CARDIOVASCULAR DISEASE IN TURKEYS

Slide Study Set #24

A Continuing Education Program Prepared

By R. J. Julian

Ontario Veterinary College
University of Guelph, Guelph, Ontario, Canada

In Cooperation with the

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of

The American Association of Avian Pathologists

AAAP
382 West Street Road
Kennett Square, PA 19348

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Introduction

Abbreviations Used:

AV: atrioventricular
BW: body weight
LV: left ventricle
RA: right atrium
RV: right ventricle
RVF: right ventricular failure
STC: spontaneous turkey cardiomyopathy
TV: total ventricular weight

Cardiovascular disease is an important cause of death in commercial turkeys. Spontaneous turkey cardiomyopathy (roundheart), ruptured aorta and sudden death account for more than 50% of the "normal" mortality in male (tom) turkeys. These conditions may be directly related to rapid growth rate, although other factors may also be involved. The avian heart differs from the mammalian heart in that it is cone-shaped, has a thin right ventricle (RV) and thick left ventricular (LV) wall, with a thickness ratio of 1:4. A muscular flap, rather than a mammalian valve, separates the right atrium (RA) from the RV. This muscular flap is a continuation of muscle from the right ventricular wall. Other valves are similar to those found in the mammalian heart.

Slide 1 Avian heart cut in cross section at the free edge of the right atrio-ventricular (AV) valve to show the thick LV wall, a response to high blood pressure, and the thin RV wall. The RV has been opened and reflected to show the RA. The right AV valve can be seen folding in from the RV wall (top). AV valve can be seen folding in from the RV wall (arrows).

The anterior mesenteric artery of the fowl is different from its mammalian counterpart in that it has an outer longitudinal muscle as part of the adventitia. The thoracic aorta contains more elastin and less collagen than the abdominal aorta resulting in reduced compliance and distensibility and higher

blood velocity in the abdominal aorta. Furthermore, the abdominal aorta does not have a vasa vasorum. This increased resistance to blood flow combined with high blood pressure may explain the frequency of aortic rupture in turkeys. Birds have a renal portal system (10). In turkeys, if there is sudden failure of forward flow through the kidney, blood returning from the legs may pool around the lobules and on the ventral surface of the kidney, which in birds has no capsule. This occurs in sudden death from hypertrophic cardiomyopathy and occasionally in other cases of sudden death. Cardiovascular disease in turkeys is caused by mechanical and metabolic responses more often than by infectious agents.

Necropsy of the heart

Examine the heart and vessels, serous membranes and coelomic cavities *in situ* for changes in size, shape, colour, increased fluid, fibrin and urate crystals. Dilation and congestion of the atria and veins, particularly the sinus venosus and vena cava, suggest right valvular insufficiency. Remove the heart and transect it at the midsection (at the level of the free edge of the right AV valve). Look for hypertrophy, dilation or other abnormality. Open the heart with scissors by following the path of blood flow and examine the valves, endocardium, epicardium and myocardium. The heart can be separated into its various parts for heart and heart to body weight (BW) ratios. The normal heart:BW ratio in male turkeys at 20 wks is 0.00324 ± 0.0006 (heart 0.324% of BW). The normal RV to total ventricular weight (TV) ratio is 0.185 ± 0.03 (RV 18.5% of TV) (2).

Routine histologic examination is frequently limited to a mid-section across both ventricles, but should also include a longitudinal section of the RV, valve and atria and two sections of the LV through the chordae tendineae; one from the free wall including the atria and one from the opposite side. Gross lesions in the heart, pericardium and vessels should always be examined histologically.

RESPONSE TO VOLUME AND PRESSURE

Hypertrophic cardiomyopathy

The heart muscle responds to an increased workload, as all muscle does, by hypertrophy. A volume increase, which occurs with increased oxygen requirement, valvular insufficiency, septal defects, anemia, sodium toxicity, etc., causes hypertrophy in which the heart enlarges because of increased chamber volume. The ventricular wall does not become thicker but the mass of the ventricle increases. In this form of hypertrophy, sarcomeres are added in series and fibres do not become thicker. This stage of volume load or overload is present in racehorses but is rarely seen in adult turkeys. In poults it leads quickly to dilatory cardiomyopathy.

A pressure increase occurs most frequently because of increased resistance to flow as the result of stenosis/obstruction of arteries, arterioles or capillaries, increased blood viscosity or high blood pressure (hyertension). Systemic hypertension is common in turkeys. Hypertrophy, as the result of a pressure overload, causes thickening of the ventricular wall.

Slide 2 Hypertrophy of the RV wall caused by the pressure overload of pulmonary hypertension. Normal (N) above. These three hearts were from 12-wk-old turkeys with polycythemia (increasing blood viscosity) from hypoxia caused by severe aspergillus airsacculitis.

Hypertrophy of the LV wall (concentric hypertrophy) because of systemic hypertension, causes the chamber to become smaller during diastole. Stroke volume may become so small that the heart is unable to supply the blood flow required by the body. This may be the lesion that causes sudden death in turkeys (1,9).

Sudden death in turkeys, perirenal hemorrhage syndrome

This sudden death condition may start as early as 6 weeks but is more prominent at 8-18 weeks. It occurs predominantly in the larger more rapidly growing males that have high blood pressure. In many flocks it is the most important cause of mortality in this age group. It may also occur in breeder flocks and sudden death or a similar condition causes mortality in hens following insemination until they become accustomed to being handled.

The most prominent lesions are perirenal hemorrhage, an enlarged hemorrhagic spleen, a swollen firm liver, and marked pulmonary congestion and edema. All tissues and organs are congested, and in birds that have been dead for several hours, there may be edema in the subcutaneous tissue. Perirenal hemorrhage is a non-specific lesion and occurs in other conditions. There is left ventricular hypertrophy and death is likely because of circulatory failure from concentric hypertrophic cardiomyopathy.

Slide 3 Blood under the kidney (arrows) in the retroperitoneal area in a turkey that died from "sudden death".

Slide 4 Enlarged liver, lung and spleens from turkeys that died from "sudden death". Normal (N) above.

Slide 5 Marked pulmonary congestion and edema. Normal (N) on right.

Slide 6 Varicoses and congestion (arrows) in fat and subcutaneous tissue reflected from around crop in male breeder that died from "sudden death". Presence of feed in crop shows that this turkey had not been sick.

Slide 7 Cross-section of heart showing concentric left ventricular hypertrophy. (RV 12.7% of TV) the result of a pressure overload.

Histologic lesions have been described and include generalized congestion/hemorrhage with marked congestion of kidney, liver, spleen and lung (4). Edema is reported in the lung and kidney. An increase in proliferative arterial and arteriolar lesions has also been described (8).

Losses may be associated with activity such as moving or catching birds, which require increased cardiac output (stroke rate and volume). Slowing growth rate and reducing activity may prevent mortality.

Ruptured atria

Turkeys occasionally die suddenly from cardiac tamponade following rupture (blow-out) of the left atrium.

Slide 8 Heart from a turkey that died from cardiac tamponade. There is a small hole in the left atrium (arrow).

Slide 9 Heart cut longitudinally and blood clot from pericardial sac from turkey that died from cardiac tamponade (fixed in formalin before pericardial sac was opened).

Degenerative changes

Dilated cardiomyopathy describes a condition in which the ventricular chamber is enlarged and the ventricular wall is thinned. Because the heart is larger the ventricular mass is usually increased even though the wall is thin. Dilatory cardiomyopathy is a degenerative condition in which myocytes are lost because of anoxic, inflammatory, autoimmune or other insults. It may also be caused by a volume overload. The heart responds as to a volume overload, adding sarcomeres in series. In young poults, fibres may appear thin and long with many mitotic figures. Heart muscle mass increases as a percent of BW as the heart continues to dilate. The heart muscle loses its ability to dilate and contract (both of which require muscle energy) and becomes ineffective, resulting in

heart failure. Frequently the dilation also results in valvular insufficiency, particularly of the right AV valve, and is followed by increased portal pressure and ascites. Dilatory cardiomyopathy is common in young turkeys.

In the dilatory form of hypertrophic cardiomyopathy, reduced blood flow results in ischemic myocardial degeneration, focal necrosis, edema, fibrosis, valvular insufficiency, decompensation and terminal heart failure. Degenerative changes and scarring result in cardiosclerosis and is the stage that is most frequently described in the literature (9. Fig. 5.5; 5.7). The histologic lesions are caused by anoxia, myocardial cell death, edema and fibrosis.

Slide 10 Dilatory cardiomyopathy of right ventricle in a 9-wk-old turkey that resulted in right AV valvular insufficiency, increased portal pressure, ascites and liver lesions (the cardiohepatic syndrome). (8). The RV has been opened.

Spontaneous turkey cardiomyopathy (STC), roundheart disease in turkeys

STC is a dilatory cardiomyopathy that causes mortality in poults mainly between 1 and 4 weeks of age. STC causing heart failure was first described in turkey poults about 50 years ago (7,8). STC is related to early rapid growth and the incidence may be increased by hypoxia in the embryo or young poult, high Na^+ in feed or water, cold and other management or environmental factors (6). Insufficient cardiac myoglobin may be part of the pathogenesis (8). In the initial stages it may be caused by a volume overload resulting in anoxia of heart muscle. Degeneration of individual myocardial cells is present in the heart of poults with experimentally-induced STC before dilation becomes prominent. Furazolidone-induced cardiomyopathy is similar to STC (7). STC in very young turkeys (1-2 weeks) is characterized by marked ventricular dilation and heart failure most frequently of the right side of the heart. Pale liver, congested lungs, ascites and other lesions of right

ventricular failure (RVF) may be present. In turkeys that survive beyond two weeks, LV and biventricular dilation become more prominent. Reported microscopic lesions are inconsistent. Changes of terminal myocardial exhaustion are a response to the circulatory failure and ischemia of dilatory cardiomyopathy rather than the cause. After 4 weeks of age, affected turkeys develop increased fibroelastic and occasionally fibrocartilaginous tissue in the subepicardium and endocardium of the left ventricle. The lesion is probably secondary to ventricular dilation and edema.

Slide 11 STC in 13-wk-old turkey. The dilatory cardiomyopathy in this bird is left ventricular dilation which is the common form observed in mammals. The heart becomes enlarged and elongated. There is usually marked lung edema, but little evidence of RVF.

Slides 12 & 13 STC in 8-wk-old turkey. The dilation in this poult is mainly RV and the heart does not become elongated. Normal (N) on right.

Slides 14 & 15 STC in 16-day-old poults from a flock with 2% mortality from STC. The hearts were enlarged (arrows) and many of the poults had ascites. Several died from stress-associated hemorrhage into the digestive tract. Biventricular and LV dilation were present as shown in the fixed hearts cut in cross section. Normal (N) on right.

Slide 16 STC affecting LV of 18-wk-old males at processing. Normal (N) above. There were no other lesions in these toms, but they were below average weight.

INFLAMMATION OF THE HEART

Pericarditis occurs in turkeys that have generalized bacterial infections. It is common in *Escherichia coli* infection secondary to respiratory disease or septicemia and in fowl cholera. In

early lesions, there are many heterophils and fibrin. Subsequently, lymphoid cells and macrophages predominate. And, if the turkey survives, fibrous tissue and adhesions result (9. Fig. 5.13).

Focal epicarditis has been reported on the wall of the LV of turkeys at processing. It is said to be caused by trauma from the heart touching the breast bone as it beats (9).

Slide 17 Focal epicarditis near the tip of the LV of hearts from broilers at processing (arrows). The lesion in turkeys is similar. The RV has been removed for RV:TV ratio.

Endocarditis, epicarditis and pericarditis with hydropericardium, resulting in restrictive cardiomyopathy, are seen in individual turkeys at processing and have occurred as a flock problem at processing with 1-3% of the turkeys condemned. No etiologic agent has been identified. There may be an association with diet (high fat and canola or rapeseed meal and oil) (18). Subendocardial and epicardial fibrosis are the prominent histologic lesions and suggest that a circulatory problem causing edema has initiated the reaction (9. Fig. 5.2; 5.3; 5.4). Liver lesions are frequently present as well.

Slide 18 Pericarditis, epicarditis and hydropericardium, and hepatic fibrosis, in turkeys at processing.

Slide 19 Epericarditis (arrows) on surface of heart. The pericardium (P) has been removed.

Slide 20 There is fibrosis under the epicardium (arrow) with both fibrous tissue and fibrin (F) on the surface of the heart (x 50)

Slide 21 Fibrosis and cartilaginous metaplasia (C) under the endocardium (x 100).

Myocarditis accompanies viral and bacterial septicemia (9. Fig. 5.13; 5.16; 5.17). Focal necrosis of myofibers and infiltration of mononuclear cells occur when turkeys are infected with virulent strains of Newcastle disease and influenza viruses (9. Fig. 5.15; 12,14). Endocarditis is

occasionally seen in individual birds but may occur as a flock problem due to erysipelas infection. It is most frequently caused by *Streptococcus*, but it may also be due to *Staphylococcus, Pasteurella*, or other bacteria or fungi. The lesions occur most commonly on the left atrioventricular and aortic valves.

Nutritional (16) and toxic cardiomyopathy (7, 19) may occur in turkey as well as in other birds (9. Fig. 5.11).

PATHOLOGY OF THE BLOOD VESSELS

Pressure-induced vascular lesions

Hypertensive lesions in small arteries may be caused by damage to the endothelium and leakage of plasma or cells into the intima or media. The vascular tissue may respond to this damage by hyperplasia, collagen production or protrusion into the lumen. Pressure also increases the workload on the muscular media and this may evoke a stretch-induced hypertrophy as it does in cardiac muscle (13).

Fibromuscular dysplasia

Fibromuscular dysplasia is an unusual vascular lesion that has been described in turkeys. The lesion is characterized by cushions of smooth muscle and endothelial proliferation in the lumen of arteries and may be related to pressure-induced degenerative changes in the vessels (9. Fig. 5.12). The clinical significance of these lesions is unknown, but it has been suggested that they may be occlusive and cause ischemic degeneration or necrosis in tissues or organs (5, 8). However, there is no evidence that fibiomuscular dysplasia causes ischemia.

Slide 22 Fibromuscular dysplasia in artery in spleen of hypertensive turkey that died from "sudden death" (x 500).

Hypertrophic arteriosclerosis

Arteriolosclerosis is usually associated with increased intravascular pressure, although the lesions are similar to hypoxia-induced muscularization and medial hypertrophy in the lung. Turkeys are hypertensive and their arteries and arterioles, as well as those of meat-type chickens, frequently show medial smooth muscle and intimal endothelial hypertrophy and hyperplasia that may be a response to rapid growth and intravascular pressure. The coronary and pulmonary arterioles are frequently thickened by fibroplasia and vacuolation of the surrounding adventitial tissue, medial smooth muscle hypertrophy with vacuolation and what appears to be intimal hyperplasia. It is also possible that some of these so-called lesions are artifacts of contraction (9. Fig. 5.23).

Slide 23 Arteriolosclerosis in arteries of lung of 12-week-old male turkey (x 125)

Arteriosclerosis

Plaques or damage to the tunica intima may be described as arteriosclerosis (9. Fig. 5.20). Arteriosclerosis may be part of the pathogenesis of ruptured aorta (11,14,17).

Ruptured aorta (dissecting aneurysm)

Fast-growing tom turkeys over 6 weeks of age can bleed to death from a rupture of the aorta. The highest mortality usually occurs between 12 and 16 weeks. The cause is likely associated with systemic hypertension. Many turkeys are hypertensive and high-energy diets increase hypertension. This syndrome has caused mortality of up to 20% in some flocks of male turkeys but would be less than 1% in most flocks. An aneurysm may develop into a longitudinal split in the muscular aorta in the abdomen between the external iliac and sciatic arteries. This rupture results in massive abdominal hemorrhage. Occasionally, the rupture will occur in the thoracic aorta just dorsal to the heart or other locations. The pathogenesis of aortic aneurysm is poorly understood, but the lesion

usually develops in the tunica media in an area of the aorta where there is no vasa vasorum. It has been shown that liver levels of copper are low in some turkeys dying from aortic aneurysm. Copper is important in collagen synthesis. It is also possible that purely mechanical forces may cause ruptured aorta (and ruptured atria which occur occasionally in male and female turkeys and cause death by cardiac tamponade). In many cases there is no aneurysm or plaque in the aorta or atria (9. Fig. 5.27; 5.28).

Slide 24 Clotted blood in abdominal cavity and thoracic area of turkey that died suddenly from ruptured aorta. The rupture in this turkey was in the thoracic aorta just dorsal to the heart.

Slide 25 Ruptured aorta from 21-wk-old male turkey. Several birds died while being moved and loaded on the truck to go to the processing plant. The rupture is in the abdominal aorta close to the renal artery. Note normal difference in thickness of thoracic and abdominal aortic wall. Fixed tissue. Normal (N) above.

Slide 26 Ruptured aorta in 24-wk-old male turkey (x 250). There is no evidence of atherosclerosis.

Vasculitis has been reported in several systemic viral infections such as paramyxovirus, orthomyxovirus, Highland J, arbovirus (9) and immune mediated vasculitis (3). Local vasculitis occurs in aspergillosis (lung) and erysipelas (skin). (9. Fig. 5.30; 5.33)

Slide 27 Thombosis and vasculitis in wall of arteries (arrows) caused by pulmonary aspergillosis penetrating vessel (x 300).

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