جامعة بنـها
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Model Answer

Fourth year students

25-5-2016

First question

a. Explain pathogenesis of toxoplasmosis?

- After ingestion of meat contain infective oocyst or feed contaminated with sporulated oocyst by final host or cats, the parasite pass a cycle as in coccidiosis in small intestine (intestinal life cycle), where bradyzoites enter intestinal epithelium, multiply and differentiate to male and female gametes with formation of unsporulated oocyst which are shedding in feces. Sporulation occurs after several days under suitable conditions from humidity and temperature in environment.
- Intermediate hosts are take sporulated oocyst with contaminated food, since occurs extra-intestinal life cycle where sporozoites penetrate intestinal wall and reach to blood with occurrence of parasitemia and change tachyzoites which locate intracellular, multiply and invade any cells of the body resulting in cell rupture and formation of tissue cyst of 10-50 um in diameter, these are formed in CNS, muscles and visceral organs as lung or liver.
- Placenta and fetus in pregnant animals are invaded by tachyzoites resulting in placentitis, neonatal mortalities and abortion. Prenatal infection may be occurs in pets. The parasite secretes powerful exotoxins resulting in granulomatous lesions.

2. **List diseases of camel causes cutaneous lesion and determine the epidemiology and control for one of bacterial origin**?

Camel pox, Papillomatosis, contagious skin necrosis, dermatophilosis, caseous lymphadenitis, Mange and ring worm

Control and epidemiology of Caseous lymphadenitis

1. Distribution: Worldwide distribution and present in Egypt.

2. Host susceptibility: Mainly sheep, goats, and occasionally horses, cattle, buffaloes, and camels.
3. Mode of transmission:

   Source of infection is the infected animal mainly the pus discharge from ruptured lymph nodes which contaminates the soil, bedding grounds. Infections occur via wounds, abrasions in the skin or even through skin contact insects help in infections.

3. Illustrate the real causes of clinical characteristic of Camel trypanosomiasis

The characteristic symptoms in trypanosomiasis Intermittent fever, anemia, emaciation and edema. Anemia is due to decrease in the life span of the erythrocytes in the circulation and decrease in the activity of the hematopoietic tissues forming the erythrocytes rather than due to destruction of the red blood cells. This comes partly from nutritional disorder and partly from toxins produced by the trypanosomes. The parasite consumes a great amount of the blood sugar producing hypoglycemia and also depletes the glycogen reserves in the body; hence emaciation is marked in spite of the good appetite. While there is decrease in the blood sugar, there is increase in the lactic acid and also an increase the euglobulin fraction.

Trypanosomes multiply in the blood capillaries and attack the cardiac muscles causing damage of the tissues. This accounts for the development of edema. Death is probably due to cachexia and heart failure.

4. Describe the clinical signs of Dourine and determine the control measures

1. Stage of edema:
   - There are recurrent low grade fever, good appetite, inflammation and edematous wellings of external genitalia with vulval edema which extended to udder and medial aspect of thigh and mucous discharges (cloudy red-yellow purulent) comes from genitalia and occasionally vaginal mucosa is

edematous, reddened & contain ulceration (nodules, vesicles and ulcers) with increase sexual desire & frequent urination.

- Enlargement of inguinal LN & abscessation of udder may appears.
- The stallion has penile and preputial swelling with paraphimosis and stallion genitallia becomes covered with vesicle, nodules and occasionally ulcers with selling of testicles and scrotum
- Lesions heals leaving area of depigmentation at genital tract, udder & perineum

2. Stage of urticarial:
    - There are cutaneous eruption or edematous cutaneous plaques or urticarial swellings known as (dollar plaques) which are circumscribed elevated or flat swellings of 2:10 cm in diameter and 1 cm thick contain serous fluid rich in trypanosomes, present on different parts of animal body and it may persist for 2:4 days and suddenly reappear on the other part of the body
    - Cutaneous plaques are considered pathognomonic
3. Stage of paralysis:
    - It is the last stage and begins with muscular paralysis of neck (facial paralysis) & progress to muscles of back & hind limb with incoordination, hyperesthesia, lameness, muscular atrophy, ataxia, stumbling, knuckling and finally complete paralysis with recumbency and death.

Control

Detection of infected stallion before breeding by CFT and positive one is castrated and discard from breeding with prophylactic treatment of all breeding mare at mating with berenil

Second question:

1. **Explain pathogenesis of equine tetanus?**

2. The spores of C. tetani are unable to grow in normal tissue or even in wounds if the tissue remains at the oxidation-reduction potential of the circulating blood. Suitable conditions for multiplication occur when a small amount of soil or a foreign object causes tissue necrosis. The bacteria remain localized in the necrotic tissue at the original site of infection and multiply. As bacterial cells undergo autolysis, the potent neurotoxin is released. The neurotoxin is a zinc-binding protease that cleaves synaptobrevin, a vesicle-associated membrane protein. Usually, toxin is absorbed by the motor nerves in the area and passes up the nerve tract to the spinal cord, where it causes ascending tetanus. The toxin causes spasmodic, tonic contractions of the voluntary muscles by interfering with the release of neurotransmitters from presynaptic nerve endings. If more toxin is released at the site of the infection than the surrounding nerves can take up, the excess is carried off by the lymph to the bloodstream and thus to the CNS, where it causes descending tetanus. Even minor stimulation of the affected animal may trigger the characteristic muscular spasms. The spasms may be so severe as to cause bone fractures. Spasms affecting the larynx, diaphragm, and intercostal muscles lead to respiratory failure. Involvement of the autonomic nervous system results in cardiac arrhythmias, tachycardia, and hypertension.

2. When you suspect foal shigellosis and how can you confirm?

3. The incubation period is short, morbidity rate may be high 25%, mortality rate due to uremic coma, may reach to 100%. Course of the disease is few (24 h-7 days).
4. Foals may be sick at birth or show signs from within few hours of birth up to 3 days of age. There is a sudden onset of fever, prostration, and diarrhea, occasionally dysentery and rapid respiration, and the foal cease to suck.
5. Foals which are sleepy or comatose at birth or soon afterwards occurs commonly. These called sleepers may be aroused but quickly revert to a comatose state.

Death within 24 hours is usual. Occasional foals show severe abdominal pain in the early stage of the disease.

Foals that survive the acute, febrile phase develop arthritis with swollen joints and lameness within 1-2 days. Death usually occurs in these more protracted cases during the period between the second.

Laboratory diagnosis:

Samples: Rectal swabs, swabs from other organs as kidney, joint and cervix of mare, blood and serum.

Laboratory examinations:

- **Direct microscopic examination** of staining smear to detect the bacilli.
- **Isolation of the organism** from suspect material on specific agar as S. S. agar.
- **Serological examination**.

3. Tabulate the clinical difference between epizootic and ulcerative lymphangitis

Epizootic lymphangitis	**Ulcerative lymphangitis**
The first symptom is a painless, freely moveable intradermal nodule, approximately 2 cm in diameter. This nodule enlarges and eventually bursts. In some cases, the lesions may be small and inconspicuous, and heal spontaneously. More often, the skin ulcers grow, with cycles of granulation and partial healing followed by new eruptions. The surrounding skin is edematous at first, and later becomes	The hind legs from the hock downwards are the most common affected site. The affected leg becomes swollen, hot and slightly painful. These signs are usually associated with lameness (when lesions are in close proximity to joints) and development of nodules in the subcutaneous tissues especially around the fetlock. Lesions are of different sizes and may be large 5-7 cm in diameter. These lesions may

thickened, hard and variably painful. The skin over the nodules may be fixed to the underlying tissues. The regional lymph nodes can be enlarged, but fever is uncommon. The infection also spreads along the lymphatics, causing cord–like thickening and further skin involvement. These cycles of exacerbation and partial healing gradually resolve, leaving only a scar. The process usually takes about 2-3 months.	rupture discharging small amount of creamy green pus which may be blood-stained. The ruptured lesions may heal within 2-3 weeks. The horse may be affected for months or even years before development of any severe general disturbances

4. **From pathogenesis of equine strongylosis, how can you conclude the clinical signs**

   Larvae are most pathogenic which result in arteritis and thrombosis resulting in partial or complete ischemia and necrosis or gangrene, thickening of arterial wall, aneurysm, intususception, bleeding due to rupture of intestinal wall nodules and verminous encephalitis. Adult worm result in blood sucking and intestinal ulceration due tissue feeding by the worms. The toxins secreted by the worm may be cause blood hemolysis.

Third question

1. **Describe the clinical signs of rabies in dog and plan your line of control**

A. Prodromal phase:

There are changes in normal behavior, mild fever, salivation, ataxia, digestive disorder as stop eating and drinking, localized pruritus especially at site of bite may result in self mutilation due to bite and scratch, this phase is still for 2-3.d in dogs

and for about one day in cats, this phase may be followed by paralytic or furious phase.

B. Furious or excitative phase (mad dog syndrome) :

- There are hyperexcitment, hyperesthia, friendly dogs become sullen, aggressive, irritable, very restless or troubled viciously, biting or aggressive attack other animals, peoples and any animates object without warning, apprehensive and fearful and facial expression is one of alertness and anxiety with dysphagia or difficulty in swallowing, excessive salivation, unequal dilatation of pupils, increased corneal reflex initially then lost, eyes are congested, unblinking and glazed and fear of natural enemy is lost.
- Infected dogs frequently roam streets and highways or wander aimless and has running fits and going to great distance (40 km), rabid dog have desires to escape from home or to hide, tetanic like erection of ears and tail, change of normal bark or howling to characteristic low-pitched hoarse howling or wolve-like howl with marked sexual stimulation and bitches come in estrus at unusually times.
- Depraved appetite where rabid dog chew the wires and frames of their cages resulting in breaking of teeth or swallow forign materials as soil, stones, metal and grass, mandible sags due to lack of muscles tones, protrusion of tongue, intermittent fine muscular tremors followed by spasmodic contraction (convulsine seizures) and muscular incordination with or without extensive skin anesthesia, ascending paralysis, coma and death due to respiratory failure and rabid dogs rarely live beyond 10. days after onset of signs but about of 0.05 % of infected dogs may be survive clinical disease.

C. Paralytic (dumb) phase **:**

Rabid animal is quiete and not irritable and do not bite untill provoked, progressive ascending motor paralysis of the throat and masster muscles usually with profuse

salivation, inability to swallow and dropping of lower jaw. The paralysis progress rapidly to all parts of the body with coma and death in a few hours.

2. **How can you differentiate on basis of field data between feline panleukopenia and feline infectious peritonitis**

feline panleukopenia	**feline infectious peritonitis**
Ip 2-10 days, morbidity rate up to 50%, and mortality rate up to 80 %in the young fever Cat assume typical "panleukopenia posture" with the sternum and chin resting on the floor, the feet tucked under the body Dehydration Abdominal pain, diarrhea, constipation and vomiting Dilated pupil and retinal degeneration Ataxia from cerebral hypoplasia in kitten infected inutero and crying Late stage, progressive weakness, hypothermia, and death	IP is about 4 months, morbidity rate is high and mortality rate is low Depression and stunted growth Icterus Abdominal and pleural effusion Palpation of abdomen reveal abdominal masses within omentum Ocular findings, uveitis, keratitis Neurological findings

3. **Describe the clinical forms of canine leptospirosis and how can you control the disease?**

IP 1-2 w, morbidity rate is variable and mortality rate is low

Peracute

fever, depression and anorexia

Abdominal and renal pain and reluctance to move

Vascular injury with hematemesis, melena, epistaxis and petechiation

Terminally, hypothermia, shock and death

Subacute and chronic form

Fever, depression, anorexia, abdominal pain and vomition

Paraspinal hyperesthia due to muscular, meningeal and renal inflammation

Peticheal and echymotic hemorrhage

Deterioration in renal function as anuria or oliguria and frequent urination

Uremic breath due to chronic nephritis

Scanty feces with melena

Signs of hepatitis as change of fecal color from brown to gray and icterus

Occasionally, abortion, uveitis and meningitis may be occur

Fourth Question

1. **When you suspect African horse sichness and how can you confirm?**

2. Acute form (pulmonary or dunkop form):

Morbidity rate varies according to the number of insect vectors during the outbreak. Mortality rate is up to 90% after 2-5 days incubation with a short course from few hours to 2 days. This form is the most common form in the acute outbreaks among susceptible horses. Clinically, it is characterized by high fever (40-41°C), cough, conjunctivitis, nasal discharges, ocular discharges, and pulmonary edema which results in respiratory distress, dyspenea, and spasmodic cough. Appetite is good until the affected horse collapses and dies.

3. Subacute form (cardiac or dikkop form):

It is the common form in horses in enzootic areas. It is characterized by long incubation period up to 3 weeks and the course lasts for several days. Clinical manifestations include fever for 4-8 days, non purulent conjunctivitis, and cyanosis

which commonly affect tongue and gums. The critical and characteristic sign is the bilateral bulging of edematous swelling overlying the supraorbital fossa, eyelids, and the surrounding tissues. As the disease progresses, the temperature return to normal and the subcutaneous edema extends to involve the whole head, neck, and brisket. These edematous swellings are doughy, painless, and cold. The tongue becomes swollen and blue in color. Mild colic and restlessness are common. Death occurs within 8 days from the onset of the manifestations in about 50% of cases.

4. Mixed form:

Both (pulmonary and cardiac) forms may develop in the same animal. The infestations of this form include the pulmonary and cardiac signs. It ends fatally.

5. Horse sickness fever or mild form:

It is more common in donkeys. It is characterized by fever for 1-5 days, anorexia, slight conjunctivitis, and dyspenea.

Laboratory examinations:

- Isolation and identification of the virus: The virus can be isolated by intracranial inoculation of the suspected samples into suckling mice. Death of mice usually occurs within 2-7 days post inoculation. Also, inoculation into cell culture can be used such as Vero and MS cells. The cells usually show CPE (cell rounding and detaching) within 2-5 days after inoculation. The virus identification is based on FAT. The differentiation of the type is based on type specific antisera from rabbits and subsequent virus neutralization in baby mice.
- Detection of the viral antibodies: CFT, FT, AGID, and NT are available for detection of the antibodies in the serum, but only NT is type specific.

4. explain pathogenesis of equine infectious anemia and how can you confirm the disease

Following infection, the virus passes to blood streams and then localizes in spleen and liver. It replicates and destructs erythrocytes. This destruction results in

congestion then icterus, edema, and progressive anemia. In the early stage, spleen contains high amount of hemosiderin and in the late stage, liver contains high amount of iron.

1. Proper management and hygiene:

   Any infected cases should be recorded and notification of the authority should be occur. The infected clinically normal animals should be identified by Coggin test and they should be destroyed. The suspected materials such as contaminated food, water, semen, uterine fluid, and milk should be hygienically disposed. The introduction of the infected cases to free areas should be restricted. The swampy areas should be drained and insects should be controlled by fly repellents and by using screened stables. Great care should be taken to avoid the transmission of the disease by the surgical instruments by proper sterilization by boiling or autoclaving.

2. Vaccination:

   Vaccines are available but are not in general use. Inactivated virus vaccines are safe.

3. **when you suspect equine habronemiasis and how can you treat the case?**

- Clinical forms :

A. Gastric habronemiasis :

   - Poor condition and coat with variable appetite which is often depraved. Large tumors result in pyloric obstruction and gastric dilatation or distention and perforation may occurs resulting in the appearance of depression, fever, pain and heat on the left side just behind costal arch. Mild to moderate colic occurs when intestinal stenosis occurs. Marked anemia if spleen is involved.

B. Cutanous habronemiasis or summer sore:

- Commence as pruritis and formation of rapid developing small papules or hard nodules with eroded scab covered centers with little discharge and some irritation. Individual lesions may increase up to 30 cm in diameter within few months and the center is depressed and composed of coarse red granulation tissue which is cover with grayish necrotic membrane and the edges are raised and thickened and the lesions may regress in colder weather and recur in the following summer.
- The lesions are present in parts of the body where skin wounds or excoriation are most likely to occur and where horses can not remove the vectors flies as on face below medial canthes of the eyes, middling of abdomen, prepuce and pens and less commonly on the legs, withers, fetlock or coronary bands.

C. Conjunctival habronemiasis :

- The lesions are present on the the eye lids and may reach to 5 mm in diameter. Conjunctivitis with small yellow necrotic mass about 1 mm diameter under the conjunctiva and may be accompanied by soreness and lacrimation and not respond to bacterial conjunctivitis therapy.

Treatment :

- Gastric form, gastric lavage with 5-10 liters of 2 % sodium bicarbonate to remove excess mucus followed by carbon bisulfide 2-5 ml/ 45 kg, b/w, orally, this result in effective removal of H.muscae and H.majus but H.megastoma residing in tumor are not affected. Trichlorphon may reduce infestation.
- Cutanous form, not respond to standard wound treatments, application of 2-10% formalin or 10% chromic acid locally with trichlorphon (Neguvon) in dose of 25 mg/ kg b/w I/V in 1 liter 5% dextrose, for two doses with one week interval or ivermectine. Surgical excision of skin lesion may be indicated.

Conjunctival form, systemic trichlorphon with local application of antibiotic anti-inflammatory eye ointment

Fifth question:

Strangles

Diagnosis:

A. Field Diagnosis**:** It is based on contagious nature of the disease among young horses and clinical signs of fever, lymphadenopathy of the throat lymph nodes with nasal discharges.

B. Laboratory diagnosis**:**

Samples: Nasal discharge, pus, nasal swabs, blood & paired serum.

Laboratory examinations:

- **Direct microscopic examination of stained smear**, the organism is Gram positive cocci exist in pair shapes.
- **Isolation of organism** on blood agar, and identification of the colony by staining or biochemical reaction.
- **Serological examination** as passive hemagglutination test.
- **Animal inoculation** as mice, pus inoculation will kill it in 24 days with evidence of acute septicemia.

Treatment:

- Isolation of infected animal and treat as soon as possible as following:

1. Medical treatment:

- Specific treatment is pencillin, first dose in a combination of crystalline and procaine pencillin of 200 and 500 IU/kg BW respectively. The second dose is procaine pencillin alone at 24 h intervals for 3-5 d.

- Cases with purpura hemorrhagica should be treated by anti-inflammatory, antihistaminic, calcium therapy, diuretics and blood transfusion.

2. Surgical treatment:
    - Iodine or icthyol ointment locally on enlarged nodes or hot fomentation to ripen the abscess with daily surgical dressing of the abscess after pus evacuation. Tracheotomy if there is dyspnea or compression on pharynx.
3. Hygienic treatment:
    - Rest of infected animal, provide soft palatable diet, clean separate water and food bucket and keep nostrils and muzzles clean with frequent removal of discharges and washing with antiseptic.