University of Mosul
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Summary:

Clostridial Infections

In avian species following diseases are caused by Clostridium bacteria.

1. Ulcerative enteritis

2. Necrotic enteritis

3. Gangrenous Dermatitis

4. Botulism

Clostridia infections are not common in birds and are not a significant source of

infection for man and animals

ULCERATIVE ENTERITIS(UE):

Ulcerative enteritis (UE) is an bacterial infection of young chickens and turkeys characterised by sudden onset and rapidly increasing mortality. The disease was first seen in enzootic proportions in quail, and was therefore called “quail disease”.

It has since been established that many avian species other than quail are susceptible. Therefore, the earliest name has been replaced by ”ulcerative enteritis

A new species of clostridia- C. colinum.

Epizootiology

Ulcerative enteritis is found in most avian species, but quail are most susceptible.

The disease is also reported in toucans and ratites.

Clinical Signs :

Birds dying in the acute stage show no clinical signs. In sub acute phase birds

are depressed with ruffled feathers. Chronic stages show atrophy of muscles

and extreme emaciation. In chickens mortality range from 2 to 10% and recovery

is common. Quail have 100% mortality.

Cross Lesions:

- The most important lesions are found in the intestine, liver and spleen.
- First, there are small, circular to lenticular mucosal ulcers affecting the small intestine, caeca and upper large intestine.
- The ulcers may penetrate as deep as the serosa, which may become perforated and result in peritonitis. The ulcers may coalesce to form large areas with a pseudo membrane.
- Small ulcers have a hemorrhagic border, which may be seen on the serosal and mucosal surfaces.
- A haemorrhagic border is less frequently found in larger lesions. Lesions with raised edges may also be found.
- In the liver, usually there are yellowish to grey necrotic lesions of varying size. The spleen is enlarged and haemorrhagic.
- Quail with acute disease may show haemorrhagic enteritis of the duodenum, with small red spots visible on the serosal surface

Diagnosis

1. Gross lesions in the intestine and liver are suggestive for clinical diagnosis.

2. Confirmation is done by crushing a necrotic piece of liver between two

slides, fixed by heat and stained with Gram stain. Large Gram positive

rods with spores confirms the diagnosis.

3. Complement fixation and FAT are rapid methods of diagnosis.

4. Differential Diagnosis: From the diseases that cause similar lesions;

Coccidiosis, Histomoniasis, Necrotic enteritis (no infectivity to quail),

Hemorrhagic syndrome and Inclusion body hepatitis

Treatment, Prevention and Control

Antibiotics such as streptomycin in drinking water, chloromycetin and bacitracin

in feed have been found useful. Strict biosecurity measures

NECROTIC ENTERITIS

Necrotic enteritis (NE) is found worldwide, and is a disease of increasing importance owing to ever increasing restrictions on the preventive use of in-feed antimicrobials.

The disease is most common in broiler chickens and meat turkeys. Broilers aged 2-5 weeks are most frequently affected. It is also fund regularly found in layers, mostly in pullets and young birds kept on litter.

Epizootiology

- The disease is caused by Clostridium perfringens type A or C in the large intestine and caeca, and subsequent migration to small intestine where it produces toxins.

- Alpha toxin produced by C.perfringens types A and C, and beta toxin produced by type C, are believed responsible for intestinal and mucosal necrosis, the characteristic lesion of the disease.

- Both have been detected in faeces of chickens with NE. Predisposing factors include outbreaks of coccidiosis, especially mild or subclinical, changes in diet, and inadequate cleaning of houses, utensils and equipment.

PATHOGENESIS

- The presence of the organisms in the intestine alone is not sufficient to induce necrotic enteritis.The following two requirements have been proposed for induction of necrotic enteritis.

o The presence of some factor causing damage to the intestinal mucosa, and

o The presence of higher than normal numbers of intestinal C. perfringens organisms.

- Bacterial cells adhere to damaged epithelium and denuded lamina propria where they prolifetae and induce coagulative necrosis.

- Attraction and lysis of heterophils granulocytes as well as further tissue necrosis and bacterial proliferation proceed rapidly.

- The alpha-toxin, a necrotizing toxin produced by all the toxin types. This toxin destroys cell membranes by recognition and hydrolysis of membrane phospholipids. Toxins may also enter the blood stream, causing systemic effects and death.

Clinical Signs

- Acute NE is characterised by increased mortality but few visibly sick birds, indicating that affected birds die rapidly.

- The signs include depression, decreased feed intake, and reluctance to move, ruffled feathers and diarrhoea.

LESIONS

- The characteristic gross lesion of necrotic enteritis is a pseudo membrane attached to the intestinal mucosa, primarily the small intestine.

- The mucosa of the caecal pouches are not changed but the caecal tonsils and adjoining narrow segments of the caeca may occasionally be affected.

- The membrane may be white, yellow, green, brown or red. A yellow or green discolourations is the most commonly found.

- The affected gut segment may be dilated and soft with fluid contents, or the wall appear turgid and rigid with dry and sparse luminal contents.

- A detached pseudo membrane is occasionally found in the gut lumen. The contents of the caecal pouches are often dark and dry.

- Birds dying with NE often show a dark liver with dilated gall bladder, pale kidneys with prominent lobular outlines, and dark and dry pectoral musculature indicating dehydration.

The body condition depends on the course of disease. Birds dying with acute NE are in good bodily condition

Diagnosis

Careful microscopic examination of smears and scrapings of the affected part

of the intestine is essential to differentiate necrotic enteritis from coccidiosis. In

dual infection with coccidiosis (E. brunetti and E. maxima which produce

similar lesions) culture for clostridia may help in diagnosis

Treatment and Prevention

Antibiotics such as streptomycin, penicillin and bacitracin are useful in treatment

and prophylaxis. Biosecurity is helpful in prevention.

GANGRENOUS DERMATITIS

Gangrenous dermatitis occurs worldwide in chickens and turkeys.The disease is characterized by a sudden onset, sharp increase in mortality, and gangrenous necrosis of the skin over the wings, thighs, breast, and head.

It occurs sporadically in chickens 4-16 wk old, affects broiler and layer replacement stocks, and occasionally causes outbreaks in turkeys

Etiology

The exact cause of gangrenous dermatitis is still not well known. At least two organisms are implicated. C. perfringens type A and C. septicum, but in many outbreaks S. aureus and E. coli are also involved. Since clostridia and Staphylococci are the common inhabitants of skin, infection usually follows wounds and injuries that cause initial necrosis, necessary for the growth of clostridia. The staphylococci help to produce anaerobic condition for clostridia growth. The disease occurs in 4-16 weeks old chickens and turkeys. Mortality does not exceed more than 5%.

Clinical Signs and Lesions

- The first sign is usually a sudden dramatic increase in mortality in the affected flock.
- Overall mortality is 10-60%. Affected chickens are extremely depressed, lethargic, and prostrate, and die within 8-24 hr.
- Red to black patches of moist, gangrenous skin are seen over the breast, abdomen, wing tips, or thighs.
- Feather loss or sloughing of the epidermis is common. When clostridial infection occurs, palpation of the affected areas often reveals crepitation due to gas bubbles in the subcutis and musculature.
- At necropsy, there is an accumulation of gaseous, serosanguineous fluid in the subcutis, and the musculature has a pale cooked appearance.
- The liver and spleen are enlarged and may contain infarcts or pale focal areas of necrosis.
- The kidneys are usually swollen, and the lungs may be congested and edematous or necrotic.
- Atrophy of the bursa of Fabricius may be found in birds that were exposed to infectious bursal disease virus in the first few weeks after hatching.

- Histopathologic demonstration of gangrenous necrosis with numerous coccoid bacteria or large, gram-positive rods with or without spores in affected tissues is sufficient to confirm a clinical diagnosis

Treatment and Control

Drinking water treatment with streptomycin, penicillin or bacitracin is useful.

The prevention of injuries and wounds would reduce the incidence of gangrenous

Dermatitis.

BOTULISM(Limber Neck, Western duck sickness)

- Botulism is caused by exotoxin of rapidly proliferating C lostridium botulinum.
- The disease affects poultry worldwide. There are 8 antigenically different toxigenic grouping (A, B, C-alpha, C-beta, D, E, F and G).
- Almost all outbreaks in poultry are caused by type C-alpha. Occasionally, however, types A, B, and E are involved. Botulism toxins are among the most potent toxins known.
- Type C toxin is produced under anaerobic conditions. Type C-alpha cultures produce three toxins: CI, C2, and small amounts of type D toxin.

Epizootiology

The organism itself does not produce disease, but its presence in the intestine

causes its rapid multiplication in a dead and putrefying carcass. The toxin is

produced in such carcass. Botulism occurs by eating toxin-containing carcass.

Maggots and fly larvae feeding on such carcass become toxic. High level of

toxin is also produced in mud, decaying vegetation and anaerobic organic matter

such as feed. Sometimes in broilers C. botulinum produces toxin in the intestine,

which get absorbed in the body, producing outbreak of botulism (toxico-infectious

botulism). Most species of birds are susceptible to botulism, but severe outbreaks

have been among the waterfowls

Clinical Signs and Lesions

Birds become sick within a few hours to 1 or 2 days after ingestion of toxic

material. There is weakness and in the coordination of the legs and wings due

to paralysis of muscles. Mostly noticed in the neck muscles, causing

characteristic “limber neck”, a name sometimes used for the disease.Birds usually die in coma, from respiratory failure. If a small dose of toxin is ingested, recovery takes place after mild signs of in coordination,Death results from cardiac and respiratory failure. There are no gross or microscopic lesions.

Diagnosis

Characteristic paralytic signs in the absence of gross lesions suggest botulism.

Diagnosis can be confirmed by the demonstration of toxin, by injecting serum

or extract of intestinal content from sick bird, intraperitoneally (0.3ml) into mice.

Positive cases develop paralysis in 1-2 days

Treatment

Administration of C. botulinum type C antitoxin controls mortality. Antibiotics with vitamin supplement are useful in toxico infectious botulism.

Prevention

Good management and hygiene normally prevent outbreaks of botulis