Pathomorphological changes in the organs of chickens infected spontaneously by the species *Salmonella pullorum* on private farms in Chernivtsi region


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The article describes the pathological and histological changes in the liver, heart, lungs, kidneys, and spleen. The changes were detected in chickens aged 10 and 14 days infected with microorganisms of the species *Salmonella pullorum*. Eggs for incubation were obtained from different family flocks, and incubation was performed in a single incubator. The initial clinical signs of the disease appeared in chickens aged 7 days and included diarrhea, increased water consumption, lameness, and mass concentration of chickens near the heat sources. Nervous phenomena in the form of circle walking, and partial blindness were observed in some cases. The pathological autopsy revealed hepatomegaly with sharp change in the organ colour, diapedetic hemorrhage under Glisson’s capsule, and diffuse miliary necrosis. Greyish-white nodular lesions of the lungs and heart, dystrophic changes in the kidneys, and deposition of uric acid salts in the ureters were characteristic features. Diffuse coagulation necrosis, massive perivascular infiltration by heterophilic lymphocytes and stasis were observed in the liver. Changes in the heart were characterized by significant infiltration by mononuclear cells and heterophils, which led to atrophy, necrosis, and replacement of cardiomyocytes by connective tissue cellular elements. Delymphatization and necrosis of the lymph nodes were pronounced in the spleen.

Keywords: hepatomegaly; military necrosis; cardiac necrosis; delymphatization; lymphoid nodules; heterophilic infiltration.

Introduction

Poultry breeding, especially as a commercial venture, grows rapidly every year in Ukraine. It is considered one of the key elements in elaborating economic and food security strategy. Supporting poultry production is important for domestic agricultural producers in terms of expanding exports of poultry meat, egg powder, and eggs to the EU countries. Ultimately, it also results in improving the welfare of the Ukrainian population. However, like every industry, poultry deals with its peculiar challenges. Seasonal viral diseases, especially Newcastle disease and avian influenza cause significant damage. Bacterial diseases, especially those caused by microorganisms of the genus *Salmonella* (family Enterobacteriaceae) also prevent poultry farming from achieving optimal results. Within the genus, salmonellae are differentiated by serotypes. This depends on their antigenic structure, i.e. the availability and combination of two main antigenic complexes: somatic O-antigen and flagellar H-antigen (absent in immobile strains). According to antigenic affinity, salmonellae are divided into 52 groups including more than 2,300 serotypes, of which more than 230 are isolated from birds and more than 700 from humans (Shivaprasad, 2000; Arny et al., 2004). Poultry salmonellosis causes significant economic losses due to the high mortality rate and temporary production downtime with the implementation of all quarantine measures (Calnek et al., 1997; Khan et al., 1998; Wigley et al., 2002). The spread of salmonellosis both in private homesteads and industrial facilities has occurred due to the increase in the number of small poultry farms, incubation of eggs from different family flocks, and violation of basic biosecurity principles on the part of the rural population working in commercial poultry farms. The age prevalence of poultry salmonellosis in the EU countries is most often registered in industrial flocks of adult laying hens and less often in young chickens up to 10 days of age (Rosu et al., 2007; Saha et al., 2012; Wang et al., 2020). In Ukraine, monitoring studies of salmonellosis in small farms and private homesteads are not conducted carefully, which complicates greatly the epizootiological situation in some regions of the country.

*Salmonella* is a poly-pathogen, but the poultry diseases are caused by *S. enteritidis*, *S. pullorum*, *S. gallinarum* in most cases and *S. typhimurium*. *S. heidelberd*, *S. anatum*, *S. haifa*, *S. infantis* less often. Chickens are natural hosts for both *S. pullorum* and *S. gallinarum* and these can remain in the organism without causing any clinical manifestation of the disease (Snoeyenbos et al., 1991; Nazir et al., 2012). The overall resistance potential of the organism declines in case of virological pressure, violation of zootechnical parameters, improper feeding, and inappropriate use of anti-bacterial drugs. As a consequence, favourable conditions for the development of pathogenic microflora emerge, including *Salmonella* microorganisms. Typhus is a disease caused by a microorganism of the species...
Clinical signs of the disease in chickens during their first week of life were somewhat different. They did not lose appetite for six days and were lively. However, massive signs of depression and refusal to feed appeared from the 7th day. Some chickens stood motionless with their heads down, others walked in circles with their necks turned or, lying on their backs, showed nervous disorders, i.e., they threw their heads back and walked in circles. Some chickens stood motionless with their heads down, while others were dystrophically altered. The muscle layer was thickened. The pathological autopsy revealed fatty liver degeneration with miliary nodules (Fig. 5a, b). Massive diffuse coagulation necrosis of lymphoid nodules (Fig. 4a, b) and white pulp delimitation were found in the chickens’ spleen. This testified to the immunosuppressive state. The lung tissue vessels were full of blood, with their walls fibrous and swollen. Parabronchids were infiltrated by heterophilic lymphocytes and plasma cells (Fig. 4c). The alveoli and peribronchial epithelial tissue was preserved. There was significant cellular infiltration of lymphocytes, histiocytes, and macrophages in the peribronchial connective tissue (Fig. 4d).

Morphologically, massive areas with necrosis of cardiomyocytes and pronounced proliferation by heterophilic lymphocytes were found in the heart muscle (Fig. 5a, b). Stagnation and hemorrhage into the interstitial tissue were typical in the kidneys. The lymphocytes and heterophiles were located between the dystrophically altered tubules in most chickens (Fig. 5c). Diffuse hemorrhagic infiltration of the mucous layer, desquamation of the apical part of the villi, and cellular infiltration of the mucosal plate were observed in the rectum (Fig. 5d). Most crypts were necrotized, while others were dystrophically altered. The muscle layer was thickened. Analyzing the variability of pathomorphological changes recorded in the chickens aged 10 and 14 days at various farms, the following relationship was found: 65% of the chickens suffered from hepatomegaly with diapedetic hemorrhage and multifocal necrosis in the organ’s parenchyma and cardionecrosis with heterophilic infiltration; 25% had fatty degeneration of hepatocytes, 36% had acute venous hyperemia of the lungs with necrotic foci and perivascular hemorrhagic infiltration, 17% suffered from pneumonia, 13% had catarhal hemorrhagic enteritis and colitis (Table 1).

Discussion

Our research results correlate with the reports of other scientists (Basu et al., 1975; Arora et al., 2015), who recorded 89.5% of poultry deaths in the Indian states of Karnataka, Maharashtra, and Tamil Nadu caused by microorganisms of the species S. pullorum. Clinical manifestations of the disease, such as depression, anorexia, greenish-yellow diarrhea, and nervous phenomena in the form of the head tilt were detected in 2001 during the salmonellosis outbreak in the state of Tripura in eastern India. The pathological autopsy revealed fatty liver degeneration with miliary parenchyma necrosis. S. pullorum was isolated from the gallbladder. Similar changes in the liver were described by Banet (Banet et al., 2008; Belih et al., 2016) in adult laying hens infected with S. gallinarum.

Chistihi (1985) also recorded a bronze colour of the liver with diapedetic hemorrhages and necrotic foci. Chistihi et al. (1985) and Hafeeji et al. (2001) found necrotic foci in the heart, hemorrhages and miliary necrosis in the spleen, acute venous stasis, and nodular lung damage caused by S. pullorum. Stagnant phenomena with hemorrhages in a mucous membrane were expressed in the intestine.
Fig. 1. Morphological changes in the organs of chickens infected with Salmonella pullorum:

a – liver dystrophy; diffuse diapedetic hemorrhages under the liver capsule in chickens aged 10 days;
b – hepatomegaly; necrosis outbreaks (arrow);
c – liver; diffuse foci of necrosis in chickens aged 10 days (arrow);
d – diffuse necrotic foci in the lung parenchyma (arrows)

Fig. 2. Morphological changes in the organs of chickens infected with Salmonella pullorum:

a – the heart; diffuse necrotic foci in the myocardium (arrows); b – the heart: massive focus of necrosis (arrow); c – sharply enlarged and dystrophically altered kidneys of chickens aged 10 days (arrows); d – the spleen of chickens aged 10 days: hemorrhages under the capsule (arrow)
Fig. 3. Pathohistological changes in the liver of chickens infected with *Salmonella pullorum*: 

- **a** – multifocal coagulation necrosis of hepatocytes with further fibrosis;
- **b** – the liver of chickens aged 10 days: 1 – hepatocyte necrosis; 2 – heterophilic infiltration; 3 – dilation of intraparticle capillaries;
- **c** – perivascular infiltration by heterophils, plasma cells, and lymphocytes;
- **d** – the liver of chickens aged 14 days: 1 – colonies of microorganisms; 2 – heterophilic infiltration, plasma cells accumulation, lymphocytes; 3 – necrosis of hepatocytes; hematoxylin and eosin.

Fig. 4. Pathohistological changes in the spleen and lungs of chickens infected with *Salmonella pullorum*: 

- **a** – the spleen: diffuse coagulation necrosis of lymph nodes;
- **b** – the spleen: delymphatization of lymphoid nodules with the development of coagulation necrosis;
- **c** – the lungs: acute congestive hyperemia: 1 – massive infiltration of bronchial pairs by heterophils, plasma cells, and lymphocytes; 2 – perivascular edema;
- **d** – the lungs: 1 – acute congestive hyperemia of the parabronchi, lymphoid infiltration; 2 – perivascular edema; hematoxylin and eosin.
Fig. 5. Pathohistological changes in the organs of chickens infected with *Salmonella pullorum*: 

- **a, b** – the heart of chickens aged 10 days: 1 – heterophilic infiltration of cardiomyocytes; 2 – cardiomegaly; 
- **c** – the kidney of chickens aged 14 days: 1 – duct epithelial dystrophy, stasis; 2 – cellular infiltration; 
- **d** – the rectum: acute catarrhal hemorrhagic desquamative colitis; hematoxylin and eosin

**Table 1**

<table>
<thead>
<tr>
<th>Typical pathohistological changes in the chickens’ organs</th>
<th>Farm 1. The number of dead chickens, 10 to 20 days (n = 1750)</th>
<th>Farm 2. The number of dead chickens, 10 to 20 days (n = 2560)</th>
<th>Farm 3. The number of dead chickens, 10 to 20 days (n = 2150)</th>
<th>Farm 4. The number of dead chickens, 10 to 20 days (n = 3068)</th>
<th>% of the dead chickens</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fatty liver degeneration with miliary necrosis</td>
<td>–</td>
<td>640</td>
<td>538</td>
<td>767</td>
<td>25</td>
</tr>
<tr>
<td>Hepatomegaly with hemorrhage under the capsule and heart necrosis</td>
<td>1137</td>
<td>1664</td>
<td>1397</td>
<td>1995</td>
<td>65</td>
</tr>
<tr>
<td>Catarrhal hemorrhagic enteritis</td>
<td>227</td>
<td>–</td>
<td>–</td>
<td>399</td>
<td>13</td>
</tr>
<tr>
<td>Pneumonia</td>
<td>297</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>17</td>
</tr>
<tr>
<td>Acute venous hyperemia of the lungs with necrotic foci</td>
<td>–</td>
<td>921</td>
<td>774</td>
<td>1104</td>
<td>36</td>
</tr>
<tr>
<td>Splenomegaly</td>
<td>–</td>
<td>51</td>
<td>–</td>
<td>–</td>
<td>2</td>
</tr>
<tr>
<td>Spot hemorrhages in the spleen parenchyma</td>
<td>455</td>
<td>–</td>
<td>559</td>
<td>797</td>
<td>26</td>
</tr>
<tr>
<td>Renal dystrophy</td>
<td>630</td>
<td>–</td>
<td>–</td>
<td>1104</td>
<td>36</td>
</tr>
<tr>
<td>Liver multifocal necrosis</td>
<td>1487</td>
<td>2176</td>
<td>1827</td>
<td>2607</td>
<td>85</td>
</tr>
<tr>
<td>Perivascular cell infiltration</td>
<td>1260</td>
<td>1843</td>
<td>1548</td>
<td>2208</td>
<td>72</td>
</tr>
<tr>
<td>Heterophilic infiltration of the small intestinal mucosa</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>460</td>
<td>15</td>
</tr>
<tr>
<td>Necrosis of the splenic lymphoid nodules</td>
<td>1137</td>
<td>1664</td>
<td>1368</td>
<td>1994</td>
<td>65</td>
</tr>
</tbody>
</table>

The pathohistological study performed by Hossain (Hossain et al., 2006) indicated the development of fatty degeneration of the hepatocytes, congestion phenomena combined with hemorrhage, isolated necrotic nodules with infiltration of leukocytes, mainly mononuclear cells and heterophiles. Wigley et al. (2001), Garcia et al. (2010), Kaoud et al. (2018) reported nodular heart disease and significant mononuclear cell infiltration leading to atrophy, necrosis, and replacement of muscle tissue with connective tissue elements.

**Conclusion**

A pathomorphological study revealed the characteristic changes that occurred during the infectious process. They included bacteremia with subsequent parenchymal diffusion of microorganisms into the internal organs and the progression of changes inherent in the granulomatous inflammatory reaction. Thus, the combination of typical signs in the localization of pathological processes was noted from the morphological point of view. They include focal myocarditis, multifocal hepatocyte confluent necrosis, fatty degeneration of hepatocytes, intraparenchymal multifocal proliferates in the lungs, hemorhagic desquamative colitis, and dystrophic changes of the nephrothelium of the renal tubules.

**References**


