Ascites (Pulmonary Hypertension Syndrome) in Poultry

Ascites is the accumulation of fluid inside the abdominal cavity as the consequence of increased pressure in the pulmonary arterioles.

Ascites results from an inability of the pulmonary vasculature to cope with increased oxygen requirements in metabolically challenged birds. Genetic predisposition, coupled with very rapid growth during the first weeks of life, often underlies this condition.

Occurrence

Ascites occurs worldwide, especially at high elevations, where the partial pressure of oxygen is reduced. However, the disease can also be present at sea level, especially in very rapid-growing birds (raised on diets that maximize growth rate) under conditions that increase metabolic rate or lead to difficulty in gas exchange (concomitant respiratory disease).

Clinical signs

Cyanotic comb may be the easiest clinical sign to spot (Figure 1). Wing veins may look very congested. Birds will be clearly reticent to exercise. Enlarged abdomen (containing ascitic fluid; Figure 2) is seen in advanced cases.

Pathogenesis

Gas exchange takes place in specialized tissue in the lungs. Here, blood capillaries in the lungs are in close contact with very thin air capillaries (the avian counterpart of mammalian alveoli). At this gas exchange surface, air and blood get extremely close and are separated only by extremely thin tissue. This intimate proximity allows gases to passively diffuse from air to blood and vice versa. As the red blood cells travel through the gas exchange zone, hemoglobin molecules contained within these cells release carbon dioxide (mostly in the form of bicarbonate) and bind oxygen. The blood concentrations of these two gases equilibrates almost perfectly with their concentrations in the air capillaries.

In a normal individual, blood exits the gas exchange zone with its hemoglobin fully saturated with oxygen. Under certain circumstances (exercise, extremely rapid growth, or extreme ambient temperatures), a larger supply of blood is needed to oxygenate the body. In mammals, pulmonary blood vessels can easily dilate to accommodate the extra blood flow. In chickens, this is not the case. Their pulmonary blood vessels are quite rigid, and increased blood flow leads to increased blood pressure. In non-compliant blood vessels, high blood pressure translates into increased speed of flow. The faster the blood flows through the gas exchange area, the less time it has to completely saturate the hemoglobin with oxygen (diffusion limitation; Julian, 1993; Wideman et al., 2007).

When blood with hemoglobin that is not completely saturated with oxygen reaches the systemic circulation, a series of
responses is initiated. Notoriously, vasoconstrictors in the pulmonary arterioles are released at a rate that overwhelms the production of vasodilators. Consequently, the diameter of the arterioles conducting the blood to the gas exchange surface is reduced. At the same time, the amount of blood pumped by the right ventricle tends to increase to compensate for the lack of systemic oxygen; if blood is to keep flowing, the pulmonary arterial pressure must go up. This is what marks the beginning of pulmonary hypertension syndrome (ascites). A positive feedback cascade develops: more oxygen is needed systemically, more blood is pumped, the right ventricle increases the pumping pressure to accomplish the task, blood velocity increases through the pulmonary arterioles, less time is available for gas exchange, more under-saturated blood reaches the left atrium, more vasoconstrictors are produced in the pulmonary arterioles, increasing the pulmonary pressure even further (Julian et al., 1987; Wideman et al., 2007; Lorenzoni et al, 2008).

If this cycle persists, the muscular walls of the right ventricle dilate to the point that the right atrio-ventricular valve cannot completely seal. This results in blood regurgitation (at high pressure) towards the cava vein during every diastole. Increased venous pressure is transmitted to the hepatic sinusoids (which normally work under very low pressure), which then degenerate. Plasma starts leaking through the degenerated blood vessels, followed by accumulation of the ascitic fluid in the abdominal cavity (Julian et al., 1987).

**Necropsy Findings**

In full ascites, the abdomen is distended and filled with a variable amount of ascitic fluid. The liver may be enlarged and is frequently covered with a layer of coagulated fibrin. Hydropericardium is also a common necropsy finding. One key finding is that the right ventricle is distended, giving the whole heart a flaccid appearance. Not every bird undergoing sustained pulmonary hypertension will have large amounts of ascitic fluid in the abdominal cavity. The amount of fluid is related to the chronicity of the events rather than their magnitude.

**Diagnosis**

Even in the absence of abdominal fluid, a careful necropsy can pinpoint ascites. In a dissected heart, the size ratio of the right ventricle to the left ventricle plus the cardiac septum must be < 0.28. Higher values indicate dilation of the right ventricle, which is indicative of sustained high pulmonary arterial pressure (ascites).

**Prevention**

Reduce growth rate. This can be achieved with feed restriction or by feeding a diet with low nutrient density. Manage ambient temperature closely. Avoid having birds outside their zone of thermal comfort. Birds panting or huddling together are indicative of hot and cold ambient temperatures, respectively. Increasing the dark period to 6-8 h can also have a positive effect. Control respiratory diseases and manage litter properly. High ammonia and dust can lead to inflammation of the airways, which will further tax the gas exchange process.

**References**


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Penn State College of Agricultural Sciences research and extension programs are funded in part by Pennsylvania counties, the Commonwealth of Pennsylvania, and the U.S. Department of Agriculture.

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