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Exertional Rhabdomyolysis in horses (Azoturia, Tying up, chronic intermittent Rhabdomyolysis)

Etiology

The cause of most cases of exertional rhabdomyolysis is not determined.

Anomalies of muscle metabolism including polysaccharide storage myopathy of quarter horses, and draft breeds, mitochondrial myopathy, and defects in sarcolemmal function are identified in a small proportion of cases.

Other putative causes include:

Hypothyroidism

Hormonal imbalance

Vitamin E and selenium deficiencies.

Electrolyte abnormalities including: sodium and potassium deficiency

High carbohydrates diets

Exercise at irregular intervals

Viral infections

Epidemiology

Exertional rhabdomyolysis of athletic horses occurs as a sporadic disease world wide.

Outbreaks of rhabdomyolysis not associated with exercise in horses at pasture are reported.

The disease is almost always associated with exercise, more than 50% of cases in thorough bred race horses become evident during or immediately after exercise, and most of remaining cases are evident after a short period of rest (horses) after exercise.

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Occurrence of the disease is unpredictable – a horse working strenuously for weeks without a problem can develop the disease after routine exercise bout.

Exertional rhabdomyolysis is thought to occur more commonly in young female horses, although epidemiological evidence of this is lacking.

A genetic linkage with susceptibility to the disease is present in standard bred trotters in Sweden, and there is a familial incidence of the disease in thorough bred race horses.

Pathogenesis

The underlying cause is usually not known, but the inciting cause is almost always exercise .however, exertional rhabdomyolysis is not, contrary to earlier suggestions, associated with excessive lactate accumulation in muscle or systemic lactic acidosis.

Whatever the inciting cause, the end result is loss of integrity of the cell membrane of muscle cells, with subsequent muscle dysfunction and death .cell contents, including myoglobin and enzymes, are released into surrounding tissues and enter the blood.

Myoglobinuria results in myoglobinuric nephropathy and renal failure in severe cases.

Clinical findings

They are variable and range from poor performance to recumbency and death.

Signs may be mild and resolve spontaneously within 24h or severe and progressive.

The usual presentation is a young (2-5 year olds) female race horse with recurrent episodes of stiff gait after exercise.

The horse does not perform to expectation and displays a short stepping gait that may be mistaken for lower leg lameness.

The horse may be reluctant to move when placed in its stall, be apprehensive and anorectic and frequent shift their weight.

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More severely affected horses may be unable to continue to exercise , have hard and painful muscles (usually gluteal muscles) , sweat excessively , be apprehensive , refuse to walk , and be tachycardiac and tachypneic .Affected horses may be hyperthermic .

Signs consistent with abdominal pain are present in many severely affected horses.

Deep red urine (myoglobinuria) occurs but is not a consistent finding.

Severely affected horses may be recumbent.

Clinical pathology

Mildly or in apparently affected horses will have moderate increase in serum creatine kinase (CK), Aspartate aminotransferase (AST) and Lactate dehydrogenase (LDH) activity.

Serum myoglobin concentration increase markedly during exercise in affected horses, and decline within 24-48 hr.

Severely affected horses are often hyponatremic, hyperkalemic, and hypochloremic.

Azotemic (increase serum urea nitrogen and creatinine concentrations and acidotic or alkalotic. hemoconcentration. increase serum total protein concentration.

Differential diagnosis

Ear tick (otobius megnini).induced muscle cramping.

Polysaccharide storage myopathy of quarter horses

Cassia occidentalis toxicosis

Hypertkalemic periodic paralysis

Laminitis

Colic

Pleuritis

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Aorto- iliac thrombosis

Treatment

The treatment chosen depend on the severity of the disease.

The general principles are rest, correction of dehydration, and electrolytes abnormalities.

Prevention of complication including nephritis and laminitis and provision of analgesia

Mildly affected horse (heart rate < 60 bpm, normal rectal temperature and respiratory rate, no dehydration) may be treated with rest and phenylbutazone (2.2 mg /kg, orally or IV every 12 hr for 2-4 d).

Horses should be given mild exercise with incremental increases in workload as soon as they no longer have signs of muscle pain.

Access to water should be unrestricted.

Severely affected horses (heart rate > 60 bpm, rectal temperature > 39 °C, 8-10% dehydrated, reluctant or unable to walk) should not be exercised, including walking back to their stalls, unless it is unavoidable.

Isotonic poly ionic fluids such as lactated Ringer's solution, should be administered IV to severely affected horses to correct any dehydration and to insure a mild diuresis to prevent myoglobinuric nephropathy.

Less severely affected horses can be treated by administration of fluids by nasogastric intubation.

Urine should be alkalinized by administration of sodium bicarbonate (1.3% solution IV or 50-100g of sodium bicarbonate orally every 12 h) to minimize the nephrotoxicity of myoglobin.

Affected horses should not be given diuretics.

Analgesia

Phenylbutazone (2.2 – 4.4 mg /kg IV or orally, every 12-24 h).

Flunixin meglumine (1 mg / kg IV every 7h).or ketoprofen (2.2 mg / kg IV every 12 h).

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Mild sedation (acetylpromazine 0.02-0.04 mg /kg IM). Or Xylazine 0.1 mg /kg IM, both with butorphanol 0.01 to 0.02 mg / kg may decrease muscle pain and anxiety.

Recumbent horses should be deeply bedded and repositioned by rolling every 2-4 h. Severely affected horses should not be forced to stand.