



Tikrit University
College of Veterinary Medicine

Ulcerative Enteritis

Subject name: Poultry Diseases

Subject year: 2024-2025

Lecturer name: Ismael I. Hasan

Academic

Email: ismailhasan@tu.edu.iq



Lecturers link



Ulcerative Enteritis



Summary

Ulcerative enteritis (UE) is a disease caused by *Clostridium colinum*, a Gram-positive, sporulated, anaerobic rod. The disease is most commonly recognized in young quail, although cases also occur in chickens, turkeys, and several other avian species. It is transmitted by the fecal–oral route and produces acute, sub-acute, or chronic UE and rarely colitis.

Classification

Clostridium colinum was initially classified as *Corynebacterium perdicum*. Peckham reproduced UE with this organism and fulfilled Koch's postulates. 16S rRNA sequence analysis places *C. colinum* in subcluster XIV-b with 6 other *Clostridium* spp. It is most closely related to *C. piliforme*, the agent of Tyzzer's disease.

Morphology and Staining

Clostridium colinum is a Gram-positive rod, occurring singly as straight or slightly-curved rods, 3–4 μ m by 1 μ m, with rounded ends. Sporulation is rare on artificial media, but if present, spores are oval and subterminal. Sporogenic cells are much longer and thicker than nonsporulating cells.

Growth Requirements and Colony Morphology

Clostridium colinum is fastidious in its growth requirements. It needs an enriched medium and anaerobic environment. The best isolation medium is tryptose-phosphate agar (Difco) with 0.2% glucose, 0.5% yeast extract, and 8% horse plasma. Pre-reduced plates are inoculated with material from liver, intestinal, or splenic lesions and incubated anaerobically for 24–48 hours at 35 °C–42 °C. The resulting colonies are 1–2mm in diameter, white, circular, convex, and semi-translucent, with filamentous margins. The organism may also grow on pre-reduced blood agar incubated anaerobically. Growth in liquid medium can be detected as early as 12–16 hours postinoculation. Actively growing cultures produce gas, but for no longer than 6–8 hours, after which growth settles to the bottom of the tube. Subcultures should be made from actively growing broth cultures still producing gas. *C. colinum* commonly shows subterminal enlargements but these are not usually obvious spores.

Susceptibility to Chemical and Physical Agents

Clostridium colinum is, by its production of spores, highly resistant to chemical agents and physical insults. Yolk cultures may remain viable for at least 16 years at -20°C , and they survive heating at 70°C for 3 hours, 80°C for 1 hour, and 100°C for 3 minutes.

Pathogenesis

After oral infection, *C. colinum* adheres to the intestinal epithelium, producing the characteristic lesions in the small intestine and, occasionally, proximal colon. The organism then may migrate to the liver via portal circulation, producing the foci of hepatic necrosis frequently seen in cases of UE. Very little is known about the basis of virulence of *C. colinum*. The role of a toxin in the pathogenesis of UE has been suggested, but not demonstrated. The genome of *C. colinum* has not been characterized

Transmission

Horizontally

Incubation Period:

1-3 days

Clinical Signs:

- 1- Diarrhea, which is initially watery but may become hemorrhagic
- 2- Listless and humped up, with eyes partly closed and feathers dull and ruffled.
- 3- Notable emaciation, with atrophy of pectoral muscles, is seen in birds affected for a week or longer.
- 4- Birds may die from acute disease with no premonitory signs.
- 5- Young quail may be subject to 100% mortality in a few days. The mortality rate in chickens typically ranges from 2% to 10%.

Gross Lesion:

- 1-Acute lesions in quail are characterized by severe ulcerative and hemorrhagic enteritis.
- 2-Variable size mucosal ulcers surrounded by a hemorrhagic halo may be visible from the mucosal and serosal side of the intestine.
- 3-Ulcers may be deep and involve the whole thickness of the intestinal wall, causing perforation and subsequent peritonitis
- 4-subacute or chronic lesions can be observed in birds surviving for several days. They consist of multiple large, roundish yellow ulcers surrounded by hemorrhages, in any part of the small

or large intestine and ceca. These ulcers later coalesce to form larger lesions and may be covered by diphtheritic membranes

5- Blood is commonly found in the gut.

6- Ulcers in ceca may have a central depression filled with firmly attached, dark-staining, soft material.

7- As in acute cases, perforation of ulcers frequently occurs, resulting in peritonitis and intestinal adhesions

8- Liver lesions are not always evident. When present, they vary from light yellow mottling to multiple large, irregular, gray, or yellow circumscribed foci

9- The spleen may be congested, enlarged, and hemorrhagic, with or without multifocal necrotic areas

Microscopic

1- Acute cases reveal erosion and/or ulceration of small intestinal mucosa, and edema, congestion and heterophilic infiltration of the mucosa and, occasionally, the submucosa and other layers of the intestinal wall

2- The intestinal lumen contains desquamated epithelial cells, erythrocytes, heterophils, cell debris, fibrin, and Gram-positive rods

3- As the lesions progress, mucosal ulcers involving villi and extending into the submucosa grow deeper.

4- Most ulcers are covered by thick pseudomembranes composed of desquamated epithelial cells, fibrin, cell debris, mixed inflammatory cells, and Gram-positive bacilli.

5- A rim of heterophils, lymphocytes, plasma cells, and macrophages surrounds the ulcers.

6- Vascular thrombosis, including a large number of Gram-positive intravascular rods in the mucosa is an almost constant finding in both the acute and chronic lesions of UE.

7- Liver lesions, when present, are poorly demarcated foci of coagulative necrosis, with minimal inflammatory reaction and occasional intralesional clumps of Gram-positive rods, randomly scattered throughout the parenchyma

Diagnosis:

1- Clinical signs and gross and histologic lesions. 2- Isolation and Identification of Causative Agent

3- RT-PCR. 4- ELISA

Differential Diagnosis

- 1-Coccidiosis,
- 2-Necrotic enteritis by *C. perfringens*
- 3- Histomoniasis

Treatment

1-Antibiotics:

A-Streptomycin at a level of 60 g/ton of feed or 1 g/gal of water gives complete protection when administered prophylactically

B-Bacitracin methylene disalicylate can be used at 200 g/ ton for control of UE in quail.

C-Furazolidone, chlortetracycline, penicillin, ampicillin, and tylosin.

D-Prevention of coccidiosis with monensin and salinomycin, combined with tylosin

2-Anti-inflammatory:3-Supplements: 4-Local antiseptics