

Pathogenesis of Experimental *Salmonella* Gallinarum Infection (fowl typhoid) in Broiler Chicks

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THIS study aimed to investigate the pathogenesis of locally isolated *Salmonella* Gallinarum biovar Gallinarum in experimentally infected Broiler chicks. One hundred and forty Broiler chicks -one day old- were randomly divided into two groups. The first group was kept as a control group. The second group was inoculated with *S. Gallinarum* biovar Gallinarum at a dose 0.2ml of sterile saline containing 3×10^8 CFU/ml through crop gavage. After inoculation, all experimental birds were kept under strict daily observation for recording clinical signs and mortality rate. Tissue samples were collected from dead and sacrificed chicks of each group at the 12th h, 1st, 2nd, 3rd, 4th, 7th, 14th, 21st and the 28th day post infection (dpi). *Postmortem* examination revealed severe congestion of internal organs. Microscopical findings illustrated congestion, thrombosis, focal hemorrhages, aggregation of bluish masses of bacterial colonies scattered in the hepatic parenchyma and thickening of hepatic capsule with bile duct hyperplasia. Fibrinous and granulomatous pericarditis were seen. Pulmonary congestion and thrombosis with focal hemorrhages and enteritis with desquamation of villar epithelium and goblet cells activation were observed. In conclusion, experimental inoculation of chicks with *S. Gallinarum* biovar Gallinarum was characterized by early septicemic lesions followed by perihepatitis, serofibrinous pericarditis and enteritis appeared after the 2nd dpi.

Keywords: Fowl typhoid, *Salmonella* Gallinarum, Pathogenesis, Pathology.

Avian salmonellosis has been considered by many investigators all over the world to be of special significance due to its danger to human beings and poultry farms (Carli *et al.*, 2001).

Salmonella Gallinarum biovar Gallinarum (*S. Gallinarum*) infection causes fowl typhoid, an acute disease which affects primarily chickens and turkeys, but pheasants, quails, and guinea-fowl are also susceptible (Shivaprasad, 2000 and Casagrande *et al.*, 2014). It is considered as one of the most important septicaemic bacterial diseases associated with serious problems and economic losses among affected chickens (Evans, 2011). These losses are represented by high mortality rate in baby chicks, retardation in growth, adverse effect on egg production of infected laying birds and low fertility and hatchability of eggs laid by carriers (Saha *et al.*, 2012). Mortality caused by *S. Gallinarum* biovar Gallinarum may be higher than 80% in Broilers (Paiva *et al.*, 2009) and may reach almost 100% in inoculated young brown layers (Oliveira *et al.*, 2005) and may reach 100% in some infected flocks (Uzzau *et al.*, 2000). The Common symptoms are depression, weakness, ruffled feathers, weight loss, 50-70% drop in egg production, prostration, apathy, drooped wings, loss of appetite, dehydration, and greenish-yellow to bloody diarrhea (Freitas Neto *et al.*, 2007).

In chickens, infections with *S. Gallinarum* biovar Gallinarum (*S. Gallinarum*) and *S. enterica* subspecies *enterica* serovar Gallinarum biovar Pullorum (*S. Pullorum*) cause septicaemic fowl typhoid and Pullorum disease, respectively (Barrow & Freitas Neto, 2000 and OIE, 2012). *S. Gallinarum* biovar Gallinarum produced lesions in chicks, indistinguishable from those associated with pullorum disease. Many pathological changes were reported during the course of acute fowl typhoid infection in Broiler chicks, including macrocytic hypochromic anaemia (Fotouh *et al.*, 2014), hepatitis, splenitis, typhlitis, omphalitis, myocarditis, ventriculitis, pneumonia, synovitis, peritonitis and ophthalmitis (Shivaprasad, 2000).

The present study aimed to investigate the pathogenesis of acute fowl typhoid in Broiler chicks –one day old- experimentally inoculated with locally isolated *S. Gallinarum* biovar Gallinarum regarding to clinical signs, mortality rate, gross lesions and histopathological findings.

Material and Methods

This study was carried out according to the guidelines for animal experimentation, and approved by the Institutional Animal Care and Use Committee, National Research Centre, Dokki, Giza, Egypt.

Isolation and identification of S. Gallinarum biovar Gallinarum

It was performed from the intestine and liver of diseased chickens showing high mortalities, diarrhea, ruffled feathers and anorexia (Finegold and Martin, 1982, Quinn *et al.*, 1994 and Swayne *et al.*, 1998). Suspected *Salmonella* isolates were identified serologically using the slide agglutination test in the Laboratories of Ministry of Public Health, Cairo, Egypt (Neville and Bryant, 1986).

Experimental Chicks

One hundred and forty of one-day-old Hubbard Broiler chicks were used in this experiment. These chicks were put under the required hygienic conditions, fed on a balanced pelleted feed, supplied with clean tap water in sufficient quantities and not supplemented with antimicrobial agents until the end of the experimental period.

Preparation of the inoculum (cultured strain)

Twenty-four hours pure cultures of isolated *S. Gallinarum* biovar *Gallinarum* were suspended in sterile saline solution using McFarland opacity tube No. 1 (Bailey and Scott, 1990). The approximate cell density in this dilution was 3×10^8 CFU/ml (Colony Forming Units/ml).

Experimental design

One hundred and forty -one day old- chicks were randomly divided into two groups. The first group (N=50) was kept as a normal control, each chick was inoculated with 0.2 ml of sterile saline through crop gavage. Each chick of the second group (N=90) was inoculated with isolated *S. Gallinarum* biovar *Gallinarum* at a dose of 0.2 ml of sterile saline containing 3×10^8 CFU/ml via the same route (Christensen *et al.*, 1996). After inoculation, all experimental birds were kept under strict daily observation for recording clinical signs and mortality rate. From dead and sacrificed chicks, tissue samples were collected from each group for bacteriological and histopathological examinations.

Bacteriological examination

All necropsied chicks were exposed to bacteriological examination. Moreover, re-isolation and identification of the inoculated *S. Gallinarum* biovar *Gallinarum* was also performed from the liver and intestine at 7th, 14th, 21st and 28th day post inoculation (dpi). Identification of the re-isolated *S. Gallinarum* biovar *Gallinarum* was depending on morphological characters and biochemical properties (Finegold and Martin, 1982).

Pathological studies

At 12 hr, 1st, 2nd, 3rd, 4th, 7th, 14th, 21st and 28th dpi, *Postmortem* findings were detected and tissue specimens from liver, heart, lungs and intestine were collected from dead and sacrificed birds, fixed in 10% formol saline, dehydrated, cleared and embedded in paraffin blocks. Paraffin sections of 5 μ thickness were prepared, stained by H&E, and examined microscopically for detection of histopathological alterations (Bancroft *et al.*, 1996).

Results

Clinical signs, mortality rate and bacteriological examination

During the experimental period, no abnormal signs or mortalities were recorded in normal control chicks. However, clinical signs following the experimental inoculation of chicks with *S. Gallinarum* biovar *Gallinarum* (20%

from all infected chicks) were represented by dullness, ruffled feathers, droppings, huddled together, white pasty diarrhea, loss of appetite, emaciation and depression. The mortality rate was 24.40% during the period of experiment (Table 1). *S. Gallinarum* biovar *Gallinarum* was re-isolated from chicks after 7th, 14th, 21st and 28th dpi.

TABLE 1. Mortality rate in the control and inoculated chicks with *Salmonella Gallinarum* biovar *Gallinarum* during the experimental periods.

Groups	Mortalities after <i>Salmonella Gallinarum</i> biovar <i>Gallinarum</i> inoculation (days)												Mortality rate
	12hr	1	2	3	4	5	6	7	8-14	15-21	22-28	Total	
Control (N=50)	0	0	0	0	0	0	0	0	0	0	0	0	0
Infected (N=90)	0	0	5	3	2	4	0	2	4	1	1	22	24.4%

Pathological findings

Postmortem examination

At 12th hpi and 1st dpi, there was congestion of the liver, heart, lungs and intestine and multiple reddish spots on hepatic surface. At 2nd dpi, congestion of the liver, lungs and heart with mild thickening of the pericardium in some chicks was observed. At 3rd dpi, there was congestion of the liver and heart associated small necrotic foci on the hepatic surface with whitish membrane covering the liver and heart in some chicks. At 4th dpi, congestion of the liver, heart and intestine with whitish membrane covering the heart was seen. At 7th dpi, thickening of the pericardium with increased pericardial fluid and mild enlargement of the liver was detected. At 14th dpi, thick white sheath covered the heart with turbid pericardial fluid and congestion of lungs was observed. At 21st dpi, congestion of the lungs and liver, thickening of the pericardium and hepatic capsule together with white necrotic foci on hepatic surface was seen. At 28th dpi, severe thickening of the pericardium with increased and turbid pericardial fluid and caseated material in lung was observed.

Histopathological examinations

In the liver: At 12th hpi, there was congestion of portal blood vessels (BVs) with multiple areas of diffuse hemorrhages replaced the hepatic parenchyma (Fig.1a), thrombosis of some portal vessels with diffuse vacuolation of hepatocytes and focal area of inflammatory cellular infiltration (ICI) in-between degenerated hepatocytes. Bluish masses of bacterial colonies mostly around BVs were noticed. At 1st dpi, vacuolar degeneration of hepatocytes and bile duct epithelium with accumulation of cellular debris and desquamated epithelium in the lumen were observed. At 2nd dpi, there was congestion of hepatic sinusoids

with vacuolar degeneration and fatty change of hepatocytes, focal aggregation of lymphocytes in-between hepatic parenchyma and hyperplasia of bile duct epithelium forming newly formed bile ductules (Fig. 1b). At 3rd dpi, diffuse fatty degeneration of hepatocytes and focal areas of hepatic necrosis associated with thickening of hepatic capsule with ICI was detected. At 4th dpi, portal BVs were congested with fatty change of some hepatocytes. At 7th dpi, there was congestion of central veins with thrombosis of some portal vessels, small focal areas of ICI (macrophages) in-between the hepatocytes (Fig. 1c) and fatty degeneration of hepatocytes. Moreover, hydropic degenerative changes associated with hyperplasia and/or desquamation of the epithelium lining of some bile ducts were seen. At 14th dpi, congestion of central veins, portal BVs and hepatic sinusoids with fatty degeneration of hepatocytes with ICI of hepatic parenchyma forming microscopic nodules (Fig. 1d) and thrombosis of some portal veins were seen. At 21st dpi, there was multiple areas of hepatic degenerative changes particularly fatty change of hepatocytes and focal areas of coagulative necrosis (Fig. 1e) and severe perihepatitis characterized by thickening of hepatic capsule with ICI and deposition of fibrin threads (Fig. 1f). At 28th dpi, congestion of hepatic sinusoids, central veins and portal BVs and accumulation of eosinophilic cellular debris mixed with ICI, obstructed partially the lumen of bile duct were detected.

In the heart: At 12th hpi, there was congestion of myocardial BVs and mild pericarditis with aggregation of bacterial colonies inside the pericardium (Fig. 2a). At 1st dpi, congestion of myocardial BVs with hyaline degeneration was observed. At 2nd dpi, there was thickening of the pericardium with mononuclear aggregation and bacterial clumps inside it (Fig. 2b) and inter-muscular ICI (macrophages). At 3rd dpi, severe thickening of pericardium due to deposition of fibrin threads and ICI that extended to underlying muscles were seen (Fig. 2c). In some hearts, the interstitium was expanded by oedema and low numbers of ICI in-between cardiac muscle fibers with focal area of granuloma. At 4th dpi, there was severe pericarditis with ICI and deposition of fibrinous exudates, myocarditis characterized by congestion of myocardial BVs with degenerative changes in cardiac muscles, and ICI between the cardiac muscles (Fig. 2d). At 7th dpi, pericarditis and fibrinous pericarditis with ICI aggregation and deposition of fibrin threads and granulomatous pericarditis were seen (Fig. 2e) and hyaline degeneration of the myocardium were noticed. At 14th dpi, there was severe thickening of the pericardium due to accumulation of fibrinous exudates containing ICI. At 21st dpi, severe thickening of the pericardium due to granulomatous reaction characterized by central area of caseous necrosis was seen (Fig. 2f). At 28th dpi, severe thickening of the pericardium due to fibrinous exudates and ICI (macrophages) was detected. Myocardial muscle fibers showed atrophy and myomalacia. Granuloma formed from focal area of caseous necrosis surrounded by inflammatory cells was observed.

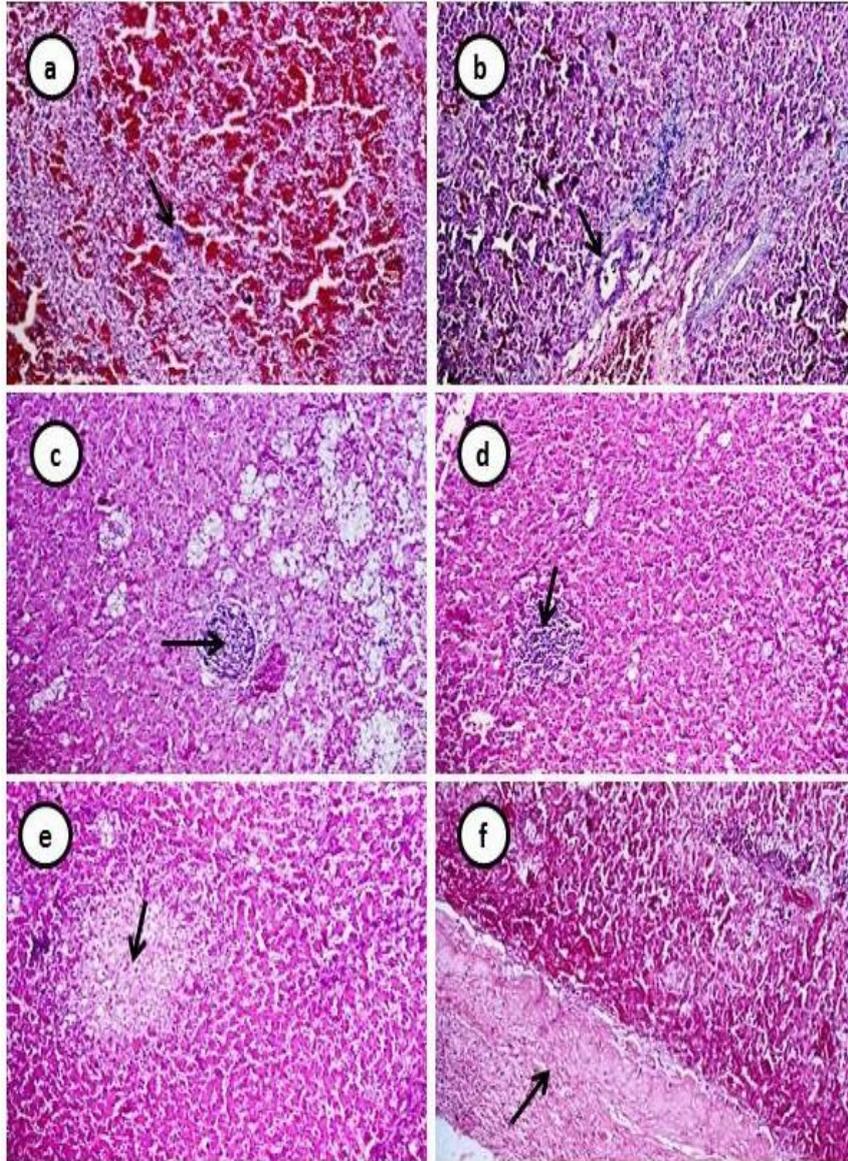


Fig.1. Liver of chick post inoculation (pi) with *Salmonella Gallinarum* biovar *Gallinarum* showing: (a) diffuse hemorrhage and bacterial clumps (arrow) within the hepatic tissue at 12th hpi, (b) hyperplasia of bile duct represented by newly formed bile ductules (arrow) at 2nd dpi, (c) focal areas of mononuclear inflammatory cells (arrow) and multiple areas of fatty degeneration at 7th dpi, (d) aggregation of inflammatory cells forming microscopic nodule (arrow) at 14th dpi, (e) focal area of coagulative necrosis (arrow) at 21st dpi, (f) severe perihepatitis (arrow) at 21st dpi. (H&E ×200)

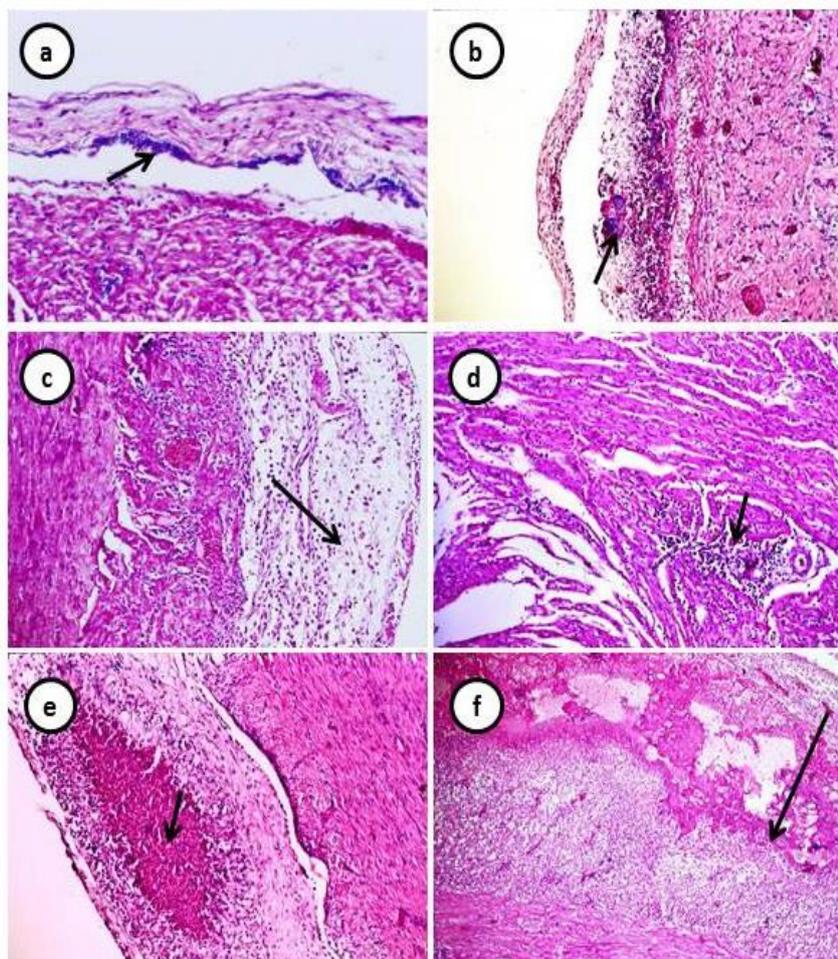


Fig. 2. Heart of chick post inoculation (pi) with *Salmonella Gallinarum* biovar *Gallinarum* showing: (a) mild pericarditis with bacterial clumps (arrow) inside pericardium at 12th hpi (H&E \times 400), (b) pericarditis with bacterial clumps (arrow) inside pericardium at 2nd dpi, (c) pericarditis with inflammatory cellular infiltration (arrow) at 3rd dpi, (d) focal inflammatory cellular infiltration (arrow) in-between cardiac muscles at 4th dpi, (e) granulomatous pericarditis (arrow) at 7th dpi, (f) severe thickening of pericardium (arrow) due to granulomatous reaction at 21st dpi. (H&E \times 200)

In the lungs: At 12th hpi, congestion of pulmonary BVs with perivascular hemorrhage and oedema with ICI (lymphocytes) was seen (Fig. 3a). At 1st dpi, there was accumulation of inflammatory exudates in the lumens of some parabronchi. At 2nd dpi, perivascular oedema was prevalent. Large areas of pulmonary alveoli were consolidated with ICI (Fig. 3b). Multiple focal areas of

pulmonary caseated granulomas were detected. These granulomas composed from central necrosis surrounded by large number of heterophils followed by wide zone of ICI (macrophages) were observed. At 3rd dpi, there was congestion of pulmonary BVs and perivascular oedema, with ICI and desquamation of epithelium of some bronchioles (Fig. 3c). Cellular debris accumulated in lumen of some parabronchi. At 4th dpi, congestion of pulmonary BVs with ICI in the pulmonary tissue was prevalent. In some lungs, perivascular oedema and hemorrhages with leukocytic aggregation (macrophages) were seen (Fig. 3d). At 7th dpi: fibrinous exudates obstructing the lumen of bronchioles (Fig. 3e). Focal areas of hemorrhage mixed with ICI in the subepithelial connective tissue of the bronchioles with desquamation of the bronchiolar epithelium in the lumen were observed. In some lungs, perivascular oedema with ICI (macrophages), while in other lungs, many granulomas characterized by focal areas of caseous necrosis were detected. At 14th dpi, there was congestion of interlobular BVs and inter-alveolar capillaries associated with perivascular and interlobular oedema. Consolidated alveoli filled with eosinophilic fluid containing desquamated epithelium, while in other lungs, the consolidated areas of alveoli contained fibrinous exudates mixed with ICI were seen. At 21st dpi, there was congestion of BVs in the pulmonary alveoli and areas of hyperplasia and desquamation of their lining epithelium with ICI in the lumen of some bronchioles. At 28th dpi, large area of caseous necrosis surrounded by ICI was detected (Fig. 3f). Desquamation of the lining epithelium of some bronchioles with peribronchiolar ICI was seen.

In the intestine: At 12th hpi, extensive ICI of intestinal mucosa with dilatation of some intestinal glands was seen (Fig. 4a). At 1st dpi, there was congestion of intestinal BVs with desquamation of the lining epithelium of intestinal villi (Fig. 4b). At 2nd dpi, slight hyperplasia and desquamation of lining epithelium with activation of goblet cells the intestinal mucosa was observed (Fig. 4c). Mucous degeneration of intestinal villi and hydropic degeneration of epithelial cells of Luberkin crypts were seen. At 3rd dpi, mild enteritis, congestion of BVs, activation of goblet cells and desquamated epithelium mixed with ICI in the intestinal lumen were seen (Fig. 4d). At 4th dpi, there was congestion of intestinal BVs with ICI in lamina propria and submucosa with activation of goblet cells and presence of desquamated epithelium mixed with ICI in the lumen. At 7th dpi, vacuolar and hydropic degeneration of the lining epithelium of intestinal villi (Luberkin crypts) with mild lymphocytic infiltrations of lamina propria were detected. Desquamation of lining epithelium of intestinal villi and presence of bluish mucous in intestinal lumen were seen. At 14th dpi, there was degeneration of intestinal epithelium with ICI in lamina propria associated with focal desquamation of lining epithelium. At 21st dpi, destruction and desquamation of the epithelial lining of intestinal villi with ICI (lymphocytes and few macrophages) in mucosa and submucosa, activation of the goblet cells among the impact epithelium were seen (Fig. 4e). At 28th dpi, there was severe ICI in lamina propria, hyperplasia of lining epithelium of intestinal villi and crypts of luberkin crypts was prevalent with destruction of intestinal villi and presence of eosinophilic debris in lumens (Fig. 4f).

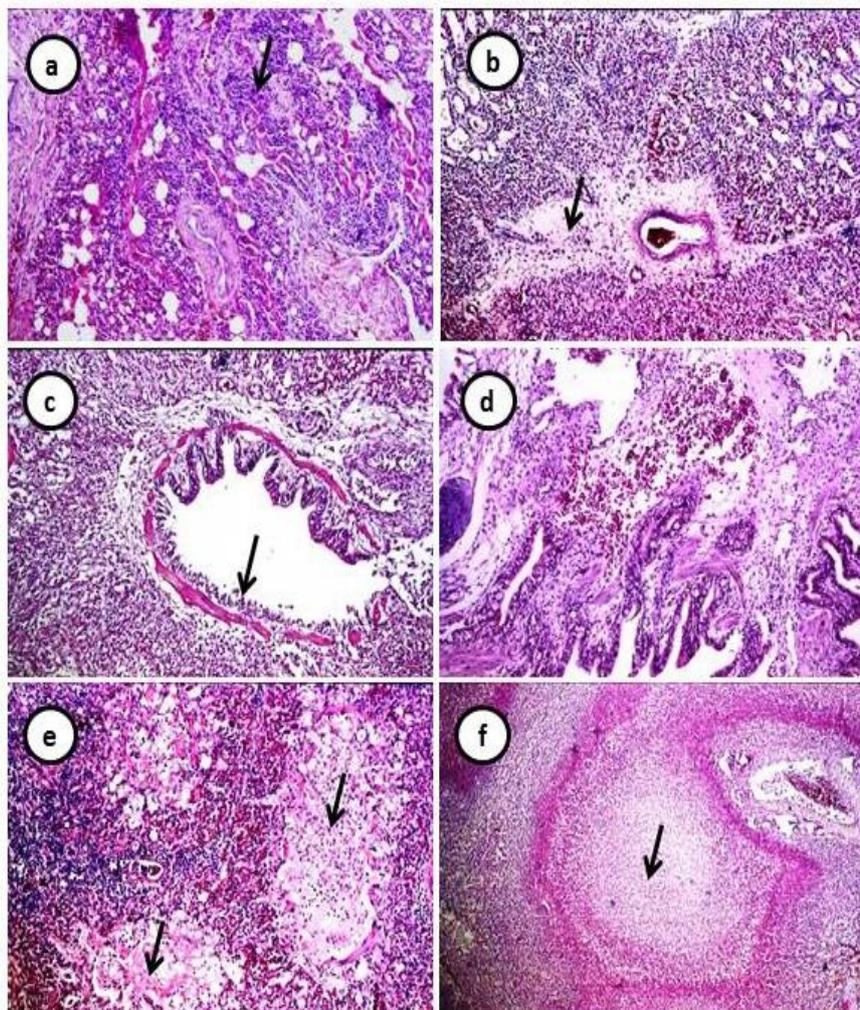


Fig. 3. Lung of chick post inoculation (pi) with *Salmonella* Gallinarum biovar Gallinarum showing: (a) severe mononuclear cellular infiltration of the interstitium and alveoli (arrow) at 12th hpi, (b) perivascular edema (arrow) and severe inflammatory cellular infiltration mainly mononuclear cells of the pulmonary alveoli at 2nd dpi, (c) desquamation of lining epithelium of bronchiole (arrow) and inflammatory cellular infiltration of the pulmonary tissue at 3rd dpi, (d) focal hemorrhage with few leukocytes aggregation particularly mononuclear cells at 4th dpi, (e) severe inflammatory cellular infiltration and accumulation of fibrinous exudates in lumen of bronchioles (arrow) at 7th dpi, (f) large area of caseous necrosis (arrow) surrounded by inflammatory cells at 28th dpi (H&E ×200).

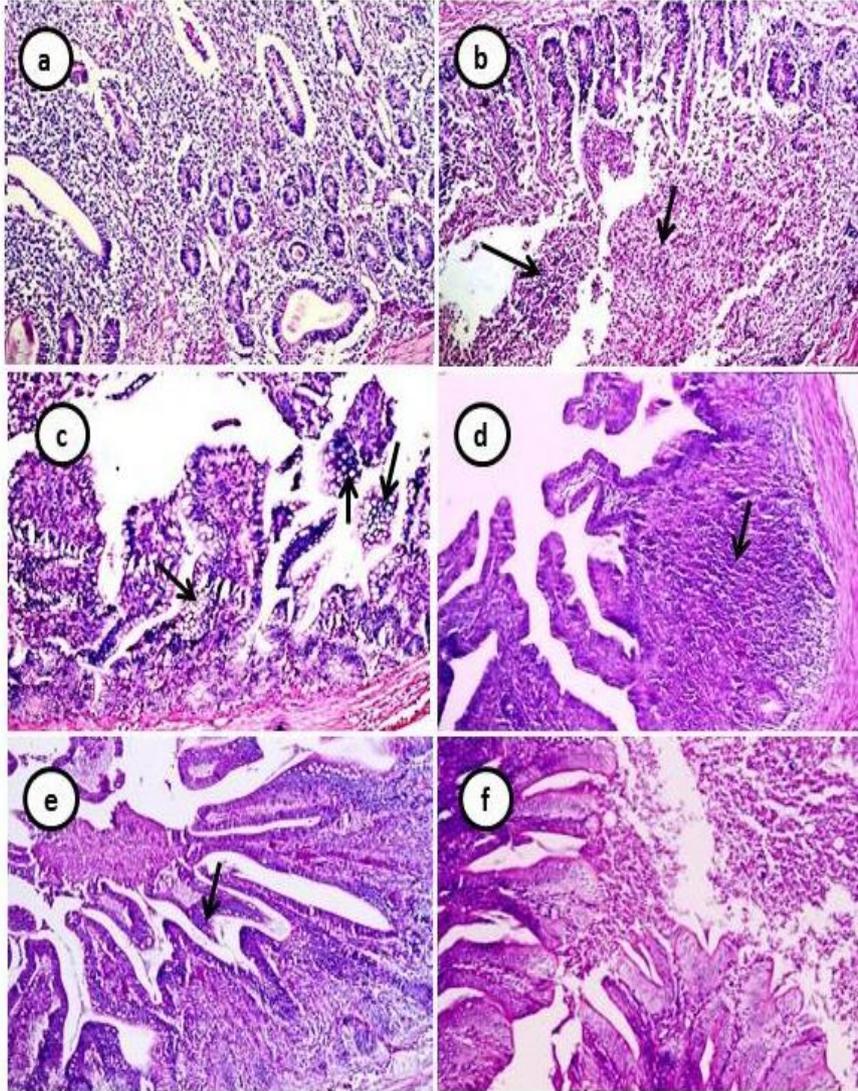


Fig. 4. Intestine of chick post inoculation (pi) with *Salmonella Gallinarum* biovar *Gallinarum* showing: (a) severe mononuclear cellular infiltration of lamina propria at 12th hpi, (b) accumulation of desquamated epithelial cells mixed with inflammatory cells in lumen (arrow) at 1st dpi, (c) desquamation of lining epithelium and activation of goblet cells (arrow) at 2nd dpi, (d) severe inflammatory cellular infiltration of lamina propria (arrow) at 3rd dpi, (e) desquamation of the epithelial lining of intestinal villi (arrow) and activation of goblet cells at 21st dpi, (f) destruction of intestinal villi and presence of eosinophilic debris in lumen at 28th dpi. (H&E \times 200)

Discussion

In the present study, the clinical signs following *S. Gallinarum* infection in chicks were dullness, ruffled feather, dropping, huddle together, white pasty diarrhea, loss of appetite, decrease in feed intake and depression. These clinical signs began to appear at 2nd dpi suggesting that the incubation period of the disease was about 24hrs. The mortality rate was 24.4%, while the mortality might reach 100% in some flocks of chicks infected with *Salmonella enterica* (Uzzau *et al.*, 2000). The different mortality results may be depended on virulence of *Salmonella* spp., age of chicks, and the route of infection (Kallapura *et al.*, 2014a & 2014b).

Postmortem examination showed that the characteristic gross lesions of experimental *S. Gallinarum* infection in chicks were septicemic in nature and represented by severe congestion of internal organs, liver, heart, lungs and intestines. Moreover, the liver showed multiple reddish spots on hepatic surface at 12th hpi and 1st dpi, perihepatitis and necrotic foci on the hepatic surface at 3rd dpi. These findings were previously reported (Casagrande *et al.*, 2014). Pericarditis was observed from the 2nd dpi till the end of the experiment. Similar observation was recorded in chickens experimentally infected with *S. Gallinarum* (Prasanna and Paliwal, 2003). The virulence of *S. Gallinarum* could be attributed to the presence of virulence plasmid (Bailey and Scott, 1990). The probable mode of pathogenesis is that, following ingestion, the organisms localize in the intestine from where they gain entrance to the blood stream, giving rise to bacteremia that results in pericarditis, hepatitis and enteritis. This suggestion was supported by isolation of *S. Gallinarum* from livers and intestines of most sacrificed and dead birds at different periods.

In the liver, microscopical findings were vacuolar and hydropic degeneration and fatty change. Congestion and thrombosis of some portal BVs with ICI (mononuclear) were seen. These findings were reported in chickens experimentally infected with *S. Gallinarum* (Prasanna and Paliwal, 2003). Hepatic cellular necrosis was reported at 3rd and the 21st dpi. Similar findings were observed in laying hens infected with *S. Gallinarum* (Freitas Neto *et al.*, 2007). Focal ICI forming microscopic nodules were observed in-between hepatic parenchyma and portal area at 12th hpi, 2nd, 7th and 14th dpi. Multiple areas of scattered hemorrhage in hepatic parenchyma were seen at the 12th hpi. Thickening of hepatic capsule due to ICI was observed at 3rd and 21st dpi. Hyperplasia of bile ductal epithelium was predominant which could be attributed to direct mitogenic effect of *S. Gallinarum* endotoxin. This finding was observed partially in chicks experimentally infected with *S. Typhimurium* (Gheith, 2008). Bluish masses of bacterial clumps were observed in hepatic parenchyma mostly around the BVs at the 12th dpi.

Pericarditis was the most prominent cardiac lesion that characterized by thickening of the pericardium due to fibrinous exudation and ICI. Pericardial and

myocardial BVs were congested. Bluish masses of bacterial clumps inside the pericardium were observed at 12th hpi and 1st dpi.

Severe congestion of the pulmonary BVs with perivascular oedema and hemorrhages were prevalent in most lungs. These findings were previously recorded in chicks experimentally infected with *S. Typhimurium* (Gheith, 2008) and also observed in Japanese quails naturally infected with *S. Gallinarum* (Casagrande *et al.*, 2014). Mononuclear cell infiltration in the bronchioles and parabronchi and the whole pulmonary tissue was consolidated with ICI. Similar findings were recorded by Dhillon *et al.* (2001). Multiple focal areas of pulmonary granulomas and caseous necrosis were observed.

Catarrhal enteritis was observed. These findings were observed in chicks (Paiva *et al.*, 2009) and Japanese quails (Casagrande *et al.*, 2014). Vacuolar and hydropic degeneration of intestinal villi epithelium and ICI were recorded. The same lesion was reported in 40-day-old chicks experimentally infected with *S. Pullorum* (Henderson *et al.*, 1999).

In conclusion, the experimental infection of chicks with *S. Gallinarum* biovar *Gallinarum* was characterized by early septicemic lesions followed by perihepatitis, serofibrinous pericarditis, perihepatitis and enteritis appeared from the 2nd dpi. Moreover, granulomatous lesions in the heart were detected at the end of the experiment.

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(Received 22/11/2016;
accepted 5 /12/2016)

دراسة مراحل المرض للعدوى التجريبية بميكروب السالمونيلا جلينرم (تيفويد الطيور) في كتاكيت التسمين

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أجريت هذه الدراسة بهدف وصف التغيرات الباثولوجية العينية والمجهريّة المصاحبة للعدوى التجريبية في الكتاكيت عن طريق التجريع بالفم لعنزة السالمونيلا جلينرم والمعزولة محلياً من حالات دجاج تسمين وبياض مصابة طبيعياً بمرض تيفويد الطيور. أجريت الدراسة على عدد ١٤٠ كتكوت تسمين هيرد عمر يوم واحد قسمت عشوائياً إلى مجموعتين، المجموعة الأولى كمجموعة ضابطة، والمجموعة الأخرى تم تجريعها بعنزة السالمونيلا جلينرم عن طريق الفم بأسترة إلى الحويصلة بجرعة ٠.٢ مللي محلول ملحي معقم يحتوي على $(3 \times 10^8 \text{ CFU/ml})$. وضعت جميع طيور التجريبية تحت الملاحظة اليومية لتسجيل الأعراض ومعدل الوفيات، وقد تم جمع عينات أنسجة كبد وقلب ورئة وأمعاء من الكتاكيت الميتة والمذبوحة من كل مجموعة بعد ١٢ ساعة، ١، ٢، ٣، ٤، ٧، ١٤، ٢١، ٢٨ يوماً من بداية العدوى. وقد أظهر الفحص الإكلينيكي للكتاكيت بعض الأعراض المرضية على شكل فقدان الشهية وبلادة مع إسهال أبيض عجيني والتجمع تحت مصدر الحرارة وكانت نسبة النفوق ٢٤.٤ %، وأظهر الفحص العيني تغييرات على شكل إحتقان شديد بالأعضاء الداخلية في المرحلة الأولى من التجربة كما شوهدت إلتهايات بالأغشية المحيطة لكل من القلب والكبد في نهاية التجربة، بالإضافة الى ذلك فقد لوحظت مناطق حمراء اللون على سطح الكبد، وبالفحص المجهرى وجد إحتقان وجلطات مع بؤر نزيقية وتغيرات تنكسية ونخر في الكبد وتجمعات من الخلايا الإلتهاية في كل من الكبد والقلب، كذلك شوهدت بعض المستعمرات البكتيرية في كل من الكبد والقلب، كما سجلت في بعض الحالات إلتهاب كبسولة الكبد وإلتهاب فبريني بغشاء التامور وإلتهاب مخاطى بالأمعاء، كذلك شوهد زيادة في عدد الخلايا الطلائية المبطنة للقنوات المرارية بالكبد، وبالنسبة للرئة شوهد إحتقان وجلطات وبؤر نزيقية وتورم حول الأوعية الدموية، كما لوحظ العديد من الأورام الحبيبية ونخر متجين في نسيج الرئة. ونخلص من هذه التجربة بأن العدوى التجريبية للسالمونيلا في كتاكيت التسمين تميزت بالتسمم الدموى في وقت مبكر يليه إلتهاب التامور المصلي الفبريني، وإلتهاب حول الكبد والأمعاء بعد اليوم الثاني للعدوى. وعلاوة على ذلك تم الكشف عن إصابات حبيبية في القلب في نهاية التجربة.