COCCIDIOSIS IN CHICKENS

SLIDE STUDY SET #7

A CONTINUING EDUCATION PROGRAM PREPARED BY

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CONTINUING EDUCATION COMMITTEE

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INTRODUCTION

Severe coccidiosis outbreaks are seen less frequently in chickens today due to the almost universal use of preventive anticoccidials in feed. Clinical outbreaks still occur frequently enough that every poultry diagnostician needs some training in recognition of gross lesions and the parasitic stages of the parasite. Oocysts and other stages are easily seen under the microscope in temporary smear mounts. Since histopathological studies are seldom required for diagnosis, they will not be described.

Outbreaks may occur due to (1) improper mixing of the anticoccidial drug, (2) drug resistance or a lack of drug efficacy against certain species or strains, (3) lack of immunity in birds which are usually maintained without an anticoccidial after they mature, or (4) reactions to improper use of a planned immunization program (Coccivac). In each case an accurate diagnosis, preferably with identification of the species, may provide a great service to poultry producers in correcting their coccidiosis control programs or in suggesting flock treatment.

CLINICAL FEATURES

Flock History and Signs

Age incidence. Coccidia require 6 to 8 days to complete their life cycle in the chicken. Signs of the disease do not appear until the 4th, 5th or 6th day post infection. The most severe outbreaks generally occur in chickens between three and six weeks of age, but onset may occur at any age, even after pullets are laying eggs. In the latter case, oocysts of the species causing the outbreak have only recently been introduced into the house.

Breed incidence. Coccidiosis appears in all breeds of layer replacement and broiler stock. In some cases, greater susceptibility of Leghorn strains has been demonstrated, but breed or strain differences are not of great magnitude.

Seasonal incidence. There is no major difference in seasonal incidence if birds are reared in confinement. Oocyst sporulation is inhibited by cool temperatures. Freezing or hot temperatures will kill the oocysts. If birds are reared on range, severe outbreaks occur during periods of warm, moist weather.

Mortality. Although mortality up to 30% has been occasionally experienced, such outbreaks are rare with present management programs. During such outbreaks, most of the mortality occurs within a period of 2 to 3 days on the 5th to the 8th day after exposure. Treatment started after signs of the disease appear is usually too late to protect the bird in which the disease is recognized. In a large house the disease may spread slowly from

one end to the other since most chickens do not move about extensively. In such situations, prompt drug treatment may come in time to protect a part of the flock receiving late exposure.

Influence of complicating diseases. Marek's disease and Infectious Bursal Disease Agent (Gumboro disease) may affect the immune mechanism which would ordinarily provide protection for the bird against coccidiosis. Since Marek's disease is often diagnosed after the presence of coccidiosis has been recognized, coccidiosis has sometimes been thought to be the primary agent responsible for transmission and development of Marek's disease. A more likely explanation is that early unrecognized Marek's disease has upset the immune mechanism which would normally have prevented appearance of clinical signs of coccidiosis.

Housing and management considerations. Although "sanitation" has often been recommended to poultry producers as a method of coccidiosis control, disinfectants and other means of destroying the oocysts have little effect in preventing outbreaks of the disease. Moisture control in the litter represents the one sanitary procedure which should be recommended to producers, since freshly passed oocysts require suitable moisture, temperature and aeration conditions to sporulate. Wet spots will almost always be discovered in a house where a coccidiosis outbreak has occurred. Leaky roofs or watering devices are often found after an outbreak has occurred. Although outbreaks have occurred after fecal accumulations in cage or slat-floor management, coccidiosis is primarily a disease of birds being produced on litter.

Clinical signs. The first signs of coccidiosis in a flock may be huddling, chilling or hiding behind feed cans during acute stages of the disease. The birds frequently present a shivering appearance, but there are no measurable decreases in body temperature until just before death, Wet, bloody or mucus-containing droppings are common signs. A flashlight may be required to detect these signs if the house is dark. A characteristic odor frequently accompanies an outbreak and experienced poultry producers can sometimes detect the disease from the odor on opening the house door. Chicks sometimes let out an anguished cry during acute attacks of diarrhea.

Clinical course. Birds surviving a severe case of coccidiosis will usually recover their appetites within a period of one to two weeks. Weight loss due to dehydration will rapidly be recovered, but infected birds never completely catch up on weight gains with uninfected controls. If an outbreak occurs in layers, egg production may be resumed and approach normal in two to four weeks. Rarely birds may be permanently stunted by rupture of the cecum which may produce a non-fatal peritonitis.

Coccidiosis versus coccidiasis. Coccidial infection is so common in chickens that frequently it may go undetected. If there are no clinical signs and no economic losses attributable to the infection, the condition has sometimes been called coccidiasis. These mild infections assist the bird in building immunity which will later provide protection. Undetected outbreaks may produce

economic losses due to weight loss or a decrease in skin pigmentation.

Species of coccidia. Nine different species of coccidia have been recognized in the United States. Six of these are considered of sufficient importance that they are regularly recorded in diagnostic laboratories. These are Eimeria acervulina, E. brunetti, E. maxima, E. mivati, E. necatrix, and E. tenella. The other three (E. hagani, E. mitis and E. praecox) will not be described in detail. E. hagani has never been adequately described for easy recognition. E. mitis and E. praecox are relatively less pathogenic. Although some writers have called these latter three nonpathogenic, any species may cause sufficient damage to preclude use of the work nonpathogenic in characterizing them.

A diagnostic chart (see center section of attached Research Report 163 by Reid, 1973) provides the easiest method of comparison and gives a key to rapid identification. The zone parasitized is shown in red, while pathognomonic characteristics are printed in red. These characteristics include macroscopic lesions, microscopic characteristics such as length and width of oocysts, together with other descriptive characteristics. Additional copies of the chart which may be removed and posted for laboratory use are available (University of Georgia, College of Agriculture, Experiment Stations, Athens, Georgia, 30602). Fuller descriptions of individual species are reviewed by Reid (1972).

Slide 1. After the bird has been opened in the usual fashion for post-mortem examination, locate the attachment of the duodenum

to the gizzard and gently free the intestine throughout its entire length by tearing it loose from the mesenteries for close inspection. The bile and pancreatic ducts will need to be cut free. Under a bright light (a dental light has been found useful in our laboratory), examine first the serosal surface for petechiae, plaques, or whitish colonies, or peculiarities in normal coloration. The intestines should be split open with scissors or an enterotome and intestinal contents examined for color and consistency. Push the contents aside to examine carefully the mucosal surface, again looking for petechiae, whitish streaks or plaques, hemorrhaged areas, color changes, presence of necrotic zones, and in the cecal area, presence of cores. Suspicious areas suggesting coccidial infection require making a smear and examining scrapings or fecal material under the microscope (see page 7 of Research Report No. 163).

Slide 2. In examination of the duodenal loop note the presence of characteristic lesions on both the unopened intestine (serosal surface) and the opened intestine (mucosal surface). Plaques are generally visible from both surfaces, but are more easily distinguished on the mucosal surface. These plaques are large and well differentiated in mild infections. They are sometimes elongated transversely like the rungs of a ladder. In severe infections, they may be coalesced so that individual plaques may be difficult to identify. Many of the slides in this series are marked with +1 to +4 which represents a lesion scoring system of increasing coccidiosis severity more fully described elsewhere

(Johnson and Reid, 1970, 1972; Reid and Johnson, 1974). #1 = +1 as a lesion score for E. acervulina.

Slide 3. A more severe infection shows similar plaques, but they are more numerous and coalescing has occurred (+3). Such an infection causes weight losses in broiler chicks, and pathogenicity is more severe than in the mild infection shown in Slide 2. A mild infection (+1 lesion score) causes no mortality or weight loss and may be of economic importance only to producers interested in skin pigmentation. Mild coccidiosis from any species prevents absorption of carotenoids which are deposited in the skin and produce the yellow color preferred in some market areas.

Slide 4. In the same duodenal loop area, severe infection (+4) of E. acervulina is shown. The distinctive lesions are obscured by the coalescence, and presence of coccidial infection could be entirely overlooked. An experienced diagnostician may recognize the roughened surface of the mucosa, whereas, individual lesions may not be identified. The appearance of the intestinal wall is grayish in color, and large numbers of oocysts will be found in the scraping from the mucosal surface in mild, moderate, and severe infections. Some thickening may be seen or felt with the fingers.

Slide 5. In extremely severe infections the entire mucosa may be reddened (+4). Such a condition is rare under field conditions, but was produced in the laboratory by administering several million oocysts. In our laboratory, heavy breeds are more susceptible to this type of infection than Leghorns.

Slide 6. Eimeria mivati lesions are similar to those of E. acervulina. The parasite invades the duodenal loop, and lesions may differ somewhat in shape. Sometimes these are more rounded; whereas, with Eimeria acervulina they may be elongated transversely. Oocyst size averages 1 or 2 microns smaller with this species than with Eimeria acervulina (see chart in Research Report 163). The two species do not produce cross immunity to each other and thus are listed as separate species by most American investigators. Because of the difficulty in distinguishing between E. acervulina and E. mivati, many diagnostic laboratories list them together as the Eimeria acervulina-mivati type. Several British investigators list E. mivati as a subspecies of E. acervulina.

SLIDE 7. This slide shows a mild or moderate infection (+2) of Eimeria mivati with somewhat rounded plaques. An infection this severe would cause some weight losses in broilers.

SLIDE 8. A severe infection of Eimeria mivati shows no distinctive lesions. Plaques are so numerous that they have completely coalesced. Differences in color and in thickening of the wall of the intestine may be noted. Compare the appearance of the opened and unopened intestine with that of the unparasitized control above.

SLIDE 9. Eimeria maxima and E. necatrix are two species which normally develop in the mid-intestinal area. Eimeria maxima produces petechiae (pinpoint hemorrhages) four to six days after oocysts have been taken in by the chicken. Petechiae appear deep in the submucosa and are best seen from the serosal surface. The

mucosal surface in mild infections may show no distinctive lesions, but the increased mucous secretion may show a pinkish color due to mild hemorrhaging.

SLIDE 10. Note the reddish mucus in the open section of the E. maxima infected intestine. A well-differentiated yolk sac diverticulum provides a convenient marker for the area most frequently parasitized by this species. Bleeding, which is most severe on the 5th and 6th days post infection, is usually associated with the growth of the macro and microgametocytes. These parasitic stages may appear in deep scrapings of the mucosa inside parasitized host cells.

SLIDE 11. This slide shows a more severe infection with more numerous petechiae and some swelling (often spoken of as ballooning).

SLIDE 12. Scrapings of the intestine on the 6th or 7th day will show the appearance of mature unsporulated oocysts. These have a distinctive brownish-yellow color, and they are considerably larger (21.5 to 42.5 microns) than the oocysts of the other species. Such an oocyst is seen between 50 and 60 on the ocular scale. There are several smaller oocysts of Eimeria mivati in the field. They measure 11.1 to 19.9 microns in length.

SLIDE 13. This slide shows a sporulated oocyst positioned to measure the width of the oocyst. This specimen measures 13 ocular spaces. The calibration factor for this microscope under oil immersion is 1.6. Thus, 13 x 1.6 = 20.8 microns in width. If the ocular were positioned to measure length, this oocyst would cover

15 ocular spaces. the calibration factor of 1.6 x 15 = 24 microns long. These dimensions (24 microns long x 20.8 microns wide) fit into the ranges on the table for E. maxima. For further directions including the calibration of the microscope, see p. 16-18 of Research Report 163.

SLIDE 14. This slide shows a severe case of intestinal coccidiosis caused by Eimeria necatrix. This species normally infects the midintestinal area and in this case is severe enough to cause marked distension. This condition is often called "ballooning". The mid-intestine is clearly marked by the yellowish papilla or the yolk-sac diverticulum. This structure is often used as a convenient landmark to locate the mid gut area.

SLIDE 15. Eimeria necatrix. This closeup view shows the ballooned intestine above and the more normal-sized intestine at the level of the yolk sac diverticulum. Petechiae are apparent on the surface, and on close inspection you will see the characteristic yellowish plaques.

SLIDE 16. A milder infection of E. necatrix on day 7 shows presence of reddish petechiae visible both on the serosal and the mucosal surface. A few of the yellowish plaques are seen on the exterior surface, but they are located too deep to be seen on the mucosal surface. These latter structures are made up of aggregations of second generation schizonts. A scraping of one of these yellowish plaques mounted on a slide may show a colony of the large schizonts. The plaque will need to be located from the outer surface, but scraped free on the mucosal surface by making a deep

gouge with the scalpel.

SLIDE 17. The second generation schizonts are often found in clusters and may measure 50 to 66 microns in diameter. No other intestinal species has such large schizonts, although large ones are also produced in the cecum by E. tenella. This unstained aggregation can often be spotted under low power of the microscope.

SLIDE 18. High power of oil immersion may be required to distinguish distinctive structures found in the schizont. If the schizont is mature (5th day), the interior of the schizont may appear to be crowded with banana-shaped second generation merozoites. A ruptured schizont releases the merozoites, which then move about with a characteristic undulating movement.

SLIDE 19. Severe hemorrhaging in the mid-gut area shows in the short area opened on the intestine as shown in the upper section. Below, the intestine stretches (between) the two ceca. The ceca have been opened and show clotted blood. This blood has come down from the hemorrhaged area of the mid-gut and is not produced by the cecal walls. Presence of blood here may confuse the diagnostician into an E. tenella misdiagnosis. However, if the cecal contents are removed, the cecal epithelium appears normal even though the oocysts of E. necatrix may already be developing in this area. Oocysts of E. necatrix develop only in the cecal area after second generation merozoites have passed down. Oocysts found in the mid-gut area represent another species and indicate an infection caused by two or more species of coccidia.

SLIDE 20. In the lower intestine between the two ceca a

moderate infection of E. brunetti is revealed after the intestine is opened. Thin, longitudinal, reddish streaks appear on the mucosal surface. Some blood is present in the mucus to the left. Ceca are generally normal except when there is some indication of dehydration which occasionally is noted with this species. Mild infections of E. brunetti are much more frequent in the field than are severe infections. Investigators frequently overlook the milder infections of Eimeria brunetti which produce weight losses in the host. The only sign of gross lesions may be a roughened area detected by the use of the dissecting instrument or by the finger on the mucosal surface.

SLIDE 21. The classical coagulation necrosis from a very severe E. brunetti infection is rarely seen in field infections. Sloughing of the entire mucus membrane may appear. There may be a close association between E. brunetti and Ulcerative Enteritis infections (Davis, 1973). Note the dehydrated appearance of the cecal contents in the upper cecum which is open. The lower cecum shows the unopened condition.

SLIDE 22. Moderate to severe cecal coccidiosis produced by E. tenella infection is shown in the upper slide. This represents about the 6th day of infection. Hemorrhaging is most severe on the 5th day with coagulation on the 6th and the whitish core filled with oocysts being typical of the 7th day. The normal cecal contents (below) show the brownish color and the soft, pasty fecal material. The ceca are elongated compared to E. tenella infected ceca. Infected ceca show thickening of the wall as well as

distension due to partial blockage in the neck region.

SLIDE 23. This slide shows a close-up view of a very mild infection (+1). Single petechiae on the mucosal surface are the only lesions seen.

SLIDE 24. A severe infection on the 7th day shows a gangrenous extension at the distal end of the lower, unopened cecum. The upper, opened cecum shows coagulated blood and necrotic material, which contains an accumulation of numbers of whitish (microscopic) oocysts. The infection has extended out into the intestinal area between the ceca as indicated by blood and necrotic materials.

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