**Metabolic disorders of horses**

**Hypocalcemic tetany of mares**

**Synonym: lactation tetany, eclampsia, transit tetany**

**1. Patient profile and history:**

There are two classic presentations of this condition:

**a.** **Lactation tetany**, which is seen in heavy milking draft horses at approximately 10 days post foaling or 1-2 days post weaning.

**b.** **Transit tetany**, which is described in lactating or nonlactating animals' transported long distances.

**2. Etiology and pathogenesis:**

**a.** Hypocalcemia is thought to be responsible for the clinical signs, although either hypomagnesemia or hypermagnesemia may be seen in some cases. Hypocalcemia may be produced by heavy lactation, transport, hard physical work, or no apparent cause.

**b.** SDFs are thought to be the result of diaphragmatic contractions synchronous with the heart beat and caused by changes in the excitation potential of the phrenic nerve because of electrolyte imbalances.

**3. Clinical findings:**

- In both presentations, severely affected animals exhibit tetany and incoordination. Horses are apprehensive and sweating with dilated nostrils, tachypnea, and synchronous diaphragmatic flutter (SDF).

- Muscular fibrillations and a rapid, irregular pulse are observed.

- Affected horses are unable to eat, drink, or swallow, and it may not be possible to pass a nasogastric tube. Clinical signs advance to recumbency with tetanic convulsions.

**4. Diagnostic plan and laboratory tests:**

- The diagnosis is made on the basis of clinical and subjective findings and confirmed by serum calcium levels.

- There is hypocalcemia (1-1.5 mmol/L). Serum magnesium values are variable, but hypomagnesemia may be seen with transit tetany (0.4 mmol/L).

**5. Differential diagnoses**:

 Include tetanus, laminitis, enteritis, and colic.

**6. Therapeutic plan:**

 Calcium solutions are administered intravenously (commercial

preparations contain 8 g of calcium per 500 ml). Responses to therapy are gradual, lessening the signs of tetany and the voiding of large volumes of urine. The response is usually good in individual animals.

**7. Prevention:**

Oral calcium supplementation or increasing dietary availability of calcium may be considered in heavily lactating mares.

**Hyperkalemic periodic paralysis (HYPP)**

**1. Patient profile and history:**

HYPP is a genetic disease of Quarter horses, Appaloosas, American point horses, and Quarter horse crosses. Gene frequency is highest in one pedigree of these breeds (i.e., affected horses are all descendants of a single Quarterhorse sire). Animals most frequently observed with the condition are well-muscled males age 2-3 years. The history and observed findings may indicate periods of prolonged recumbency or cutaneous abrasions.

**2. Etiology and pathogenesis:**

 HYPP is similar to the human condition and has been transmitted as an autosomal dominant trait that is most likely from a single sire. The disorder is produced by failure of ion transport across the skeletal muscle cell membrane due to an abnormality in the sodium channel. In horses, this has been localized to a point mutation that changes an amino acid (e.g., phenylalanine, leucine) in the subunit of the sodium channel protein.

a. Defective sodium channels remain open after membrane depolarization, allowing excessive inward sodium movement and heightened membrane depolarization. Simultaneously, normal sodium channels may be inactivated, preventing normal action potentials from developing. This creates muscular weakness.

b. Hyperkalemia may be secondary to increased release of potassium as potassium channels open to depolarize the muscle membranes. Hyperkalemia also may occur if potassium is less able to enter the myocytes, resulting in serum accumulation.

**3. Clinical findings:**

a. Clinical attacks may be triggered by chilling, transportation, exhibitions at shows, and other stressors. In heterozygotes, the most common and earliest clinical sign is muscle fasciculations, followed by muscle spasms of the face, jaws, and legs. Weakness and recumbency follow. Death may ensue due to respiratory or cardiac failure, but this is rare. Recovery may take several minutes to several hours. Animals are normal between episodes.

b. Many horses have increased respiratory rates during attacks and may show stridor if laryngeal or pharyngeal muscles are affected. Marked and persistent dyspnea may occur in homozygous foals. There are reports of HYPP attacks following

anesthesia.

**4. Diagnostic plan and laboratory tests:**

a. The condition may be diagnosed subjectively by clinical findings and objectively by elevated serum potassium levels. Serum potassium is usually elevated to between 6 mmol/L and 8 mmol/L during attacks. These elevations only persist for ***1-2*** hours. Serum should be separated from clotted samples as soon as possible to prevent red blood cell leakage of potassium into the serum.

b. Definitive diagnosis is based on a gene probe for HYPP-type sodium channel DNA. The test is based on analysis of whole blood and is available commercially.

c. A potassium chloride challenge test also has been used to diagnose the condition; however, this test is difficult to interpret and may be fatal in some horses. Potassium is administered orally at 0.1 **g** KC1 per kg, and both clinical signs and blood potassium levels are monitored. If the horse shows signs of muscle fasciculations and hyperkalemia, the test is discontinued. If results are negative, the test the test is discontinued. If results are negative, the test may be repeated up to four more times, increasing the administered potassium by 0.025 g KCl per kg every challenge up to a total potassium dose of 0.2 g/kg in adults or 0.15 g/kg in foals or weanlings.

d. Electromyography of suspect horses is practiced for immediate diagnostics and is quite reliable (90% reliability).

5. Differential diagnoses for presenting clinical signs include colic, trauma, and exertional rhabdomyolysis.

6. Therapeutic plan

a. Emergency treatment consists of the intravenous administration of 5% dextrose (2 ml to 6 ml/kg) together with sodium bicarbonate (1 mmol/kg to 2 mmol/kg) or 23% calcium gluconate (0.2 ml/kg). Dextrose and bicarbonate will move potassium back into cells. Calcium counteracts the effects of hyperkalemia.

b. Long-term therapy involves removing high-potassium feeds from the diet (e.g., protein supplements, bran, sweet feeds) and feeding a diet low in potassium (e.g., whole grains, grass hay).

c. Medical therapy may be used, including acetazolamide (a potassium-wasting diuretic) at 2 to 3 mg/kg orally, 2 or 3 times a day. The dose may be decreased over time until the lowest effective dose is established.

7. Prevention:

 a- There is no cure for HYPP, which is a genetic disease and is inherited as an autosomal dominant trait. HYPP has been diagnosed more frequently during the last few years most likely because the carriers of the condition have been used heavily as sires. Because of the characteristics these carriers have (e.g., well-proportioned, heavily muscled appearance), the disease probably has been unknowingly propagated.

b- Owners should be counseled about parentage identification and DNA gene probe testing. HYPP-positive horses should be removed from breeding use.

**Exertional (post-exercise) rhabdomyolysis in horses**

**Synonym: Myoglobinuria, azoturia, tying up syndrome, Monday morning disease**

a. Patient profile and history

(1) The most common presentation of the condition (also called equine paralytic myoglobinuria, tying-up syndrome, and azoturia) is in horses after unaccustomed exercise or insufficient training.

(2) There may be a familial basis to the disease in certain breeds (e.g., Quarter horse), but the condition is common among all breeds and is overrepresented in young, female animals.

(3) The condition may be chronic and recurring in certain animals.

**b. Etiology and pathogenesis:**

**(1) Etiology:**

(a) Predisposing factors include heavy musculature, irregular exercise, high grain diet, and a nervous disposition. There is degeneration of muscle cells and muscle bundles associated with unaccustomed exercise [e.g. Monday morning disease).

(b) The condition is not associated with the vitamin E or selenium status of the animal.

**(2) Pathogenesis:**

Recent studies have indicated that the pathogenesis may be a glycogen storage disorder with a possible autosomal-recessive pattern of inheritance. However, there may be multiple causes for the accumulation of glycogen and polysaccharide.

**c. Clinical findings:**

- There is a sudden onset of muscle soreness, which may range from stiffness (a rigid stance) to recumbency, and the animal sweats profusely after exercise. There is swelling and rigidity of various skeletal muscle groups.

- Myoglobinuria is evident clinically.

**d. Diagnostic plan and differential diagnoses:**

- Clinicians should rely on history, clinical findings, and laboratory results to establish the diagnosis.

- For chronic or **re**lapsing cases, a muscle biopsy may be useful in differentiating the clinical picture from that of lameness.

**e. Laboratory tests:**

- There is an increased CPK level (5000-1 0,000 IU/L)

- Myoglobinuria.

**f. Therapeutic plan:**

(1)Early therapy is essential, including rest, analgesics, and supportive therapy (e.g., bedding, intravenous fluids if necessary to increase excretion and dilute myoglobin).

(2) Sodium bicarbonate intravenously is warranted in cases with acidemia.

**g. Prevention:**

The only recommended prevention includes training modifications and attention to diet. Overconditioned animals are more prone to Exertional rhabdomyolysis. Dantrolene sodium has been used to slow calcium release and decrease muscular contraction.