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LECTURE NOTES

COURSE: VCM 503: COMPANION ANIMAL MEDICINE

(EQUINE SECTION)

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TOPICS:

- **DIABETES (MELLITUS & INSIPIDUS)**
- **AZUTORIA**
- **POISONING**
- **NUTRITIONAL DEFICIENCY**

LECTURE I: DIABETES MELLITUS (D.M)

INTRODUCTION

Diabetes Mellitus is a chronic disorder of carbohydrate metabolism due to relative or absolute insulin deficiency. It is a very rare condition in horses.

AETIOPATHOGENESIS

The pathogenic mechanism responsible for decreased insulin production and secretion are multiple but usually they are related to destruction of islet cells, secondary to either severe pancreatitis or selective degeneration. Most cases of equine diabetes mellitus (in the literature) are actually cases of secondary diabetes associated with Cushing's diseases primary D.M is associated with pancreatic destruction secondary to chronic pancreatitis of non-specific origin e.g. streptococcus infection ("Bastard Strangles") or Corynebacterium Spp obscesses or pancreatic destruction associated with strongyle migration

(generally strongylus equines).

CLINICAL FINDINGS

Weight loss, polydipsia, polyuria, intense hyperlipidemia and high blood levels of cholesterol, trighycerides and glucose are common clinical findings when the condition is observed/encountered. The condition is most likely to occur in old horses and may be due to pancreatic injury related to migration of strongly larvae.

PROGNOSIS AND MANAGEMENT

Prognosis is poor guarded. Treatment may involve a combination of weight reduction diet (high fiber, high complex CHO) and insulin.

II. DIABETES INSIPIDUS

Diabetes insipidus is caused by reduced secretion of anti diuretic hormone (ADH). When target cells in the kidney lack the biochemical machinery necessary to respond to secretion of normal or increased circulating levels of ADH, nephrogenic

Neurogenic diabetes Inspidus occurs when the physiologic mechanism of the diseases is either a failure of production of or diminished release of vasopresin from the hypothalamus and posterior pituitary gland in response to sufficient increases in phornia osmolality and or diminution of blood volume. These conditions are not common in horses.

AETIOPATHOGENESIS

Some lesions responsible for the destruction of ADH synthesis or secretion in hypophyseal D.I include large pituitary neoplasm particularly cyst or inflammatory granuloma and traumatic injury to the skull with haemorrhage and glia proliteration in the neurohypophyseal system.

CLINICAL FINDINGS AND DIAGNOSIS

Polyuria, polydipsia are the predominant clinical signs reported in D.I. Rapid & severe dehydration and depression is expected in horses with D.I if water is unavailable for 12hrs or more. In addition, hyposthenuria (specific gravity <1.006) occurs with neurogenic D.I even in the face of dehydration.

The two syndromes of D.I can be separated by measuring plasma vasopressin after water deprivation. (vasopressin increases with nephrogenic D.I) and response to exogenous vasopression (0.20 u/kg 1m should result in substantial increase in urine concentration for 2-12 hours and a decline in water consumption for the same with neurogenic D.I).

MANAGEMENT

Treatment of D.I is directed at managing the polyurra/polydipsia. With Neurogenic D.I, recovery may occur of the disorder is a result of a

factor such as trauma of the posterior pituitary, since additional secretary neurons may be generated within 6 wks. Hormone replacement could be used as a treatment for congenital neurogenic D>I but cost would likely prevent this.

Management of both types of D.I involve control of sodium & water intake sodium sodium be restricted to minimally acceptable dietary levels while adequate water should be available to dehydration.

III. **AZOTURIA**

Synonyms/related conditions

- Paralytic myoglobinuria
- Typing-up or cording-up
- Monday morning disease
- Exertional myopathies.
- Polymyositis *Sacral Paralyers
- Desioruophlic myositis * Black water
- Muscular dystrophy
- White muscle disease.

INTRODUCTION

Azoturia refers to excess nitrogen compounds (Urea) in the urine. This was thought to be so because of the dark reddish brown colour of the urine produced during the acite stage of the condition but

subsequently found to be myoglobin and hence the name paralytic myoglobinuria.

The term equine exertional myopathies encompasses the various muscle related conditions (as listed above).

AETIOPATHOGENESIS

The actual causes is unknown. Although it has be associated with exercise after a period of rest during which feed has not been restricted, it has also been seen in horses on pasture. The myositis may arise from direct trauma or as a part of a syndrome including secondary deficiency of vite and absence of protective level of selection in diet.

CLINICAL FINDINGS AND DIAGNOSIS

The ---------- is usually sudden and dramatic, there is profuse sweating, muscular stiftness, difficult movement, and trembling. Rapid pulse and respirant (with dialeted nostril). and in severe cases myoglobinuria, recumbency and nervous signs involvement. Increased serum activities of AST and CK are useful indicators of the extent of muscle damage.

Prognosis depends on the extent of muscle damage. It is good for standing animal; faily good for recumbent ones due to loss of use of hindquarter provided they remain quiet and pulse returns to normal within 24hrs. prognosis is poor when nervous signs are involved.

MANAGEMENT

Keep the patient as quiet as possible while attempts should be made to keep the animal standing. Sedatives/tranquilizers are indicated to relieve pain and restlessness. Selenium and Vit. E injection are also favourable symptomatic therapy is often required.

IV. POISONING

INTRODUCTION

Toxicology is the study of poisons on biologic system, including their chemical properties and biological effect.

A poison is any solid, liquid or gas that interferes with life processes, ranging from the molecular level, to the organism level, to the population level. Poisoning is a state of being poisoned.

The term toxin refers to poisons produced by a biologic source (e.g. veioms. Plant toxins) while the toxic agent is referred to as a toxicant. Toxicosis, poisoning and intoxication are synonymous terms for the disease produced by a toxicant.

Poison is any substance that on ingestion, inhalation, absorption, application, injection or development within the body, in relatively small amount may cause structural damage or functional disturbance. While poisoning is the morbid condition produced by a poison.

As compared with other domesticated animal, cases of poisoning

are less commonly encountered in horses.

When poisoning is suspected in horses, the 1st thing to do is to identify possible sources of the toxicant and exposure to such sources rapidly eliminated while systemic symptomatic therapy is instituted for the affected horse/population.

CAUSES/SOURCES OF POISONING

Sources of possible poisoning varied in horses. It ranges pasture contamination with substances like lead, insecticides etc to accidental administration e.g. chemical ingestion or overdose of otherwise helpful drugs to even malicious administrationstion or overdose of otherwise helpful drugs to even malicious administration.

FACTORS AFFECTING THE ACTIONS OF POISONS

i. Absorption the physical and chemical nature of the toxicant determines its solubility which in turns influences its absorption. The route of administration will also affects its absorb. Gaseous substances renders absorption earliest etc.

ii. Exposure the dose is of primary concern however, the exact intake of poison is seldom known. Durant and frequency of exposure are also important.

iii. Biologic factor: This varies with different species reacting differently to a particular poison. Age, size, body weight, route of

metabolism and elimination.

DIAGNOSIS OF POISONING

- Adequate history
- Clinical evaluation including laboratory anslysis (whole blood, serum, urine, milk, ingesta, feed, plants, water etc).
- Post mortem/Neucropsy if the animal dies.
- Clinical signs: General: anorexia, dehydrat, depressor, emaciation, dyspnea
- CNS- Hyperexictability, muscular twitching, incoordinate, paresis
- GIT- diarrhea, salivat, colic abnormal mout/ataxia
- Blood
- Liver

List of plants, chemicals other toxicant/poisoning.

- Plants: accidental feeding (overgrazing, drought, use of herbicides, masking plant in hay etc.

- Crotalaria spp - Bracken fern (Ptefidium aquilinum
  - Lantana camera
  - Strichnine
  - Locoweeds: Asragalus & Oxytropis spp
  - Consult your toxicology notes.

* Chemical
  - Lead
  - Copper
  - Cyanide
  - Fluoride etc.

* Mycoxins (Aflatoxins)
  - Aspergillus flavus
  - Pelicillium

- Snake bite/venona (zootoxins)

  Rattlesnakes- crotalus spp

* **Bee Stigs**

GENERAL APPROACH TO MANAGEMENT OF POISONING

- Prevention of further absorption
  - Remove topical toxicant by thorough washing with soap and water
  - Give laxative (mineral oil, magnesium etc $S0_4$)
  - Activated charcoal (1-2g/kg) is effective in absorbing a wide variety of compounds and usually detoxicant
  - Administer nonspecific detoxicant (¼)
- Calcium gluconate (20%) 100-500ml
- Dextrose (500%) 500 –100ml
- Sodium thiosuplhate (25%) 150-500ml
- **Supportive Therapy**
  - Its necessary until toxicant has been metabolized and

eliminated

- Types of 5 therapy will depend on the animal's clinical

Condition E.g

- Control of conclusive seizure
- Maintenance of respiration
- Treatment of school
- Correction of electrolyte and fluid loss
- Control of cardiac dysfunction
- Alleviation of pain etc

Specific Antidotes

- When the actual poisoning is established and its antidote known, it should be administered.