

Clostridial Infections

Clostridial infections in poultry have 4 pathological forms:

1. Ulcerative enteritis is caused by *Clostridium colinum*.
2. Necrotic enteritis is caused by *C.perfringens*.
3. Gangrenous dermatitis is caused by *C.perfringens* and *C. septicum*.
4. Botulism is caused by *C. botulinum*.

Toxins produced by the organism are responsible for the pathology diseases. The organisms have no ability to induce the disease by itself unless co-factors exist, such as dietary ingredients or changes, severe stress, other infectious agents, coccidiosis, or immunosuppressive infections such as infectious bursal disease or chicken infectious anemia.

Ulcerative Enteritis (Quail Disease)

Definition:

Ulcerative enteritis (UE) is an acute *C. colinum* infection in young chickens, turkeys, and game birds that characterized by sudden onset and rapidly increasing mortality. The disease is named quail disease as it first seen in quails.

Etiology:

C. colinum is a gram-positive, anaerobic, non-motile, spore-forming rod. The recommended medium for isolating is tryptose-phosphate agar supplemented with 0.2% glucose and 0.5% yeast extract and enriched with 8% horse plasma. Plates are inoculated with material from liver lesions and incubated anaerobically for 1-2 days at 35-42°. The colonies are 1-2 mm in diameter, white, circular, convex, and semi-translucent and have filamentous margins.

Natural and Experimental Hosts:

UE is affecting a wide range of avian species, but natural outbreaks were seen in quails, grouse turkeys and chickens. Ulcerative enteritis is more frequently seen in young birds. It occurs in chickens 4-12 weeks, turkeys 3-8 weeks, and quail 4-12 weeks of age. Outbreaks in chickens often accompany or follow coccidiosis, chicken infectious anemia, infectious bursal disease, or stress conditions.

Transmission

UE is transmitted through droppings; birds become infected by ingesting contaminated feed, water, or litter. The organism produces spores, resulting in permanent contamination of premises after an outbreak has occurred.

Incubation Period

In quail, the acute form of UE results in death within 1-3 days

Clinical Signs

1. Birds are found dead without exhibit any clinical signs.
2. The birds are usually well muscled and have feed in the crop.
3. Quail shows watery, white droppings.
4. The infected birds become listless and humped up.
5. General signs such as partly closed eyes and dull ruffled feathers.
6. Extreme emaciation is seen in birds affected 1 week or longer.
7. Mortality in young quail may be as high as 100%, while it is 2-10% in chickens.

Necropsy:

1. Hemorrhagic enteritis in the duodenum with punctate hemorrhages may be visible through the serosa in the intestinal wall.
2. Ulcerations may be extensive resulting in eroded intestinal wall and then perforating the intestines inducing peritonitis.
3. Ulcers are usually lenticular in shape, that may be coalesced to form large necrotic, diphtheritic patches
4. Liver lesions are seen as yellow mottling to large, irregular yellow areas along the edges.
5. The spleen may be congested, enlarged, and hemorrhagic.

DIAGNOSIS

1. Case history.
2. Clinical signs.
3. Characteristic necropsy lesions.
4. Isolation and identification of organism (enriched tryptose-phosphate agar).
5. Fluorescent antibody (FA) was found to be highly specific for diagnosis of UE.
6. Agar gel immunodiffusion (AGID) test has also been used.

Differential Diagnosis

The diseases must be differentiated from coccidiosis, necrotic enteritis, and histomoniasis. Frequently, coccidiosis in chickens, turkeys, and pheasants

TREATMENT

Chlortetracycline (CTC), Amoycilline and Tylosin are used for control infection. Ulcerative enteritis can be prevented and/or controlled through lincomycine feed additive.

Necrotic Enteritis

Definition:

Necrotic enteritis (NE) in domestic chickens is the disease caused by *C. perfringens* which has been reported from most areas of the world where poultry is produced. Moreover, *C. perfringens* has also been associated with NE in turkeys.

Etiology:

C. perfringens types A or C. Alpha toxin produced by *C. perfringens* types A and C and beta toxin produced by *C. perfringens* type C are responsible for intestinal mucosal necrosis. *C. perfringens* can be isolated readily on blood agar plates incubated anaerobically at 37°C overnight. *C. perfringens* colonies on blood agar (with rabbit, human, or sheep blood) are surrounded by an inner zone of complete hemolysis and an outer zone of discoloration. The organisms are short to intermediate, gram-positive rods without spores. Most strains ferment glucose, maltose, lactose, and sucrose; do not ferment mannitol; and variably ferment salicin. Principal products of fermentation are acetic and butyric acids. Gelatin is hydrolyzed; milk is digested; and no indole production occurs. Growth on egg yolk agar demonstrates the presence of lecithinase and the absence of lipase production. Subculturing on egg yolk agar plates, one-half of which have been spread with *C. perfringens* antitoxin, and incubating anaerobically overnight will produce a zone of precipitation around colonies on control sides of the plate and little or no precipitation on sides spread with antitoxin.

Clinical Signs:

1. Marked to severe depression, decreased appetite, reluctance to move, diarrhea, and ruffled feathers.
2. Clinical illness is very short followed by death.

Necropsy

1. The lesions are usually confined to the small intestine, primarily jejunum and ileum.
2. Intestines are friable and distended with gas.
3. The mucosa is lined by a loosely to tightly adherent yellow or green pseudomembrane.
4. Flecks of blood may be seen
5. Hepatitis, characterized by swollen, tan colored livers with necrotic foci.

Diagnosis:

1. Case history.
2. Clinical signs.
3. Characteristic necropsy lesions.
4. Isolation and identification of organism.
5. Fluorescent antibody (FA).
6. Agar gel immunodiffusion (AGID).

Differential Diagnosis

The diseases must be differentiated from ulcerative enteritis (UE) and *Eimeria brunetti* infection. Ulcerative enteritis is caused by *C. colinum*; characteristic gross lesions are multiple areas of necrosis and ulceration in the distal small intestine and ceca and areas of necrosis in the liver while lesions of NE usually are confined to jejunum and ileum with little or no involvement of ceca or liver. *E. brunetti* infection causes gross lesions similar to those produced by *C. perfringens*; however, microscopic examination of fecal smears, impressions, or intestinal sections should demonstrate the presence or absence of coccidia. Finally, NE and coccidiosis often occur simultaneously in a flock.

Treatment and Prevention:

Amoxicilline, tylosin, lincomycin and oxytetracycline have been shown to be effective in preventing and controlling NE. Probiotics such as *Lactobacillus acidophilus* and *Streptococcus faecium* reduce the severity of NE. Control of coccidiosis should be applied.

BOTULISM

Definition:

Botulism is an intoxication caused by exotoxin of *Clostridium botulinum*. Synonyms are “limberneck” and “Western duck sickness”. Free-ranging and confinement-reared poultry and feral birds can be affected. Most avian cases are caused by *C. botulinum* type C.

Etiology

C. botulinum is a gram-positive, spore-forming bacterium capable of elaborating potent exotoxins under appropriate environmental conditions. The species consists of a diverse group of anaerobic bacteria including 4 cultural (I—IV) and 8 antigenically different toxigenic groupings (A, B, C alpha, C beta, D, E, F, and G). Cases of botulism in chickens, ducks, pheasants, and turkeys in natural or commercial settings have been caused primarily by the type C toxigenic group.

Incubation Period

With high levels of toxin, disease appears within hours. With low toxin doses, onset of paralysis occurs within 1—2 days.

Transmission

C. botulinum type C is distributed worldwide wherever large populations of wild and domestic birds are found. Type C organisms readily grow in the gastrointestinal tract of birds and are considered obligate parasites. Presence of organisms in the gastrointestinal tract of wild and domestic birds and resistance of spores to inactivation, favor spread of this organism.

Clinical Signs

1. Clinical signs of botulism in chickens, turkeys, pheasants, and ducks are similar.
2. In chickens, flaccid paralysis of legs, wings, neck, and eyelids are predominant features of the disease.
3. Paralytic signs progress cranially from the legs to include wings, neck, and eyelids.
4. Affected birds are found sitting and are reluctant to move.
5. Wings droop when paralyzed.
6. Limberneck, the original and common name for botulism, precisely describes the paralysis of the neck.
7. Because of eyelid paralysis, birds appear comatose and may seem dead.
8. Gasping has been observed.
9. Death results from cardiac and respiratory failure.

Diagnosis

The differential diagnosis of botulism is based on clinical signs and lack of gross or microscopic lesions. Definitive diagnosis requires detection of toxin in serum, crop, or gastrointestinal washings from morbid birds.

The mouse bioassay is a sensitive and reliable method for confirming heat-labile toxin in serum. Groups of mice are inoculated with suspect serum samples. Other mice receive samples treated with type-specific antiserum. If toxin is present in the sample, signs and death of mice given

untreated samples usually occur within 48 hours. Mice inoculated with specific antitoxin will be protected.

An antigen-capture ELISA assay for *C. botulinum* type C toxin was able to detect 0.25ng/ml toxin compared to 0.12ng/ml detection using the mouse bioassay.

The mild form of the disease must be differentiated from Marek's disease, drug and chemical toxicity.

Botulism in waterfowl must be differentiated from fowl cholera and chemical toxicity. Lead poisoning of water birds commonly is confused with botulism.

Isolation of *C. botulinum* requires anaerobic culturing of samples inoculated into cooked-meat medium and incubated anaerobically at 30°C. After 3—5 days' incubation, toxin can be detected using the mouse bioassay with specific typing antitoxins.

Treatment

It is difficult. Treatment of affected broiler flocks with sodium selenite and vitamins A, D3, and E reduced mortality. Antibiotics including streptomycin (1g/L in water), or periodic chlortetracycline treatments also reduced mortality. Inoculation with specific antitoxin neutralizes only free and extracellularly bound toxin and might be considered for treating valuable birds in zoologic collections which considered impractical in commercial poultry, duck, or pheasant outbreaks.

GANGRENOUS DERMATITIS

Definition

It is a severe necrosis of muscle and subcutaneous of furtherless skin areas of breast, wings, legs and abdomen in chickens due to infection with *C. perfringens* and *C. septicum*. It is also observed in turkey breeder hens from wound infections occurring during mating. Gangrenous dermatitis also has been given a variety of names including necrotic dermatitis, gangrenous cellulitis, gangrenous ermatomyositis, avian malignant edema, gas edema disease, wing rot, and blue wing disease (as a complication with CIA).

Etiology

Causes of GD are *C. septicum*, *C. perfringens* type A and *Staphylococcus aureus* either singly or in combination. *C. septicum* should be cultured anaerobically on blood agar plates containing 2.5% agar and incubated for 1-2 days at 37°C. *C. septicum* ferments glucose, maltose, lactose, and salicin but not sucrose or mannitol. Principal products of fermentation are acetic and butyric acids. Gelatin is hydrolyzed. Milk is not digested, and indole is not produced. Growth on egg yolk agar demonstrates an absence of lecithinase and lipase production. Spores are oval and subterminal in location.

Clinical Signs

1. Birds show different degrees of depression, incoordination, inappetence, leg weakness, and ataxia.
2. The course of illness is short (less than 24 hrs) and the birds are found dead.
3. Mortality rate 1-60%.

Necropsy

1. Presence of dark, moist areas of skin, usually devoid of feathers, overlying wings, breast, abdomen, or legs.
2. Extensive blood-tinged edema, with or without gas (emphysema), is present beneath affected skin.
3. Underlying musculature is discolored gray or tan and may contain edema and gas between muscle bundles.
4. Discrete white foci (necrosis) in the liver.
5. In turkeys, tail cellulitis (bubbly tail), edema and vesicle-like lesions were present laterally and ventrally around the tail.

Diagnosis

1. Detection of the typical gross lesions.
2. Isolation and identification of the causative agent.
3. In field cases of GD, because occurrence of GD due to other infectious agents affecting immune systems of the bird, diagnosis of the underlying etiology is necessary such as IBD virus, avian adenoviruses, CAV and reoviruses must be determined.

Differential Diagnosis

GD must be differentiated from a variety of skin conditions. Dermatitis caused by mycotic agents (*Candida albicans* and *Aspergillus fumigatus*) can be differentiated from GD by demonstrating fungal elements in impression smears or tissue sections, and by isolating and identifying the agent.

Ulcerative dermatitis (“breast burn”) of broiler chickens and plantar pododermatitis of turkeys are conditions characterized by erosions and ulcers accompanied by acute inflammatory changes over the breast, hock, and plantar surface of the feet.

A strong correlation between wet or poor litter and these conditions is present

Treatment:

1. Outbreaks of GD are treated effectively by chlortetracycline, oxytetracycline, erythromycin and amoxicilline.
2. Other line of treatment is the usage of copper sulfate in the water and chlortetracycline or furoxone in the feed.
3. However, in many instances,

Prevention:

1. Thorough cleaning and disinfection of the house and floor have helped resolve farms with historical problems.
2. Preventive measures are applied to control immune-suppressive diseases.
3. The use of water acidifiers and acidification of litter.
4. Management procedures to improve litter condition, reduce moisture and bacterial levels in the environment, and minimize trauma are useful adjuncts to treatment.