



Benha University
Fac Vet Medicine
Animal Med Dept
Vet. Internal Medicine
General Medicine Exam
Vet. Pharmaceuticals and Biological preparation
program



Time allowed: 2 hrs.
Date: 22-5-2016
Total marks: 50 marks

جامعة بنها
كلية الطب البيطري
قسم طب الحيوان
الامراض الباطنة
برنامج الأدوية البيطرية والمستحضرات البيولوجية

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Please answer all questions

Answer model

1- Describe the clinical signs of the following:

- a. Guttural pouch diseases
GP mycosis

(6 marks)

- a-Dysphagia due to involvement of pharyngeal branches of hypoglossal and vagal nerves.
b-laryngeal hemiplagia(roaring disease) due to involvement of laryngeal branches of vagus.
c-Facial paresis due to involvement of facial nerve.
d-Horner's syndrome (ipsilateral facial sweating and hyperthermia, smaller palpebral fissure, mild miosis) due to involvement of sympathetic neurons.

-The clinical signs of guttural pouch empyemia are:

- 1- Chronic toxaemia (manifested by pyrexia)
- 2- distension of one or both pouches.
- 3- pain on swallowing and dysphagia
- 4- Coughing
- 5-Intermittent mucopurulent or purulent nasal discharge especially when animal lowers its head.

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GP tympany

A congenital defect of closure of the Eustachian tube that allows the pouch to fill with air and distended obviously and to an enormous size.

2- Diabetes mellitus in dogs

(6 marks)

- 1- The onset of D.M. is insidious.
- 2- Polyuria and polydipsia are the prime clinical signs.
- 3- Polyphagia is often present.
- 4- Bilateral cataract (opacity of lens), corneal opacity and retinopathy.
- 5- Weight loss and emaciation (due to muscle wasting).
- 6- Digestive disorder if pancreatic parenchyma has been damaged particularly due to pancreatitis.
- 7- In the terminal ketoacidotic stage (blood acidosis phase) there are:
 - a) Acetone odour may be detected on the breath.
 - b) Neuropathy due to direct toxic effect of ketogenic acids on CNS.
 - c) Inappetance, increased rate and depth of breathing, vomiting (2-3 times per 48 hours).
 - d) Severe dehydration (may lead to acute renal failure) and listlessness
 - e) The terminal episode is diabetic coma and death.

3- Outline the causes and pathogenesis of the following:

a. Azoturia in horses

(6 marks)

Etiology and pathogenesis :

1-Carbohydrate Overloading

- The classical presentation is feeding the draught horse on full working ration during resting at weekend and when the horse returns to work several days later it suffers an attack of the disease.
- The muscle glycogen accumulates during the rest period and when used during exercise it produces excessive lactic acid in a rate higher than removal by blood vessels leading to lactic acid accumulation.
- This causes local tissue damage (myopathy) and constriction of the blood vessels, resulting in decreased blood flow to the tissues and further reduction in lactic acid removal.

- Accumulation of sarcolactic acid in muscles produces swelling and hardening of muscle (hard board-like).

2-Local Hypoxia

- Certain types of muscle fibers are larger, have greater glycogen stores and fewer surrounding blood vessels than others.
- Local hypoxia (lack of oxygen supplied by the blood) may increase the lactic acid production in these fibers.
- However equine rhabdomyolysis normally occurs at the start of exercise, when these fibres would not yet be working and the condition is not usually seen in horses with other conditions causing impaired circulation

3-Thiamine Deficiency

- Thiamine (one of the Vitamin B complex) involved in the metabolism of waste products from muscle activity.
- A deficiency, therefore, could lead to a build up of these waste products and hence, lactic acidosis.

4-Vitamin E and Selenium Deficiency

- This theory is based on reports of success at preventing further episodes following supplementation.

5-Hormonal Disturbances

- Reproductive hormones, thyroid hormones and cortisol have all been implicated in equine rhabdomyolysis.

6-Electrolyte Imbalances

- Chronic sodium and/or potassium deficiencies may be involved in chronic equine rhabdomyolysis.

Pathogenesis

- 1- During exercise the large store of glycogen formed during the period of rest in the muscles metabolized to sarcolactic acid.
- 2- Accumulation of lactic acid leads to:
 - a- Degeneration of the muscles and liberation of myoglobin (muscle haemoglobin)
 - b- Swelling of muscle because lactic acid is hydrophilic.

b. Constipation in dogs

(6 marks)

Causes:

1. Dietary and environmental causes: indigestible material (wool, hair, bones) mixed with the faeces causes impaction of the colon.

2. Mechanical obstruction:

a. Intraluminal:

1. Tumor of colon or rectum.
2. Perineal hernia.
3. Rectal diverticulum.

b. Extraluminal:

1. Enlarged prostate gland or prostatitis.
2. Healed pelvic fracture → narrowing of pelvic canal.
3. Tumor in the pelvis or prostate gland.

3. Painful defecation:

1. Anal sacculitis or abscess.

2. Perianal fissure.
3. Anal spasm.
4. Rectal foreign body.
5. Proctitis.
4. Neurologic:
 1. Central:

Due to C.N.S dysfunction or spinal cord dysfunction.
 2. Intrinsic colonic nerve dysfunction → megacolon.
5. Metabolic
 1. hypothyroidism
 2. hyperparathyroidism
 3. hypokalemia
4. Debility → general muscular weakness.
6. Drugs:
 - Anticholinergics
 - Antihistaminics
 - Diuretics
 - Barium sulphate used for radiographic examination.

Pathogenesis:

Usually colon functions to absorb sodium (in the proximal part) and the associated water → hard faeces.

The lower part of colon used for storage of the absorbed sodium (not returned to lumen except if the mucosa inflamed to the hard faeces causes irritation to the mucosa).

4- Plan the line of diagnosis for the following:

- a. Equine colic

(6 marks)

(a) Acute colic

Acute colic with severe pain may be caused by:

- 1- Acute gastric dilatation resulting engorgement with grain.
- 2- Impaction of the ileocecal valve resulting from feeding finely chopped indigestible roughage..
- 3- Impaction of the small colon with foreign materials.
- 4- Accumulation of gas (Flatulent colic) due to ingestion.
- 5- Intestinal accidents including volvulus; intussusception and strangulation (MECHANICAL COLIC).
- 6- Enteritis, especially that caused by the ingestion of sand (SAND COLIC)
- 7- Hemorrhage into intestinal wall as occurs in purpura haemorrhagica and anthrax.

(B) Subacute Colic

Subacute colic includes 2 common forms of the disease.

(a) obstructive colic:

caused by impaction of the cecum or colon (pelvic flexure) with :

- 1- Undigested fiber.
- 2- Dry, firm mass of ingesta.

(b) Spasmodic colic :

Caused by increased gut motility (violent irregular peristaltic movement) and usually following.

- 1- Periods of excitement.

- 2- Drinking of cold water after exercise.
- 3-As a consequence of severe hunger.
- 4- Sudden change of food.
- 5- irritation of the gut by unsuitable food stuffs.
- 1- Verminous aneurysm (Recurrent colic) caused by strongylus vulgaris larvae (verminous thromboembolic colic)
- 2- old age.
- 3- Debility.
- 4- poor teeth.
- 5- Dietetics error (feeding indigestible roughage).
- 6- 5-Phytobezoars.
- 7- Enteroliths .

Clinical findings :

- The clinical signs in different types colic are much the same, varying only in the severity
 - Pain observed being almost continuous in acute cases and intermittent in subacute cases. The following manifestation of pain may be observed:

- 1- Restlessness manifested by kicking the belly and rolling.
- 2-Looking at the flank (**FLANK WATCHING**) is a common sign especially, in cecal tympany and impaction.
- 3- Lie down carefully and get up slowly, especially in flatulent colic.
- 4- Often adopting a dog-sitting posture especially in acute gastric dilatation.
- 5- Affected horses may adopt other abnormal postures, including saw horse attitude and lying on the back.
- 6- In the most severe cases there is
 - profuse, patchy sweating
 - Sobbing respiration
 - Signs of shock including rapid pulse and clammy skin
 - The horses movements are so violent that self physical injury occurs.
- 7- Auscultation of the abdomen is helpful in diagnosis.
 - In flatulent colic, there are high – pitched "gassy pings"
 - In spasmodic, there are loud gut sound (continuous borborygmi).
 - in obstructive colic, the normal sounds are decreased or absent.
- 8- Rectal examination is essential in diagnosis :
 - In flatulent colic, there is gaseous distention of intestinal loops.
 - No abnormalities are detectable in spasmodic colic.
 - Impaction of cecum or colon are readily palpable.
 - In impaction of ileocecal valve, a cylindrical mass in the terminal part of ileum high up in the right flank.
 - In case of verminous colic, the obstructed a.v. may be palpable and slack, distended loops of intestine can be found.
 - Small intestinal accidents are characterized by fluid-filled loops of gut of appropriate size.
 - Hernias into the inguinal canal are best palpated per rectum, as the intestine may not protrude as far as the scrotum .
- 9- Projectile vomiting of evil-smelling, green fluid usually result from an obstruction of the intestine at any level from pylorus to the ileocecal valve.

b. Pancreatitis in dogs

(6 marks)

Causes:

1. Trauma during surgical operation.
2. Autoimmune mechanisms.
3. Metabolic abnormalities.

Symptoms:

Dogs of middle aged and obese dogs usually affected.

1. Vomition.
2. Abdominal pain.
3. Faeces usually: contain blood.
4. Jaundice may occur when bile due to either occluded by inflammation or digested by enzymes.
5. Shock may follow
6. Hypoglycemia, increase insulin production and hypocalcaemia (Ca^{++} combined with fat in peritoneal cavity) are detected on laboratory examination.
7. Secondary bacterial infection may occur.

Chronic relapsing pancreatitis: (may occur also in cats)

1. Repeated mild attacks of vomition.
2. Repeated mild attacks of abdominal pain.
3. Voluminous orange or clay coloured rancid smell faeces.
4. Faeces may contain undigested particles.
5. Diabetes mellitus followed.

Diagnosis:

1. History (age and obesity)
2. Symptoms: - Abdominal pain – vomition.
3. Laboratory tests:
 - A. *Faeces:*
 1. Presence of fat and undigested food.
 2. Absence of trypsin
 - B. *Serum:*
 1. Hyperlipemia.
 2. Elevated amylase and lipase.

5-

- a. Prescribe the treatment protocol for COPD in horses (4 marks)

1- The provision of fresh air, as the horse should be kept permanently in the open air.

2- Avoid exposure of the horse to dust, therefore wood chippings or saw dust should be used for bedding instead of straw.

3- Corticosteroids, such as dexamethazone 25mg/ animal In every 2nd day for up to 2 weeks may give remarkable results because of their antinflammatory used as an aid in the temporary treatment of COPD such as:

a- isoprenaline inhalation is a sympathomimetic drug which stimulate beta-1 (cardiac) and beta2 (smooth muscle) receptors causing cardiac stimulation and bronchial muscle relaxation (temporary relied for 1-2 hrs.)

b- Turbutaline inhalation is a sympathomimetic drug which exhibits action selectively in smooth muscle receptors (beta-2) causing bronchodilation with no cardiac stimulation for 1-2 hours.

c- Clenbuterol HCL(long acting bronchodilator) is a beta-2 sympathomimetic has no untoward effect on circulatory system of exercising horses.

d- Atropine is a parasympatholytic drug given In at rate of 0.02 mg/kg B.w.

5- Antibiotics are used in treatment of COPD in horses, but there is only limited clinical evidence of their value. E.g. penicillin procaine 25.000

I.U/kg B.W. daily for 2 weeks (sulphamethazine has also been recommended).

b. You are called to examine an 8-years old mare with muscular fibrillation and tremors. Difficulty in urination and defecation were observed. Plan your line of diagnosis, differential diagnosis and treatment? (10 marks)

The disease could be:

- 1- Lactation tetany of mares
 - 2- Hyperlipemia
 - 3- Encephalomalacia
- The most common is lactation tetany