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Morphological studies on natural ascites syndrome in broiler chickens

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ABSTRACT

The pathology of ascites syndrome in 55 male and female broiler chickens aged 26 to 44 days was studied and compared with normal controls. At necropsy, gross changes included dark breast muscle, marked abdominal distention, presence of 40-260 ml clear yellow fluid with clots of fibrin in the abdominal cavity, hydropericardium, cardiomegaly and pericardial haemorrhages, and congestion in lungs, liver, kidneys and intestines. The liver was shrunken with greyish capsule. The histopathology revealed congestion, dilatation of parabronchi and adjacent air capillaries, hypertrophy of smooth muscle of parabronchial walls, numerous small-to-large hyaline cartilaginous foci and, in some cases, osseous foci in parenchyma of lung. Congestion, oedema and myofiber degeneration of heart, pronounced dilatation of sinusoids, atrophy and degeneration of hepatocytes, marked thickening of capsule and in some cases fatty change of liver; congestion of glomeruli and urate deposits in the lumina of collecting tubules in the kidney; congestion, disappearance of white pulp and mild to severe thickening of capsule in the spleen, congestion, follicular depletion, intrafollicular cyst formation in the bursa of Fabricius were also seen.

Key words: morphology, histopathology, ascites syndrome, broiler chickens, Iran

Introduction

Ascites syndrome is an economically important problem with high morbidity and mortality mainly affecting broiler chickens the world over. It is defined as an excessive accumulation of serous fluid in the abdominal cavity, leading to carcass condemnation (MAXWELL et al., 1986; CALNEK et al., 1991). Ascites syndrome has been reported from broilers raised in high and low altitude areas (HALL and MACHIACO, 1968; CUEVA et al., 1974; LOHR, 1975; NEUMANN et al., 1975; MAXWELL et al., 1986a).

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Several situations are known to influence the occurrence of ascites in broiler chickens, such as atmospheric hypoxia, housing environment, respiratory diseases, rapid growth rates, high-energy rations, toxins, nutritional aspects and feed additives (ANDERSON et al., 1986; JULIAN and WILSON, 1986; HOERR, 1988; SHANE, 1988; WIDEMAN, 1988). To our knowledge, as yet there have been no systematic studies of the morphologic changes associated with this syndrome in Iran. The purpose of this study was therefore to define the gross and histopathological lesions of ascites syndrome that occur in broiler chickens raised at low altitude, in the Fars Province of Iran.

Materials and methods

Fifty-five 26- to 44-day-old broiler chickens (34 males and 21 females) of a commercial broiler breeder strain with clinical signs of marked abdominal swelling suggestive of an ascitic syndrome were collected from various farms in the Shiraz area, Iran. Twenty birds of similar age and strain from the same farms, but without clinical signs of ascites, served as controls. The birds were vaccinated at 3 weeks old against infectious bronchitis and Newcastle disease. The birds were maintained on a standard commercial broiler starter and grower diet.

A post-mortem examination was carried out on each bird which died or was killed during the growing period up to 44 days of age. In those birds with distended abdomens, ascitic effusion was aspirated by a 50 ml syringe and dispelled into a measuring cylinder, measured, centrifuged and used to make smears. Smears were stained with Giemsa and examined for their cellular content. The thorax and abdomen were exposed, photographed and examined. Pieces of right ventricle of heart, of liver, lung, kidneys, spleen, pancreas, skeletal muscle, small and large intestines, bursa of Fabricius, were selected for histopathological examination. The tissues were fixed in 10% buffered formalin, embedded in paraffin, sectioned at about 5 μm, stained with haematoxylin and eosin (HE) and periodic acid Schiff (PAS) and studied microscopically.

Results

Gross pathology

Post-mortem examination of affected broilers showed mild to severe abdominal distension with congestion and darkness of thigh and breast muscles (Fig. 1). Occasionally, subcutaneous oedema was also present. A most striking finding was an accumulation of either clear or straw

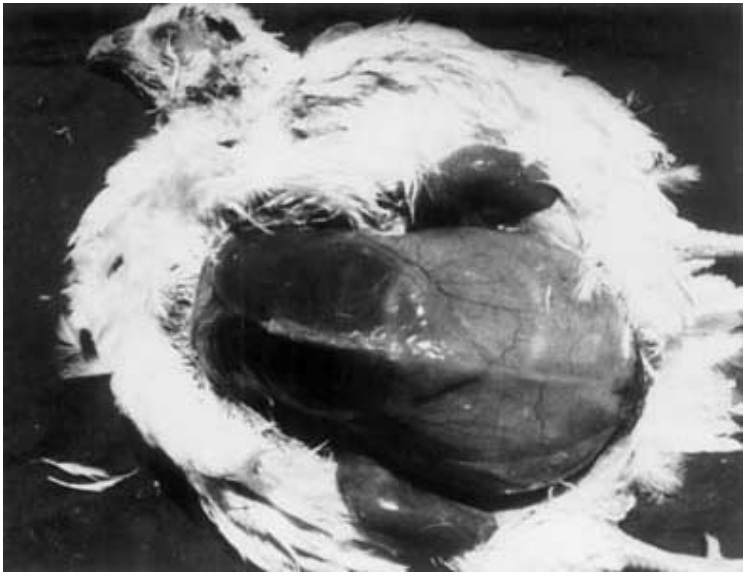

Fig. 1. Broiler with distended abdomen and congested skeletal muscle caused by ascites

coloured effusion, with or without gelatinous clots in the abdominal cavity. All birds yielded ascitic effusion, which measured between 40 to 260 ml (Table 1). The cellular content was composed mainly of red blood cells, lymphocytes and a few macrophages. Of the ascitic birds, 62% were males.

Table 1. Comparison of ascitic fluid measurement in male and female broilers

Ascitic effusion (ml)	Male % (N=34)	Female % (N=21)	Total % (N=55)
41-84	38	24	62
85-128	16	13	29
129-172	5	2	7
173-216	-	-	-
217-260	2	-	2

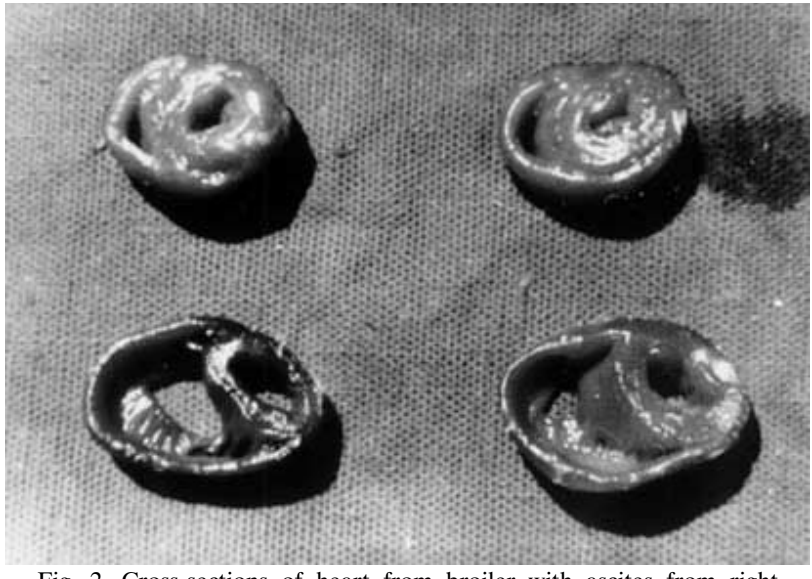

Fig. 2. Cross-sections of heart from broiler with ascites from right ventricular failure (below) and from normal broiler (above). There is hypertrophy of the right ventricular wall and dilation of the chamber.

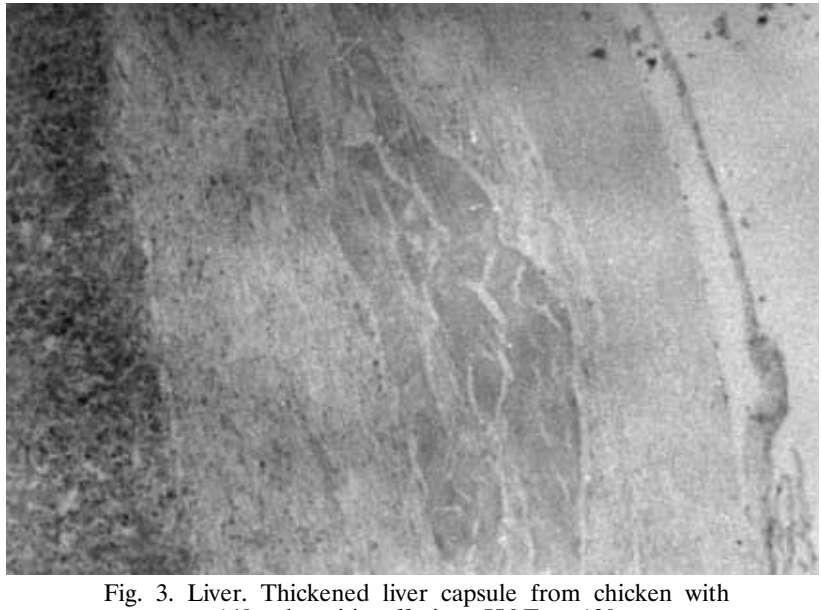

Fig. 3. Liver. Thickened liver capsule from chicken with 140 ml ascitic effusion. H&E; ×120

Pericardial effusion, sometimes with yellow fibrin clots, was seen. Birds with ascites showed cardiomegaly, dilatation of caudal vena cava, marked right-sided distention (Fig. 2), and petechial haemorrhages on the epicardium. Livers were frequently covered with a thin to thick film of yellow to greyish gelatinous fibrin clots which formed adhesions with the rib cage and other adjacent organs. Livers sometimes were congested (nutmeg liver) and swollen; in some cases they were shrunken, with thickened capsule and irregular edges. Kidneys were often congested and enlarged. In some cases they contained whitish urate deposits. Lungs, spleens and intestines, as well as other organs, were considerably congested.

Histopathology

Liver. Frequently, the liver capsule was thickened about 2-10 times normal due to fibrosis, oedema and fibrin clots (Fig. 3). In some cases there was severe congestion and a marked dilation of the sinusoids that caused atrophy of hepatocytes and fibrosis of parenchyma. Accumulation of inflammatory and immune cells consisting of lymphocytes, macrophages and a few heterophils were associated with the portal triads, especially perivascular areas. Occasionally, foci of degenerating hepatocytes around these areas were seen. In addition, centrilobular fatty changes together

Table 2. Incidence and severity of microscopic lesions in the livers of affected chickens

Type of lesion	Severity	Male (N=34)	Female (N=21)	Total (N=55)
Microscopic lesions	high	25	13	38
	moderate	27	16	43
	few	9	9	18
Capsule thickening	high	16	5	21
	moderate	25	15	40
	few	20	18	38

with other lesions were sometimes observed. Table 2 shows the incidence and severity of these lesions.

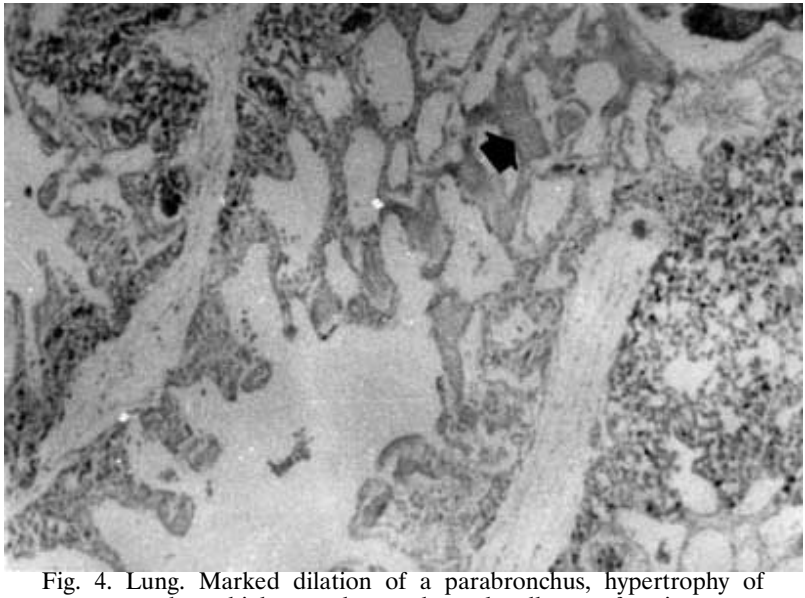

Fig. 4. Lung. Marked dilation of a parabronchus, hypertrophy of parabronchial smooth muscle and collapse of atria and air capillaries in a broiler chicken. H&E; ×120

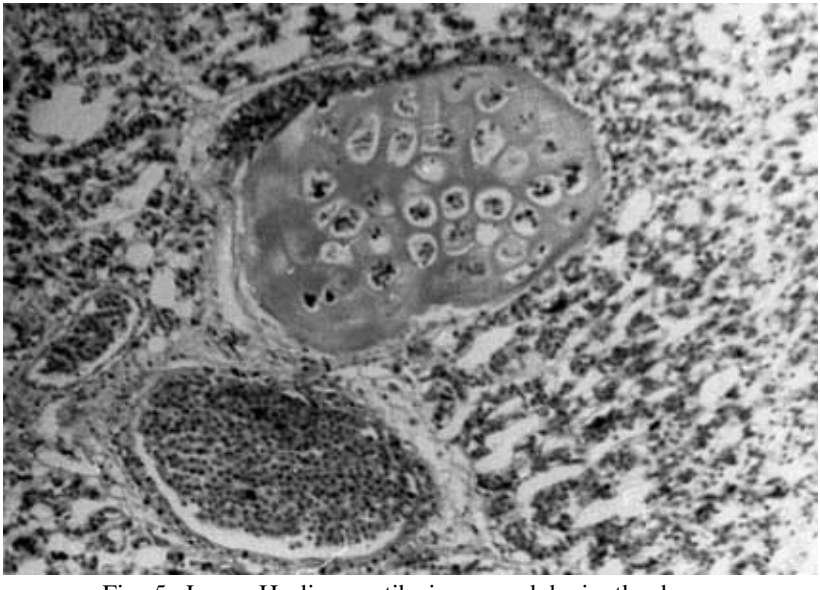

Fig. 5. Lung. Hyaline cartilaginous nodule in the lung of a broiler chicken with ascitic syndrome. H&E; ×200

Lung. Excessive dilation of the tertiary bronchi and related air capillaries associated with moderate to severe congestion were seen in several lungs. Some parabronchi walls showed fibrous thickening and accumulation of few adipocytes which were surrounded by lymphocytes, and sometimes heterophils. Thickening of walls of many blood capillaries and prominent hypertrophy of smooth muscles of parabronchi walls were seen (Fig. 4). Pulmonary oedema with the existence of serous fluid in parabronchi and related air capillaries were observed. A small to large number of hyaline cartilaginous foci were commonly seen in the lungs of

Table 3. Incidence and severity of cartilaginous nodules in the lungs of ascitic broiler chickens

Foci of hyaline cartilage	Male (N=34)	Female (N=21)	Total (N=55)
High	25	13	38
Moderate	22	15	37
Few	15	11	26

Fig. 6. Lung. Fibrocartilage and osseous foci (arrows) in parenchyma of lung of a broiler chicken with ascitic syndrome. H&E; ×200

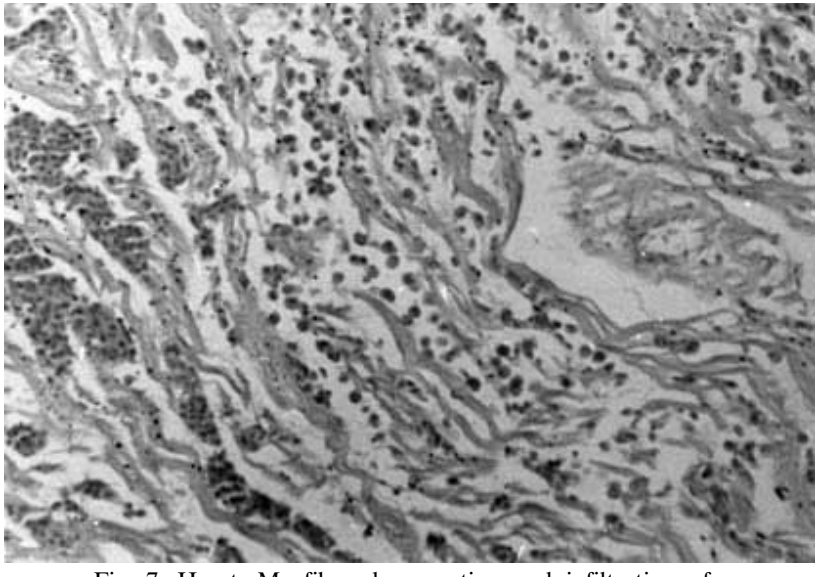

Fig. 7. Heart. Myofibre degeneration and infiltration of heterophils and lymphocytes between myofibrils from a chicken with 130 ml ascitic effusion. H&E; ×200

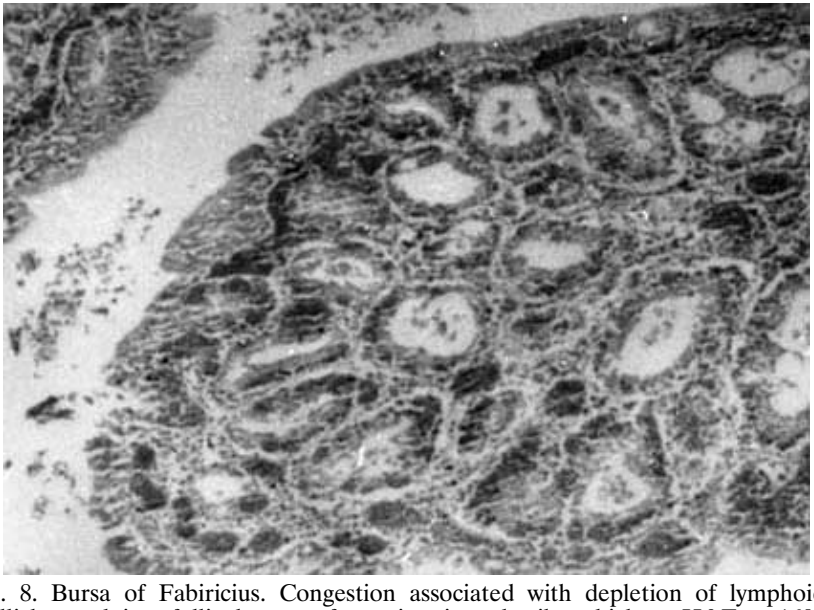

Fig. 8. Bursa of Fabiricius. Congestion associated with depletion of lymphoid follicles and intrafollicular cyst formation in a broiler chicken. H&E; ×160

all affected birds (Fig. 5) as shown in Table 3. Also, in some cases fibrous and osseous foci were diagnosed in parenchyma of lungs (Fig. 6).

Heart. The heart was severely affected in many cases with fibrous thickening of the pericardium. Oedema, myofibre degeneration and infiltration of heterophils and lymphocytes between myofibrils frequently occurred in the myocardium (Fig. 7).

Kidney. In kidney, many glomeruli appeared congested and the PAS-stained sections revealed thickened basement membranes. The proximal convoluted tubules generally appeared normal, but collecting tubules were dilated with urate deposits in the lumina. Scattered foci of lymphocytes were present in the interstitial tissue of some kidneys.

Other tissues. Spleen showed passive congestion, mild to severe thickening of capsule and to some extent disappearance of white pulp. The small and large intestine showed severe congestion of the villous heads, together with lymphocytes, heterophils and plasma cells in the submucosa. Congestion, oedema, paleness of nuclei and disappearance of cross striations were also seen in skeletal muscles. Lesions in the bursa of Fabricius showed severe congestion associated with depletion of lymphoid follicles, formation of intrafollicular cysts and interfollicular oedema (Fig. 8). No changes were seen in pancreas except for mild congestion.

Discussion

Ascites is a significant cause of mortality in many flocks of growing broiler chickens and the incidence appears to be increasing (CALNEK et al., 1991). This syndrome may result from vascular damage, increased vascular hydraulic pressure, or blockage of lymph drainage (JULIAN, 1983). The main causes of ascites in broiler chickens are usually chronic passive congestion caused by right ventricular failure (JULIAN and WILSON, 1986) and hepatic fibrosis secondary to hepatitis (JULIAN, 1988; CALNEK et al., 1991). The results of this study correspond closely with those reported earlier at high and low altitudes (CUEVA et al., 1974; NEUMANN et al., 1975; MAXWELL et al., 1986; WITZEL et al., 1990). In this study, male broiler chickens were more susceptible than females, probably because of their higher metabolic requirement for oxygen. It is possible that fast-growing male broilers have insufficient pulmonary diffusion capacity; anything that interferes with oxygen transfer, breathing ability, or cardiac output might cause the hypoxia which results in pulmonary arterial hypertension and right ventricular hypertrophy. Myocardial changes, together with infiltration of heterophils and lymphocytes, was a common finding in the heart observed in the course of

the present investigations. These cells were rarely seen in control birds. These histological changes were similar to those seen by WILSON et al. (1988) and WITZEL et al. (1990). Infiltration of inflammatory cells into the myocardium has been described in other cases of heart failure in birds (HALL and MACHIACO, 1968; VAN VLEET and FERRANS, 1982).

In the present study there was an increase in the number of cartilaginous nodules in the lungs of ascitic birds compared with control birds. Ascites is a disease of fast growing broiler chicks and is positively related to the growth of birds (JULIAN et al., 1987) and a positively correlated growth rate with the number of nodules (SARANGO and RIDDELL, 1985). Rate of growth may be another possible explanation for the increase in the number of nodules in ascitic broilers. The occurrence of cartilaginous and osseous nodules in the lungs of birds has been reported by several researchers (KALINER, 1976; JULIAN, 1983; WIGHT and DUFF, 1985; SARANGO and RIDDELL, 1985; MAXWELL et al., 1986; MAXWELL, 1988). Several hypotheses have been presented as to their origin, such as: 1. Inhalation of dietary bone meal (INNES et al., 1956) that was disproved by WIGHT and DUFF (1985), 2. Areas of ectopic primary bone formation (BORST et al., 1976), 3. Single embryonic cells of cartilage that entered the circulation from the leg bones and became trapped in the lungs and subsequently developed in lungs (JULIAN, 1983), 4. Embolic chondrocytes derived from abnormal cartilage (SARANGO and RIDDELL, 1985), 5. The result of abnormal embryonic induction of cartilaginous or mesenchyme germ cells that had become dissociated from adjacent bronchi (WIGHT and DUFF, 1985), and 6. In a possibly hypoxic environment pulmonary fibrosis develops which could lead to an increase in the number of nodules found in the lungs (MAXWELL, 1988). It is possible that they may interfere with pulmonary blood flow.

Congestion in the lungs and kidney glomeruli, together with infiltrations by inflammatory cells described by HALL and MACHIACO (1968) in altitude-reared birds and MAXWELL et al. (1986), resemble the changes seen in this study. Changes in livers were also similar to the studies of LOHR (1975), SANGER et al. (1985), MAXWELL et al. (1986), and WILSON et al. (1988).

In conclusion, the present results closely resemble those found in ascitic birds at high and low altitudes. Although the specific etiology of ascites syndrome could not be determined, it appeared that this condition was not likely to be the result of any of the commonly recognised causes of right heart failure and ascites in broilers.

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SAŽETAK

Patologija sindroma ascitesa je istraživana u 55 muških i ženskih tovnih pilića u dobi od 26 do 44 dana i uspoređena je s normalnim kontrolnim pilićima. Makroskopske promjene nađene pri obdukciji uključivale su: tamne prsne mišiće, izraženo proširenje trbuha, nazočnost 40-260 ml bistre žute tekućine s ugruščima fibrina u trbušnoj šupljini, hidroperikard, kardiomegaliju i perikardijalna krvarenja, te kongestiju pluća, jetre, bubrega i crijeva. Jetra je bila skvrčena sa sivkastom kapsulom. Histopatološki je nađena kongestija, proširenje parabronha i pridruženih zračnih kapilara, hipertrofija glatkih mišića parabronhalne stijenke, brojni mali do veliki centri hijaline hrskavice, a u nekim slučajevima i okoštale centre u parenhimu pluća. Na srcu su nađeni kongestija, edem i degeneracija mišićnih vlakana, a u jetri izražano proširenje sinusoida, atrofija i degeneracija hepatocita, značajno zadebljanje jetrene ovojnice i u nekim slučajevima masne promjene jetre. Glomeruli su bili punokrvni, a u šupljinama sabirnih bubrežnih kanalića su se nalazili urati. Slezena je bila punokrvna s gubitkom bijele pulpe, te blagom do znatno zadebljalom ovojnicom. U Fabricijevoj burzi je nađena punokrvnost, nestajanje folikula i nastajanje cisti u folikulima.

Ključne riječi: morflogija, histopatologija, sindrom ascitesa, tovni pilići, Iran