Investigation of Enteritis and Elevated Mortality in Young Guinea Fowl

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Summary
Increased mortality in young guinea fowl, raised commercially for meat, was investigated. Mortality was preceded by running, abnormal stance and gait, pasty vents, anorexia, and inactivity. On investigation, Farm A flock 4 had a cumulative mortality over 36% by 3 weeks of age. Necropsy, histopathology, bacteriology, virology, and parasitology were performed. Coccidiosis was diagnosed; astrovirus and rotavirus were also present in affected keets. In contrast to Farm A, Farm B had no elevation in mortality; flocks there were also positive for astrovirus and rotavirus but negative for coccidiosis. Prophylactic prevention in future flocks with amprolium has eliminated this early mortality peak. This is suggestive that a heavy coccidial infection or combination of coccidiosis and enteric viral infection caused the enteritis and increased mortality in these guinea fowl.

Introduction
Commercial guinea fowl production, while common in Europe and Africa, is present to a limited extent in the United States. In comparison to turkeys and chickens, little is known about guinea fowl diseases, especially in the US. Causes of enteritis in guinea fowl can be bacterial, viral, or protozoal. Based on European data, the common species of coccidia that infect guinea fowl are Eimeria meleagrisae and E. grenieri2,7 resulting in stunted growth, ruffled feathers, anorexia, diarrhea2,7, and even mortality in the case of E. meleagrisae2,7. Other species of Eimeria have also been recently identified in guinea fowl11. No data is available from the US on guinea fowl coccidia prevalence or prevalence. Astroviral infections as a cause of guinea fowl enteritis have been reported from Europe2,4 while rotavirus has been isolated from Italian cases, but this virus has not been shown to produce clinical signs experimentally8. The following case investigates enteritis, stunting, and mortality in a flock of guinea fowl in North Carolina.

Case History
Farm A had 8 flocks of guineas of different ages in a single 600 x 40 foot house divided into 75 foot sections. Approximately 2,000 birds were placed per flock, flocks were separated by 6 weeks of age and divided by an approximately 4 foot tall chicken wire fence. Shared water and feed lines ran half the length of the house. Litter was changed approximately once per year. Farm B was similar except it was 300 x 40 foot house and divided for growing 4 flocks instead of 8. Farm B was not affected and served as a control. Four flocks from Farm A and two flocks from Farm B ranging in age from 14 days to 56 days were evaluated.

Results
Gross Pathology: Keets were emaciated with atrophied breast muscle and little pericardial fat (Figure 7). After the keets were removed, 70% of the birds had an enlarged gall bladder that extended past the liver margin (Figure 8). Adrenal glands were enlarged in 75% of the birds. Several birds had bursal tumors (Figure 9, arrow). Gross lesions were not identified in the intestines or other organs.

Histopathology: Small intestinal coccidiosis was found with marked bacterial overgrowth, large amounts of plant material, and enteritis (Figures 10, 11). Coccidial coccidiosis was present in most birds with some having very severe lesions (Figure 12). The bursa of Fabricius had bacterial overgrowth. No significant lesions were identified in the liver, sciatic nerve, brain, adductor muscle, lung, heart, spinal cord or kidney.

Clinical Presentation
Farm A presented with increased mortality in flock 4, which was 2 weeks old when initially examined. Daily percent mortality started increasing around day 10 with 1.25% and finally peaked on day 14 with 8.8% mortality. Compared to normal keets (Figure 4), approximately 3% of the keets in flock 4 exhibited the following clinical signs; abnormal stance and gait, lethargy, and apparently distended abdomen. The abnormal stance was characterized by keets standing upright with their keel perpendicular to the ground (‘penguin-like’); the birds also ambulated in this position (Figure 5). Affected birds usually had pasty vents (Figure 6). According to the grower, if these birds were not culled, they advanced to lateral recumbency, paddled in circles, and soon died. Roughly 1.5% of the birds were not showing abnormal gait but did show general weakness and inactivity. These birds were hunched and huddled in groups.

Methods
Affected keets from Farm A flock 4 were taken to NCSU College of Veterinary Medicine for further diagnostics. The keets were humanely euthanized by cervical dislocation and a routine necropsy was conducted. Samples were collected for histopathology, bacteriology, parasitology, virology, and electron microscopy. Liver, duodenum, jejunum, ileum, cecum, sciatic nerve, brain, bursa of Fabricius, adductor muscle, lung, heart, spinal cord and kidney were collected for histopathology. These samples were fixed in 10% neutral buffer formalin, 1 gram of fecal material was mixed with 10 mL of sodium nitrate solution; 1 gram of fecal material was mixed with 10 mL of solution and centrifuged for 10 minutes before being examined microscopically. Bacteriology was performed on pooled fecal samples that were collected from Farm A flock 4. Samples were tested for Salmonella via direct culturing on XLT4 and brilliant green agar with novobiocin. Eimeria samples were collected for histopathology. These samples were fixed in 10% neutral buffer formalin, whereas the electron microscopy samples were stored in 10% neutral buffer formalin.

Results
RT-PCR revealed astrovirus in intestinal samples from all flocks on both farms and rotavirus in the youngest flock (2 weeks of age) on each farm. All flocks were negative for reovirus and coronavirus. Neither Salmonella nor Campylobacter species were isolated. No virus particles were visualized on electron microscopy.

Discussion
Histopathology and fecal flotation strongly suggested coccidiosis as the cause of the increased mortality and enteritis in Farm A. Based on microscopic analysis of oocysts it is most likely that the keets were infected with both E. meleagrisae (lacking a microplor) and E. grenieri (microplor).1 Other species of Eimeria have also been recently identified in guinea fowl11. The possibility of involvement of other coccidia species is possible, as there is limited information on coccidia in guinea fowl in the US. Further characterization, isolation, and speciation of the coccidia in this case is warranted. The abnormal ‘penguin-walking’ was most likely due to weakness and emaciation caused by coccidial enteritis. Dysbacteriosis was most likely due to coccidial overgrowth, which damaged the intestinal mucosa altering the luminal environment and allowing for bacterial proliferation.

Treatment
Amprolium via the drinking water was given for treatment (0.024% solution) and prevention (0.012% solution) of coccidiosis. Withdrawal time for this drug must be considered, and it must legally be extended beyond the 0 day withdrawal for chickens and turkeys due to its off-label use in guinea fowl. The consequences of coccidiosis were long-lasting in this flock with severe uniformity problems through processing.

References