OBSERVATIONS ON PATHOLOGICAL ALTERATIONS ASSOCIATED WITH SPONTANEOUS CARDIOMYOPATHY OR "ROUND HEART DISEASE" IN IMPORTED BREEDS OF TURKEY POULTS

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ABSTRACT
Spontaneous cardiomyopathy or round heart disease of turkeys was noticed in the imported breeds of heavy broad-breast turkey poults breed (B6 & T9). Most deaths from the disease were detected during the first 3 weeks of life. The mortality rate was ranged from 5 to 15 %. The most sticking and consistent lesion found at necropsy was enlargement and distortion of the heart due to dilatation of both ventricles. Ascites and hydropericardium were present in some cases. Lungs were congested and edematous. The liver, intestine, spleen and kidneys occasionally showed evidence of congestion. Microscopically, congestion, degeneration and vacuolation of myofibers resulting in damage and disappearance of some muscle fibers were markedly seen in the heart. The livers of the affected turkeys revealed portal fibrosis, bile ductal hyperplasia, hepatocytic vacuolation and intracytoplasmic eosinophilic globules in the hepatocytes. The histopathological changes in the lungs, intestine, spleen, kidneys and brain were also recorded. Moreover, the pathogenesis of the condition in relation to the pathological findings were discussed.
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INTRODUCTION

Circulatory disturbances in turkeys are likely to become increasingly prevalent because of economic need to continue to produce fast-growing breeds of turkeys. The consequences of breeding for rapid growth have undoubtedly affected the occurrence of circulatory-related mortality in commercial turkey flocks. Spontaneous turkey cardio-First Scientific Conference of Fac. Vet. Med., Moshtohor, Sept. 1-4, 2004, Benha- Ras Sedrmyopathy, also known as round heart disease, is a prevalent circulatory disturbance affecting turkeys particularly raised at moderate to high altitudes (9). A condition in turkeys with enlarged and rounded hearts was first described by Magwood and Bray in 1962 (16). It has continued at a low incidence since 1962 (2,4), but over the past several years the incidence has been increasing and occasionally may be quite high (20,24). Since then possible genetic, management factors and nutritional causes of the round heart syndrome have been implicated (4, 14, 15,20,21,) . Recently, round heart disease was noticed in turkey farms of Egypt associated with the imported breeds of turkey poults, causing sudden death and high mortality. Therefore, the purpose of the present study was to describe the pathological changes accompanied with spontaneous cardiomyopathy in the Egyptian turkey farms.

MATERIALS AND METHODS

Five outbreaks of spontaneous cardiomyopathy or round heart disease of turkeys has occurred in the imported breeds of heavy,broad breast turkey poults (B6&T9). The diseased and dead birds were allowed for post mortem examination. Tissue specimens were collected from different body organs (heart, liver, lungs, kidneys, intestine, spleen and brain) and fixed in 10 % neutral buffered formalin. Paraffin sections were prepared at 4-5 microns and stained by hematoxylin and eosin for histopathological examination. Periodic Acid–Schiff (PAS) stain was used for glycogen demonstration according to Drury and Wallington (8). Moreover, routine bacteriologic culture from the affected birds revealed negative results.

RESULTS

Signs &Mortality: Most deaths from round heart disease were detected during the first 3 weeks of life, with mortality peaking at the end of the 2nd week .The mortality rate was ranged from 5 to15%. After 3weeks of age, mortality was sporadic. The majority of poults died suddenly and did not manifest any premonitory signs , but some had ruffled feathers, drooping wings and a general unthrifty appearance.
**Gross lesions:** The most striking and consistent lesion found at necropsy was enlargement, distortion and discoloration of the heart (Fig.1). Enlargement of the heart was due to dilation of both ventricles particularly the right ventricle which was more severely affected than the left one. Cardiac musculature was pale and coronary vessels were markedly distended with blood. The normal tapered appearance of the heart was lost and the apex presented a blunt or rounded appearance (Fig.2). The pericardial sac was mostly distended with amber-coloured transudate and ascitic fluid was present in some cases. In older pouls, the pericardium was thickened grayish white and the enlarged hearts were due to marked hypertrophy of the ventricles in addition to dilatation (Fig.3). Lungs were often oedematous. The examined livers were enlarged and congested and the intestines were hyperaemic and the mesenteric blood vessels were markedly dilated and engorged with blood (Fig.4). Moreover, spleen and kidneys sometimes showed evidence of congestion or often appeared unchanged.

**Histopathology:**

**Heart:** The histopathological examination of the heart revealed congestion of myocardial blood vessels and intramuscular capillaries (Fig.5). Focal areas of haemorrhage and hyalinization of some cardiac muscle fibers evidenced by loss of cross striation and pyknosis of the nuclei were seen (Fig.6). Focal areas of coagulative necrosis of some muscle fibers were also detected (Fig.7). The main microscopic lesions in heart muscle were vacuolation of the myofibers (Fig.8). This lesion frequently involved perivascular and subendocardial tissue (Fig.9). In more advanced cases, some muscle fibers had disappeared and were replaced by the vacuoles (Fig.10). Moreover focal areas of fibroblastic proliferation and leucocytic aggregations mainly lymphocytes and macrophages were also detected in the myocardium in few cases (Fig.11). The pericardium in some examined cases was thickened due to oedema and leucocytic infiltration mainly lymphocytes.

**Liver:** The central, portal veins and blood sinusoids were congested with blood. Clear intracytoplasmic vacuoles were commonly seen in the hepatocytes (Fig.12). Intracytoplasmic eosinophilic globules were also detected in the hepatocytes in many cases (Fig.13). These globules stained dark red with PAS stain (Fig.14). Small focal areas of coagulative necrosis were observed in the hepatic parenchyma in few cases (Fig.15). The portal areas in some cases revealed fibrous tissue proliferation and congestion of the portal veins with hyperplasia of the biliary epithelium forming new bile ductules (Fig.16).
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Lungs: Microscopical examination of the lungs revealed congestion of the interstitial blood vessels and perialveolar capillaries. Oedema of the peribronchiolar and interlobular connective tissue with lymphatic dilatation were characteristic (Fig.17). Perivascular oedema was also prominent. Moreover, eosionophilic exudate in the lumen of some bronchioles and small focal lymphocytic aggregation in their wall were also detected (Fig.18).

Kidneys: the renal blood vessels and intertubular capillaries were dilated and ingorged with blood in some of the examined cases. Intertubular hemorrhages were observed in few cases (Fig.19). Moreover, most cases had degeneration of epithelial cells of the renal tubules represented by vacuolar and hydropic degeneration.

Intestines: The mucosal and serosal blood vessels and capillaries were dilated and filled with blood. Desquamated epithelial cells mixed with lymphocytes and eosinophilic exudate were present in the lumen of the small intestines in some cases.

Spleen: Congestion of the splenic blood vessels, focal areas of extravasated blood and lymphoid depletion were detected in the spleen in some cases (Fig.20).

Brain: Congested blood vessels, neuronal degeneration and status spongiosus of the cerebellum were also seen in some cases.

DISCUSSION

Since little is known about round heart disease, especially basic facts, the objective of this study was to search for lesions in the heart and other organs in turkeys showing the condition. To our knowledge this is the only report on the occurrence of round heart disease of turkeys in Egypt. Although the incidence of this disease in the field is unknown, communication with poultry scientists and veterinarians would suggest that it is fairly widespread. Perhaps two facts should be considered here, first, because the disease occurs during the first few weeks after hatching and second, because mortality is generally low, most pouls that die from this disease may be discarded without a postmortem examination. The poultryman probably considers them as normal poult mortality and does not become concerned since the economic loss is primarily the initial cost of the poult. For this reason the disease may be more widespread than is realized.

The exact etiology of spontaneous cardiomyopathy is unknown. A genetic influence was demonstrated by breeding trials (1,4). Clinical observations indicate that the incidence of spontaneous cardiomyopathy is increased at high altitude and with cold weather (9). Raising turkeys under hypoxic condition resulted in a high incidence of
spontaneous cardiomyopathy (14). Moreover, some outbreaks of the condition have been associated with hypoxia during incubation of the eggs (3). It is probable that an increased oxygen requirement associated with rapid growth and cold may increase the incidence of spontaneous cardiomyopathy (12). The production of cardiomyopathy in turkey poults by oral administration of furazolidone was studied (6,10,11). Furazolidone resulted in changes in enzymes, contractile proteins and membranes in the myocardium, consistent with a role of tissue hypoxia in the cardiac lesions (18). Also, round heart disease in turkey poults has been associated with high levels of dietary salt (15). Myocardial changes were similar to furazolidone-induced cardiomyopathy. A mechanism was suggested where sodium ions may cause hypertension and also influence the electrical contractile activity of the heart muscle (19).

In our observations, the most consistent gross finding in the affected turkeys was a marked enlargement and distortion of the heart. It is interesting that the early changes in young poults consist principally of right and left ventricular dilatation, whereas in older birds marked hypertrophy of the ventricles occurs. This is probably not significant, but it is logically the result of peripheral resistance and the heart’s adaptation. On the other hand, these cardiac changes were accompanied with venous congestion of the different body organs. The increased blood pressure in the veins, liver, and abdominal vessels forces plasma fluid (oedema) out of the vessels, particularly the fenestrated sinusoids of the liver, into the peritoneal spaces, where it is called ascitic fluid (13). Furthermore, severe pulmonary oedema as a result of pulmonary hypertension would result in hypoxic respiratory failure and death and would explain the increasing incidence of death without ascites in many cases of round heart disease.

The present study revealed that the microscopic changes in abnormal hearts were non-specific and included congestion, haemorrhages, degeneration and vacuolation of myofibers, resulting in damage and disappearance of some muscle fibers. Previous studies recorded abnormal accumulation of cytoplasmic glycogen and myofibrillar lysis in the myocardium of turkey poults with spontaneous round heart disease (5,7). The detection of fibroblastic proliferation and leucocytic aggregation in few cases could be explained as a method of repair of cardiac muscles. On the other hand, bile duct hyperplasia, portal fibrosis, focal necrosis and vacuolation of the hepatocytes with the presence of intracytoplasmic PAS-positive globules were seen in the liver in this study. Similar observations were reported by Calne et al., (1). Simpson et al. (22) demonstrated that the intracytoplasmic hepatic vacuoles did not
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stain as lipid in frozen section. Furthermore, Meirom et al. (17) found by immunofluorescent technique that the intracytoplasmic hepatocyte globules contain alpha globulins.

Histopathological lesions in the lungs, intestine, kidneys, spleen and brain could be secondary to the congestive heart failure. However, the detection of lymphoid depletion in the spleen may be supported by Staley et al., (23) who mentioned that the immune system plays an integral part in the development of the congestive cardiomyopathy of round heart disease.

The round heart problem in turkeys is unsolved to date. Detailed histopathological study of the heart and other organs has been disappointing in that it failed to yield information on the proper etiology of the condition. However, our results of the myocardial and hepatocytic vacuolation with the presence of PAS-positive globules in the hepatocytes corroborate previous suppositions which pointed toward a glycogen infiltration of tissues such as the heart as the primary changes of spontaneous cardiomyopathy in turkeys. In view of the current evidence it is possible that the poults which develop the problem have a metabolic defect as the basic mechanism underlying the development of round heart disease.

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 أجربت هذه الدراسة على فشل القلب التلقائي أو مرض القلب المكور حيث شهدت حالات منه بين قطعان كتاكيت الرومي المستوردة ذات الصدر العري (ب6،ت9). و كانت أغلب الوفيات خلال الثلاثة أسابيع الأولى من العمر حيث تراوحت نسبة الوفيات من 5 إلى 15 %, و أظهرت الصفة التشريحية أن تضخم و تشوه القلب نتيجة أنساع غرف القلب هي أهم الإصابات العينية, كما لوحظ في بعض الحالات انسكاء عام و ارشاح يغشاء التامور أو احتقان في كل من الرئتين، الكبد، الأمعاء، الطحال والكلى. و أظهر الفحص المجهرى وجود احتقان و تغمرات فاسدية في الألياف العضلية للقلب مع احتقان بعض هذه الألياف, و بالنسبة للكبد فقد شهد تليف بالمناطق البالبية و زيادة في عدد الخلايا المبطنة لل끈وات المرارية مع وجود فجوات فاسدة, وكرات صغيرة حامضية الصبغة داخل خلايا الكبد. كما سجلت التغييرات الهيستوبولوجية في الرئتين, الأمعاء, الطحال, الكلى و المخ. وقد نوقشت ميكانيكية حدوث هذه الحالة و علاقتها بالتغييرات المرضية المصاحبة لها.