Avian Clostridial infections

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Clostridial Infections

- The genus *Clostridium* includes the Gram-positive, spore-forming, catalase-negative, anaerobic bacilli.
- Many of them have been recovered from environmental and clinical specimens of wild and domestic avian species.
- Pathogenicity of clostridia is mediated through potent endotoxins.
- Clostridial diseases may occur as soft tissue infections, intoxications and toxico-infections.
- The economically significant avian clostridial diseases were necrotic enteritis, ulcerative enteritis, botulism and gangrenous dermatitis.

Necrotic Enteritis

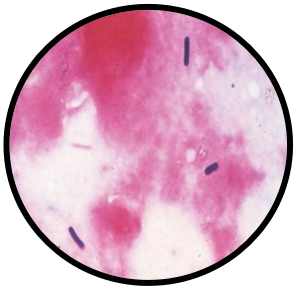
- It is a sporadic acute bacterial disease primarily of chickens and turkeys, although other avian species can be affected.
- Sudden death, friable distended intestines and severe necrosis of the intestinal mucosa characterize the disease.

Occurrence:

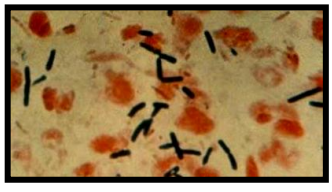
- Chickens 2-10 weeks of age raised on litter.
- Turkeys that are 7-12 weeks of age. Both species usually have some predisposing enteric conditions.
- It has been reported in wild and domestic ducks and geese.

Etiology

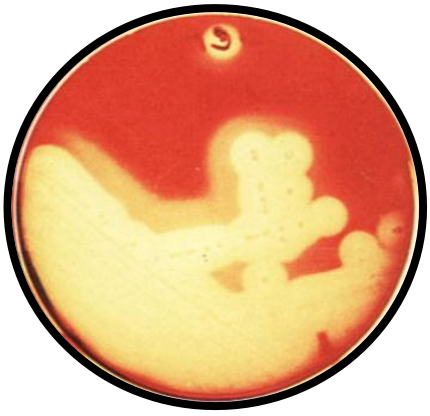
- *Clostridium perfringens* (type A or C) and their toxins are the cause of necrotic enteritis.
- It is anaerobic, Gram-positive rods and produce double-zoned hemolysis on blood agar plates (inner beta-hemolytic zone and outer zone of alpha-hemolysis).
- The presence of lecithinase and absence of lipase can be observed on egg yolk agar.
- Alpha toxin is produced by *C. perfringens* type A and C and beta toxin is produced by *C. perfringens* type C and is responsible for the mucosal necrosis.
- *C. perfringens* are normal inhabitants of the intestinal tract.



Gram positive Clostridium in tissue smear



Gram positive Clostridium, spore forming bacilli and filament shape



inner beta-hemolytic zone and outer zone of alpha-hemolysis

McClung Toabe egg yolk agar (lecithinase and lipase production)

Predisposing conditions

- Intestinal mucosal damage is necessary for the clostridia to proliferate and produce sufficient toxin.
- Coccidiosis, ascaridia migration, hemorrhagic enteritis in turkeys, and severe *Salmonella* infection are predisposing conditions for mucosal damage.
- Severe immunosuppression from infectious bursal disease or turkey hemorrhagic enteritis frequently precedes necrotic enteritis.
- Sudden changes in feed formulation such as addition of high levels of high energy-high protein diet as fishmeal; barley, oat or wheat may results in disturbed intestinal flora.
- Acute terminal complication of primary intestinal diseases.

Clinical signs

- The acute onset of depressed, ruffled birds occurs.
- Depression, in appetence, reluctance to move and diarrhea.
- These birds rapidly progress to death with severe dehydration.
- There is rapid increased mortality. Mortality rates up to 15% have been reported in chickens.
- Excessive water consumption, water-filled crops.
- Foul-smell, brown, or black or bloody-stained feces.

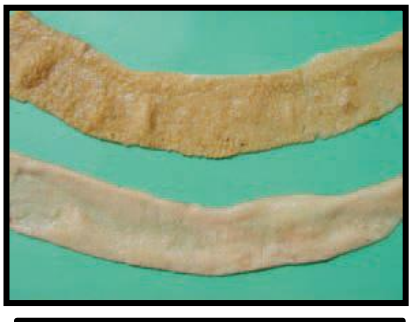
"Pasting" of the vent of a bird with diarrhea from necrotic enteritis. Often chickens may die before any diarrhea is observed.

Gross lesions

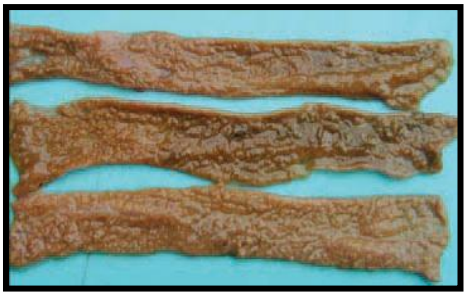
- Lesions are usually found in the mid-small intestines, which are distended and friable.
- Intestinal contents consist of foul-smelling brown fluid and a brownish diphtheritic membrane covers the mucosa.
- Severe dehydration with darkening of the breast muscle and swelling and congestion of the liver may be present.

NE intestinal Lumen Is Filled With Brownish Watery Content, Mixed With Gas Bubbles

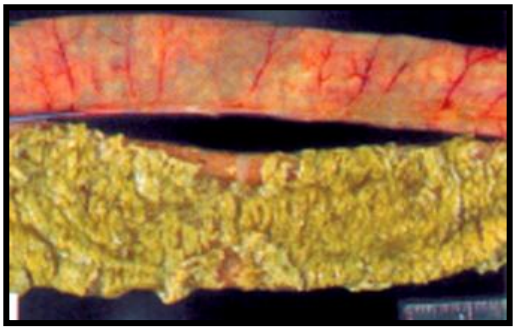
NE Multiple Petechial Haemorrhages Could Be Perceived Through The Wall In Different Areas Along The Small Intestine

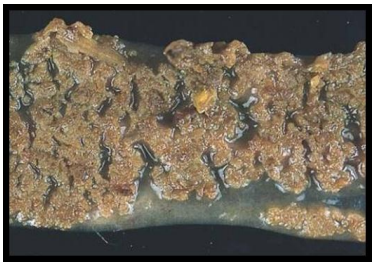


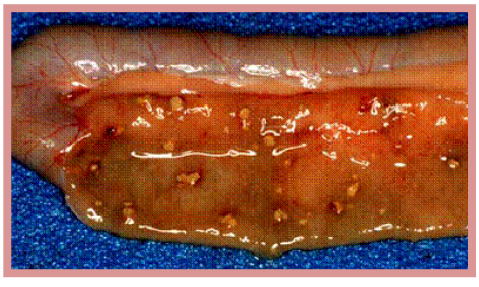
Necrotic Mucosa Acquires A Greyish-Creamy Or Greenish Appearance. Sometimes The Mucosa Has A Flannelette Blanket-Like Appearance



NE The Mucosa Has A Linear Pattern Similar To The Bark Of A Tree

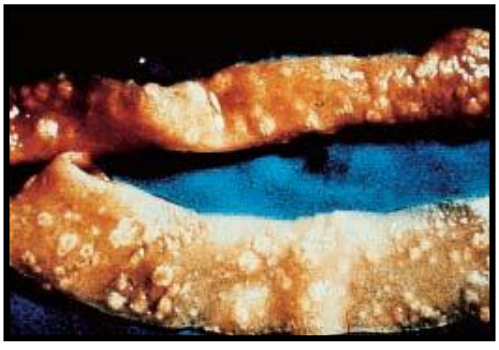


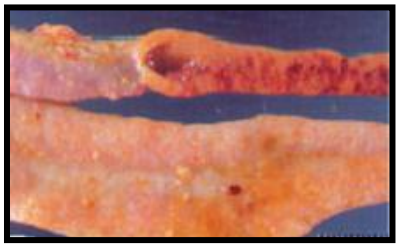


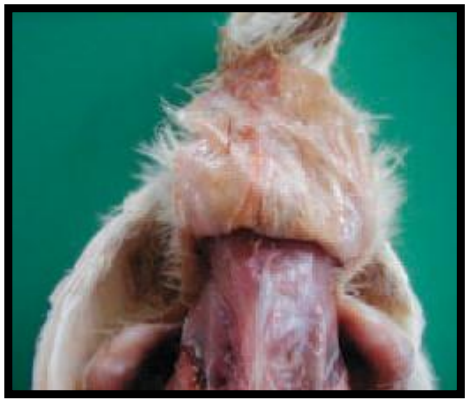


Opened intestine from a broiler chicken with necrotic enteritis. The intestinal wall is thin, the lumen contains blood-tinged fluid and there are white areas of ulceration.

Opened intestine from a broiler chicken with severe necrotic enteritis. The surface of this intestine is covered with a thick "membrane" of dead tissue giving it a "turkish-towel" appearance.







Severe dehydration of skin underlining

NE Marked Congestion Of Liver, Responsible For Its Dark Red To Black Appearance, Is Present

(A) Mild to moderate necrotic enteritis in a 5-week-old broiler chicken with concurrent coccidiosis. Note the hyperemia and diffuse necrosis of the mucosa with multifocal ulcerations.
(D) Large, Gram-positive rods will attach to the villi tips early in the infectious process and are later associated with areas of necrosis.

Diagnosis

- Intestinal mucosal appearance and typical history of acute and severe increase in mortality is strongly suggestive of necrotic enteritis.
- Histopathologically, there is heavy clostridial colonization of the villous epithelium.
- Identification of a predisposing factor is necessary for successful treatment.

Isolation and culture media:

- *C. perfringenes* can be isolated from intestinal contents and mucosal scrapings. Specimens should be fresh and processed as soon as possible.
- Anaerobic blood agar plates can be used for isolation.
- McClung-Toabe egg yolk agar should be used to detect lecithinase and lipase production by isolates.
- For determination of toxin types, isolates should be cultured in cocked meat medium containing 1% glucose.

Mice inoculation (mouse protection test)

- Culture should be centrifuged and supernatants tested for toxins.
- To 1.2 ml aliquots of supernatants, 0.3 ml of *C. perfringens* types A and C antitoxins are added, followed by neutralization at room temperature for 30 minutes. Injection of 0.5 ml intravenously into pairs of mice.
- A third group of mice receives supernatant mixed with normal horse serum as control.
- Mice are observed for 24 hours.
- If the isolate is type A, mice inoculated with supernatants neutralized by type A antitoxins will survive.
- If the isolate is type C, only mice receiving supernatants neutralized by type C antitoxin will survive.

Control

- Good management practices of cleaning and disinfections of poultry houses prior to bird replacement are essential.
- All predisposing factors must be controlled.
- Administration of appropriate feed medication such as growth promoters and prophylaxis feed additives may be warranted.

Growth promoters:

- The purpose of growth promotion has Been curtailed. NE has been treated with
  - Lincomycin, bacitracin, and tylosin **in water.**
  - Bacitracin, lincomycin, virginiamycin, and avoparcin, **in feed**.
- As The overall use of **Antimicrobials** for enteric health **has decreased**, the evaluation of use of other products (**prebiotics, probiotics, phytogenic compounds, etc**.) **has increased in an attempt to mitigate the increasing risk of NE**.

Digestive Enhancer

- Feed additive, antibacterial represent 44.7% of total drugs used via feed.
- Non or weak absorption, without residual effect.
- Increase absorption of feed by reduce wall thickening.
- Affect mainly Gram-positive organisms.

Prebiotics

- The use of prebiotics such as yeast wall extracts, which stimulate growth of beneficial intestinal flora, have produced inconsistent results. Others, however, have shown protective effects with the use of these products

Probiotics

- Probiotics have been shown to lessen the impact of NE in laboratory challenge.
- Competitive exclusion products have been shown to decrease the incidence and severity of NE in experimental challenge.
- Bacillus subtilis spores competitively exclude C. perfringens from broiler chicks and Bacillus-colonized chicks have increased bodyweight, feed efficiency, and intestinal integrity compared with controls.
- Other direct-fed microbials, including Lactobacillus fermentum, L. acidophilus, Enterococcus faecium and B. licheniformis have been shown to produce similar effects in NE- challenge models.

Phytogenic Compounds

- Phytogenic compounds have been evaluated for their efficacy in reducing the incidence and severity of NE.
- Anise oil, citral, and essential oil blends have all been shown to be effective in providing protection from C. perfringens challenge.

Treatment

- Determination of predisposing condition will dictate specific medication.
- Water-soluble penicillin-derivatives or semisynthetic penicillin (Ampicillin and Amoxicillin) as a flash medication for 5-7 days.
- Bacitracin, Rifamycin, Tetracyclines and lincomycin can be used.

Ulcerative Enteritis
Quail Disease

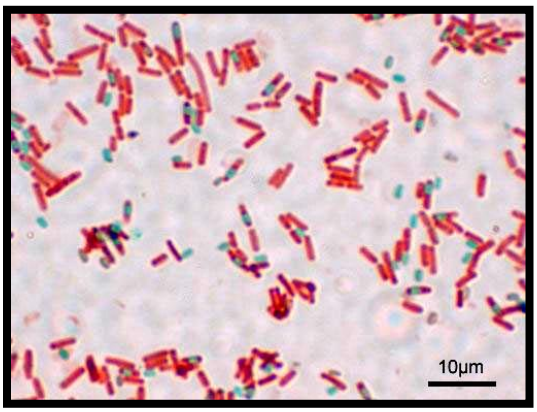
- Ulcerative enteritis, is an acute bacterial infection originally referred to as quail disease, is caused by *C. colinum*.
- Turkey poults and young chickens are frequently infected.
- It is characterized by ulceration of intestinal tract and focal or diffuse hepatic necrosis.

Occurrence

- The disease is highly contagious especially among quail and occurs frequently in adult quail.
- Young birds are affected more frequently than adult.
- It is recorded to infect pigeons, pheasants and partridges.
- It occurs frequently in association with other diseases, including coccidiosis, chicken infectious anemia, *Mycoplasama gallisepticum* infection, and internal or external parasitism.

Etiology

- *Clostridium colinum*, is a Gram-positive, anaerobic, spore-forming bacillus.
- The organism is very resistance. It withstand boiling for 3 minutes or 70C for 10 minutes, hence boiling suspected material is useful in killing other contaminating bacteria during isolation trials.
- The preferred medium is tryptose-phosphate-glucose broth or agar with 8% horse plasma. Cultures are incubated an-aerobically.
- *C. colinum* also grows in the yolk sac of 5-7-day-old inoculated chicken embryos.



Endospores stained by Malachite green (endospore staining)

Gram positive bacilli

Spread and transmission

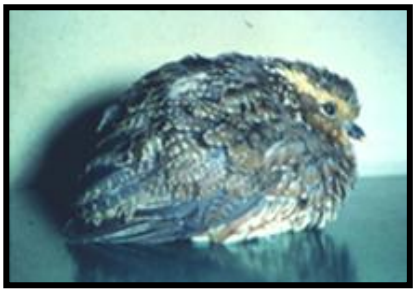
- *C. colinum* is spread primarily through droppings of acutely affected or recovered carrier birds and persists in the soil for many months.
- Interspecies transmission can occur among susceptible birds.
- Infection can spread by flies that feed on infectious droppings.
- The surviving birds become chronic carriers.

Clinical signs

- In most species signs are similar, include listlessness, humped appearance, retracted neck, drooping wings, partially closed eyes, ruffled feathers, diarrhea, anemia, and perhaps bloody feces.
- In quail, white watery droppings are distinctive.
- In chicken flocks, a course of 2 or 3 weeks is common and then the chickens recover slowly.
- Sudden death may occur without signs being apparent, especially during onset. Birds that die suddenly may be well muscled and fat, especially in quail.
- Mortality may be very high with quail, up to 100% within a few days.
- Mortality in chickens seldom exceeds 10%.

Lesions

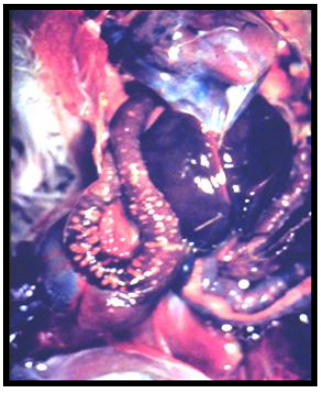
- There are deep ulcers scattered throughout the intestine, including ceca, and the ulcers may be numerous enough to coalesce.
- Deep ulcers often detected through the serosa of the unopened intestine and may penetrate it to induce peritonitis.
- The intestine may contain blood similar to Coccidiosis.
- Acute cases have severe enteritis of the small intestine.
- Emaciation with atrophy of breast muscles may be seen in birds with prolonged course of the disease.
- The liver is usually contains large yellow or focal yellow lesions or both. The lesions tend to be colorful and distinctive.
- The spleen is often enlarged and may be hemorrhagic.



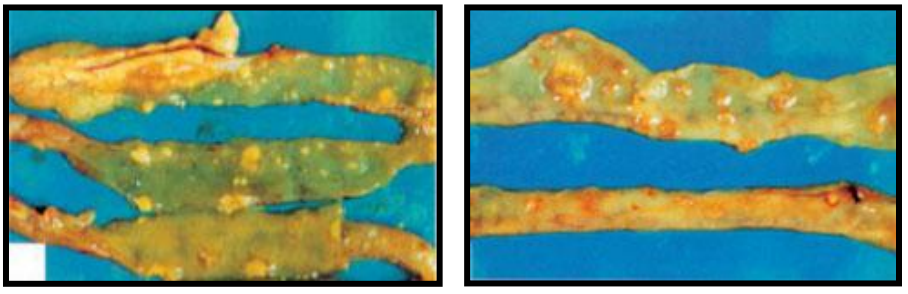
Quail showing humped appearance

bloody feces

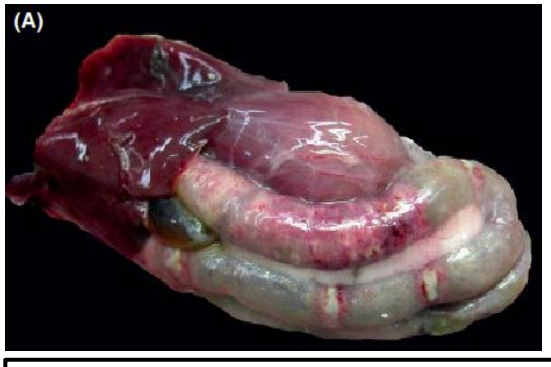
The intestine may contain blood similar to coccidiosis



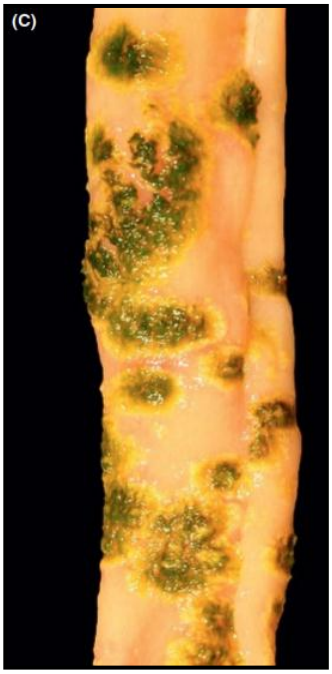
Multifocal ulcerative enteritis (*Clostridium colinum in quail)*

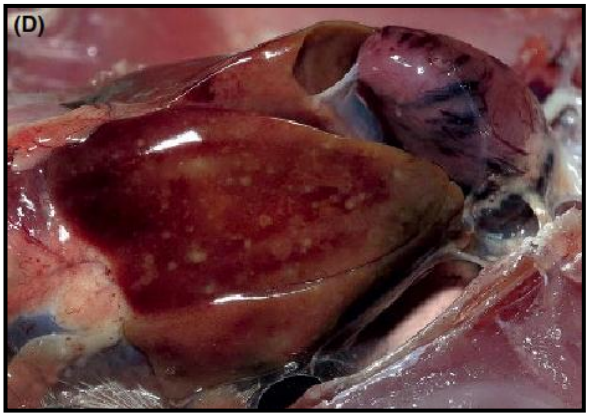


Severe ulceration of intestinal mucosa (ulcers may be numerous enough to coalesce).

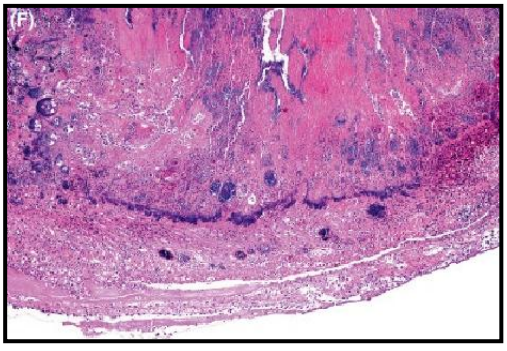


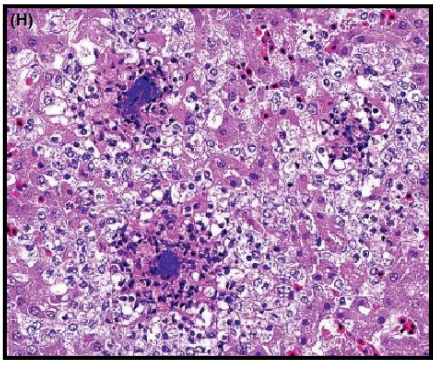
Ulcerative enteritis lesions in quail.
(A) Ulcers visible from serosal surface in small intestine. Note hyperemia and hemorrhage around ulcers. A few pin point lesions are also visible on the surface of the liver.
(B) Acute ulcers on the mucosal surface of the small intestine; some of them hemorrhagic and/or surrounded by a hemorrhagic rim.





(C) Chronic ulcers seen on the mucosal surface of the small intestine; notice diphtheritic membranes covering the ulcers.
(D) Multifocal areas of necrosis in the liver.





(F) Mucosal and submucosal ulcers showing numerous bacterial colonies typical of Clostridium colinum.
(H) Microscopic appearance of liver lesion. Note the multifocal areas of necrosis without distinct separation from normal tissue, minimal inflammatory response and bacterial colonies.

Diagnosis

- **Clinical signs and lesions**: Typical intestinal ulceration and colored liver lesions are strongly suggestive.
- **Stained impression smears** made from liver cut surface may reveal the rod-shaped bacillus with its subterminal spore.
- **Isolation and identification**.
- **Serological identification:**
  - Fluorescent antibody test.
  - GDPT has been used to detect soluble *C. colinum* antigens in intestinal contents, but cross-reactions with *C. perfringens* types A and C are a problem.
  - Complement fixation test has been used to detect carrier birds.

Differential diagnosis

- *C. colinum* must be differentiated carefully from *C. difficile* and *C. perfringens*.
- Coccidiosis often is present in the same bird and assessing the relative importance of the two diseases may be difficult.
- Ulcerative enteritis should be differentiated from histomoniasis.

Botulism
Limber neck
Western duck sickness

- It is a sporadic and highly fatal intoxication, occurs worldwide in free-ranging birds, domestic and wild avian species, caused by toxins of *Cl. botulinum*.

Occurrence

- In birds, botulism occurs frequently in chickens, captive pheasants and wild ducks.
- Except for vultures, most birds are susceptible.
- Most outbreaks in birds occur in semmi-mature or mature chicken flocks.
- Botulism seldom occurs in well-managed commercially raised poultry.

Etiology:

- It is caused by intoxication with one of 8 neurotoxins produced by the anaerobic bacillus *Clostridium botulinum* (A, B, C alpha, C beta, D, E, F, and G).
- Most of avian outbreaks have been caused by toxin type C.
- Although rare, botulism of type A and E has occurred in small chicken flocks fed human feed waste.
- *C. botulinum* toxin is more potent than cobra venom when inoculated s/c in guinea pigs.
- Source of performed toxin include feed, carcasses, maggots or decaying plant material.
- In some outbreaks the **toxin production can occur *in vivo* (toxico-infectious botulism).**

Clinical signs

- Mortality in chickens and turkeys may reach 40%, and has been reached 100% on pheasant farms.
- Including a characteristic flaccid paralysis that beginning the legs and progresses cranially to wings, neck and eyelids.
- In mild intoxications, only leg paralysis may be observed, and if forced to move, birds appear lame.
- Birds may sit with necks and wings outstretched and legs tucked underneath, unable to rise.

- The eyelids may droop or be closed completely and birds may appear comatose.
- Diarrhea with increased urates may be seen in broilers.
- Recovering birds usually regain muscle control in the eyelids first, then in neck and wings, and finally in legs over a 3-5-day period if given ready access to feed and water.
- Fine tremors of muscles and feathers occur in some birds.
- In many chickens feathers tend to be loose and easily pulled from the neck area.


Botulism in chickens showing paralysis of wing and lower eyelid, difficulty breathing caused by partial paralysis of respiratory muscles, and ruffled hackle feathers.


Lesions

- Most diseased birds are free of gross lesions.
- Mild enteritis is very rare in birds that lived for sometimes.
- The upper part of GIT especially crop may contained putrid food or maggots but is usually empty.

Diagnosis

- Clinical signs are highly suggestive.
- It depends on demonstration of neurotoxin in serum, liver, spleen, crop and GIT.
- Specimens should be collected from live birds showing clinical signs to avoid postmortem toxin production from *C. botulinum* in intestine.
- Isolation of *C. botulinum* from tissues of affected birds is not definitive evidence for diagnosis of botulism, because the organism has been isolated from healthy birds.
- Cold gelatin diluent containing PBS and 0.2% gelatin may be used to extract specimens for toxin assays.

- Chopped meat-glucose-starch (CMGS) medium may be used as an enrichment medium.
- Serologic detection in the host: It is of little value because the intoxication dose of neurotoxin is less than the dose required to produce detectable level of antitoxin or antibodies. ELISA and PCR for toxin detection have achieved some success.
- Mouse bioassay for toxin in the serum of affected birds is confirmatory.
- **Differential diagnosis:** Clinically the disease should be differentiated from MD, ND, AE, and chemical intoxication. Also Fowl cholera in waterfowl.

Control

- The disease can be avoided by preventing access of poultry to any source of toxin.
- Sick and dead birds should be picked up regularly because they are a common source of toxin.
- Type C toxoid can be used to immunize birds.
- Avoid raising ducks on shallow water.
- The prophylactic use of selenium and antibiotics has been effective on broiler farms where botulism is enzootic.

Treatment

- Antitoxin can be given to valuable affected birds, either monovalent (type C) or polyvalent (types A&C).
- It is important that the treated birds have access to fresh, clean, nonalkaline water.
- Treatment of flocks with sodium selenite and vitamins A, D and E has been reported to reduce mortality.
- Treatment with bacitracin, streptomycin, chlortetracycline and penicillin has also been reported to be efficacious.

Gangrenous Dermatitis
Necrotic Dermatitis
Gangrenous Cellulitis
Avian Malignant Edema
Wing Rot

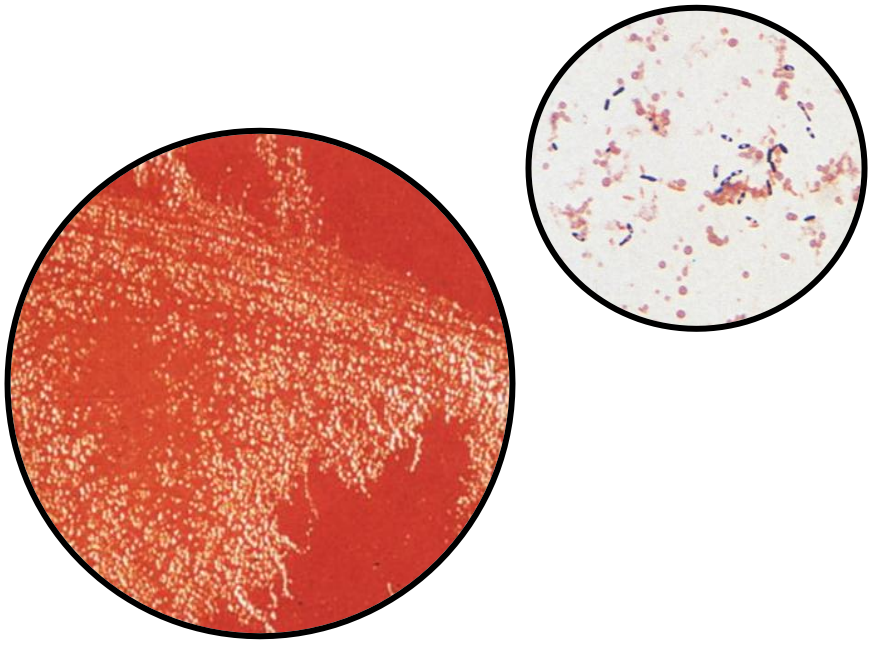
- It is a disease of young growing chickens and turkeys (up to 20 weeks of age) characterized by necrotic areas of the skin and by severe underlying infectious cellulites.

Occurrence

- Most outbreaks have occurred in chickens 4-16 weeks old.
- Young birds may be poorly feathered.
- Outbreaks often occur in excessively warm, humid houses.
- Affected flocks may be immunologically deficient.

Etiology

- Primary skin lesion, secondarily invaded by various bacteria including *Clostridium sp.* (especially *C. septicum*), *Staph. Sp.*, *Strept. sp.* and *E. coli*.
- On blood agar plates incubated anaerobically, colonies are circular, slightly raised with irregular margins, translucent, gray and beta-hemolytic.



Mode of infection and Predisposing factors

- Cutaneous wounds probably occur initially as a result of cannibalism, and mechanical trauma. Bacteria invade the traumatized skin and underlying tissue and their toxins or metabolites cause cellulites. Septicemia and toxemia follow, leading to death.
- Immunosuppression secondary to IBD or CIA increases the susceptibility of affected flock.
- Other factors may enhance susceptibility to infection such as aflatoxicosis, nutritional insufficiency or poor sanitation.

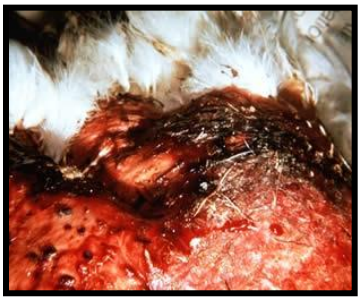
Clinical signs

- A sudden, sharp increase in mortality is often the first indication of onset. Mortality varies but can be quite high.
- Sick birds are depressed, and sometimes prostrate or lame.
- Skin lesions, often crepitant, are apparent in live and dead birds.
- The course of the illness is often less than 24 hours

Lesions

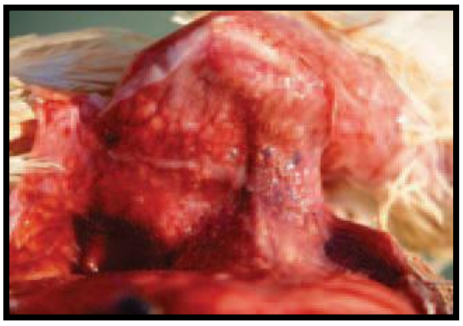
- There are scattered patches of darkened, gangrenous skin.
- Cutaneous sloughing or feather loss in affected areas.
- Marked emphysematous or serosanguineous cellulites underlies some skin lesions.
- Swelling and infarction may be apparent in parenchymatous organs. There may be foci of necrosis in the liver.
- Severe atrophy of the bursa of fabricius is usually present.

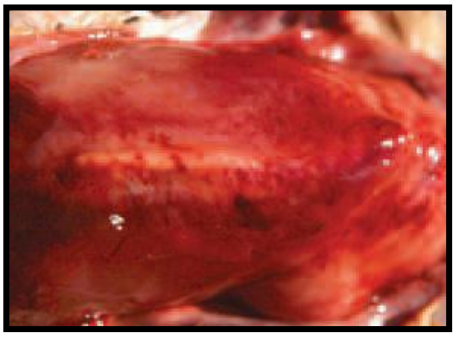


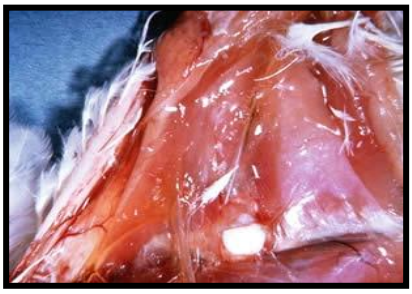
Scattered patches of darkened, gangrenous skin







Scattered patches of darkened, gangrenous skin




Skin lesions, often crepitant, are apparent in live and dead birds

Characteristic subcutaneous congestion and emphysema associated with gangrenous dermatitis in a chicken.

Swelling and infarction may be apparent in parenchymatous organs. There may be foci of necrosis in the liver

Diagnosis

- History, clinical picture and P.M. lesions give a tentative diagnosis.
- Smear or histological sections of affected tissues will reveal bacteria.
- Isolation of bacterial agent from affected tissues.

Control

- Avoid causes of trauma, and cannibalism.
- Application of vaccination programs for IBD and CIA to avoid immunosupression.
- Eliminate all stresses of the birds (parasitism, malnutrition, coccidiosis, etc.).
- Cleaning and disinfections of the houses may be helpful.
- Cheap grade salt is used on the soil at a rate of 60-63 lb/1000 f^2.
- Administration of appropriate feed medication such as growth promoters and prophylaxis feed additives may be warranted.

Treatment

- Determination of predisposing condition will dictate specific medication.
- Amoxacillin as a flash medication (20 mg/kg/b.w) for 3-4 days is effective treatment.
- Penicillin, tetracyclines, or other fast acting antibiotics such as flouroquinolones.
- Water treatment with copper sulfate or drinking water acidification with citric or propionic acid have been used to reduce, but not eliminate, GD-associated mortality in flocks.