Benha University	Anthority for	Kein Alan Surance and the sura
Fac Vet Medicine	Time allowed: 3 h Date: ^Y 4-5-2016	جامعة بنها كلية الطب البيطرى قسم طب الحيوان الامراض الباطنة
Animal Med Dept Vet. Internal Medicine	Total marks: 50 marks	قسم طب الحيوان
Complementary Exam Food quality and control program	بة الاغذية ===============================	الامراض الباطنة امتحان تكميلى – برنامج جودة ومراقب ====================================
Answer model		
Please answer all questions		
1- Describe the clinical signs o	-	
a. Anal sacculitis in dog Scooting	gs	(5 marks)
Constipation Hard feces		
Biting of back		
2- Diabetes insipidus in dogs		(5 marks)
-polyuria -polydipsia		
-urine is watery with low Sp	Gr.	
3- Outline the causes and path	nogenesis of the followin	g:
Cuching diagona in d	-	(E. 100 e. 11/10)

a. Cushing disease in dogs (5 marks) Hyperadrinocorticism

- Excess of cortisol which may or may not be due to excessive ACTH

- 1. Increased secretion of ACTH occurs in about 85% of cases causing bilateral adrenocortical hyperplasia.
- In majority of these (85% cases) an autonomously secreting neoplasmas (carcinoma or adenoma) of ACTH-secreting cells of 'adenohypophysis' is responsible.
- The other cases of the 85% cases) where there is over-production of ACTH (idiopathic cortisol hyperplasia) are considered to be due to some 'functional' defect in the hypothalamus or adenohypophysis producing insensitivity to the usual negative feed-back control.
- 2. Most of the remaining 15% of cases due to autonomously secreting neoplasm (carcinomas and adenoma) of the adrenal cortex.
- Due to the negative feed-back effect, ACTH secretion is minimal and the cortex of the other gland is atrophied. Long term administration of glucocorticiods in high doses also produces this disorder (Latrogenic Cushing's syndrome) and again there is suppressed ACTH production and consequently bilateral adrenocortical atrophy.

b. Pancreatitis in dogs

Causes:

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

- 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.
 - 4- Plan the line of diagnosis for the following:
 - a. Enteritis in dogs

History

- 1. Physical causes:
 - a. Overeating
 - b. Spoiled food.
 - c. Indigestible material (wool, hair, bones).
 - d. Allergy to egg milk or horse meat.
- 2. Chemical causes:
 - Caustic solutions \rightarrow lead, arsenic, thallium, Hg and phenol.
- 3. Infectious:
 - a. Distemper
 - b. Leptospirosis.
 - c. Panleukopenia.
 - d. Toxoplasmosis.
 - e. Parasitic.
 - -Coccidiosis.
 - -Giardiasis.
 - -Entamoeba.
 - -Balantidium.

<u>Clinical findings:</u>

1. Diarrhoea.

(5 marks)

(5 marks)

- 2. Tenesmus especially on palpation.
- 3. Abdominal pain especially on palpation.
- 4. Temperature may or may not elevated.
- 5. Tense abdomen.
- 6. The animal may lie outstretched fore limbs with its sternum or ground (cold place) or may lie in a praying position (elbow and sternum on the ground while hind limbs were standing).
- 7. Borborygmus (loud peristaltic sound).
- 8. Dehydration, electrolyte depletion, or acidosis.
- 9. Faces: fluidy

Offensive odour May be black or tar-like \rightarrow HB May be streaked with blood.

b. Addison's disease

(5 marks)

Reduced output of both glucocorticiods and mineralocortcoids due to:

- 1. Bilateral cortical in 90% of cases and mechanism (idiopathic Addison's disease).
- 2. Destruction of the cortices (less common causes) by neoplastic metastases, amyloidosis, chronic inflammation and fibrosis, as well as, thromobosis of the adrenal vessels.
- 3. Following treatment of Cushing's disease with mitotane or adrenalectomy.
- 4. Destructive lesions of the pituitary or hypothalamus, resulted in reduced ACTH secretion and "secondary" hypoadrenocorticism, with a reduced secretion of glucocorticiods alone.
- 5. The sudden cessation of long-term corticosteroid administration which has suppressed ACTH production will have the same short-term effects.

Clinical signs:

The clinical features of Addison's disease are nonspecific and easily confused with those of renal G.I.I. diseases.

- 1. If the disease onset suddenly, there is shock, collapse, and renal failure (Addisonian crisis = acute cases).
- 2. If the disease develop progressively (chronic cases), there is "waxing and warning" of signs:
- a. Dehydration and weight loss due to fluid loss.
- b. Reduction in blood pressure, cardiac output and weak pulse.
- c. Polyuria and polydipsia in some animals.
- d. Anorexia, abdominal pain, vomiting and diarrhoea with trembling due to lack of glucocorticiods.
- e. Depression and muscular weakness in chronic cases.

Diagnosis:

- a. History:
- b. Clinical signs
- c. Lab. Diagnosis:
- Differential WBCS:
- 1. Eosinophilia occurs in some, but not all, affected animals.
- 2. Absolute lymphocytosis.
- Blood chemistry
- 1. Hyponatraemia due to increased excretion of sodium, resulted from adlosterone deficiency.
- 2. Azotaemia due to increased blood urea and creatinine.
- 3. Hyponatraemia may, but rarely, be present.
- Specific diagnostic tests:
- "ACTH stimulation test" is most valuable diagnostic test which can be routinely performed.

5-

a. Describe the treatment of diabetes mellitus in dogs (5 marks)

a- Mild diabetes mellitus:

1-Reduction of carbohydrate intake is sufficient to control hyperglycaemia and gluosuria i.e, control by change to semidiabetic diet.

2- If the dog is obese reduce fat intake additionally i.e. protein and low in carbohydrate is advocated, consisting of approximately 80% meat and 20% carbohydrate (rice or biscuit) and fed at rate of 30 gm /kg b.wt. / day.

b- Moderate diabetes mellitus:

In addition to the previous diet restriction, an oral hypoglycaemic drug to - stimulate insulin secretion may be needed

for example:

1- Sulphonyluras (most commonly employed).

2- Tolbutamide (but markedly hepatotoxic in the dog).

3- chloropamide (preferable to use)

c- Severe DM (diabetic ketonaemia and ketonuria)

1- In addition to the before mentioned diet restriction, routine i.e. daily S/C insulin for successful treatment and does usually ranges from 5-50 units daily depending on the severity of the disease and size of the dog. Insulin *should* be given several hours prior to feeding.

2- A depot insulin injected S/C with a 24-hours action is preferable for example:

- Lente insulin (a mixture of insulin zinc suspensions).

- Isophane insulin (known as neutral protamine hagedorn = NPH.

- Protamine zinc insulin (PZI).

N.B.:

Soluble insulin (crystalline insulin) injection with a rapid, short acting effect, is best reserved for the emergency treatment of ketoacidotic coma (2 units /kg b.wt. for dogs up to 20 kg and above that weight I unit/kg B.wt. half I /V and half I / M repeated every 4 hours) till ketonuria disappear, then depot I.

3- All insulin preparations are available in strength of 40 or 80 Units /ml., except soluble insulin is also available in the strength ,of 20 units / ml.

- Insulin should be kept refrigerated and the bottle well shaken before withdrawing a dose.

4- Disposable insulin syringes, with needles attached, are preferable than glass-barreled syringe which require sterilization before use, with risks of infection and insulin inactivation by heat or spirit. Additionally insulin syringes permit greater accuracy in measuring the doses, because it is graduated on the basis of 20 units per ml (therefore when using 40 units / ml insulin,) the true dose is twice-in units-that shown on the, syringe).

Urine samples after overnight fasting must be tested first before insulin treatment with glucose-keto-urine strips (unaffected by ascorbic acid) to avoid hypoglycaemic coma due to insulin over dose (preceded by signs of drowsiness, weakness and ataxia). This condition can be overcomed by oral 1-2 table spoonful of honey.

d- Dangerous diabetes mellitus: (Diabetic ketoacidosis)

- Treatment similar to the sever D.M., but give crystalline soluble insulin, and additionally fluid therapy must be adopted as follow:

1-Lactate Ringer's' solution, but may give rise to" paradoxical acidosis" of C.O.

2- Sodium bicarbonate 4.2% is used in rate of 2.5 ml / kg b. wt.,

followed after 4 hours by potassium chloride 0.20% and sodium

chloride 0.9% injections to overcome fall in potassium

(hypokalaemia) after correction of acidosis and K+ accompanies

glucose into cells as insulin is given.

E.C.G valuable monitoring indicates the presence of hypokalaemia by depressed T-waves and prolonged QT-interval.

3- Atropine sulphate 0.05 mg / kg b. wt. 4 times daily and limitation of oral fluid to decrease the pancreatic exocrine cases of " diabetic pancreatitis".

b. Clinical case: You are called to examine a dog with temp 40 °C. the dog suffered from anorexia and stiffness in gait. Plan your line of diagnosis and treatment. (15 marks)

The case could be:

- 1- Tonsillitis
- 2- Stomatitis
- 3- Artherittis Most suspected is tonsillitis