

1970

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Larry L. Jackson
Iowa State University

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Recommended Citation

Jackson, Larry L. (1970) "Equine Azoturia, A Clinical Review," *Iowa State University Veterinarian*: Vol. 32 : Iss. 1 , Article 6.
Available at: https://lib.dr.iastate.edu/iowastate_veterinarian/vol32/iss1/6

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Equine Azoturia, A Clinical Review

Larry L. Jackson, D.V.M.*

Introduction

Azoturia, also known as paralytic myoglobinuria and paralytic myohemoglobinemia is a disease of the equine, occurring during exercise, after a period of inactivity, while on full rations. The primary lesion noted is muscle degeneration resulting in release of myoglobin into the blood plasma. This circulating myoglobin is filtered into the urine by the kidneys resulting in clinical signs of myoglobinuria. Secondary lesions involving the kidneys result from their increased filtration of myoglobin, thus clouding the clinical picture of this disease.

History

Clinical cases of azoturia invariably are presented for treatment with similar histories as to the onset of the disease. The horses affected are usually in good physical condition and hardened to physical labor. They invariably have been given a period of rest during which a high energy ration is fed at high intake levels. When the animal is then put back to work it suddenly becomes very rigid in its movements and appears to be in great pain.

Clinical Signs

Clinical examination reveals groups of muscles to be swollen and in a state of tonic spasm. Disturbances in gait are noted as a result of this spasm. If the disease is severe the animal may be unable to move or may go down and be unable to rise. Dark brown urine is passed as a result of kidney filtration of circulating myoglobin.

Etiology

The etiology of azoturia although not fully understood is metabolic in character. The breakdown in normal metabolism occurs in the pathways designated as glycogenolysis and glycolysis. These metabolic pathways are concerned with the conversion of stored glycogen to glucose and finally to pyruvate. The pyruvate thus produced normally enters the tricarboxylic acid cycle and is metabolized aerobically to produce energy. In the normal animal a small amount of the pyruvate produced can be anaerobically metabolized to lactic acid which diffuses into the extracellular fluid and is stored until that time when oxygen is again available. The stored lactic acid can then be converted back to pyruvate which can be utilized in the tricarboxylic acid cycle.

It should be realized that all metabolic pathways discussed above are reversible and much more intricate than has been indicated. Many steps in these pathways have been intentionally omitted here to simplify this discussion.

In the horse with azoturia extremely large muscle stores of glycogen are present due to the high energy rations fed. When the horse is called upon to work and thus needs energy, the conversion of glycogen to pyruvate and on into the tricarboxylic acid cycle occurs normally as long as sufficient oxygen is available for this aerobic conversion. When the oxygen supply becomes insufficient due to the great amount of substrate (glycogen) being mobilized, the pyruvate is anaerobically converted to lactic acid. It is this lactic acid, which in high concentrations is irritating to muscle cells, that causes cell rupture and muscle necrosis to occur. It is evident that as necrosis occurs the tissues involved become more anaerobic. This

* Dr. Jackson is an Instructor in Veterinary Clinical Sciences, Iowa State University, Ames, Iowa.

causes the metabolism to produce more lactic acid thus initiating a vicious cycle of abnormal metabolism resulting in the clinical manifestations of azoturia.

This explanation of the etiology of azoturia is the classic one and is valid except for one point. What is the underlying cause of decreased oxygen tension which allows the pyruvate to lactic acid conversion to become abnormal? That is, why should one horse out of several of the same physical condition and on the same ration have a decrease in cellular oxygen supply while the other horses do not? A search of existing literature is not effective in answering these questions. Perhaps some coenzyme or cofactor plays a role in the etiology of azoturia. The biochemistry involved in the etiology of azoturia probably should be investigated in seeking the answers to these questions.

Clinical Pathology

Although the etiology of azoturia is still in some doubt the clinical signs are well known and recognizable, and clinical pathological tests have been worked out to help establish a diagnosis. Many research projects have been carried out to establish which of the serum enzymes involved are the most reliable in confirming a diagnosis of azoturia. It is generally agreed that serum glutamic oxaloacetic transaminase and serum creatine phosphokinase determinations are the most reliable. Both of these enzymes are cofactors in the aerobic pathways of glycogen metabolism. If their serum levels increase it is an indication that the aerobic pathways are nonfunctional. Serum creatine phosphokinase determination seems to be the most accurate test for azoturia as its function is almost entirely involved with muscle metabolism in the horse. Serum glutamic-oxalacetic transaminase determination, although the enzyme is present in the liver of the horse as well as muscle, is also a reliable test.

Serum creatine phosphokinase and serum glutamic-oxalacetic transaminase disappear at different rates from the serum of a horse, the serum creatine phosphokinase level decreasing the most rapidly. It is possible to use this fact as a

tool for prognostication by determining serum creatine phosphokinase and serum glutamic-oxalacetic transaminase serially and comparing their attrition rates.

Treatment

The horse with azoturia should be placed in a box stall and all exercise should be avoided thus terminating the demand for energy production by glycogenolysis and glycolysis. Analgesics may be administered to alleviate the severe pain of azoturia. Antihistamines are also indicated. The administration of thiamine hydrochloride and vitamin E appear to be beneficial, especially in early or mild cases. Ancillary treatments such as intravenous or oral administration of electrolyte solutions are indicated in severe cases and of course antibiotics would be beneficial in preventing bacterial invasion of the damaged musculature and kidneys. Some authors recommend the administration of sodium bicarbonate orally. This tends to buffer the accumulation of lactic acid in the musculature and also to prevent the formation of tubular casts in the damaged kidneys. If the affected horse does not succumb to the pain and shock of an acute attack or later die as a result of irreversible kidney damage, the recovery from azoturia is usually uneventful.

It should be obvious that the best treatment for azoturia is its prevention. This can be accomplished simply by decreasing the horses dietary intake during periods of decreased energy demand.

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