CARDIOVASCULAR DISEASES OF CHICKENS

I. ASCITES OR PULMONARY HYPERTENSION SYNDROME

DEFINITION

Ascites secondary to pulmonary hypertension syndrome (PHS) is one of the most important causes of mortality in broiler chicken flocks. It is associated with rapid growth and a high metabolic rate.

OCCURRENCE

Ascites occurs worldwide in rapidly growing broiler chicken flocks.

HISTORICAL INFORMATION

Ascites was first reported in 1968 in broiler chickens raised at a high altitude. However, the incidence of ascites caused by PHS, where broilers are grown at a low altitude, has increased over the past several years and coincides with genetic and nutritional improvements that resulted in better growth rate and feed conversion.

ETIOPATHOGENESIS

Four pathophysiological mechanisms are recognized to cause ascites: increased hydrostatic vascular pressure, decreased oncotic pressure, increased capillary permeability, and impaired lymphatic drainage. Although numerous chemical toxicities have been reported to cause ascites in broiler chickens through one of these mechanisms, the most common form of ascites in fast-growing broiler chickens is caused by increased hydrostatic vascular pressure.

Rapid growth, elevated metabolic rate, and therefore a high oxygen demand impose an increased workload on the heart. This, combined with the insufficient pulmonary capillary capacity of the modern broiler chicken, aggravates the pulmonary hypertension and further precipitates right ventricular hypertrophy.

Hypertrophy is soon followed by dilation, right ventricular failure, passive congestion, and then ascites. This process is accelerated in birds because of an anatomical particularity. The right atrioventricular valve is a muscular flap, an extension of the right ventricular wall. Any hypertrophy of the latter affects the valve and its apposition against the septum, facilitating venous regurgitation, passive congestion, and ascites.

CLINICAL SIGNS

Clinically affected broiler chickens are smaller than normal and depressed with ruffled feathers. Severely affected birds show abdominal distension with reluctance to move, respiratory distress, and cyanosis.

LESIONS

1. Hypertrophy and dilation of the right ventricle [Fig. 1: Ascites; AAAP] with or without accumulation of straw-colored ascitic fluid in the peritoneal cavities [Fig. 2: Ascites; NCSU], and a generalized passive congestion are characteristic of ascites secondary to PHS.

2. Hydropericardium, protein clots in the ascitic fluid, and a fibrotic liver [Fig. 3: Ascites; AAAP] may be present in chickens with chronic PHS.

3. Microscopic lesions show generalized passive congestion.
DIAGNOSIS

Macroscopic lesions are diagnostic.

If mortality in a flock is abnormally high, look for causes decreasing oxygen availability to the broiler chicken (poor ventilation, high altitude, concomitant respiratory pathology, etc.), or increasing oxygen needs (rapid growth, cold rearing temperature stimulating the metabolic rate).

Other pathological mechanisms can be involved in the development of ascites, and toxicities due to sodium, phenolic compounds, coal-tar derivatives, and dioxin, among others, might also be considered.

CONTROL

Lowering the oxygen requirement by slowing the metabolic rate will reduce, and if severe enough, prevent ascites. A variety of feed restriction and light programs have been used or recommended. The goal is to find a program that will maintain feed efficiency while reducing metabolic rate without increasing days to market.

TREATMENT

There is no treatment.

II. SUDDEN DEATH SYNDROME OF CHICKENS

DEFINITION

Apparently healthy fast-growing broiler chickens, mainly males, die suddenly after a short terminal wing-beating convulsion. Dead birds are found lying on their back. This is a common cause of “normal mortality” in a flock.

OCCURRENCE

This condition occurs from 1-8 weeks of age in most intensive broiler-growing areas of the world. The incidence in a flock varies from 0.5% to more than 4% in some cases. Sixty to 80% of the affected birds are males.

HISTORICAL INFORMATION

This syndrome has been recognized for 30 years and has been described as acute death syndrome, heart attack, flip-over, dead in good body condition, and lung edema.

ETIOLOGY

The cause is unknown but this condition affects highly performing broiler chickens. It is suggested that death is the result of ventricular fibrillation secondary to a possible imbalance of metabolites or electrolytes. It is classified as a metabolic disease and the incidence appears to be affected by genetic, environmental, and nutritional factors.

CLINICAL SIGNS

There are no premonitory signs. Large healthy broiler chickens will start to convulse and wing flap, and rapidly die lying on their back.
CARDIOVASCULAR DISEASES OF CHICKENS

LESIONS

Birds are in good body condition with a full digestive tract. There is red and white mottling of the breast muscle, the ventricles of the heart are contracted, and the auricles dilated with blood. Lungs might be congested secondary to postmortem blood pooling. There are no specific histopathologic lesions.

DIAGNOSIS

Dead birds appear healthy and there are no lesions except the findings described above.

CONTROL

Various feed and light regimens have been tried with little success in decreasing the incidence of sudden death without decreasing feed conversion.

TREATMENT

There is no treatment.

III. ROUND HEART DISEASE OF CHICKENS

This myocardial degeneration used to affect mature chickens (> 4 months of age) but has not been diagnosed in commercial poultry flocks for years. Birds die with a bilateral ventricular hypertrophy and dilation. Histopathology reveals myocardial fatty infiltration. The etiology is unknown.