Avian Coccidiosis

In poultry, coccidial infection causes parasitic enteritis in multiple parts of the intestinal tract, leading to poor performance, loss of pigmentation, diarrhea, and mortality in severe cases.

Coccidiosis is caused by protozoan parasites in the genus *Eimeria*. These parasites are host-specific, and many species occupy a specific segment of the intestinal tract. There are nine species of coccidia described in chickens, and several other species have been described in turkeys, ducks, and geese. Coccidiosis may be one of the most common and costly diseases in the poultry industry. The costs are associated with prevention, control, loss of performance, and mortality, especially when the disease is complicated by other infectious agents such as *Clostridium perfringens*. Vaccination, preventative medication, and good management practices can help control the disease.

**Clinical signs**

Under heavy infections, birds may appear depressed with ruffled feathers. Diarrhea and dehydration may be present. Blood and/or clear to bright orange mucus may be present in the feces. Affected birds may appear pale in comparison to unaffected birds. Body weight and feed conversion may also be affected in heavy infections with more pathogenic strains of coccidia.

**Occurrence**

Coccidiosis occurs worldwide. It is especially prevalent in high-density flocks and in flocks where animals have direct contact with their feces. Virtually every poultry flock raised on litter is affected by coccidiosis to some degree, depending on flock density, quality and amount of bedding material, and immune status of the flock. Animals raised in cages with no contact with fecal material do not develop the disease. However, it is important to note that if caged animals have access to manure (due to poor design or lack of maintenance of the cages), the disease will develop.

**Transmission**

Coccidia is transmitted via oral ingestion of contaminated feces. Following ingestion, the parasite undergoes its complex life cycle within the intestinal tract, during which it replicates and ruptures the intestinal cells. The infected animal then sheds oocysts in the feces. After a few intestinal cycles of the parasite, birds can develop long-lasting, but species-specific, protective immunity. Challenge with a different species will lead to infection and disease, depending on the number of oocysts consumed.

**Diagnosis**

Gross lesions are typically sufficient to diagnose coccidiosis. If necessary, a mucosal scrape can be taken for microscopic examination. Oocysts are easily seen under the light microscope. Lesions, which are variable depending on the species of coccidia, are described below.

**Chickens**

*E. acervulina* is the most common species found in commercial operations, which may be due in part to the easily identifiable lesions. In light infections, gross lesions are seen in the duodenum, in the form of white plaques measuring 2-3 mm (Figure 1). In heavy infections, the lesions may go beyond the duodenum and appear smaller and more densely packed. There may be mucoid fluid in the intestine.
E. maxima is not easy to diagnose based on gross observations. Most lesions occur in the jejunum and ileum. Small petechiae, visible through the serosa of the intestine (Figure 2), are often seen (although the presence of these lesions is not pathognomonic). Inside the lumen, it is common to observe abundant yellow/orange mucus with or without blood (Figure 3). The intestine is sometimes described as having a ballooned appearance because some sections of the intestine remain flaccid while others retain a more robust intestinal tone. Because the characteristic lesions are not always easy to identify, a mucosal scrape should be taken for microscopic examination.

E. tenella infects the ceca. It can be recognized easily due to the characteristic nature of the lesions. In light infections, small petechia cover the mucosa (Figure 4). In heavy infections, free blood or yellow, cheesy cores in the cecal lumen are common (Figure 5). The ceca may appear distended and the lumen filled with clotted blood and sloughed mucosa.
Figure 5. Hemorrhagic cecum of a broiler chicken. Note the thickened wall of the cecum. These lesions are compatible with severe infection with *E. tenella*. Photo credit: Dr. Irene Rojas.

*E. necatrix* produces lesions in the small intestine. The lesions appear in older chickens compared to the other species of *Eimeria*; this infection is uncommon in young broilers. When birds are kept over 10 weeks of age, *E. necatrix* can be a concern. The intestine may appear ballooned, and the lumen is often filled with increased mucus and necrotic, sloughed mucosa. In dead birds, black and white spots, referred to as "salt and pepper" lesions, can be seen through the serosa.

**Turkeys**

*E. adenoeides* infects the ceca. In light infections, there is edema and petechia in the ceca. In heavy infections, a core with caseous material develops in the cecal lumen. *E. gallopavonis* localizes caudal to Meckel’s diverticulum (beginning of the ileum). The intestine is edematous, and a white caseous exudate develops in the lumen. *E. meleagrimitis* affects the upper portion of the small intestine. It produces edema, with large amounts of fluid and mucus in the lumen.

**Differential diagnoses**

- Bacterial enteritis, viral enteritis

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**Control**

Contrary to popular opinion, coccidia are not highly resistant organisms at the level of individual parasites or oocysts. The number of coccidia in the environment quickly decreases with very high humidity, low oxygen tension, high ammonia concentration, and time outside a host (over 2 weeks). However, as a population, coccidia are extremely resistant. A reduction in parasite numbers is not difficult to achieve, but complete elimination of the parasite from a production site is virtually impossible. In most poultry commercial operations, rigorous disinfection is not a conventional strategy for preventing the disease.

There are two common techniques to prevent coccidiosis: vaccination and preventive medication. Birds can receive a vaccine for the relevant species at one day of age. Vaccination introduces a mild coccidia challenge (with peak intestinal lesions around 12 days of age) which may lead to immunity against field strains of the parasite. Drugs known as ionophores (monensin, narasin, maduramicin, salinomycin) or chemicals (clopidol, robenidine, diclazuril, nicarbazin) may be administered in the diet to control infection. Drugs in the chemical group tend to be very effective, but development of drug resistance is rapid (with the notable exception of nicarbazin). Ionophores are less effective at suppressing all stages of parasite replication, and some shedding of the parasite still occurs. However, this is advantageous for development of natural immunity, and resistance develops very slowly, if at all. Thus, ionophores can be used for long durations with limited pressure to change the product. Chemicals should only be used sporadically, for a short period of time, to avoid development of resistance.

Management practices are also very important for preventing coccidiosis. Coccidia oocysts are ingested due to litter pecking behavior. Maintaining a thick, dry layer of litter effectively dilutes the fecal material with the bedding, resulting in a reduced number of coccidia ingested during litter pecking. The consumption of a limited number of oocysts stimulates immunity against coccidiosis.

**Treatment**

Treatment is reserved for when prevention has failed. Amprolium is one of the more popular drugs for the treatment of coccidiosis. Sulfonamides, such as sulfadimethoxine, can be administered in the drinking water for treatment under the direction of a licensed veterinarian. Appropriate drug withdrawal is necessary for meat birds. This medication is not suitable for laying hens. Before medicating a flock, consult a current version of the Feed and Additive Compendium or FARAD for current information on approved products.

**References**
