Equine Laminitis (Founder)

Its acute degeneration of the sensitive laminae of the hoof

Etiology…
1- Excessive eating of large quantities of lush green food
2-grain overload & Ingestion of large quantities of soluble carbohydrate
3-Retained placenta
4-Colic with persistent pawing
5- Obesity (over weighting) & lameness with increased weight bearing on one limb
6- Excessive work on hard surfaces
7- Corticosteroid induce laminitis
8- Standing for periods of days during transportation
9- Systemic illnesses induce laminitis such as metritis, pleuropneumonia, diarrhea & anterior enteritis

Pathogenesis…..
The basic lesion of laminitis is the separation of the sensitive laminae of the third phalanx from the inter digitating laminae lining the internal surface of the hoof, so that the third phalanx drops through the hoof and comes to rest on the sole,
The exact mechanism is unknown but , It is speculated that a pain - hypertension-vasoconstriction cycle develops in horses with acute laminitis
A-The pain associated with the laminar degeneration causes release of vasoconstrictor substances such as the catecholamines, angiotensin II, and vasopressin. These substances then cause peripheral vasoconstriction with a subsequent reduction in blood flow to the foot (Ischemia with micro thrombus formation also speculated), and systemic hypertension will follow.
B-Loss of the connection between the third phalanx and hoof allows the third phalanx to rotate within the hoof capsule, and to displaced ventrally (sink) within the hoof as a result of weight transmitted through the third phalanx.
C-Rotation of the third phalanx causes the sole to be pushed downward or'dropped', and the point of the toe of the third phalanx may penetrate the sole.
D-Serum accumulates in the space created by degeneration of the laminae and displacement of the third phalanx and there is breakdown of the white line

The disease occurs in 3 distinct phases…..
1-a developmental stage in which lesions are detectable in the sensitive laminae but during which there are no clinical signs
2-the acute phase from the development of the first clinical signs through to rapid resolution or to rotation or ventral displacement of the third phalanx;
3- The chronic stage evidenced by rotation of the third phalanx with or without ventral displacement and characterized by persistent pain.

Clinical findings ….
Acute laminitis ….
Develops rapidly may be with one hour & manifested by..
1- Signs of the disease are mostly related to pain in the feet, All hooves may be affected, but ore commonly the fore feet are affected and hind feet are spared.
2-difficult movement with repetitive and frequent shifting of weight from one foot to the other& There is acharacteristic shuffling gait

3-the animal show anxiety, accompanied by muscle fasciculation, sweating, a marked increase in heart rate to as high as 75/min, rapid,shallow respiration, and a moderate elevation of body temperature.
4- There is a characteristic posture with all four feet (Saw hourse)being placed forward of their normal position with lowering of the head and arching of the back, and some horses may be recumbent for long periods.
5- The diagnostic signs in laminitis include pain on palpation around the coronet and , a marked withdrawal response when hoof testers are applied to the hoof.
6- The intensity of the increase pulse in the palmar digital artery.
7- In horses in which the third phalanx is displaced distally, a concavity may be palpable at the coronary band.

Chronic laminitis ….manifested by…
1- there is separation of the wall from the sensitive laminae and dropping of the sole.
2- The hoof wall spreads and develops marked horizontal ridges, and the slope of the anterior surface of the wall becomes accentuated and concave.
3- Horses with chronic or refractory laminitis may continue to feel much pain, lose weight, and develop decubitus ulcers over pressure points because of prolonged recumbency.
4- Loss of integrity of the sole and disruption of the white line may allow infection to develop in the degenerate laminae.
5- infection may spread to involve the pedal bone, causing a septic pedal osteomyelitis.
3- the animal becomes lame easily with exercise and may suffer repeated, mild attacks of laminitis.
4- Radiographs of more severe or advanced cases will demonstrate rotation of the pedal bone within the hoof. Chronic or refractory cases may have osteopenia of the pedal bone with proliferation of bone at the toe.

Treatment…
Acute laminitis is an emergency and treatment should be started without delay, and the most important principles for treatment are ,,,
1-Removal of the causative agent or treatment of the inciting disease

- The inciting disease should be treated firstly and attempt made to remove any causative agent. Horses with traumatic laminitis should be rested and housed in stalls that have well bedded

2-Pain relief and minimization of inflammation.

- the use of non- steroidal anti-inflammatory drugs to break the pain-hypertension- peripheral vasoconstriction cycle that may be important in the pathogenesis of laminitis is advisable by using ,,

a-Phenylbutazone,at doses of 2.2-4.4 mg/kg intravenously or orally every 12-24 hours, is an effective analgesic in cases of mild to moderate laminitis. Higher doses (6.6 mg/kg every 12-24 hours) may be required in severe cases. However, the potential for phenylbutazone toxicosis, evident as colic, gastrointestinal ulceration, nephrosis, hypoproteinemia, leukopenia, and hyponatremia.
b- Flunixin meglumine (1 . 1 mg/kg, 1M or I.V every 8-12 hours)
c- ketoprofen (2.2 mg/kg, 1M every 12-24 hours).
d- Dimethyl sulfoxide (DMSO) 1 g/kg, intravenously as a 10% solution in isotonic sodium chloride.

Note: the administration of corticosteroid is contraindicated

3-Vasodilation of blood vessels in the foot. by using

- Acetylpromazine at dose rates ranging from0.01 to 0.05 mg/kg, 1M, every 6-12 hours

4-Prevention of formation of micro thrombi in dermal capillaries..by using

a-Aspirin which reduces platelet aggregation in normal horses by blocking formation of thromboxane A2,Asprine is administered at a dose of 10 mg/kg orally every 48 hours.

b- Heparin 40 to 80 mg/kg IV or subcutaneously every 8-12 hours for 3-5 days.

5-Prevention of rotation or distal displacement of the pedal bone by Mechanical support to provide pain relief and to prevent rotation or distal displacement of the pedal bone.

6- Promotion of keratinization and hoof growth by using

Methionine oral dose rate 10 g/day for 3 days followed by 5 g/day for 10 days.

7- Antibiotics may be indicated to prevent secondary infection of the degenerate laminae.

8- local therapy such as ice baths of the hoof or having the horse stand in cold water ,some time advisable

Purpura hemorrhagica

Its acute and non-contagious disease of horses characterized by vasculitis

Etiology …

The common association of the disease is with Strep. equi infection of the upper respiratory tract, causing deposition of complexes of antigen and immunoglobulin in the walls of capillaries and small blood vessels. The disease appears to be immune complex-mediated and due to a type III hypersensitivity reaction.

Epidemiology …

1-Purpura hemorrhagica is non-contagious, sporadic disease of horses.It has been recorded rarely in pigs and cattle.

2-The incidence is highest when extensive outbreaks of strangles occur,

3-There is a suggestion that the disease might be associated with an adverse reaction to therapeutic drugs or vaccination against strep.equi .

Pathogenesis …

The basis of the disease process is an aseptic vasculitis of capillary walls that is accompanied by extravasation of plasma and blood into the tissues. blood clothing factors were affected in sever cases .

Clinical findings …

1- Affected horses are depressed and have reduced or absent appetite

2-high fever with increase heart rate

3- Extensive subcutaneous edematous swellings are the characteristic sign of the disease, They occur most commonly about the face and muzzle, but are often present on other parts of the body and are not necessarily symmetrical in distribution.

5- The swellings may appear suddenly or develop gradually over several days. They are cold and painless,pit on pressure,& it may be tightly distended and even ooze red serum

6- Swellings on the head or throat may cause pressure on the pharynx with subsequent dyspnea and dysphagia.

7- Extensive edema of the limbs occurs in almost all cases.

8- Submucous hemorrhages occur in the nasal cavities and mouth, and petechiae may be present under the conjunctiva.

9- Hemorrhage and edema of the gut wall may cause colic but in most cases there is no diarrhea or constipation.

10- Severely affected skin, and especially that of the legs, may slough and leave granulating wounds.

11- Infarctive purpura hemorrhagica some times occur and manifested by, infarction of multiple tissues including the gastrointestinal tract and muscle, affected horses have signs of colic and muscle swelling.

12- The course of the disease is usually 1-2 weeks and many animals die from blood loss, dyspnea due to laryngeal or pharyngeal swelling, and secondary bacterial infections.

Treatment …it include

1- Reduction of inflammation of the blood vessels.. which done by

- A-Corticosteroids such as dexamethasone (0.05-0.2 mg/kg,IV or 1M every 24 hours) or prednisolone (0.5-1 mg/kg, 1M or IV every 24 hours) .

- B- Non-steroidal anti-inflammatory drugs (phenylbutazone 2.2 mg/kg orally or IV every 12 hours, or flunixin meglumine 1.1 mg/kg orally or IV every 12 hours) may reduce inflammation and provide some analgesia.

2- Removal of the source of the antigenic stimulus..by

- penicillin (procaine penicillin, 20 000 IU/kg, 1M every 12 hours,or potassium penicillin, 20000 IU/kg, IV every 6 hours) until the clinical signs resolve.

3- Supportive care…included bandaging of swollen limbs, care of wounds, , and intravenous fluid adpministration.

Snake bite

Its an important accident affected domesticated animals .the most common snakes are the Crotalus spp such as rattlesnakes and pit vipers , the elapid snakes including the cobra, mamba ,and Australia's snakes including tiger snakes and common brown snakes.

Epidemiology …

1-The incidence of snakebite is controlled by the geographical distribution of the snakes and their numbers.

2-the morbidity rate in farm animals is low, although a mortality rate may reach to 20%

3-Most snakebite accidents occur during the summer months and bites are mainly affected parts which attach the ground.

Pathogenesis …

- The effects of snakebite (envenomation)depend upon the size and species of the snake, the size of the bitten animal and the location of the bite, particularly with reference to the thickness of the hair coat and the quantity of subcutaneous fat.
- As a general rule the venom is injected by fangs which leave a bite mark comprising a row of small punctures with two large punctures outside them. An exception is the coral snake, which must chew to inoculate the venom
- The important pathogenic effect include

1-Neurotoxins, causing flaccid paralysis,pupillary dilatation and paralytic respiratory failure

2-Cyto-toxcisity, which are associated with tissue necrosis, including platelets,leading to intravascular coagulation

3-Hemolysins, leading to a hemorrhagic tendency

4-Myotoxins, causing muscle necrosis and myoglobinuria.

Clinical findings …

1-Bites are associated with a local swelling which develops rapidly and is associated with severe pain, usually sufficient to produce signs of excitement and anxiety.

2-Bites about the head may be followed by swellings of sufficient size to cause dyspnea. If sufficient neurotoxin has been injected a secondary stage of excitement occursand followed by marked dilatation of the pupils, salivation, hyperesthesia, tetany,depression, recumbency, and terminal paralysis.

3- In small animals, death may occur due to asphyxia during convulsions in the excitement stage of the disease.

4- In animals that recover there is usually local sloughing at the site of the swelling,and secondary bacterial infection may developed

Clinical pathology …

An ELISA for identification of venom in blood, urine or other body tissue or fluid

Treatment …

1-the application of a tourniquet proximal to a limb bite site.

2-a firm pressure bandage is applied over the bite to restrict the distribution of the venom via the lymphatics and retain it in the site and prevent systemic effects

3-Excision of the bite site is recommended

4-Systemic treatment should include antivenin, antibiotics, and antitoxin

5-ACTH,cortisone, and antihistamines. These drugs have been found to be valuable as a protection against possible anaphylaxis after treatment with antivenin,