Spontaneous Coronary Arteriolsclerosis in Broiler Chickens
Toshiaki MASEGI, Katsuya SATO, Masashi KAWADA, Tokuma YANAI, and Katsumoto UEDA
Department of Veterinary Pathology, Faculty of Agriculture, Gifu University, I–1 Yanagido, Gifu 501–11, Japan
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ABSTRACT. Arteriolsclerotic changes were found in 202 out of 203 randomly collected heart specimens of broiler chickens. These chickens were reared in a poultry farm and killed at a poultry meat processing plant at 60 days of age with healthy appearance. Cellular intimal thickening due to a proliferation of smooth muscle cells of small arteries or arterioles were found mostly in the innermost layer of the left ventricle. Severity of the thickening varied from slight to severe. In 2 cases, myocardial necrotic changes were found in the proximity of severely affected small arteries and arterioles.—KEY WORDS: arteriolsclerosis, broiler chicken, intimal thickening.


Although vascular sclerotic lesion in poultry has been studied extensively [1, 5–7], few reports have been published on arterial changes of broiler chickens. We previously noted that arteriolsclerotic changes were frequently found in the myocardium of broiler chickens that had died or had been culled during growth [2]. In the present paper we describe a high prevalence of arteriolsclerotic changes in the heart vessels of commercial broilers obtained from a processing plant. Broiler chickens were examined from a farm which reared about 4500 Arbor-Acre breed chickens from September to November, 1989. There was no history of acute communicable diseases or overt intoxications during the rearing period in the flock. Chickens were sent to the processing plant at 60 days of age in November 1989, and 203 hearts were obtained randomly. Information on the sex and other gross lesions of their organs or carcasses was not available.

After examination for gross changes, the hearts were fixed in 10% formalin and cut transversely at three levels: the base, middle and apex of the ventricles. Histologic sections were obtained from these cut planes after embedding in paraffin. Sections were stained with hematoxylin-eosin (H.E.), Van Gieson, Gomori’s aldehyde-fuchsin, or phosphotungstic acid hematoxylin. Recording the degree of severity of arteriolsclerosis was made as described previously [2]. The most severely affected artery was selected from each heart under a microscope, and the figure of the transverse section of the artery was illustrated on a tracing paper (40 g/m²) at x 100 magnification using a magnifying apparatus. The paper was cut along with the line of internal elastic membrane and inner surface of the endothelial lining. And then, the pieces of paper were weighed. Occlusion rate was figured out by percentage of area of lumen to area inside of the internal elastic membrane.

All the hearts had well-developed epicardial adipose tissue around coronary vessels. Pericardial fluid was increased slightly in some cases. In 17 cases the right atrial cavity was moderately dilated. Histologically, varying degrees of arteriolsclerotic changes were found in 202 of 203 samples. Affected arteries were those less than 200 μm in diameter in the cross sections, and several to more than 10 affected ones were found per section. The vascular changes ranged from very slight edematous swelling of the endothelium which was found only in a small number of cases to vascular thickening due to increased numbers of smooth muscle fibers (Fig. 1) in small arteries and arterioles located in the myocardial interstitium. There was no marked increase of collagen or elastic fibers in the majority of thickened vessels. According to the morphometry performed on every cardiac samples, 28 cases (13.8% of the total) had occlusion rate of less than 50% (Fig. 2). The majority (117 cases; 57.6%) had an occlusion rate of between 51 to 90% (Fig. 3). In 57 cases, an occlusion rate of more than 91% (Fig. 4) was seen, comprising 28.1% of the total. Seven cases in the severe lesion group had completely obstructed arterioles. As shown in Fig. 5, these arteriolsclerotic changes were found mostly in the innermost muscle layers surrounding the left ventricle, and fewer numbers of the right ventricle in the interventricular septum were affected.

Myocardial scarring foci were found in two cases. Scars were found near the apex of the heart and were accompanied peripherally with myocardial necrotic areas. Heterophil infiltration in the interstitium of the myocardium was found in 135 cases, and fibrous thickening of the

Fig. 1. Cellular intimal thickening of a myocardial small artery. H. E. stain, × 250.
Fig. 2. Slight degree of arteriosclerosis (occlusion rate of less than 50%) in a myocardial small artery. Elastica Van Gieson stain, × 330.

Fig. 3. Moderate degree of arteriosclerosis (occlusion rate, 50–90%) in a myocardial small artery. Elastica Van Gieson stain, × 330.

Fig. 4. Severe degree of arteriosclerosis (occlusion rate of more than 90%) in a myocardial small artery. Elastica Van Gieson stain, × 330.

Fig. 5. Location of myocardial small arteries showing arteriosclerotic changes. Approximate locations of the affected arteries observed in all cases were spotted in a schema at the three examined levels of the heart.
pericardium in 45 cases. Prevalence and site of the latter two inflammatory lesions had no relation to the severity and location of the small arterial and arteriolar changes. However, the myocardial scar lesion was located in close proximity to the severely affected small arteries and arterioles.

Intimal thickening of the myocardial small arteries and arterioles in the present cases closely resembled that described as an early change of coronary arterial lesions in chickens and turkeys [3], and in experimentally produced arteriosclerosis of cockerels [4]. The moderate to severe degree of arteriolosclerotic changes in the present cases corresponded to the lesions found in 9-week-old cases of our previous study [2]. The incidence of intimal thickening in the present cases (99.5%) was greater than that in the previous study (71.5% average of various aged birds and 84.0% in 9-week-old birds). Broiler chickens used in the previous investigation were growth-retarded while those in the present cases were well-nourished. The high body weight-gain in the present groups presumably relates to the higher incidence of the lesion in the present than in the previous cases. In both groups, intimal lesion was limited to intimal thickening. Fibrous thickening was rarely seen. This is probably because the age of chickens was only up to 60 days. A point of interest is the topography of preferential site of these affected small arteries and arterioles in the myocardium. Affected small arteries and arterioles were concentrated around the left ventricle. Broiler chickens which are expected to grow fast and reach body weight of around 3 kg within about 60 days may require blood circulation beyond capacity of their heart, and this situation may trigger the small arterial and arteriolar lesion of the left ventricle. The overworking load of the heart relevant to the body weight may increase with age. This consideration on the cause of myocardial small arterial and arteriolar lesion may coincide with the age-related incidence of the lesion and also higher incidence in healthy broilers than that in growth-retarded ones. The present study disclosed that almost every broiler chickens develop myocardial arteriolosclerotic lesion by the age of 60 days, although we cannot explain the reason of presence of exceptional ones spared the lesion at this age.

Another interesting finding in the present study is the association of myocardial necrotic lesion with severe arteriolosclerotic changes, although the association was only found in two cases. Similar necrotic changes of myocardium associated with intimal thickening were seen in the previous study [2].

REFERENCES