Diseases of muscles

Azoturia

Tying up = Monday morning disease = Paralytic myoglobinuria
Sporadic exertional rhabdomyolysis in horse

❖**Def.:** Disease of horses, occurring during exercise after a period of inactivity on full rations, It is characterized by myoglobinuria, muscular degeneration & lumber paralysis.

❖**Incidence** → Draft horses & Race horses fed heavily on grain

❖**Causes**

1. Abnormalities in metabolic function
2. Overfeeding on rest day or high-carbohydrate diets
3. Dietary deficiency of vitamin E & selenium
4. Hypothyroidism
5. Sodium or potassium deficiency
6. Viral infection

✓**The most common cause is:**

- Performing exercise of unaccustomed intensity or duration, which can result in metabolic exhaustion & hyperthermia.
- Prolonged period (days to weeks) of rest in a horse previously accustomed to regular exercise.

❖**Pathogenesis**

▪Large stores of glycogen → in muscles during a period of rest

▪The glycogen is rapidly metabolized to → lactic acid

▪Lactic acid accumulated in the muscles in the form of **sarcolactate**

▪**Lactate** → is very toxic leading to swollen of the muscles → muscular hyaline degeneration and coagulation of muscle proteins.

▪Liberation of myoglobin → myoglobinemia → myoglobinuria & **red brown urine**

▪Coagulative necrosis of muscle fibers → hard, stiff & painful swelling of the large muscle

▪The gluteal muscles → most commonly involved → animal unable to stand and lie down (Sternal or lateral recumbency)

▪Pressure on the sciatic & other crural nerves → degeneration of the rectus femoris and vastus muscles

▪Excessive myoglobin → high pressure on the kidney tubules → degeneration of the renal tubules and formation of cast then nephrosis or nephropathy develops leading to → azoturia & uremia

▪Death due to septicemia or myohemoglobinuric nephrosis and uremia or due to degeneration of myocardium

❖Clinical finding

A. Acute form

1. Signs develop **15 minutes to 1 hour** after the beginning of work or exercise
2. Profuse sweating, stiffness of gait.
3. The signs may disappear in a few hours if the horse is allowed to rest immediately but the condition usually progresses to recumbency
4. Severe pain & repeated attempts to rise
5. Rapid respiration, week rapid pulse, congested mucus membrane & temperature may rise up to 40.5 ^{o}C
6. One limb or all four limbs may be affected.
7. The gluteal muscles is stiff & hard
8. **The urine is deep reddish brown or coffee color**
9. Appetite and water intakes are often normal, constipation may occur due to decrease in peristaltic movement

B. Sub-acute form

1. Signs are mild and myoglobinuria is **absent**
2. There is lameness, and great restriction of movement of the hind limbs
3. The horse first assuming a **dog sitting position** followed by lateral recumbency then death.

❖Diagnosis

1. Case history (rest, overfeeding)
2. Clinical signs
3. Urine analysis → Urine is dark red & contains myoglobin
4. Serum analysis → Elevated serum creatinine phosphokinase & serum glutamic oxalacetic transaminase (GOT).
5. Electrocardiograph is diagnostic in case of myocardial degeneration

❖Differential diagnosis

1. **Laminitis** → no discoloration of urine
2. **Myositis** → develops slowly & affects the muscles of the jaw

Myositis	Azoturia
بعد التمارين بربع ساعه بيحصل عرج	
بيختفى بعد شوية (disappear signs)	مع مرور الوقت بيزيد سوء

3. **Rheumatic myositis** → No discoloration of urine & respond to treatment with antirhumatic
4. **Vitamin E & selenium deficiency** → no discoloration of urine & decrease in serum selenium and vitamin E
5. **Iliac thrombosis** → can be detected by rectal palpation.
6. **Tetanus** → Prolapse of the 3rd eye lid, locked jaw, history of old wound
7. **Colic** → Fits of colic with normal colored urine

8. **Blood parasites** → Positive blood film

Hemoglobin urea	Myoglobinuria	Hematuria
Centrifugation		
▪ No sediment ▪ Bright red	▪ No sediment ▪ Dark red (**coffee color**)	▪ Clear sediment ▪ Bright red

❖Treatment

A. Hygienic treatment

1. Complete rest
2. Application of fresh heavy bedding
3. Turn the animal every **4 hours** to avoid hypostatic congestion
4. **Avoid** giving any **nitrogenous food** during the course of treatment
5. Application of **hot fomentation** especially on the hind quarter

B. Medical treatment

1.Sedative & anti-inflammatory → Novalgin 50 % 20 - 30 ml or dexamethasone

2.Antacid → sodium bicarbonate → to keep the urine alkaline & prevent myoglobin precipitation in the renal tubules

3.Electrolyte or glucose solution to maintain a high rate of urine flow & avoid tubular blockag

4. Vitamin E, selenium injection

5. Narcotic drugs → by stomach tube

6. Insulin, calcium preparation, antihistaminic drugs

7. Purgative

8. Diuretic or catheterization

❖Prognosis

1. Good if the animal remains standing
2. Recovery occurring in 2 - 4 days
3. Recumbency is usually followed by fatal uremia and septicemia.

❖Control

1. Reduce the grain ration to half on rest day and when the horse is getting no exercise
2. Daily exercise to minimizes the diseas

Diseases of muscles

1-myasthenia:

is skeletal muscle asthenia(weakness) inflammatory(myositis) and non inflammatory (myopathy)

General causes of myoathenia:

1-Ischemia e.g iliac thrombosis in horse

2-Metabolic effect on muscle fiber e.g hypo k hypo ca

3-nutritinal cause e.g vit E&SE deficiency

4- general toxemia and many plant toxins

5-F.M.D ,Black leg and Blue tounge

MYOPATHY

Non inflammatory degenerative change cch by muscle weakness, hyaline degeneration in muscle fiber, myoglobinuria and biochemical elevation of some muscle enzymes e.g

CPK

Aspartate aminotransferase

causes of myopathy

1-nutritional deficiency of vit E and SE

2- post exercise rhabdomyolysis occure as in equine paralytic myoglobinurea

3-Equine atypical myopathy cause by hypoglycin

4-Metabolic:hyperkalemic periodic paralysis in equine

5-Inherited myopathies

Pathogenesis

1-hyaline degeneration and coagulative necrosis
2-sudden death as in acute form of vit E and se deficiency
3-muscle weakness,pain, recumbancy, stiffgait and inability to move
4-myoglobinuria (dark brown urin) e.g azoturea
5-elevation of muscle enzymes as CPK

Clinical signs

1-sudden onset of muscle weakness
2-normal body temperature or slight elevated in sever cases
3-animal apper to be in pain but remain alert
4 respiratory and circulatory insufficiency
5-myoglbinuria and myoglobinemia
6-tachycardia and irregularity
7-death in 24 h

Treatment

-complete rest of animal
-pain killer
-treatment of systemic change
-fluid therapy
-nutrition correction