Pathology and colonization of internal organs after experimental infection of broiler chickens with *Salmonella* Gallinarum through oral or intraperitoneal routes

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Keywords

Broiler chicken – Chick – *Salmonella enterica* serovar Gallinarum – Typhoid – Experimental infection – India.

Summary

This paper describes pathological changes and the frequency of isolation of *Salmonella enterica* subsp. *enterica* serovar Gallinarum (O: 9, 12) from internal organs in broiler chicks experimentally infected through oral or intraperitoneal routes. The experiment was conducted on 110 one-week-old chicks divided into three groups: the CR group (30 chicks) was kept uninfected and served as control, the OR group (40 chicks) was inoculated orally with *Salmonella* Gallinarum (10^9 organisms/ml), and the IP group (40 chicks) was infected intraperitoneally with *Salmonella* Gallinarum (10^9 organisms/ml). Three birds from each group (dead or sacrificed) were observed at 3, 5, 7, 14, 21, 28, 35, and 42 days postinfection for evaluation of gross and histopathological changes in visceral organs, and for frequency of isolation of *Salmonella* Gallinarum from internal organs. Gross and histopathological changes were compared between infected groups by measuring mean lesion scores. The gross and histopathological changes in visceral organs, although similar in both infected groups, were more severe and observed at earlier stages of infection and in more birds in the IP group. There was however no significant difference between the two infected groups in the frequency of isolation of *Salmonella* Gallinarum from internal organs, even in fecal sheddings. It was therefore concluded that the intraperitoneal route should be primarily considered for inducing *Salmonella* Gallinarum infection in experimental trials.

■ INTRODUCTION

Fowl typhoid (FT) is a septicemic disease of poultry that causes considerable economic losses through mortality and increased morbidity. Infection of birds of all ages, in the field or experimentally, can result in very high mortality (5, 10). The disease is caused by the gram-negative bacterium *Salmonella enterica* serovar Gallinarum (31), a member of the Enterobacteriaceae family which is widely distributed throughout the world (34). *Salmonella* Gallinarum is highly adapted and seldom causes significant problems in hosts other than chickens, turkeys and pheasants (30, 34). No difference in susceptibility to *Salmonella* Gallinarum has been observed between local and commercial chickens (26). It was formerly known as *Shigella gallinarum*, when first isolated by Klein in England in 1889 (30). The disease was called fowl typhoid in 1902 (31).

FT has been eradicated in the commercial poultry production of developed countries, but is still a major problem in developing countries (22). In India, FT has long been plaguing the poultry industry, causing heavy economic losses due to mortality in young and adult chickens. Since it was first reported by Cooper and Naik (9) in India, the incidence of FT is on the increase and illustrated by the fact that *Salmonella* Gallinarum alone accounted for 32% of *Salmonella* of avian origin typed at the National Salmonella Centre (Veterinary), Izatnagar, India, from 1987 to 1995 (14). *Salmonella* Gallinarum has been found to be the predominant serotype and the major cause of mortality in poultry in India (28, 32).

Various strategies, i.e. novel antibiotics, vaccines, immunotherapeutics and antimicrobial feed additives, are currently explored to control *Salmonella* infection in poultry (3, 4, 23). The birds are

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routinely experimentally infected by *Salmonella* Gallinarum to evaluate efficacy of different drugs and vaccines. The alimentary tract is the natural route of *Salmonella* infection in poultry. Following oral ingestion, *Salmonella* penetrates the mucosal epithelium of the small intestine, interacting with columnar epithelium cells and microfold cells. *Salmonella* has been shown to survive and replicate within macrophages residing in the lymphoid follicles in the intestines. Macrophages have been found to play an important role in the dissemination of *Salmonella* to organs of the reticulo-endothelial system such as the liver, spleen and bursa (17). The experimental reproduction of FT in adult chickens *via* oral *Salmonella* Gallinarum challenge requires a very high titer as well as treatment with some reagents to reduce the effects of gastric juice (6, 35). This inherent difficulty in the reproduction of FT has been an obstacle to the experimental evaluation of vaccines as well as to understanding FT outbreaks in the field (6). Alternate routes, i.e. intraperitoneal and respiratory routes, have also been reported to induce experimental FT (5, 6). However, the comparative study of *Salmonella* Gallinarum infection through intraperitoneal and oral routes is scanty. Thus we explored the feasibility of using the intraperitoneal route in experimental birds as a cost effective alternative model to study FT further. The clinico-hematobiochemical changes have already been described in a previous paper (29); this article focuses on the gross and histopathological lesions and the isolation of the bacteria from visceral organs.

■ MATERIALS AND METHODS

Experimental birds and their management

The study was carried out in the experimental house of the Department of Veterinary Pathology, Faculty of Veterinary Science and Animal Husbandry, Shere-Kashmir University of Agricultural Sciences and Technology, Kashmir, India, with 110 unsexed one-day-old broiler chicks procured from a local hatchery. The birds were treated humanely during the whole period of the experiment and the work was agreed upon by the Institutional Animal Ethics Committee on ethical standards in animal experimentation (No AU/FVS/Estt/C-12/16638-40). The chicks used were from the same breeding flock. They were reared for a period of 49 days under strict hygienic conditions and maintained on broiler mash from day 1 till the end of experiment. Feed and water were given *ad libitum*. Bacteriological and serological examination showed that the birds were negative for *Salmonella* at the beginning of the study.

Salmonella *strain*

The *Salmonella* Gallinarum strain (from here on called "SG") used for inducing infection was isolated according to the standard method from a disease outbreak in a private broiler farm in Ganderbal area in October 2009 (21). The isolate was serotyped as *Salmonella enterica* subsp. *enterica* serovar Gallinarum with the antigenic structure O: 9, 12 by the National Salmonella and Escherichia Research Institute, Kasauli, Himachal Pradesh, India. The SG strain was selected after it was shown to be virulent following a preliminary infection experiment using seven-week-old commercial broiler chickens.

Experimental design

At day 7, before infecting the chicks with the SG strain, they were divided in three groups: uninfected (n = 30, control birds, group CR), orally infected (n = 40, group OR) and intraperitoneally infected (n = 40, group IP). The chicks of groups OR and IP were challenged with 10^9 organisms of SG strain in one millimeter of normal saline. The three groups of birds were kept separately in different rooms of the experimental house.

Pathological findings

Birds from each group were observed thrice daily (morning, noon and evening) for clinical signs and mortality. Following SG infection on day 7 (day 0 of infection), three birds from each group were euthanatized at 3, 5, 7, 14, 21, 28, 35 and 42 days postinfection (DPI) by cervical dislocation for gross and histopathological studies. However if any bird died due to infection during these specific days, the number of euthanatized birds was reduced to keep the total number (sacrificed + dead birds) equal to 3. Representative tissue samples from the liver, spleen, heart, lungs, bursa of Fabricius, kidneys and intestines were taken from both dead and euthanatized birds and fixed in 10% neutral buffered formalin. These were processed for paraffin embedding using alcohol as dehydrating agent and benzene as clearing agent. The sections were cut at 4-5 µm thickness and stained by the routine hematoxylin and eosin method (24).

Lesion scoring

Gross and histopathological lesions in birds of all groups were scored. Dead birds in OR and IP were also taken into consideration for scoring. Each of the gross and histopathological lesions in different organs was graded as mild, moderate or severe with corresponding intensity scores of 1, 2, and 3. The lesion score was determined for each of the organs in sacrificed and dead birds of a group by multiplying the gross lesion intensity by the number of birds showing that particular intensity of lesion and then by dividing the total number of birds (sacrificed + dead) examined for lesions.

Bacterial isolation and identification

The samples from the liver, spleen, heart and ceca as well as fecal samples were collected from each sacrificed or dead chick at 7, 21 and 35 DPI for bacterial isolation and identification. The samples were individually collected in Rappapart Vassiadis (Oxid, UK) and incubated at 37°C for 18-24 hours. They were then streaked onto brilliant green agar (BGA) and xylose-lysine-desoxycholate agar (XLD) and incubated at 37°C for 24 hours. The identity of suspected blank colonies from XLD and pink colonies from BGA were biochemically confirmed (40).

Statistical analysis

An analysis of variance (ANOVA) was used to test for level of significance of gross lesion scores (36). When differences were significant, Tukey test was used for comparison of mean gross lesion scores between different groups at 95% confidence level using SPSS 17 software (19). Similarly, total histopathological lesion scores were determined for each group and analyzed by ANOVA, and mean values in different groups were compared by Tukey test.

■ RESULTS

Table I shows the number of birds that died in the different experimental groups at various DPI. No mortality was recorded in the uninfected birds of the control group. Maximum mortality was observed in IP (47.50%) with most of the birds dying from 1 to 7 DPI, and peak mortality (four chicks) observed at 4 DPI. Clinical signs, previously described (29), were observed in IP as early as 12 hours after infection and in OR at 3 DPI. Recovery was noticed in OR from 21 DPI, and in IP from 15 DPI.

Table II shows the intensity of gross lesions in different organs of the experimentally-infected birds. The birds in CR did not exhibit any gross lesion in any of the organs examined throughout the

experiment. Gross lesions were first detected in the visceral organs of IP birds at 1 DPI and were not detected in OR birds until 3 DPI.

Initial changes in OR birds included congestion of visceral organs, enlargement of the liver and spleen, and thickening of intestinal mucosae. Necrotic foci on the surface of the spleen and liver were observed at 9 and 14 DPI. Other changes included a bronze discoloration of the liver and mild grayish nodular areas on the ventricular region of the heart.

In IP birds, the initial changes, in addition to congestion of visceral organs, included severe enlargement of the liver and spleen, and distention of the gall bladder. Prominent necrotic foci on the spleen were observed at 5 DPI. Small necrotic foci were visible on the liver as early as 5 DPI, however larger necrotic areas on the liver were observed in a bird which died at 10 DPI (Figure 1). Severe congestion and swelling of the kidneys were observed in IP. In this group, clearly visible grayish white nodules of various sizes were observed at 21 DPI projecting above the surface of the heart (Figure 2). In general, the gross changes in IP infected birds were similar to those observed in OR infected birds but the lesions were more severe and observed at earlier stages of infection and in a higher number of birds.

Table II also shows the intensity of histopathological lesions recorded in different organs of experimentally-infected birds. The histopathological changes in the liver of OR infected birds at 3 DPI were characterized by congestion of blood vessels, hemorrhages, and mononuclear cell infiltration around blood vessels, besides isolated foci of necrosis along with infiltration of heterophils observed at 14 DPI. IP birds showed similar lesions in the liver at 1 DPI, whereas at 3 DPI, aggregates of heterophils were observed in the parenchyma (Figure 3). There were numerous large-sized necrotic foci along with infiltration of heterophils at 7 DPI and 10 DPI (Figure 4).

Large necrosis areas causing severe depletion of the lymphoid tissue along with reticular endothelial cell hyperplasia were noticed in the spleen as early as at 5 DPI in IP (Figure 5). A similar type of lesions but with less intensity was observed in the spleen of OR chicks at 7 DPI onward (Figure 6). The heart showed degeneration of myocardial muscles at 14 DPI and 21 DPI due to infiltrating mononuclear cells, which was mild to moderate in OR (Figure 7) and extensive in IP resulting in atrophy, necrosis, and replacement of the heart muscles (Figure 8). The lungs showed congestion in the interlobular septa and hemorrhages in the parabronchi of both infected groups. The interlobular septa were infiltrated with mononuclear cells mixed with heterophils. The kidneys showed congestion, interstitial hemorrhages, mononuclear cell infiltration in the interstitial tissue along with mild degenerative changes in the tubular epithelium from 3 to 21 DPI in OR. These changes were accompanied by moderate to heavy degenerative changes in the tubular epithelium at 5 and 7 DPI in IP. In the bursa of Fabricius, a mild depletion of lymphoid tissues in the follicles along with infiltration of lymphocytes in interfollicular spaces were noticed from

Table I

Mortality pattern and number of birds sacrificed in the different groups of broiler chickens infected by oral or intraperitoneal routes with *Salmonella* Gallinarum

Days postinfection	CR		OR		IP	
	Died	Sacrificed	Died	Sacrificed	Died	Sacrificed
0	0	_	0	_	0	_
1	0	_	0	_	2	_
2	0	_	0	_	3	_
3	0	3	1	2	2	1
4	0	_	1	_	4	_
5	0	3	1	2	1	2
6	0	_	1	_	3	_
7	0	3	2	1	2	1
8	0	_	0	_	1	_
9	0	_	1	_	0	_
10	0	_	0	_	1	_
11	0	_	1	_	0	_
12	0	_	0	_	0	_
13	0	_	0	_	0	_
14	0	3	1	2	0	3
21	0	3	0	3	0	3
28	0	3	0	3	0	3
35	0	3	0	3	0	3
42	0	3	0	3	0	3
Total	0	24	9	19	19	19
Mortality (%)	0		22.50		47.50	

CR: control group; OR: chicks inoculated orally with *Salmonella* Gallinarum; IP: chicks infected intraperitoneally with *S*. Gallinarum

Table II

Number of birds showing gross and histopathological lesions of different intensities in various organs in broiler chickens infected by oral or intraperitoneal routes with *Salmonella* Gallinarum during the entire experiment[1]

	Num. of birds showing lesions of various intensities						Mean lesion score	
	OR[2] (n = 19 + 9 = 28)[4]			IP[3] (n = 19 + 19 = 38)			OR (n = 19 + 9 = 28)	IP (n = 19 + 19 = 38)
Intensity score[5]	**1**	**2**	**3**	**1**	**2**	**3**		
Gross lesion score								
Liver	10	4	1	17	10	3	0.75 ± 0.160[a]	1.21 ± 0.142[b]
Spleen	7	4	-	14	7	1	0.54 ± 0.140[a]	0.82 ± 0.135[a]
Heart	7	1	-	8	4	2	0.32 ± 0.103[a]	0.57 ± 0.144[a]
Kidneys	7	1	-	11	3	-	0.32 ± 0.103[a]	0.45 ± 0.116[a]
Lungs	6	-	-	9	-	-	0.21 ± 0.078[a]	0.23 ± 0.069[a]
Bursa	5	-	-	8	-	-	0.17 ± 0.073[a]	0.21 ± 0.067[a]
Intestines	11	2	-	10	-	-	0.54 ± 0.120[a]	0.26 ± 0.072[b]
Histopathological score								
Liver	17	9	2	14	18	6	1.48 ± 0.120[a]	2.12 ± 0.114[b]
Spleen	12	9	-	18	12	3	1.07 ± 0.145[a]	1.34 ± 0.134[b]
Heart	4	5	1	9	8	6	0.60 ± 0.173[a]	1.13 ± 0.181[b]
Kidneys	5	6	-	9	8	1	0.60 ± 0.157[a]	0.73 ± 0.153[a]
Lungs	11	-	-	17	-	-	0.39 ± 0.093[a]	0.44 ± 0.081[a]
Bursa	9	-	-	12	1	-	0.32 ± 0.089[a]	0.37 ± 0.087[a]
Intestines	11	3	2	12	2	-	0.89 ± 0.171[a]	0.42 ± 0.097[a]

[1] From day 0 of infection (i.e. at 7 days of age) until 42 days postinfection
[2] OR: chicks orally inoculated with *Salmonella* Gallinarum
[3] IP: chicks intraperitoneally infected with *Salmonella* Gallinarum
[4] Data in parentheses indicate the number of birds in the group (sacrificed + dead, respectively).
[5] 1 = mild; 2 = moderate; 3 = severe
[a,b] Means on the same row with different superscripts differ significantly ($p < 0.05$).
Note: no lesions were observed in any of the organs of the control birds throughout the experiment.

14 DPI onward in OR. In IP, in addition to depletion of lymphocytes, atrophy of bursal follicles, degenerative changes and slight metaplastic changes (in the epithelium separating the cortex from the medulla) were also evident at the early stage of infection. In OR birds severe catarrhal enteritis was observed, characterized by congestion, marked goblet cell hyperplasia, infiltration of heterophils and mononuclear cells in the lamina propria, mucosa and submucosa, and degeneration and desquamation of the epithelium. These changes were less prominent in IP birds.

Lesion scores varied between the different organs of a group as well as between the two infected groups. Mean gross lesion scores for the liver and mean histopathological scores for the liver, spleen and heart were significantly higher ($p < 0.05$) in IP than in OR birds (Table II). However, mean gross lesion scores and mean histopathological scores for the intestines were significantly higher ($p < 0.05$) in OR than in IP birds (Table II).

SG was isolated from all the cultured samples of dead chickens. No significant differences between the two infected groups in their frequency of isolation from internal organs were observed at any time postinfection. Mean SG isolation in OR was 67% from the liver, 33% from the spleen and the heart blood, 55% from ceca and 44% from fecal samples, whereas in IP it was 78% from the liver, 44% from the spleen, heart and ceca, and 33% from fecal samples (Table III).

Table III

Isolation of *Salmonella* Gallinarum from visceral organs of broiler chickens infected by oral or intraperitoneal routes at various days postinfection

	OR			IP		
	Positive samples			Positive samples		
	7 DPI	21 DPI	35 DPI	7 DPI	21 DPI	35 DPI
Liver	3/3	2/3	1/3	3/3	2/3	2/3
Spleen	2/3	1/3	0/3	3/3	1/3	0/3
Heart blood	2/3	1/3	0/3	2/3	2/3	0/3
Ceca	2/3	1/3	1/3	2/3	1/3	1/3
Feces	2/3	0/3	2/3	2/3	0/3	1/3

OR: chicks orally inoculated with *Salmonella* Gallinarum
IP: chicks intraperitoneally infected with *Salmonella* Gallinarum
DPI: days postinfection
Note: *Salmonella* Gallinarum was not isolated from any organs at any stage from the control group.

Figure 1: *Liver of a bird intraperitoneally infected with* Salmonella *Gallinarum showing areas of necrosis at 10 days postinfection.*

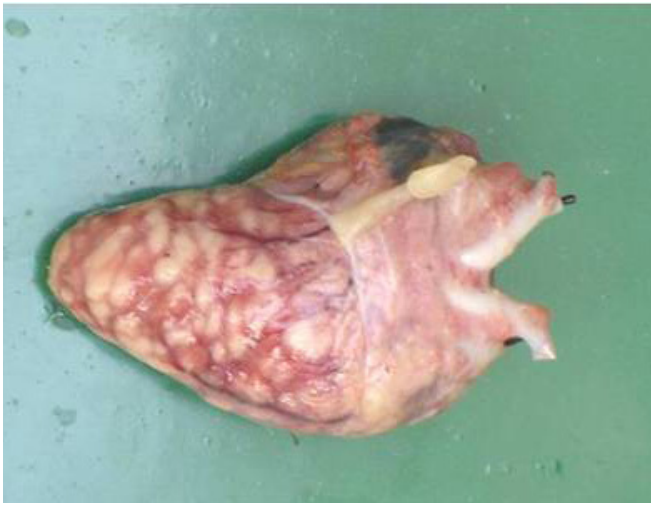

Figure 2: *Heart of a bird intraperitoneally infected with* Salmonella *Gallinarum showing white nodules at 21 days postinfection.*

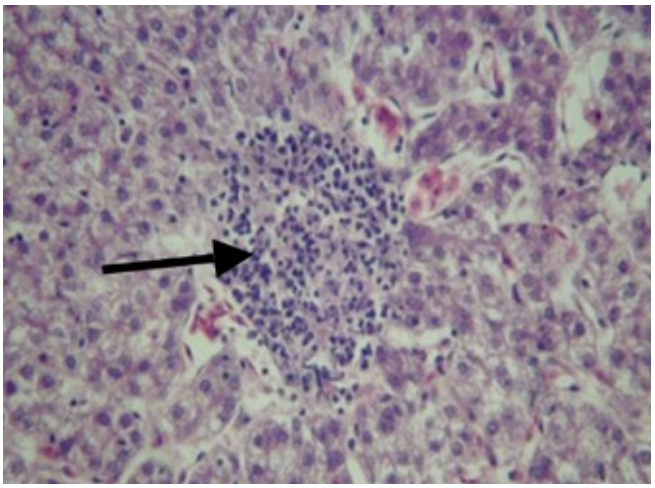

Figure 3: *Liver of a bird intraperitoneally infected with* Salmonella *Gallinarum showing aggregates of heterophils in parenchyma (arrow) at 3 days postinfection. Hematoxylin and eosin (x 1280).*

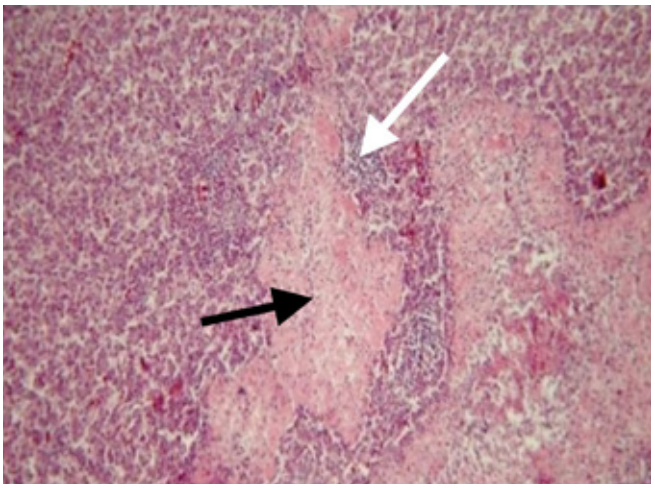

Figure 4: *Liver of a bird intraperitoneally infected with* Salmonella *Gallinarum showing large areas of necrosis (black arrow) surrounded by heterophilic infiltration (white arrow) at 7 days postinfection. Hematoxylin and eosin (x 960).*

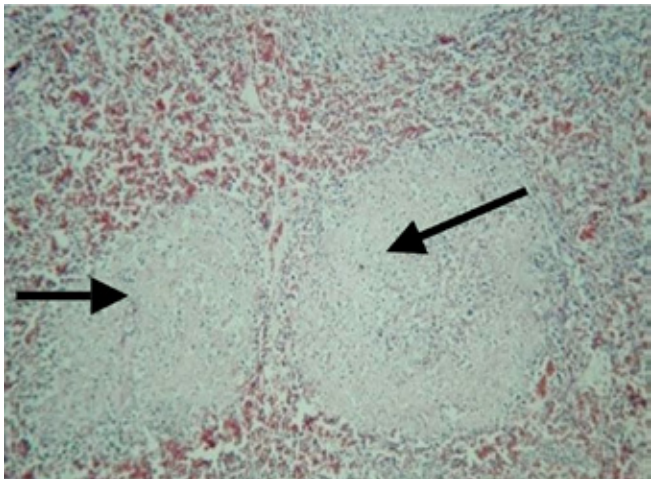

Figure 5: *Spleen of a bird intraperitoneally infected with* Salmonella *Gallinarum showing severe necrosis (arrow) and congestion along with marked depletion of lymphocytes at 5 days postinfection. Hematoxylin and eosin (x 960).*

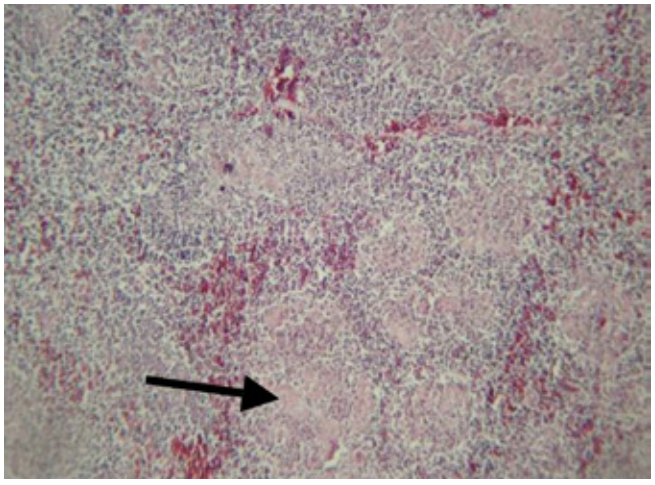

Figure 6: *Spleen of a bird orally infected with* Salmonella *Gallinarum showing depletion of lymphocytes along with areas of necrosis (arrow) at 7 days postinfection. Hematoxylin and eosin (x 240).*

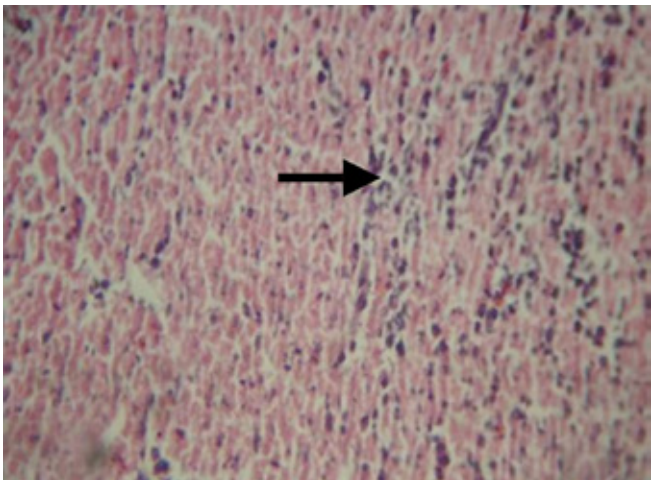

Figure 7: *Heart of a bird orally infected with* Salmonella *Gallinarum showing disruption of myocardial muscles due to infiltrating mononuclear cells (arrow) at 21 days postinfection. Hematoxylin and eosin (x 12,500).*

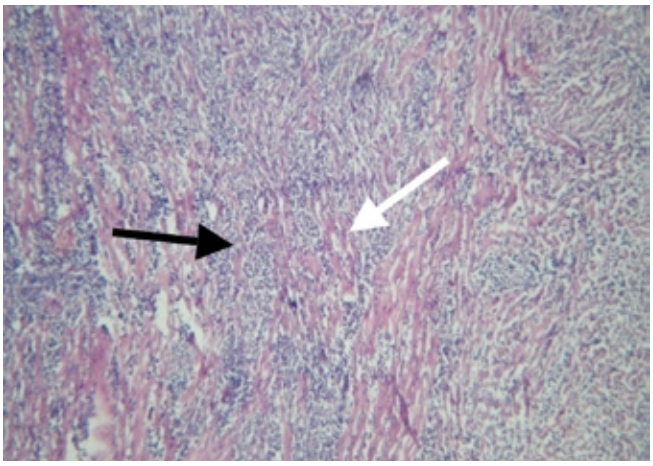

Figure 8: *Heart of a bird intraperitoneally infected with* Salmonella *Gallinarum showing fragmentation (white arrow) and replacement of myocardial fibers with heavy infiltrating mononuclear cells (black arrow) at 21 days postinfection. Hematoxylin and eosin (x 960).*

■ DISCUSSION

In the present study, FT was reproduced experimentally through both oral and intraperitoneal routes by locally isolated *Salmonella* Gallinarum strain to study various pathological alterations and the frequency of bacterial isolation from internal organs. We previously described the clinical signs, mortality and hematobiochemical changes (29). They correlate with the bacterial isolation, and gross and microscopic lesions of the disease.

The most common lesions observed were necrosis, degeneration, hemorrhages and infiltration of leukocytes, in conformity with earlier reports (12, 15, 26). However, the distribution and intensity of these lesions in various organs following the two routes of inoculation differed. The earlier appearance of clinical signs (29) and lesions in IP birds could be used to reduce expense and timing by diminishing the number of studied birds and hours of experimentation.

Gross and microscopic lesion scores suggested that the liver and spleen were the primary target organs involved in SG infection, irrespective of the route of inoculation. These observations are similar to those from Al-Shabibi (2) on *Salmonella* Typhimurium infection. Severe catarrhal enteritis, which was more prominent in orally infected birds, has also been reported by Prasanna et al. (33). The sloughing of superficial layers of villi revealed the damage to the integrity of the intestinal epithelium, resulting in the translocation of bacteria to other tissues in OR birds. Colonization of visceral organs including the liver and spleen occurs when *Salmonella* is not cleared by the host immune system, resulting in systemic infection (1). The lesions in the liver, spleen, heart, kidneys, bursa, intestines and lungs revealed the invasive potential of the *Salmonella* Gallinarum strain used and its pathogenicity.

In general, pathological changes were of less intensity in OR birds than in IP birds, as suggested by the fact that mean gross lesion scores for the liver, and mean histopathological scores for the liver, spleen and heart were significantly higher in IP birds than in OR birds. This could be because only a small proportion of the bacteria in OR (compared to IP) were able to reach visceral organs due to the antagonistic effects of low gastric pH (37) and inhibitory effects of the normal intestinal flora (8). As reported by Christensen et al. (7), viable counts of approximately 10^4 colony-forming units of *Salmonella* Gallinarum in the spleen and liver are necessary for the development of significant pathological and hematological changes. Environmental conditions (including pH, temperature and growth in chicken tissues) can also affect the expression of *Salmonella* Gallinarum virulence factors such as flagella and fimbriae, outer membrane proteins and iron uptake systems (39). The presence of B and T lymphocytes in the upper gastrointestinal tract (25, 38) and anti-*Salmonella* IgA in the crops of birds have also been reported to counter oral infection (18). These conditions make difficult the experimental reproduction of FT through the oral route. The intraperitoneal route of infection could be an alternative to overcome these difficulties in experimental trials where the oral route of infection is not essential.

Isolation of SG in the liver and spleen of dead chicks suggested that death originated from FT. No bacteria were isolated from the birds of the control group. A large degree of similarity between OR and IP birds was also observed in the frequency of isolation of samples from liver, spleen, heart blood, cecum and feces in the present study. Most of these samples were positive at one week postinfection but only a small percentage were still positive at three weeks postinfection. This observation concurs with that of Wigley et al. (41). The ability of *Salmonella* Gallinarum strain to invade the liver and spleen, although indicative of a systemic infection, has not always correlated with the frequency of fecal shedding of the pathogen. The decrease in the rate of fecal shedding of the bacterium after one week postinfection in both groups agrees with earlier findings (13, 19, 20). Ishola (20) reports that the rate of fecal shedding decreases from one to four weeks postinfection with *S. enteritidis*. The decline in the rate of fecal shedding or re-isolation from visceral organs indicates a reduction in the level of systemic infection in birds, probably through a humoral and cell mediated immune response (16, 27). Both responses peak at three to four weeks postinfection, a point that coincides with bacterial clearance (41). The percentage increase of birds shedding the organism at week 5 postinfection in both infected groups could be due to a gradual reduction in the immune response. Oral challenge at relatively low doses, as it is likely to occur in broiler chickens under natural outbreaks, may not cause systemic infection but rather intestinal carriage which is more persistent (11). The presence of more than 50% birds as silent carriers in orally infected birds in the present study indicates that the majority of birds may act as carriers for other birds.

■ CONCLUSION

From the present study, it can be deduced that the intraperitoneal route can be considered as one of the alternative cost-effective methods for inducing *Salmonella* Gallinarum infection in experimental trials of novel drugs, feed additives, etc., as the induction of FT in birds using that route revealed similar clinical signs and pathological lesions (although more severe) as those observed with the oral route.

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Résumé

Nazir S., Kamil S.A., Riyaz A., Mir M.S., Darzi M.M., Yasine A., Goudar K.S. Pathologie et colonisation des organes internes après infection expérimentale des poulets de chair par *Salmonella* Gallinarum par voies orale ou intrapéritonéale

Cet article décrit les changements pathologiques et la fréquence d'isolement de *Salmonella enterica* subsp. *enterica* serovar Gallinarum (O : 9, 12) des organes internes des poussins de chair infectés expérimentalement par voie orale ou intrapéritonéale. L'expérience a été menée sur 110 poussins, âgés d'une semaine et divisés en trois groupes : le groupe CR (30 poussins), non infecté, a servi de groupe témoin, le groupe OR (40 poussins) a été inoculé par voie orale (10^9 organismes/ml), et le groupe IP (40 poussins) a été infecté par voie intrapéritonéale (10^9 organisms/ml). Trois poussins de chaque groupe (morts ou sacrifiés) ont été examinés 3, 5, 7, 14, 21, 28, 35 et 42 jours après l'infection afin d'évaluer l'importance des lésions macroscopiques et histopathologiques, et de déterminer la fréquence d'isolement de *Salmonella* Gallinarum dans les organes internes. Les différences entre les groupes infectés ont été évaluées par la comparaison des scores moyens des lésions macroscopiques et histopathologiques. Bien que similaires dans les groupes OR et IP, les lésions des organes viscéraux ont été plus sévères, plus précoces et présentes chez un plus grand nombre de volailles dans le groupe IP. Cependant, il n'y a pas eu de différence significative entre les deux groupes infectés en termes de fréquence d'isolement de *Salmonella* Gallinarum dans les organes internes ni dans les fientes. Il a été conclu que la voie intrapéritonéale devrait être privilégiée pour induire une infection à *Salmonella* Gallinarum lors d'essais expérimentaux.

Mots-clés : Poulet de chair – Poussin – *Salmonella enterica* serovar Gallinarum – Typhoïde – Infection expérimentale – Inde.

Resumen

Nazir S., Kamil S.A., Riyaz A., Mir M.S., Darzi M.M., Yasine A., Goudar K.S. Patología y colonización de órganos internos después de una infección experimental en pollos de engorde con *Salmonella* Gallinarum por vías oral e intraperitoneal

El presente artículo describe los cambios patológicos y la frecuencia de aislamiento de *Salmonella enterica* subsp. *enterica* serovar Gallinarum (O: 9, 12) de órganos internos en pollos de engorde, infectados en forma experimental por vías oral e intraperitoneal. El experimento fue llevado a cabo en 110 pollos de engorde de 1 semana de edad, divididos en tres grupos: grupo CR (30 pollos) mantenido sin infección y sirvió de control, grupo OR (40 pollos) inoculados oralmente con *Salmonella* Gallinarum (10^9 organismos/ml) y grupo IP (40 pollos) infectados intraperitonealmente con *Salmonella* Gallinarum (10^9 organismos/ml). Tres aves de cada grupo (muertas y/o sacrificadas) fueron observadas al día 3, 5, 7, 14, 21, 28, 35 y 42 post infección, para evaluar cambios macro e histopatológicos en los órganos viscerales y asesorar la frecuencia del aislamiento de *Salmonella* Gallinarum de órganos internos. Los cambios macro e histopatológicos fueron comparados entre los grupos infectados mediante medidas de graduación promedio de las lesiones. Los cambios macro e histopatológicos en los órganos viscerales, a pesar de ser similares en ambos grupos infectados, fueron más severos y observados a un estadio más temprano de infección y en más aves en el grupo IP. Sin embargo no hubo diferencia significativa entre los dos grupos infectados en cuanto a la frecuencia del aislamiento de *Salmonella* Gallinarum de los órganos internos, incluyendo en efusiones fecales. Por lo tanto se concluye que la ruta intraperitoneal debe ser considerada ante todo para inducir la infección por *Salmonella* Gallinarum en estudios experimentales.

Palabras clave: Pollo de engorde – Pollito – *Salmonella enterica* serovar Gallinarum – Tifoidea – Infección experimental – India.