THE AVIAN CARDIOVASCULAR SYSTEM

A Continuing Education Program Prepared

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Introduction

Cardiovascular disease is an important cause of death in commercial turkeys and meat-type chickens. Spontaneous turkey cardiomyopathy (STC) (roundheart), ruptured aorta and sudden death account for over 50% of the "normal" mortality in tom turkeys. Flip-over (sudden death syndrome) and pulmonary hypertension leading to right ventricular failure cause high losses in broiler and roaster chickens. In both chickens and turkeys these condition are related directly to growth rate. The diagnosis is usually based on history and gross examination.

Other cardiovascular diseases are rare in commercial and other poultry, waterfowl, pet and wild birds, but occurs occasionally because of infectious, nutritional, toxic or unknown insults. In these cases histology or other tests may be required for diagnosis.

Normal

The avian heart differs from the mammalian heart in that it is cone-shaped, has a thin right ventricle (RV) and thick left ventricle (LV) wall (1:4). A muscular flap rather than valves separate the right atrium and ventricle (6). The muscle flap is a continuation of muscle from the right ventricle wall. Hypertrophy of the right ventricle results in thickening of the valve and valvular insufficiency.

Slide 1 avian heart cut in cross section at the free edge of the right AV valve to show the thick LV wall, a response to high blood pressure, and the thin RV wall. The right AV valve can be seen folding in from the the RV wall (top) (see also slide # 5).

Other valves are similar to the mammalian heart. A cartilaginous plaque is found in the wall of the aorta where the major vessels leave the heart (1. Fig. 5.1). Lymphoid foci and foci of extramedullary granulopoiesis are common in the myocardium of broilers (1. Fig. 1.2)..

Birds have a renal portal system (6) and the kidney has no capsule. 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.

Necropsy of the heart

Examine the heart and vessels, serous membranes and coelomic cavities in situ for changes in size, shape, colour, 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, endo-, epi- and myocardium. The heart can be separated into its various parts for heart and heart to body weight ratios. The normal heart-to-body weight ratio in

broilers decreases with age from 0.0082 at day 9 to 0.0047 at day 42 (heart 0.47% of BW at day 42). The normal right ventricle-to-total ventricle ratio is 0.20 ± 0.03 (RV 20% of TV).

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 included.

Congenital defects

A variety of anomalies have been described in the cardiovascular system in poultry, but only intraventricular and intraatrial septal defects occur with any frequency (4). Most congenital defects result in death from right ventricular failure (RVF) in the first 2-3 weeks (5).

Slide 2 Intraventricular septal defect in 16-d-old broiler. The broiler had ascites from RVF following a volume overload on the RV and pulmonary hypertension. Note enlarged right ventricle wall.

RESPONSE TO VOLUME, PRESSURE AND INJURY

Circulatory problems or injury causing edema in the myocardium or under the epi- or endocardium will result in fibroplasia and fibrosis (1. Fig. 5.2, 5.3, 5.4). Cardiac lipidosis is seen occasionally (1. Fig. 5.6).

Slide 3 Epicardial fibrosis secondary to RVF which caused chronic ascites and hydropericardium.

Hypertrophic cardiomyopathy

Response to volume and pressure. 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, anaemia, sodium toxicity, etc., causes hypertrophy in which the heart enlarges because of increased chamber volume (eccentric hypertrophy). In chickens, a volume overload may quickly lead to a pressure load on the RV. The ventricular wall does not become thicker but the mass of the ventricle does increase. In this form of hypertrophy, sarcomeres are added in series and fibres do not become thicker.

A pressure increase occurs most frequently because of increased blood flow, but also because of increased resistance to flow as the result of constriction, stenosis or obstruction of arteries, arterioles or capillaries, or because of increased blood viscosity. Hypertrophy as the result of a pressure overload causes thickening of the ventricle wall. In pressure-induced hypertrophy of the LV the chamber becomes smaller during diastole (concentric hypertrophy). Stroke volume may become so small that the heart is unable to supply the blood flow required by the body. This hypertrophy may be the cause of sudden death in turkeys.

Slide 4 Turkey heart cut in cross section from turkey that died from sudden death. The left ventricle is hypertrophied.

In pulmonary hypertension syndrome (PHS) in broilers both a pressure and a volume overload are present in the right ventricle until moderate valvular insufficiency occurs and pressure is reduced. Ascites secondary to right AV valvular insufficiency resulting in increased portal pressure is the most frequent cause of ascites in poultry. PHS causes ascites because of increased portal pressure following valvular insufficiency and RVF secondary to pulmonary hypertension (PH). PH caused by increased blood flow or increased resistance to flow in the lung results in right ventricular hypertrophy (RVH) as measured by RV:TV. (4).

Slide 5 Broiler hearts cut longitudinally to show pressure-induced hypertrophy of RV (heart on left) and both pressure and volume-induced changes in other hearts (normal on top). Note hypertrophy of right AV valve. There is atrophy of the LV caused by the hypoxia of RVF in the 2nd and 4th hearts.

Sodium intoxication results in PHS very rapidly in young chicks. The pathogenesis of the heart lesion in sodium toxicity in poults is not clear and is said to be similar to STC. It is likely a volume-induced dilatory cardiomyopathy: In both chicks and poults subcutaneous edema may occur, and testicular edema has been reported in chicks.

Degenerative changes

Dilatory cardiomyopathy describes a condition in which the ventricular chamber is enlarged and the ventricular wall is thinned. Because the heart is larger, ventricle mass is usually increased. Dilatory cardiomyopathy is a degenerative condition in which myocytes are lost because of hypoxic, inflammatory, autoimmune or other insults. The heart responds as to a volume overload, adding sarcomeres in series. In young poults which have dilatory cardiomyopathy, fibres may appear thin and long with many mitotic figures. Heart muscle mass increases as a % of body weight as the heart continues to dilate. The heart muscle becomes stiff with connective tissue and collagen and loses its ability to dilate and contract (both of which require muscle energy) and becomes ineffective, resulting in heart failure. Frequently, the dilation results in valvular insufficiency, particularly of the right AV valve and is followed by increased portal pressure and ascites. Hydropericardium and fibrous epicarditis may be present. Dilatory cardiomyopathy is common in turkeys. It is rare in chickens but is seen occasionally in male breeders.

Slide 6 Dilatory cardiomyopathy in 14-d-old poults. RV, LV and biventricular dilation are present. Normal hearts on right.

In both hypertrophic and dilatory cardiomyopathies interference with blood supply may result in myocardial degeneration, focal necrosis, edema, fibrosis, valvular insufficiency, decompensation, muscle atrophy and terminal heart failure. Degenerative changes and scarring

result in cardiosclerosis and is the stage that is most frequently described in the literature. The histologic lesions are caused by anoxia, myocardial cell death, edema and fibrosis (1. Fig. 5.5; 5.7)..

Endocardiosis

Nodules on the endocardium of chickens are common. They are most common on the chamber side of the RV valve about 3 mm from the free edge. The nodules consist of loose connective tissue and amorphous ground material and are similar to myxoid degeneration of the valve in humans (1. Fig. 5.8). They have been associated with right heart failure but are probably stretch-induced as a result of valvular insufficiency rather than the cause of the insufficiency. The incidence is increased in chickens raised at high altitude and in those with PHS.

(Slide 7) Broiler hearts separated for RV:TV ratio, showing endocardial side of RV and right AV valve. The 2 hearts on the right are from broilers with PHS. Normal on left. The RV in the centre has endocardiosis on the right AV valve. Note the white plaque of epicardial fibrosis on the tip of the heart on top right.

Roundheart disease in chickens

Roundheart disease has been used to describe distinct entities in chickens and turkeys. It has also been used for right ventricular failure which is discussed under ascites.

In chickens, roundheart disease affects birds older than 4 months and is characterized by sudden death. Hearts of affected chickens are pale and enlarged, with hypertrophy confined to the left ventricle.

(Slide 8). Chicken heart with round heart disease. The apex of an affected heart may be dimpled. Note marked hypertrophy of the left ventricle, normal on right (slide courtesy of Dr. C. Riddell).

The fibers throughout the myocardium are swollen and granular and contain fine vacuoles (1. fig. 5.9). In severe lesions, the vacuoles may coalesce, producing an apparent empty space around the nucleus and leaving a prominent cell membrane around the periphery of the fiber. The vacuoles and resulting empty spaces represent fat.

Furazolidone-induced cardiomyopathy

Excessive amounts of furazolidone in the diet will produce cardiac dilation and ascites in chickens, ducks and turkeys. Histologic lesions reported are similar to STC and are probably secondary to tissue hypoxia caused by dilation. Myocytolysis occurs as an early lesion prior to dilation in the myocardium of furazolidone-poisoned ducks (8) and STC (1. Fig. 5.10). This myocytolysis is the most likely cause of dilatory cardiomyopathy.

Cardiomyopathy and ascites have been produced by feeding poultry high levels of rapeseed

oil containing erucic acid. Erucic acid accumulates in the heart of birds, resulting in fatty degeneration of the myofibers, necrosis and fibrosis. The associated edema is presumably the result of heart failure, but it may also be due to hypoproteinemia secondary to liver damage.

A cardiomyopathy has been reported associated with potassium deficiency causing sudden death in hens near point of lay. The hearts are enlarged with both dilation and hypertrophy as occurs in PHS (7).

Ascites and hydropericardium have been described as prominent features in the toxic fat syndrome (dioxin toxicity) and chlorinated biphenyl toxicoses. Carbolinium toxicity also caused hydropericardium.

Slide 9 Broiler with ascites and prominent hydropericardium and caused by toxic fat syndrome.

In selenium/vitamin E-deficient birds, myopathy in skeletal, gizzard or intestinal muscle is generally more remarkable than that in the myocardium. However there may be heart lesions recognized as white streaks or patches and associated with hydropericardium. Metals (cobalt, lead), chemicals and poisonous plants (Cassia, Crotoloria etc.) may cause myocardial damage).

Slide 10 Myocardial degeneration and calcification of heart muscle fibres in duck that died from heart damage caused by selenium deficiency (1. fig. 5.11).

Flip-over; sudden (acute) death syndrome; death in good condition

The term sudden death syndrome has been used to describe well-fleshed broiler chickens that die suddenly with food in the gastrointestinal tract. There are no diagnostic lesions, but generalized congestion of the lung and dilation of the atria with constriction of the ventricles are significant. The deaths have been attributed to "heart attacks" but are probably the result of ventricular fibrillation.

Slide 11 The diagnosis of flip-over is based on circumstantial evidence. At necropsy there must be strong evidence the broiler was normal, healthy and eating before it died. The intestine must contain ingesta and the heart ventricles must be contracted (in fresh specimens).

INFLAMMATION OF THE HEART

Pericarditis is frequently found in birds that have generalized bacterial infections. In commercial poultry it is particularly common in *E. coli* infection secondary to respiratory disease. In early lesions there is fibrin and many heterophils are present; later lymphocytes and macrophages predominate and if the chicken survives, fibrous tissue and adhesions result.

Slide 12 Septic pericarditis in broiler hearts. The pericardium is loosely or tightly adherent to the peicardium depending on the age of the lesion. Fibrin is usually yellow but

becomes pale as fibrosis occurs.

Slide 13 Fibrin and inflammatory cells on pericardium of broiler (x 125).

Non-septic pericarditis occurs in birds with hydropericardium (1. figs. 5.2, 5.3, 5.4). In broilers and heavy turkeys focal fibrinous epicarditis, occasionally with pericardial adhesion is a frequent lesion near to the tip on the antero-ventral side of the left ventricle where it is close to the sternum. This white area of fibrosis on the epicardium is made up of proliferating and mature fibroblasts with few inflammatory cells (1. fig. 5.14)

It likely occurs secondary to localized subepicardial edema as the result of trauma when the beating heart contacts the sternum.

Slide 14 Focal pericardial adhesion on broiler chicken hearts. The RV has been cut off the heart on the left. See also slide 7.

Visceral urate deposits (visceral gout) on serous membranes may be confused with inflammatory exudate. They are most prominent on the heart and kidney.

Slide 15 Visceral urate deposits secondary to hyperuricemia from urate nephrosis (caused by dehydration) or other kidney disease may resemble pericarditis. The chalky deposits on the epicardium are gritty to touch (particularly if scrapped lightly with a scalpel blade).

Myocarditis accompanies many viral and bacterial infections of birds. Focal necrosis of myofibers and infiltration of mononuclear cells occur when chickens are infected with virulent strains of Newcastle disease and influenza viruses (1. Fig. 5.15). Similar microscopic lesions associated with intranuclear inclusion bodies have been described as a prominent feature of Derzsy's disease in goslings. Myocarditis is a prominent feature of the hydropericardium syndrome caused by adenovirus infection in broilers in Pakistan and other countries. There are also isolated reports of myocarditis caused by adenovirus, parvovirus, avian leucosis virus and other virus agents.

Slide 16 Myocarditis causes hemorrhage and pale streaking of myocardium. Hydropericardium is usually prominent. Similar gross lesions may occur with a variety of toxic and degenerative myocarditis, making diagnosis difficult.

Granulomatous myocarditis occurs in chickens with *Staphylococcus* and *Salmonella pullorum* infection (1. Fig. 5.17, 5.18) and in various birds infected with miscellaneous protozoan species. Fungal and tubercular granulomas also occur. Although the gross appearance of granulomas and granulomatous inflammation in the heart may resemble neoplasia, the microscopic appearance is usually quite diagnostic.

Slide 17 Fungal granules in heart of broiler (x 120). See slide # 28, for gross appearance.

Endocarditis is usually seen in individual birds but may occur as a flock problem. It is most frequently caused by Streptococcus but it may also be due to *Staphylococcus, Pasteurella,* or other bacteria. The lesions occur most commonly on the left atrioventricular and aortic valves. Endocarditis is commonly associated with infarcts in liver, spleen, heart and brain or if on the right AV valve with ascites.

Slide 18 Endocarditis of left AV valves (yellow mass on valves). Heart cut longitudinally in midline showing left AV and aortic values and right AV vales.

Slide 19 Endocarditis of left AV valves and septic infarcts in organs.

Slide 20 Endocarditis of right AV valve (yellow foci attached to muscular valve) causing valvular insufficiency. A volume overload RVH, RVF and ascites.

PATHOLOGY OF THE BLOOD VESSELS

Arteriosclerosis

Arteriosclerosis is common in many different types of birds. The early lesions consist mainly of collagen and connective tissue. In older birds foamy cells, extracellular lipid, cholesterol and calcification become incorporated into the plaques which then can be considered true atheromas (1. Fig. 5.21).

Slide 21 Arteriosclerosis of pulmonary artery of broiler.

The Marek's virus has been shown to induce similar vascular lesions and hypertrophic arteriosclerosis of unknown etiology is seen frequently in chickens and other birds (1. Fig. 5.25).

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 (1. Fig. 5.12).

Arteriolosclerosis (hypertrophic arteriosclerosis)

Arteriolosclerosis is usually associated with increased intravascular pressure, and the lesions are similar to hypoxia-induced muscularization and medial hypertrophy in the lung. The coronary and pulmonary arterioles may be thickened by fibroplasia of the surrounding adventitia tissue, medial smooth muscle hypertrophy and what appears to be intimal hyperplasia. It is also possible that some of these so-called lesions are artifacts of contraction.

Slide 22 Hypertrophic arteriosclerosis in pulmonary artery 12-wk-old male turkey (x 125)

Slide 23 Hypertrophic arteriosclerosis of unknown etiology in lung of meat-type chicken (x 300)

Slide 24 Hypertrophic arteriosclerosis in artery on peritoneal surface of intestine in a Canada goose (x 50)

Mineralization

Hyperuricemia results in focal uric acid crystal deposition in organs and tissues such as the wall of blood vessels. Necrosis occurs in these foci and mineralization may be present (1. Fig. 5.34)..

Vitamin D_3 toxicity causes mineralization in vessel walls as well as in kidney tubules. Chronic kidney disease may produce similar lesions.

Slide 25 Mineralization of artery walls in the proventriculus secondary to amyloidosis of the kidney (x 125).

Occasionally, dystrophic calcification will occur in the lung following inhalation of dicalcium phosphate or with moderate Vitamin D_3 overdose.

Ruptured aorta (aortic aneurysm)

Ruptured aorta may be associated with arteriosclerosis (1. Fig. 5.27; 5.28). It is also possible that purely mechanical forces (high pressure, poor strength collagen associated with low copper) may cause ruptured aorta (and ruptured atria) (5).

Emboli and thrombi

Thrombosis of vessels in birds may be secondary to bacterial emboli causing vasculitis (1. Fig. 5.29; 5.30). Fat and cartilage emboli have been reported without associated pathology. Fibrin thrombi may be found in capillaries of birds dying from systemic disease and in the vitamin E/selenium related microangiopathy of avian encephalomalacia.

Slide 26 Fibrin thrombi in capillaries of cerebellum in mild avian encephalomalacia.

Vasculitis

Vasculitis can be caused by a variety of chemical and physical stimuli from within or outside the body. Lead poisoning and mycotoxins are reported to cause vasculitis but most vascular lesions are the result of microbiological toxins, inflammatory mediators and cells (heterophils thrombocytes, basophils) or immune mediated damage. Endothelial cells in birds are phagocytic and may ingest microorganisms which then initiate vasculitis or obstruct capillaries (1. Fig. 5.35). Bacterial thrombi and some endotoxins cause vascular damage. Vasculitis may be present in pneumonic and encephalitic aspergillosis and in some cases of Mycoplasma synoviae infection (1. Fig. 5.31; 5.32;

5.33).

Slide 27 Aspergillus infection causing thrombosis and vasculitis in 10-d-old broiler (x 50).

Various types of immune mediated vasculitis have been reported or suspected. Marek's virus and mycoplasma induced vasculitis be immune mediated. Necrotic hemorrhagic hepatitis (hepatitis-splenomegaly of cage layers) frequently has vascular lesions in veins in the liver that may be immune mediated or amyloid associated (1. Fig. 5.37)

NEOPLASIA

Endothelial tumors (hemangioma, angiosarcoma, hemangioendothelioma) occur sporadically in chickens but can occasionally cause high mortality in a flock. They can be caused by the leukosis/sarcoma viruses. Hemangiopericytoma is described occasionally. Marek's disease lymphoid tumors are the most common tumors of the chicken heart. Histiocytic sarcoma, lymphoid tumors due to REV, fibrosarcoma and rhabdomyosarcoma have been reported in the heart of birds.

Slide 28 Marek's disease tumors in heart of Leghorn pullet. These lesions might resemble pullorum disease or granulomas caused by other infection.

Slide 29 Hemangiomas in skin of legs of Barred Rock chicken. Tumors were also present in the feathered areas of the skin.

Slide 30 Hemangioma in skin (x 30).

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