E and Selenium Deficiency
5. Hormonal Disturbances
6. Electrolyte Imbalances

How Does the Disease Progress?

During exercise the large store of glycogen formed during the period of rest in the muscles metabolized to sarcolactic acid. Accumulation of lactic acid leads to Degeneration of the muscles and liberation of myoglobin (muscle haemoglobin) and swelling of muscle because lactic acid is hydrophilic.

How does the illness appear?

Signs appear 15 to 60 minutes after starting an exercise regimen. The degree of muscle injury and the amount of activity both influence the clinical indicators

1. In very mild cases that receive little amount of exercise, only poor performance is observed. While in mild cases, stiffness in gait is observed.



Fig: 1 showing difficulty to stand and red arrow excessive sweating



Fig: 2 Coffee colour urine

2. In severe cases, which receive excessive exercise:
 - A. Excessive sweating, stiffness in the gait, and unwillingness to move.
 - B. Then the horse assumes a dog-sitting position followed by lateral recumbency, laying down and repeated attempts to rise.
 - C. A stronger weak pulse that accelerates, quick breathing, and a temperature rise that can reach 40.5°C v Muscles that resemble hard boards, especially the gluteal and quadriceps femoris in the hind legs.
 - D. Frequent but scanty urination.

E. Dark reddish-brown (Haemoglobinuria) urine was expelled by the horse.

The concurrent alterations in Haemato-Biochemistry

1. Muscle degeneration: Muscle degeneration is the main azoturia lesion that is seen. Myoglobin is subsequently released into the blood plasma as a result. Muscle cells contain a protein called myoglobin, which is in charge of carrying oxygen. Myoglobin comes out of muscle fibres and into the blood.
2. Myoglobinuria: The kidneys then filter the myoglobin in circulation, which is ultimately expelled in the urine. Due to the presence of myoglobin, this causes myoglobinuria, which manifests clinically as dark or reddish-brown urine.
3. Secondary kidney lesions: Secondary kidney lesions may result from the kidneys' heightened myoglobin filtration. The clinical picture of the disease may be obscured by these extra renal alterations.
4. Aspartate aminotransferase (AST), creatine phosphokinase (CPK) levels and Lactate dehydrogenase (LDH) sharply increase in seriously affected horses and myoglobinuria develops. The result of the present study was in line with finding of Quist *et al.*, 2011 and El-Ashker, 2012.

Which treatment plans are advised for the sick dogs?

1. Avoid further exercise.
2. Massage of limbs by hot application.
3. I/V injection of large quantities of fluids and electrolytes to maintain high rate of urine flow to avoid renal tubule blockage and subsequent uraemia. (Inj. RL 10 litre I/V Bid for 5days)
4. Inj. sodium bicarbonate (7.5%) 25 ml I/V daily for 5 days.
5. Non-steroidal anti-inflammatory drugs (NSAIDs) such as Flunixin and Phenylbutazone may be used to control the pain. Flunixin @ 1.1 mg /kg B.W. I/V
6. Butorphanol @ 0.02-0.04 mg /kg B.W. I/V
7. I/M injection of 0.5gm thiamine HCL daily to increase the tolerance of blood to lactic acid by increasing lactic acid metabolism.
8. Inj. prednisolone 10ml 5 vial, 10ml I/M for 5days
9. Inj. E-Care Se 10 ml I/M weakly for 3 weak
10. Powder E-Care Se 1pack, 60gm daily orally

How can the illness be identified?

1. History
2. Clinical signs
3. Laboratory diagnosis
 - A. **Urine analysis:** To identify myoglobin pigments in urine, using just spectrophotometric means of detection.
 - B. **Blood:** To determine the levels of the muscle-specific enzymes AST and CPK. The first enzyme is more specific and diagnostic than the second, but both are typically



Fig 3 Horse in lateral recumbency

increased.

The Possible Preventive Strategies

1. Reduce the working ration (particularly grains) to 0.5, the amount during the period of rest.
2. Exercise should be light at first, then progressively more intense.
3. A consistent exercise routine with plenty of time to warm up and cool down correctly.
4. To help keep your horse active, give them plenty of turnout (avoid lush grazing as part of the dietary control).
5. Be cautious if any of these measures are not feasible, such as during winter when physical activity is limited.

Conclusion:



The risk of azoturia in draft (draught) horses is sporadically affected; the disease occurs during exercise after a period of at least 2 days of complete rest on a full working ration. Thus, limited exercise and a restricted carbohydrate diet should be carried out.

References:

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