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Pathological and Molecular Studies on Some Pathogenic Bacteria in *Meleagris Gallopavo* of Hybrid Converter Breeds in Egypt

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Abstract

TURKEYS (*Meleagris gallopavo*) are prone to various pathogenic bacterial diseases that affect their production, yet little is known about their pathogenicity in hybrid converter breeds. Therefore, this study is an investigation of the most common bacterial diseases affecting turkeys and their pathological impact on the various tissues. Three turkey farms of hybrid converter breeds were checked in 2023 for any clinical signs. Recently, dead birds were necropsied, and all gross lesions were recorded. Samples from each farm were screened with real time qPCR for the presence of *Salmonella typhimurium*, *E. coli*, *Clostridium perfringens*, *Pseudomonas aeruginosa*, *Mycoplasma gallisepticum*, and *Pasteurella multocida*. Tissue samples were subjected to histopathological techniques to determine various microscopic lesions. Real time qPCR revealed that *Salmonella*, *E. coli*, and *Pseudomonas* were positive in all examined farm samples. While clostridia and *Mycoplasma* were positive in one farm sample, On the other hand, none of the tested samples were positive for *Pasteurella*. Histopathological examination revealed congestion, necrosis, and leukocytic cell infiltration in multiple organs. This histopathological evidence highlights the health risks associated with these infections in the farm. Further investigation is needed to determine the source of contamination and implement appropriate control measures to prevent the spread of these pathogens.

Keywords: Turkeys, Bacteria, Meleagris gallopavo, Pathology, Molecular.

Introduction

Salmonella is a member of the Enterobacteriaceae family and has two genetically distinct species, one of which (*Salmonella enterica*) has six subspecies determined by biochemical reaction patterns. Only one subspecies (S. *enterica* subspecies *enterica*) is associated with disease in warm-blooded animals, despite the fact that it contains over 2,500 motile and nonhost adapted serovars such as S. *enterica* subspecies *enterica* serovar Enteritidis and *S. enterica* subspecies *enterica* serovar Typhimurium [1,2]. Clinical signs may also include diarrhea with urate staining of the vent, decreased feed consumption, and huddling near heat sources. Adult birds are more resistant to infection, and diseased individuals are often

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asymptomatic, although they can pass infection on to hatchlings via eggs [1].

In acute to subacute cases, the lesions were multiple and included multifocal necrosis of hepatocytes with fibrin accumulation and heterophilic infiltration in the hepatic parenchyma. The heterophils with a few lymphocytes and plasma cells infiltrated the Periportal area. In chronic cases, especially when the heart is involved with large nodules, the liver showed chronic passive congestion with interstitial fibrosis. The spleen revealed severe congestion with fibrin exudate in acute stages, and severe hyperplasia of the mononuclear phagocytic cells in the later stages [3].

Colibacillosis refers to localized or systemic infection caused by avian pathogenic Escherichia coli (APEC) either entirely or partly [4]. In mammals, colibacillosis was primarily enteric or urinary tract disease, while in poultry, it was most often a localized or systemic disease [5]. The causative agent of colibacillosis is *E. coli* which belongs to the family Enterobacteriaceae, while other infectious and noninfectious agents either predispose the bird for infection or increase the intensity of the disease [4-7].

Clinical signs varied from inappropriate to unresponsiveness according to the type of disease produced by *E. coli*. In general, localized infection resulted in milder and fewer clinical signs than systemic diseases [4]. Forms of the disease include hemorrhagic septicemia, colisepticemia, coligranuloma (Hjarre's disease), venereal colibacillosis, swollen-head syndrome, airsacculitis (chronic respiratory disease (CRD)), peritonitis, *Coliform* cellulitis, turkey osteomyelitis complex, salpingitis, orchitis, panophthalmitis, omphalitis/yolk sac infection, and enteritis [4,7].

E. coli could cause severe infection in the upper and lower respiratory system. If the infection was localized in the upper respiratory system, swollen head syndrome produced and characterized by facial and sinus swelling [8]. While fibrinous pneumonia with fibrinous whitish yellow exudates in the air sacks was produced in case of lower respiratory infection [9]. In older cases, the intestine showed congestion, hemorrhage, desquamation, and necrosis. Focal areas of diphtheritic enteritis were presented with heterophilic infiltration and necrotic tissue over the intestinal mucosa. Villus vacuolation, hypertrophy and flattening of intestinal mucosa were recorded [5,10].

Mycoplasma gallisepticum (MG) is the causative agent of infectious sinusitis in turkeys. It belongs to the genus *Mycoplasma* (class Mollicutes). Previously, it was designated as serotype A *Mycoplasma*. It is phylogenetically classified in the Pneumoniae subgroup of *Mycoplasma*s [11,12]. Turkeys infected with MG suffered sinusitis, coughing, dyspnea, tracheal rales, listlessness, and weight loss. In complicated cases turkeys exhibited nasal discharge, foamy eye secretions, infraorbital sinuses swelling which may result in closed eyed. In some cases, turkeys displayed nervous signs including torticollis and opisthotonos. A drop in egg production was also reported in breeding flocks [11,13,14].

Gross lesions in infected turkeys consisted primarily of mucosal congestion and catarrhal exudate in the upper respiratory tract including nasal and paranasal passages, trachea, bronchi, and air sacs. Sinusitis was usually most prominent in turkeys with accumulation of mucoid to caseous exudate. Air sacs filled with caseous exudate that may be focal or diffuse. Varying degrees of pneumonia were seen. In severe cases, caseous airsacculitis and fibrinous perihepatitis and pericarditis resulted in high mortality [11,13,15].

Microscopically, thickening of mucous membranes of upper respiratory tracts due to mononuclear cells infiltration and hyperplasia of lymphoid follicles was reported. Transformation of the respiratory epithelium from ciliated pseudo-stratified columnar to nonciliated low cuboidal or squamous epithelium was described. Lungs showed pneumonic areas, lymphofollicular changes, and granulomatous lesions. Turkey brains in cases of encephalitic revealed acute to subacute encephalitis with perivascular lymphocytic cuffing, vasculitis, focal to multifocal necrosis, and leptomeningitis [11,15].

There are many species of *Pseudomonas* but *Pseudomonas aeruginosa* is the most common species causing infections, especially yolk sac infections and septicemia in poults and young chicks. In general, *Pseudomonas* is an opportunist that produces respiratory infections. It is often associated with soil, water, and humid environments [16]. Clinical signs vary depending on whether infections are localized or systemic, but in general, it may include respiratory distress, anorexia, stunting lameness, lassitude, neurologic

signs, swelling of wattles, head, and sinuses, swelling of hock joints or foot pads, diarrhea, and conjunctivitis [16,17]. Lesions of *p. aeruginosa* were various and include edema and fibrin in subcutaneous tissue with hemorrhage, exudate in affected joints, airsacculitis, pericarditis, hepatic serositis, and pneumonia. Necrotic foci in spleen, liver, kidney, and brain as well as cellulitis, sinusitis, conjunctivitis, and keratitis were recorded [16-19].

Ulcerative enteritis (UE) is a disease caused by *Clostridium colinum*. The disease affects young quail mainly but also occurs in chickens, turkeys, and other avian species. Necrotic enteritis (NE) is a disease caused by the toxins produced by pathogenic strains of *Clostridium perfringens* type A, C, and G [20,21]. Birds infected with UE suffered from diarrhea, which was watery at first then became hemorrhagic. Infected birds became listless with eyes partly closed with dull ruffled feathers. In advance cases, notable emaciation with pectoral muscles atrophy was seen. The mortality rate typically ranged from 2% to 10% in turkeys [22]. Birds with NE suffered from diarrhea, emaciation, anorexia, and dehydration with ruffled feathers. In some cases, birds were found dead without clinical signs e but with very characteristic intestinal lesions upon necropsy. Mortality rates were up to 50% [20].

Acute lesions were characterized by severe ulcerative and hemorrhagic enteritis. Ulcers were variable in size and surrounded by a hemorrhagic halo. In some cases, ulcers were deep and involved the whole thickness of the intestinal wall, causing perforation and peritonitis. Subacute or chronic lesions were characterized by multiple large, roundish yellow ulcers surrounded by hemorrhages, in any part of the small or large intestine [22].

Material and Methods

Sampling

During 2023, regular visits to three turkey farms of hybrid converter breeds in the Delta region, Egypt, were conducted. Clinical examination of diseased birds was performed and recorded. Following clinical examination, the diseased turkeys were euthanized and necropsied. Freshly dead birds were also necropsied. Representative samples were collected from ailing and dead turkeys. Clinical signs and postmortem findings were recorded. Imaging of clinical signs and gross lesions was done by digital camera. Representative tissue samples were taken from various tissues of all necropsied birds then pooled and labeled and were frozen at -20 °C until time of nucleic acid extraction for real time qPCR. Part of tissue samples with gross lesions was labelled and fixed separately in 10% neutral buffered formalin at room temperature for 12:48 hours for histopathology.

This study was approved by the ethical committee of the Faculty of Veterinary Medicine, Mansoura University, Code: Ph. D100/ for 2021.

Quantitative RT-PCR (qRT-PCR) for bacterial detection

Extraction of nucleic acids from collected samples was done by QiaAmp bacteria DNA extraction kits according to the manufacturer's protocol. Real time PCR was used to detect *Salmonella*, *Coliforms*, *Pseudomonas*, *Mycoplasma*, *Clostridium* and *Pasteurella*. The primers used were mentioned in Table 1. PCR amplifications were conducted in a 25 µL reaction containing the following mix: 1 µL of each primer (10 mM), SYBR Green PCR Master Mix (12.5 µL) (Qiagen, Hilden, Germany), sterile PCR-grade water (9.5 µL) and specific genomic DNA (2 µL). For the development of standard curves, genomic DNA obtained from pure bacterial cultures was serially diluted tenfold.

The cycling condition was conducted at 50 ◦C for 30 min, the cDNA was denatured at 94 ◦C for 15 min, and then 40 cycles of 95 ◦C for 15 s and 60 ◦C for 30 s. The Ct threshold cycle values were then plotted against the bacterial DNA copy counts to generate standard calibration curves. The standard curves represented log10 CFU/gram of the fecal contents and quantified the bacterial concentrations in each DNA sample.

Histopathology

The fixed tissue specimens were trimmed under fume hood to fit into cassettes and labeled. Tissue samples were routinely processed in ascending grades of ethanol (ethanol 70% for1hr then 95% for 1hr two time then 100% for 1hr two changes), xylene (two changes) for 1hr then embedded in melted paraffin. Serial sections of 4-5µm were cut for H&E staining and IHC using microtome then placed on clean glass slides. At time of staining, slides were dewaxed by xylene, rehydrated by descending grades of ethanol then washed, placed in hematoxylin for 3-5 min, washed by tap water and counter stained by eosin for 8 min. Finally, slides were washed in water, dehydrated by

ethanol, cleared by xylene, mounted with quick mount. The stained slides were examined by light microscope [23].

Results

Clinical signs and gross lesions

In all farms, turkeys exhibited depression, respiratory manifestations, weakness, and diarrhea in some cases, dropped wings were noted. In the first farm, gross examination revealed congested intestines, enlarged liver with necrotic foci, splenomegaly, and accumulation of fibrinopurulent exudate on the surface of heart, lung, and protonium Figure 1. In the second farm, Gross lesions consisted primarily of sinusitis with accumulation of mucoid to caseous exudate. Air sacs filled with caseous exudate that may be focal or diffuse. Varying degrees of pneumonia were observed. In severe cases, caseous airsacculitis and fibrinous perihepatitis and pericarditis resulted in high mortality Figure 2. In the third farm, lesions were characterized by hemorrhagic, distended, and friable intestine. Ulcers were variable in size and surrounded by a hemorrhagic halo. Liver lesions were minimal and included light yellow mottling to multiple large, irregular, gray circumscribed foci. The spleen was congested, enlarged, and hemorrhagic, with or without multifocal necrotic areas Figure 3.

Real-time PCR for bacteria?

The Ct value of the positive bacterial samples and count was as shown in Table 2. Ct values of amplified nucleic acid showed positive amplification signals for *Salmonella* and *E. coli* in clinically collected samples from diseased birds Graph 1. Ct values of amplified nucleic acid showed positive amplification signals for *Clostridium* in clinically collected samples from diseased birds Graph 2. Ct values of amplified nucleic acid showed positive amplification signals for *Pseudomonas* in clinically collected samples from diseased birds Graph 3. Ct values of amplified nucleic acid showed positive amplification signals for *Mycoplasma* in clinically collected samples from diseased birds Graph 4. Positive and negative control samples were included. None of the tested samples were positive for *Pasteurella*.

Histopathological examination

Histopathological findings of samples from Farm 1 are shown in Figure 4 A-I. The liver showed congestion and fatty change in hepatocytes. Tracheal wall showed destruction of epithelial lining trachea with congested blood vessels The spleen revealed fibrinous deposits and haemorrhage in red pulp. The intestine demonstrated sloughing of villi with congested blood vessels extensive necrosis in the mucosa and submucosa, with lumen filled with admixture of necrotic debris, fibrin and heterophils. The heart showed interstitial fibrosis with few leukocytic cell infiltration. Lungs showed multiple necrotic areas replacing pulmonary tissue, marked interstitial edema with heavy heterophils infiltration.

Histopathological findings of samples from Farm 2 are shown in Figure 5 A-I. The liver showed congestion with perivascular fibroplasia with leukocytic cells infiltration. The trachea showed hyperplasia of epithelial lining, edema, and leukocytic cells infiltration. congested capillaries in lamina propria. The lungs demonstrated congestion, perivascular edema, fibrosis with few mononuclear cell infiltrations. The intestine showed short blunt villi. The heart showed vacuolations of cardiomyocytes. The spleen showed congestion, and perivascular haemorrhage in red pulp.

Histopathological findings of samples from Farm 3 are shown in Figure 6 A-I. The liver showed congestion and vacuolations of hepatocytes. The trachea showed hyperplasia of the lining epithelium, congested blood vessels, and edema in lamina propria and submucosa. The lungs showed congested blood vessels with perivascular edema, severe interstitial edema with leukocytic cells infiltration, and congested blood vessels. The intestine showed congested blood vessels with loss of epithelium covering villi. The heart showed dilated blood vessels and perivascular fibrosis in heart.

Discussion

Mixed infections were not much studied in turkeys. However, recent research has shown that mixed infections can have a significant impact on the health and productivity of turkey flocks [24-26]. Understanding the interactions between different pathogens in mixed infections is crucial for effective disease management strategies. This study outlines the interactions among bacterial pathogens in three farms in the Delta region of Egypt.

In the first and third farm, diseased birds were positive for *E. coli*, *Pseudomonas*, and *Clostridium* by qRT-PCR so gross and histopathological findings were similar. Necropsied birds showed congested ulcerated intestines, enlarged liver with

necrotic foci, splenomegaly, and accumulation of fibrinopurulent exudate on the surface of heart, lung, and protonium. The ulcers and necrosis of the intestine were attributed to the replications of *Clostridium* in the intestinal mucosa agreed with Opengart and Boulianne [20], Jackson [21] . While accumulation of fibrins in visceral organs and lesions of liver and lungs may be attributed to the mixed infection with *E. coli* and *Pseudomonas* [4,9,17].

On the other hand, birds of the second farm were positive for *E. coli*, *Pseudomonas*, *Salmonella*, *Mycoplasma*, and *Clostridium*. This explains the high intensity of the clinical signs in this farm. Necropsied birds suffered sinusitis with accumulation of mucoid to caseous exudate, air sacculitis with caseous exudate, pneumonia, fibrinous perihepatitis, and fibrinous pericarditis. Although gross lesions may be resulted from the mixed infection of all the five bacteria, *Mycoplasma* and *Salmonella* played the major role in theses lesions as reported by Wijesurendra, et al. [27],Ferguson, et al. [28],Beyaz, et al. [29].

The interaction among multiple bacterial species in a host can lead to increased virulence and disease severity. It seemed that the interaction among bacteria favors the growth of each other. This was evidenced by bacterial count which was higher in the second farm than other farms. Histopathological findings confirm the high pathogenicity of these bacteria which was evident by the lesions in almost all organs. This study highlights the interaction among three to five bacterial species in hybrid converter breeds of turkeys.

Conclusion

To our knowledge, this is the first report that describes the interaction of five different bacteria species in hybrid converter breeds of turkeys in Egypt. This study provides valuable insights into the dynamics of bacterial interactions and the possible pathological findings within hybrid converter breeds of turkeys, shedding light on potential implications for turkey farming practices in Egypt. Further research is needed to fully understand the impact of these bacterial interactions on turkey health and productivity.

Acknowledgment

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Conflicts of interest

The authors declare no conflict of interest.

Funding statement

This research received no external funding.

TABLE 1. Showing the Primers used in RT-qPCR.

Primer	Nucleotide sequence (5′→3′)	Reference
Clostridium	F: AAAGGAAGATTAATACCGCATAA R: ATCTTGCGACCGTACTCCCC	[30]
Pasteurella	F: ATCCGCTATTTACCCAGTGG R: GCTGTAAACGAACTCGCCAC	[31]
Coliforms	F: ATGAAAGCTGGCTACAGGAAGGCC R: ACCATGCCGTGGGTTTCAATATT	[32]
Pseudomonas	F: AATGCGGGATGCTGCTCT R: GGTCGGTCTCCTCGAACTCTT	[33]
Salmonella	F: ACAGTGCTCGTTTACGACCTGAAT R: AGACGGCTGGTACTGATT ATAAT	[34]
Mycoplasma	F: GCTGGGTTGATTGTTGTTTCTT R: TCTTCACGTTCTTGGATCATCAT	[35]

TABLE 2. Showing the Ct, NA concentration value, and count of positive bacterial samples in three farms

Farm		*Salmonella*	*Coliform*	*Clostridium*	*Pseudomonas*	*Mycoplasma*	*Pasteurella*
Farm 1	**Count**	**Negative**	$8.76x\ 10^7$	$2x\ 10^3$	$1.5x\ 10^4$	**Negative**	**Negative**
	Ct		27.89	28.83	26.87		
	NA Conc			48			
Farm 2	**Count**	8.88 x 103	$4.05x\ 10^6$	$2.25x\ 10^3$	$4.76x\ 10^3$	$2.79x\ 10^3$	**Negative**
	Ct	20.31	28.71	30.05	27.84	28.97	
	NA Conc			45			
Farm 3	**Count**	**Negative**	$7.93x10^4$	625	$2.5x10^3$	**Negative**	**Negative**
	Ct		24.71	31.05	28.97		
	NA Conc			50			

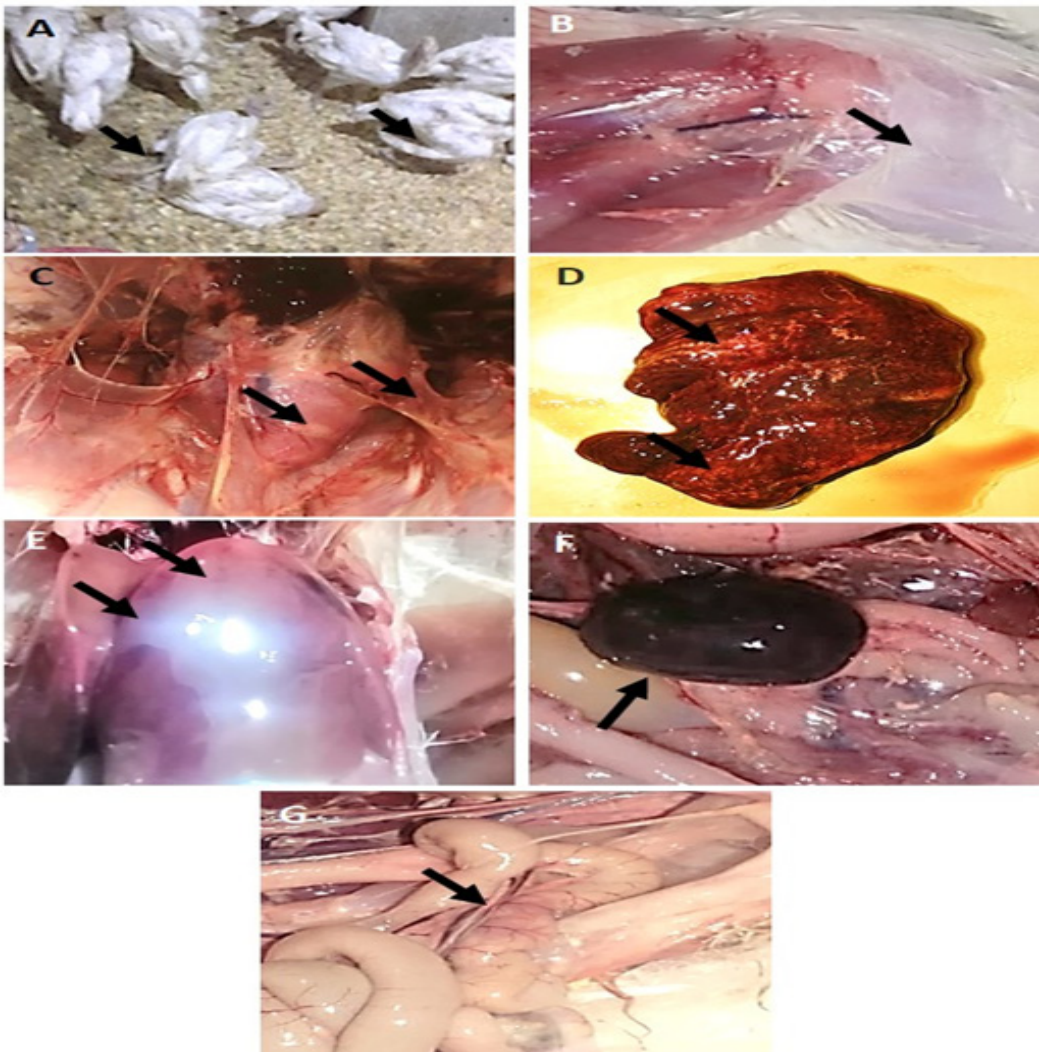


Fig. 1 (A-G). Macroscopic pictures showing clinical signs and gross findings in poults of farm 1. Turkeys are unable to stand with dropped wings (arrows) (A), inflamed joint (arrows) (B), fibrinous pericarditis (arrows) (C), dark congested lung with multiple necrotic areas (arrows) (D), enlarged liver with prominent thick capsule (arrows) (E), swollen dark spleen wall (F), and congested intestine (G).

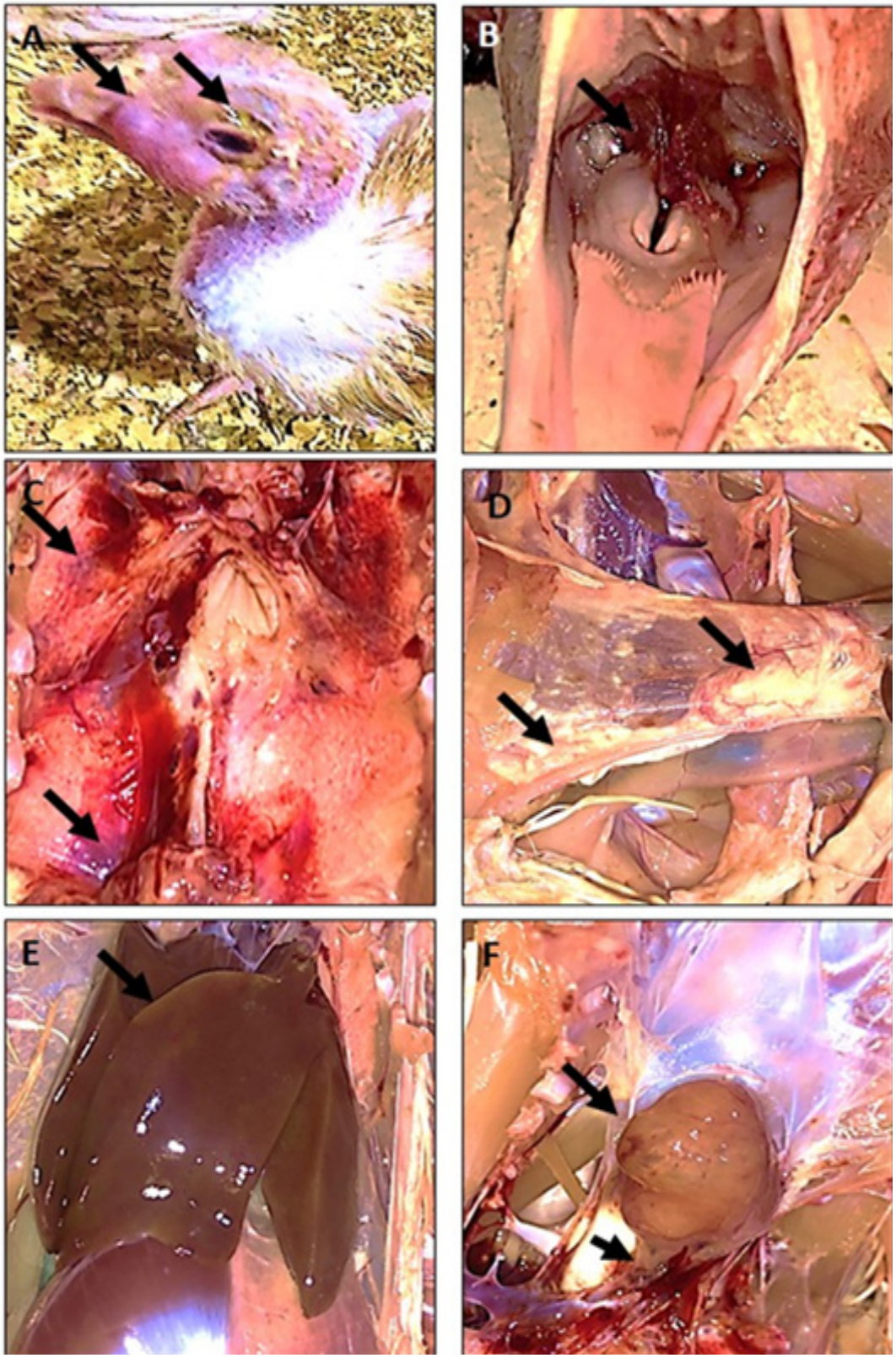


Fig. 2 (A-F). Macroscopic pictures showing clinical signs and gross findings in poults of farm 2. Poult suffering from swollen infraorbital sinuses with foamy eye secretions (A), mucoid exudate filling the buccal cavity (B), hemorrhagic lung with thickened pleura (C), caseous exudate covering the serous membranes (D), swollen liver with pale borders (E), and emphysematous heart with thick pale pericardium (F).

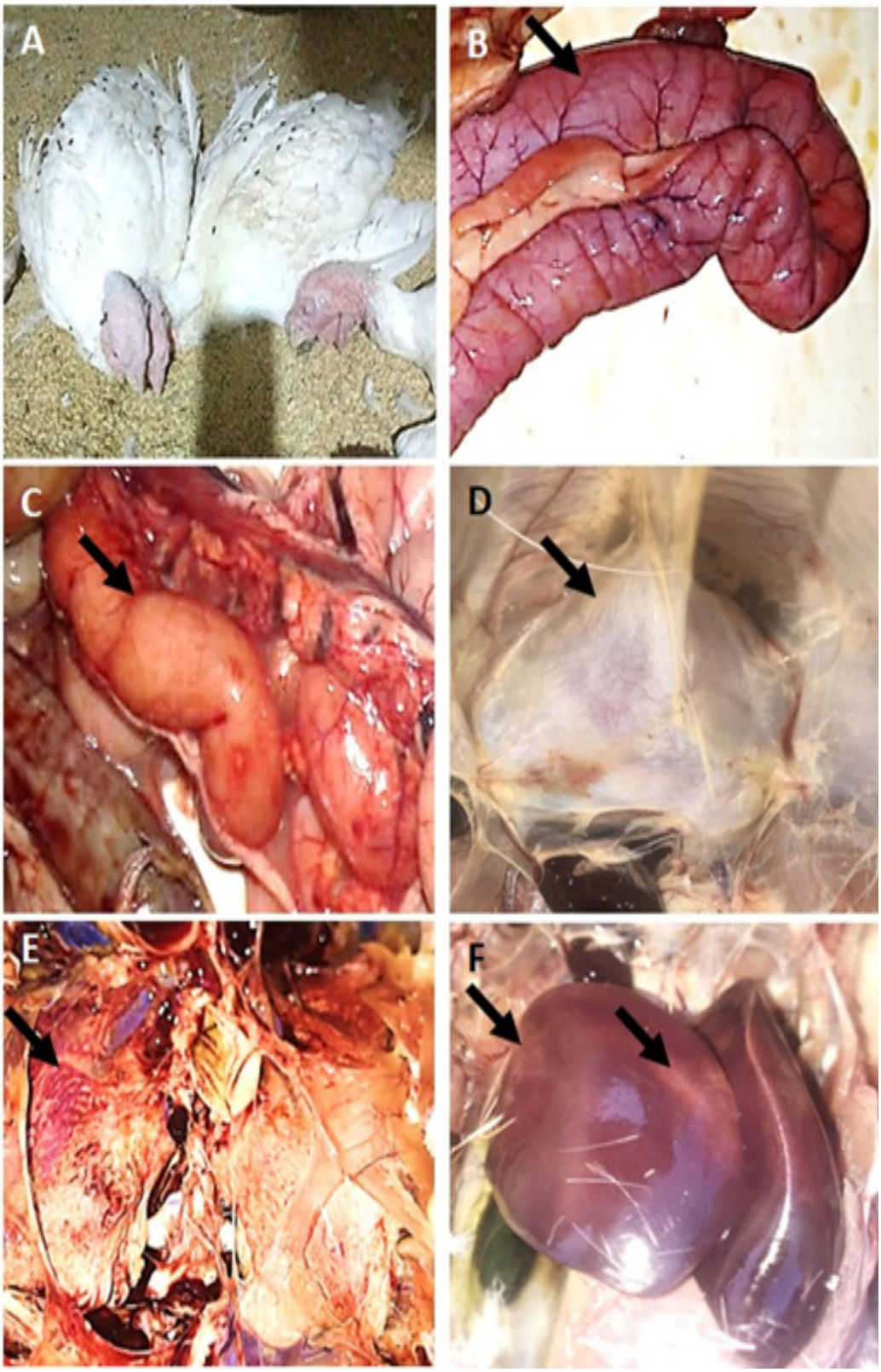
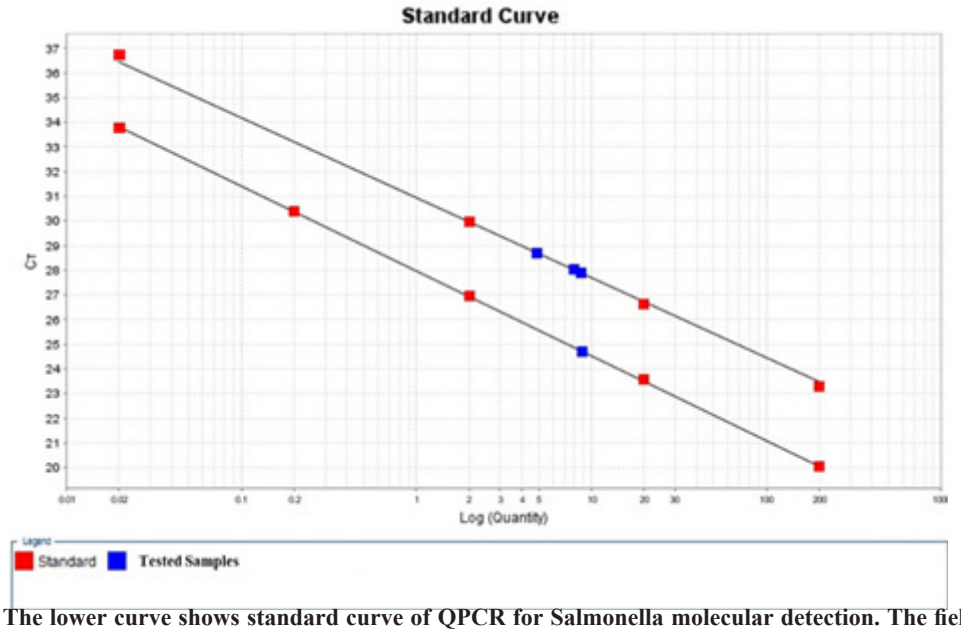
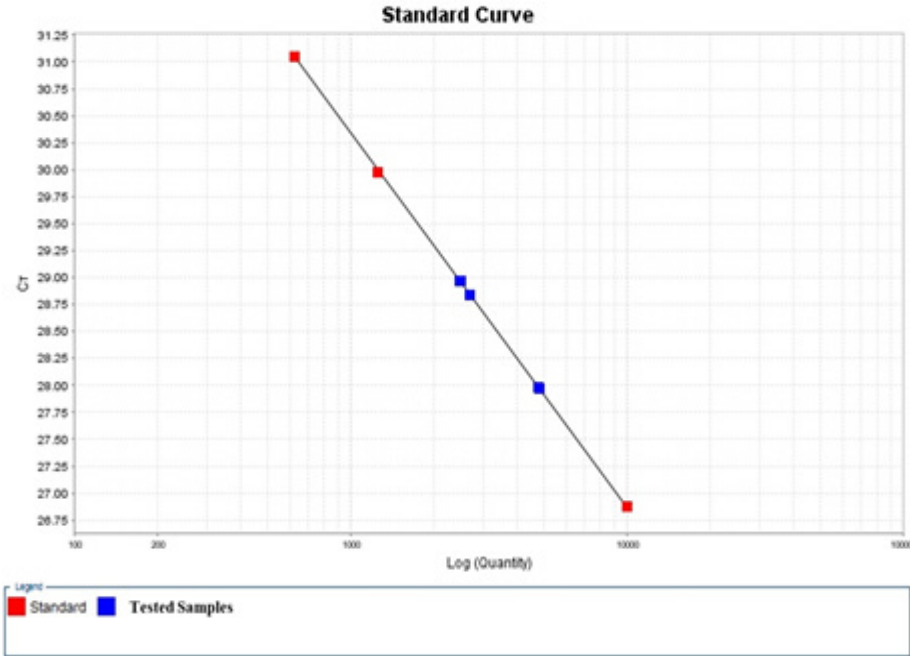


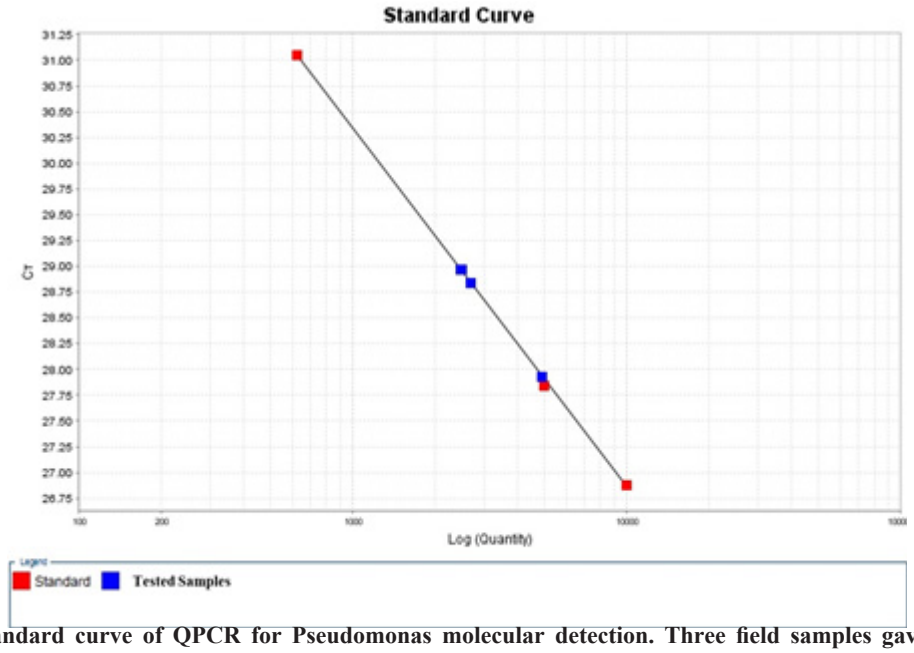
Fig. 3 (A-E). Macroscopic pictures showing clinical signs and gross findings in poults of farm 3. Depressed emaciated turkeys (A), congested intestine (B), hemorrhagic intestine (C), thick grey pericardium (D), focal congestion in the lung (E), and pale areas on the liver (F).



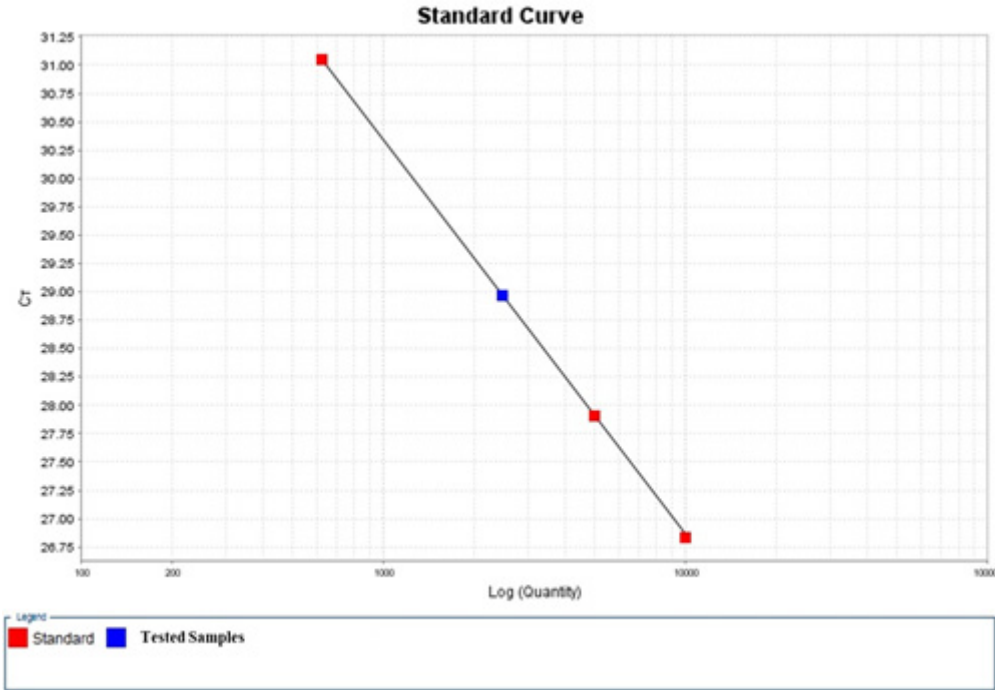
Graph 1: The lower curve shows standard curve of QPCR for Salmonella molecular detection. The field sample gave positive amplification with threshold cycles (Ct) at 20.31 (blue dot) with count = 8.88 x 103. The upper curve shows amplification plots of QPCR of E. coli molecular detection. Three field samples gave positive amplification with threshold cycles (Ct) at 24.71, 27.89 and 28.71 (blue dots) with count = 7.93x104, 8.76x 107 and 4.05x 106, respectively. The red dots represent the standard positive controls.



Graph 2: Standard curve of QPCR for QPCR of Clostridium molecular detection. Three field samples gave positive amplification with threshold cycles (Ct) at 28.83, 30.05, and 31.05 (blue dots) with count =2x103, 2.25x103 and 625, respectively. The red dots represent the standard positive controls.



Graph 3: Standard curve of QPCR for Pseudomonas molecular detection. Three field samples gave positive amplification with threshold cycles (Ct) at 26.87, 27.84, and 28.97 (blue dots) with count =1.5x 104, 4.76x 103 and 2.5x103, respectively. The red dots represent the standard positive controls.



Graph 4: Standard curve of QPCR for Mycoplasma gallisepticum molecular detection. The field sample gave positive amplification with threshold cycles (Ct) at 28.97 (blue dot) with count = 2.79x103. The red dots represent the standard positive controls.

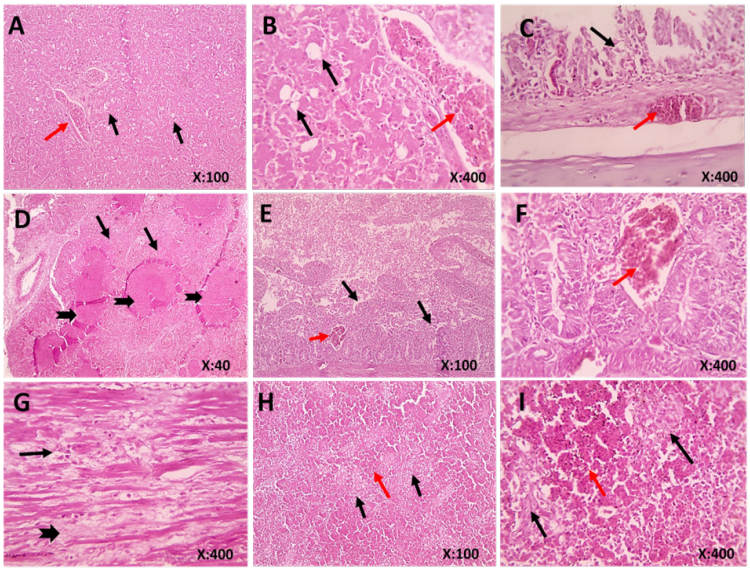


Fig. 4. Microscopic pictures of tissue samples collected from farm 1 showing congested blood vessels (red arrow) and fatty change in hepatocytes (black arrow) in liver (A&B) , tracheal wall showing destruction of epithelial lining trachea (black arrow) with congested blood vessels (red arrow) (C), Lung showing multiple necrotic areas replacing pulmonary tissue (thick black arrow), marked interstitial edema with heavy heterophils infiltration (thin black arrow) (D), Intestine showing sloughing of villi (black arrows) with congested blood vessels (red arrow) (E&F), Myocardium showing interstitial fibrosis (thick black arrows) with few leukocytic cells infiltration (thin black arrow) (G), spleen showing fibrinous deposits (black arrows) and haemorrhage in red pulp (red arrow) (H&I). H&E

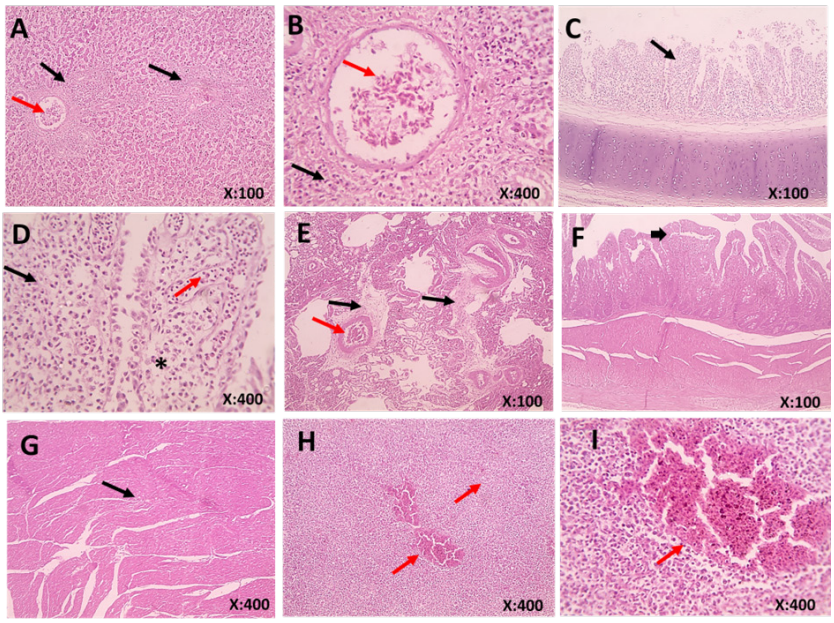


Fig. 5 (A-I). Microscopic pictures of tissue samples collected from farm 2 showing congestion (red arrow) with perivascular fibroplasia with leukocytic cells infiltration (black arrows) in liver (A&B), tracheal epithelium showing edema (*), leukocytic cells infiltration (black arrow), congested capillaries (red arrow) in lamina propria, with congested blood vessels (red arrow) (C&D), congestion (red arrows), perivascular edema, fibrosis with few mononuclear cells infiltration (black arrows) in lungs (E), short blunt intestinal villi (black arrow) (F), vacuolations of cardiomyocytes (black arrows) (G), congestion (red arrow), and perivascular haemorrhage (red arrow) in red pulp of spleen (H&I). *H&E*

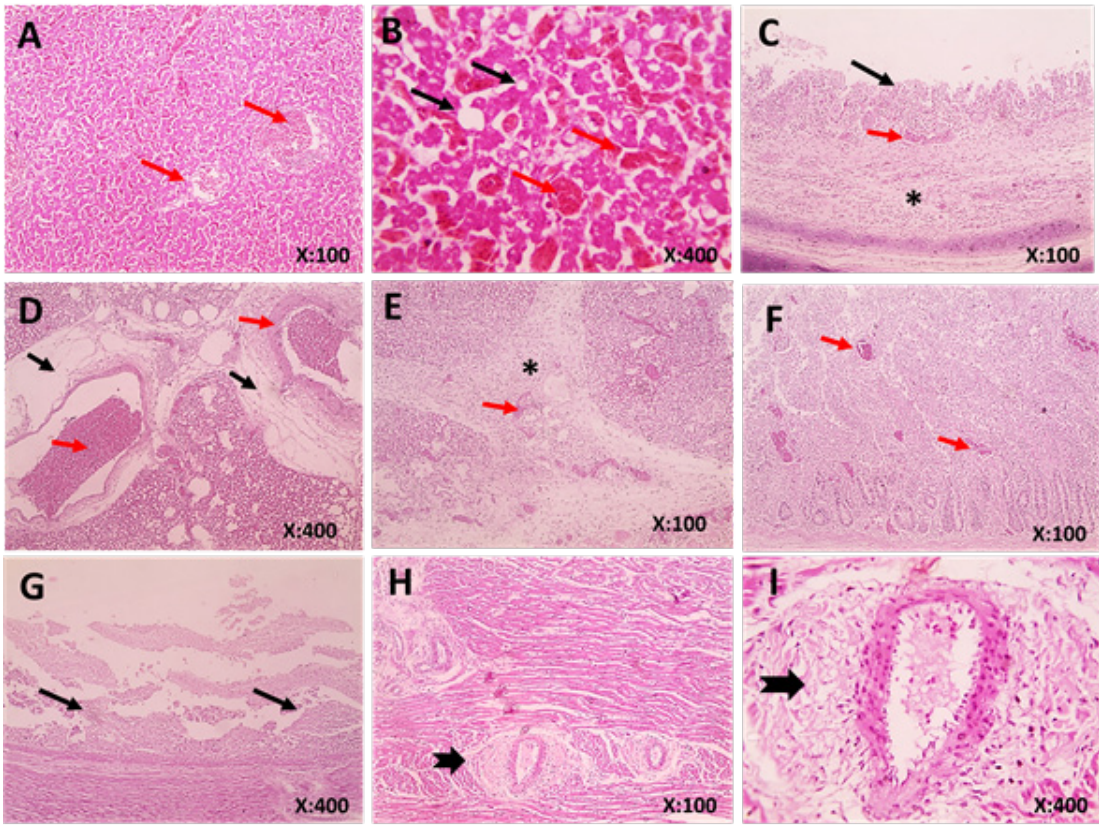


Fig. 6 (A-I). Microscopic pictures of tissue samples collected from farm 3 showing congestion (red arrows) and vacuolations of hepatocytes (black arrows) (A&B), trachea showing hyperplastic lining epithelium (black arrow), congested blood vessels (red arrows), edema in lamina propria and submucosa (*) (C), lungs showing congested blood vessels (red arrows) with perivascular edema (D), severe interstitial edema with leukocytic cells infiltration (*) besides congested blood vessels (red arrows) (E), intestine showed congested blood vessels (red arrows) with loss of epithelium covering villi (F&G), dilated blood vessels (red arrows) and perivascular fibrosis in heart (H&I). H&E

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دراسات مرضية وجزيئية على بعض البكتيريا المسببة للأمراض للسلالات المحولة الهجينة من الديوك الرومي في مصر

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إن الديوك الرومية (*Meleagris gallopavo*) معرضة للعديد من الأمراض البكتيرية المسببة للأمراض التي تؤثر على إنتاجها، ومع ذلك لا يُعرف سوى القليل عن قدرتها المرضية في السلالات الهجينة المحولة. لذلك تعتبر هذه الدراسة بحثاً عن الأمراض البكتيرية الأكثر شيوعاً التي تصيب الديوك الرومية وتأثيرها المرضي على الأنسجة المختلفة. تمت مراقبة ثلاث مزارع للديك الرومي من السلالات المحولة الهجينة في عام 2023 بحثًا عن أي علامات سريرية. وفي الآونة الأخيرة، تم تشريح الطيور النافقة، وتم تسجيل جميع الآفات الجسيمة. تم فحص عينات من كل مزرعة باستخدام qPCR في الوقت الحقيقي لوجود السالمونيلا والإيكولاي والكولستريديا والسيدوموناس والميكوبلازما والباستوريلا. تم إخضاع عينات الأنسجة لتقنيات التشريح المرضي لتحديد الآفات المجهرية المختلفة. كشف اختبار qPCR في الوقت الحقيقي أن كانت السالمونيلا والإيكولاي والسيدوموناس إيجابية في جميع عينات المزرعة التي تم فحصها. في حين كانت كلوستريديا والميكوبلازما إيجابية في عينة واحدة من المزرعة، من ناحية أخرى لم تكن أي من العينات المختبرة إيجابية للباستوريلا. كشف الفحص النسيجي المرضي عن احتقان ونخر وتسلل خلايا الكريات البيض في أعضاء متعددة. يسلط هذا الدليل التشريحي المرضي الضوء على المخاطر الصحية المرتبطة بهذه العدوى في المزرعة. هناك حاجة إلى مزيد من التحقيق لتحديد مصدر التلوث وتنفيذ تدابير الرقابة المناسبة لمنع انتشار مسببات الأمراض هذه.

الكلمات المفتاحية: الديوك الرومي؛ البكتيريا؛Meleagris gallopova ، علم الأمراض، الجزيئية.