Avian Necrotic Enteritis

Necrotic enteritis is an enteric disease of poultry characterized by patches of necrotic tissue on the intestinal epithelium. This disease can cause high mortality, primarily in juvenile broilers.

The causative agent of necrotic enteritis is the bacterium *Clostridium perfringens* (types A and C), a normal occupant of the chicken digestive tract. These bacteria are rod-shaped, Gram-positive, anaerobic, and sporulated. *C. perfringens* is frequently isolated from poultry houses. Presence of the bacteria does not necessarily indicate presence of the disease. Many factors must co-occur for necrotic enteritis to develop, including dietary factors and coinfection with coccidia. While antibiotic growth promoters effectively controlled necrotic enteritis in the past, recent trends in reducing the usage of antibiotics have changed the approach to controlling this disease. Current strategies focus on managing predisposing factors, rather than trying to eliminate the causative bacteria.

**Clinical signs**

Birds seem depressed, with ruffled feathers. Diarrhea, dehydration, and inappetence are common. Mortality may be significant (as high as 30% in untreated flocks). In subclinical necrotic enteritis, there are only subtle clinical signs, if any (hence, the term *subclinical*). Subclinical disease results in a decrease in production (reduced weight gain and increased feed conversion).

**Occurrence**

Necrotic enteritis (and subclinical necrotic enteritis) is a multifactorial disease. Several components must occur simultaneously for *Clostridium* to begin multiplying in large numbers and producing toxins (a-Toxin and Net-b) that harm the intestine. Necrotic enteritis normally occurs after infection with *Eimeria* parasites (primarily *E. maxima*), especially when birds are fed diets containing grains that promote high intestinal viscosity, such as wheat, barley, and rye. High levels of animal protein and animal fat content in the diet have also been correlated with the incidence and severity of the disease. It is believed that the excess intestinal mucus production and protein leakage associated with *E. maxima* infection and certain diets provide an adequate environment for *C. perfringens* to replicate and produce the damaging toxins. Immune suppression caused by viral infections has also been linked to necrotic enteritis. The disease tends to occur in juvenile meat-type chickens. In intensive production, the disease occurs most frequently in 16- to 28-day-old birds. Notably, in litter-raised birds, the peak coccidial challenge also occurs near this time window, depending on anticoccidial program, litter quality, and bird density. In layer-type birds, the disease is uncommon, but can appear later in life. Some *C. perfringens* isolates are much more aggressive than others, and the disease can persist on a particular farm until practices that encourage disease co-factors are addressed.

**Transmission**

*C. perfringens* is normally present in the feces of some chickens; thus, the litter pecking behavior of birds easily spreads the organism throughout the flock. However, *Clostridium* normally exists commensally in the intestine, and only under certain circumstances will it start producing the toxins that are associated with the disease.
**Diagnosis**

Lesions are fairly indicative of the disease. Under the microscope, necrosis of the tissue and inflammatory infiltrate are evident. Small rods corresponding to Clostridium are seen in close proximity to the lesions, attached to cellular debris. Culture of C. perfringens from animals whose lesions do not match to those of necrotic enteritis should not be classified as necrotic enteritis. Even abundant numbers of Clostridium (1 x10^5 CFU/g of intestinal content) may be found in healthy birds, and much greater numbers occur in diseased animals (1 x 10^7-8 CFU/g of intestinal content).

In necrotic enteritis, the epithelium of the jejunum and ileum (or portions thereof) are necrotic. Patches of the epithelium may be detached, partially detached, or loosely adherent to the basal membrane. The epithelium that is still adherent to the basal membrane appears uneven and is frequently described as having a "Turkish towel" appearance (Figure 1). Abundant mucus, liquid, and cellular debris are commonly found in the intestinal lumen. Foul odor from decaying tissue is a frequent finding. Presence of hemorrhages, seen from the serosal surface, are a common finding (Figure 2). In subclinical necrotic enteritis, there is the characteristic presence of necrotic foci in the small intestine (Figures 3 and 4). These lesions are more frequent in the jejunum, but they can extend into the caudal part of the duodenum or proximal part of the ileum. Along with the necrotic lesions, abundant mucus and cellular sloughing are found in the intestinal lumen. Presence of congestion, mucus, and/or cellular sloughing without the characteristic necrotic lesions corresponds to general enteritis and should not be mistakenly diagnosed as subclinical necrotic enteritis.

![Figure 1. Massive destruction of the mucosal surface of the small intestine of a broiler chicken affected by a severe case of necrotic enteritis. Photo credit: Dr. Lorenzoni.](image1.png)

![Figure 2. Small intestine of a broiler chicken experimentally infected with E. maxima and C. perfringens. Hemorrhages are evident from the serosal surface. Photo credit: Dr. Lorenzoni.](image2.png)

![Figure 3. Typical ulcers in the mucosa of the small intestine of a broiler chicken with subclinical necrotic enteritis. Photo credit: Dr. Lorenzoni.](image3.png)

![Figure 4. Typical ulcers in the mucosa of the small intestine of a broiler chicken with subclinical necrotic enteritis. Photo credit: Dr. Lorenzoni.](image4.png)
Control

For decades, antibiotic growth promoters (e.g., BMD, enramycin, avilamycin) have been used on a regular basis to prevent necrotic enteritis. Nowadays, with considerable pressure to decrease the use of antibiotic growth promoters, these medications are no longer a sustainable strategy to control necrotic enteritis.

Vapors of hydrogen peroxide can be used to inactivate \( C.\ perfringens \) spores. On chicken farms, heating the humid litter considerably decreases the number of viable spores. This effect is thought to be mediated by the ammonia vapors released from the litter, rather than from the relatively modest temperatures reached in such a process. However, cleaning and disinfection of the premises should not be overestimated. \( Clostridium \) is a hardy organism and is commonly present in the intestinal tracts of birds. Instead of trying to eliminate the organism from the premises, it is advised to control the environmental factors that \( Clostridium \) needs to generate the disease.

Control of coccidia should be a pillar in the strategy against necrotic enteritis. With coccidia under control, it is rare to have an outbreak of necrotic enteritis. Maintenance of good litter quality can be very helpful to control coccidiosis. It is also worth mentioning that ionophore drugs (a category of antibiotics whose primary target is coccidia) are also active against \( C.\ perfringens \). In some places, ionophores are still considered to belong to a slightly different category than antibiotics, and their use is still commonplace to reduce the incidence of necrotic enteritis.

Given that wheat, rye, barley, animal protein, and animal fat increase the incidence of necrotic enteritis, consider reducing the inclusion rate of these ingredients in diet formulations. Probiotics and acidifiers have been marketed to reduce the incidence of the disease, but the outcomes of these interventions are highly variable.

Treatment

Aggressively targeting the predisposing factors, in addition to \( Clostridium \) itself, will ensure the efficacy of the treatment. Antibiotics such as bacitracin, lincomycin, oxytetracycline, virginiamycin, and others have been proven useful in treating necrotic enteritis.

References


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