DIGESTIVE DISORDERS OF CHICKENS

I. DYSBACTERIOSIS

DEFINITION

Terminology used in Europe to describe an intestinal microflora imbalance and overgrowth characterized by enteritis and mild diarrhea.

OCCURRENCE

Dysbacteriosis is commonly observed after 21 days of age in European commercial broiler chicken flocks but can occur as early as 15 days of age.

HISTORICAL INFORMATION

There has been an increase in the number of broiler chicken flocks affected with dysbacteriosis with the ban of growth promoters in Europe in 1999.

ETIOLOGY

Over growth of an abnormal bacterial duodenal population has been demonstrated in birds affected with dysbacteriosis. *Clostridium spp.* has been shown to contribute to this overgrowth. The absence of antimicrobial growth promoters, animal protein and animal fat appear to predispose farms to the disease. Other predisposing factors may include non-specific stress, mycotoxins and systemic disease.

CLINICAL SIGNS

Dysbacteriosis is characterized by normal water consumption, humid litter, poorly formed and wet feces and a reduction in feed intake.

LESIONS

Thinning and ballooning of the small intestines accompanied by viscous or watery intestinal contents.

DIAGNOSIS

History of diarrhea, wet droppings. Elimination of any other causes of diarrhea and wet litter. Empirical therapeutic response to antimicrobial effective against *Clostridium perfringens* or other enteric pathogens might be a diagnostic indicator for both necrotic enteritis and dysbacteriosis.

CONTROL AND TREATMENT

Monitoring litter quality with a litter box might help in assessing any changes in fecal water content and alert to early signs of diarrhea. Antibiotics might be required if there is associated mortality or subsequent necrotic enteritis. Competitive exclusion products might help.

II. POLYCYSTIC ENTERITIS OF BROILER CHICKENS or RUNTING-STUNTING SYNDROME OF BROILER CHICKENS

DEFINITION

Polycystic enteritis (PE) has recently appeared in the Southeastern United States. It is characterized by large numbers of chicks with marked growth depression, watery diarrhea, and cystic enteritis.
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OCCURRENCE

PE may appear in chicks as early as 6-7 days of age, but the usual peak of the problem occurs at around 10-12 days of age, mostly during winter and spring. Farms that have short down times between flocks appear to be at higher risk for the disease. Turkeys are not known to be affected.

HISTORICAL INFORMATION

Runting Stunting Syndrome (RSS) has been recognized in chickens since the late 1970s. This condition occurs sporadically, usually with increasing severity over a year period within a given complex, then declines afterwards. During 2003-2005 a new clinical and pathological presentation appeared and caused economically significant problems in the Southeastern United States, and some countries in Asia, Middle East, and Latin America. In contrast to RSS, persistent problems with PE have been noted on specific “problem” farms as successive flocks are affected. Many research institutions are actively studying this condition, further characterizing enteric viruses, developing diagnostic tests and searching for potential vaccine candidates.

ETIOLOGY

The disease has been reproduced by placing broiler chicks on contaminated litter obtained from previously affected farms, and by gavaging birds with intestinal contents from affected chickens. These resulted in severe weight depression. Multiple viruses have been isolated with two groups commonly present; reoviruses and astroviruses. Bacteria do not appear to be involved in the disease as primary agents. Vertical transmission is considered a possibility and is being investigated.

CLINICAL SIGNS

Affected flocks show large numbers of depressed chicks huddling around feeders and drinkers within hours after placement. Litter quickly becomes damp. Feed consumption decreases, there is loss of flock uniformity and many chicks will show severe growth depression (5 up to 20%). If allowed to remain in the flock, stunted chickens do not recover. This will translate in increased need for culling, reduced livability, increased feed conversion, and days to market.

LESIONS

At necropsy, affected chicks have small livers with enlarged gallbladder, pale, dilated thin-walled intestines with watery contents and undigested food. Histologically, intestinal lesions consist of numerous large cysts involving intestinal crypts with degenerating or necrotic cells and mucin inside the lumen of these cysts. As the condition progresses, intestinal villi become shortened and clubbed.

DIAGNOSIS

History, clinical signs, and microscopic intestinal lesions are suggestive of the disease.

CONTROL

Built-up litter and short downtime may contribute to PE. Proper brooding temperature minimizes early poor uniformity and delayed growth. Heat treatment of affected houses during downtime is likely to mitigate the condition.

TREATMENT

There is no specific treatment. Good husbandry and symptomatic support of an affected flock will lessen economic losses. Severely stunted chicks will not recover and should be culled.
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III. TRANSMISSIBLE VIRAL PROVENTRICULITIS

DEFINITION

Transmissible viral proventriculitis (TVP) is a transmissible proventricular inflammation of viral etiology found in commercially raised broiler chickens and associated with increased proventricular fragility, impaired feed digestion, poor growth performances, and increased contamination and decreased efficiency at processing.

HISTORICAL INFORMATION

Within the past 15 years, commercial broiler chickens from Southeastern United States have sporadically been affected with this disease.

ETIOLOGY

TVP has experimentally been reproduced with homogenates from proventricular tissue of affected birds and a virus that is consistent with a new type of adenovirus. Presence of the virus in proventricular lesions in natural and experimentally infected birds indicate it is the cause of the disease. Chicks can be experimentally infected by oral or intracoelomic inoculation, but the natural route of infection is unknown.

CLINICAL SIGNS

Affected birds are pale and significantly smaller than uninfected flock mates. They show poor growth rate, increased feed conversion, and the passage of undigested or poorly digested feed in the feces.

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At necropsy affected broilers show proventricular enlargement, especially the isthmus between the proventriculus and ventriculus, with mottled thickened, firm walls. Attenuation of mucosal papilla where ducts from the glands open into the lumen may be seen. The mucosa appears roughened. Dilated, cystic glands are not indicative of TVP. They are a postmortem changes that occurs rapidly following death. Four lesions characterize the microscopic changes in the proventriculi: 1) necrosis of the glandular epithelium, 2) lymphocytic infiltration in the interstitium of proventricular glands and mucosa, 3) hyperplasia of ductal epithelium, and 4) replacement of lost glandular epithelium by ductal epithelium. Epithelial cell nuclei are swollen, pale, and often have prominent nucleoli. Inclusion bodies are rarely seen and cannot be relied on to provide a specific diagnosis. Lesions do not occur in other tissues.

DIAGNOSIS

TVP is difficult to identify on the basis of gross lesions. In contrast, microscopic lesions are sufficiently characteristic of provide a diagnosis. Confirmation requires demonstration of the aden-like virus by electron microscopy or a fluorescent antibody (FA) test. The FA test only works on fresh frozen tissue. Correlation between histopathology and virus presence is very high.

CONTROL, PREVENTION AND TREATMENT

There are no specific treatment, prevention, or control measures for TVP other than biosecurity measures effective against infectious agents.

IV. NECROTIC HEMORRHAGIC HEPATITIS

Although vibrionic hepatitis accurately describes a disease still seen sporadically in chickens, another hepatic disease has emerged in layers (both egg and meat types) with no regular relationship to Campylobacter infection. This disease, termed necrotic hemorrhagic hepatitis, occurs in 40-60-week-old layers and is
associated with increasing mortality over a period of several weeks (up to 0.3% per week) and a parallel
decrease in egg production (up to 20%). Affected hens are usually found dead without premonitory signs and
the major gross liver lesions include marked hepatomegaly with diffuse pallor, variable stippling/mottling with
red and yellow foci, friable consistency, and intralobar and subcapsular hematomas. The spleen is often large,
pale, and friable and the ovaries are inactive. The hepatic destruction suggests a chronic progressive disease and
there are histological changes pointing to a primary inflammatory process involving segments of portal veins.
No microbiological agent has been consistently demonstrated using aerobic, anaerobic, and microaerophilic
culture techniques, various special stains, direct electron microscopy, or chicken inoculation.