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Detecting Some Virulent Bacterial Causes of Omphalitis in Broiler Chickens

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Abstract

OMPHALITIS is the main cause of mortality and low performance in broiler chicks. Detection of some virulent bacterial causes of yolk sac infection in broilers was the aim of this study. This investigation included 45 examined broiler flocks in Delta region of Egypt, aged 1-8 days old and suffered from high mortality, depression, diarrhea and low vitality. Bacterial isolation revealed 39 *E. coli* isolates (86.6%), 7 *Salmonella* isolates (15.5%), 6 *Pseudomonas aeruginosa* isolates (13.3%) and 3 coagulase-negative *Staphylococcus* isolates (6.6%). The polymerase chain reaction test used to detect virulent genes in these isolates revealed that all 4 examined *E. coli* isolates had *iroN*, *hlyF* and *ompT* genes. The *invA, stn* and *fimA* genes were detected in 4 examined *S.* Enteritidis isolates. *Staphylococcus lentus* isolate was positive to *sea* and *tsst* genes and negative to *etb* gene, and *S. saprophyticus* isolate was positive to *sea, tsst* and *etb* genes while *S. xylosus* was positive to *etb* gene and negative to *sea* and *tsst* genes. *tox A*, *phzM* and *oprL* genes were identified in 4 tested *P. aeruginosa* isolates. Antimicrobial sensitivity testing revealed that all 4 tested E. coli isolates were resistant to ampicillin while doxycycline and imipenem showed sensitivity. While the 4 examined S. Enteritidis isolates were resistant to ampicillin but showed sensitivity to gentamycin, amikacin and imipenem. The coagulase-negative Staphylococcus isolates were resistant to clindamycin but sensitive to doxycycline, ciprofloxacin and gentamycin. P. aeruginosa isolates (n=4) were resistant to imipenem and were sensitive to amikacin, cefepime and ciprofloxacin.

Keywords: Omphalitis, Yolk sac infection, Bacteria, Virulence genes, Multi drug resistant.

Introduction

Ensuring the health and productivity of poultry is therefore essential for sustaining this industry, particularly during the critical early stages of a chick's life. Embryonic development of chickens depends heavily on nutrients derived from the yolk, which serves as the primary reservoir of nourishment. The yolk sac, analogous to the mammalian placenta, facilitates the absorption, digestion, and transfer of these nutrients to the developing embryo. However, the period following hatching, especially the first week, is fraught with challenges. This stage accounts for 30-50% of overall mortality in broiler chicks, with omphalitis emerging as a leading cause of death along-side other conditions such as avian encephalomyelitis, brooder pneumonia, and pullorum disease [1].

Omphalitis, also known as yolk sac infection (YSI) or mushy chick disease, is a non-contagious condition often linked to unhealed navels which may be caused by infectious and/or non-infectious agents. This disease is economically significant, as it not only causes increased mortality but also leads to poor weight gain, stunted growth, and inferior carcass quality in surviving chicks [2]. The prevalence of

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YSI varies between 5% and 30%, with higher incidence rates reported during colder seasons such as winter and autumn [3].

The unabsorbed yolk sac in young chicks provides an ideal medium for bacterial growth such as *Escherichia coli*, *Salmonella* spp., and *Staphylococcus* spp. which responsible for omphalitis in broiler and layer chicks [4]. Infection typically occurs when the navel fails to close properly after the yolk sac is drawn into the abdominal cavity, allowing environmental bacteria to invade the chick tissues. Poor hygiene on breeding farms and improper hatchery management exacerbates the problem [5].

The degree of the organism's virulence is based on certain virulence factors that may have acquired. These factors may be toxins or surface structures that enable the bacterium to adhere to vector cells or to attack the immune system. According to *E. coli* the five essential virulence genes carried by plasmid, hlyF (hemolysin), ompT (outer membrane protease), iroN (siderophore), iss (serum survival), and iutA (iron transport), are considered markers for APEC from other avian fecal *E. coli* [6]. All strains and serovars of *Salmonella* enterica were found to carry the *Salmonella* enterotoxin determinant (stn) gene which has been proposed to be a putative causative agent of diarrhea [7]. *Salmonella* adhesion to the host cell is an initial and most important step in the pathogenesis of salmonellosis. Fimbriae which encoded by (fimA, agfA, sefA and sefD) genes are one of the most important tools of bacterial adhesion to host cell [8]. In addition, Toxic shock syndrome (TSS) caused by Staphylococcus is an acute, life-threatening illness characterized by the sudden onset of high fever, hypotension, and a widespread erythematous rash. If untreated, it can progress rapidly to multi-organ dysfunction and potentially fatal shock [9]. P. aeruginosa also has got massive arsenal of virulence supplies such as lipopolysaccharide, elastase, alkaline proteases, pyocyanin, pyoverdine, hemolysins, phospholipase C and rhamnolipids [10]. The outer membrane proteins of P. aeruginosa (OprI and OprL) play important roles in the interaction of the bacterium with the environment as well as the inherent resistance of P. aeruginosa to antibiotics And pyocyanin produced by P. aeruginosa is a key virulence factor that causes damages to airway and lung in hosts. Conversion of phenazine-1-carboxylic acid to pyocyanin involves an extra metabolic pathway which contains two enzymes encoded, by phzM and phzS [11].

While antimicrobial therapy is commonly used to treat omphalitis, the overuse of antibiotics in both human and veterinary medicine has led to the emergence of antibiotic-resistant pathogens. This growing resistance is a global concern, with predictions suggesting that by 2050, antibiotic-resistant infections could result in 10 million deaths annually [12]. The persistent release of antibiotics into the environment due to extensive use in the veterinary sector exacerbates this issue, underlining the need for alternative strategies to combat bacterial infections.

This research aimed to unveil some bacterial causes of omphalitis in broiler baby chicks, focusing on their incidence, virulence genes and antimicrobial sensitivity.

Material and Methods

Sample collection for bacterial isolation and identification

Samples from liver and yolk sacs (n=10 per flock) in diseased broiler chicken flocks (n= 45) in Delta region, Egypt from May till October 2023. The examined flocks aged 1 - 8 days old were suffering from high mortality (3-12%) in the first 10 days of age, retarded growth, depression, huddling together, profuse watery diarrhea (pasty vent) and lameness. Samples were weighed and suspended in nutrient broth (as 1:10 dilution) and incubated at 37 C for 20 hours. Loops-full of suspension were streaked onto MacConkey agar (Oxoid, Ltd, UK), Baird parker agar (Oxoid, Ltd, UK), Cetrimide agar (Himedia, India) and incubated at 37 C for 24 hours. Further culture of suspected colonies on selective media such as eosin methylene blue agar (EMB, Oxoid, Ltd, UK), Rappaport-Vassiliadis, Xylose Lysine Deoxycholate agar media (XLD, Oxoid, Ltd, UK) were applied, and the suspected colonies were stained by Gram's stain and examined microscopically. Biochemical identification tests (indole test, methyl red Test, Voges – Proskauer reaction, citrate utilization test, urea hydrolysis test, hydrogen sulfide production test, oxidase test and catalase test) were carried out on recovered suspected colonies according to Quinn et al. [13]. This work followed the guidelines of the Ethics Committee of Damanhur University and was approved by the committee decision No. DMU/VetMed-2025/027.

Serological, virulence genes identification and antimicrobial sensitivity test

These tests was applied on four *E. coli*, four *Salmonella*, three *Staphylococcus* and four *P. aeruginosa* isolates recovered from cases which showed severe postmortem lesions with the highest mortality rates.

Serotyping of E. coli

Four isolates were selected for serological identification according to using rapid diagnostic *E. coli* antisera sets for (O) and (H) antigens (Denka Seiken Co., Japan) for diagnosis of the Enteropathogenic types and pathotype categorization was according to Bahgat et al. [14].

Serotyping of Salmonella isolates

Also, 4 *Salmonella* isolates were selected for serological identification by using *Salmonella* antisera of (Sifin Company, Germany) and interpretation of results according to white Kauffman scheme 2007 [15].

Identification of Staphylococcus isolates

VITEK2 COMPACT identification system (bioMérieux, France) was used for definite identification of 3 obtained *Staphylococcus* isolates directly from suspected colonies grew on Baird parker agar media.

Identification of P. aeruginosa

The identification of P. aeruginosa was dependent on colony morphology on cetrimide agar which is specific to P. aeruginosa then application of biochemical tests.

Virulence genes identification by using PCR test

DNA was extracted from isolates by using QIAamp DNA mini kit (Qiagen N.V, Germany) and the Oligonucleotide primers used in cPCR as shown in Table 1. Preparation of PCR Master Mix was used according to Emerald Amp GT PCR master mix (Takara, Japan) by adding 12.5 µl of Emerald Amp GT PCR master mix (2x premix), 5.5 µl of PCR grade water, 1 µl of forward primer (20 pmol), 1 µl of reverse primer (20 pmol) and 5 µl of template DNA. Cycling conditions of the two primers during PCR are shown in Table 1. Agarose gel electrophoreses (1%) applied according to Sambrook et al. [25] with some modifications. Each uniplex PCR product, negative and positive controls (20 µl) were loaded to the gel. The gel was examined under UV cabinet and photographed by a gel documentation system.

Antimicrobial sensitivity test

The antimicrobial sensitivity test was carried out according to the guidelines of Clinical and Laboratory Standards Institute [26] using Mueller-Hinton agar (Oxoid, Ltd, UK) for *E. coli*, *Salmonella* and P. aeruginosa isolates, and nutrient agar (Oxoid, Ltd, UK) for coagulase-negative *Staphylococcus* isolates. The following antimicrobial discs: ampicillin (10 µg), cefepime (30 µg), cefotaxime (30 µg), ceftazidime (30 µg), gentamycin (10 µg), amikacin (30 µg), ciprofloxacin (5 µg), imipenem (10 µg), trimethoprim-sulfamethoxazole (1.25/23.75 µg), doxycycline (30 µg), erythromycin (15 µg), linezolid (30 µg) and clindamycin (2 µg) were used. Criteria for the interpretation of the zone diameter for sensitive, sensitive dose dependent, intermediate and resistance was done and recorded as CLSI guidelines, 2020 as shown in Tables 2, 3, 4.

Results

Postmortem examinations

Internally, the examined chicks showed retained putrefactive yolk sac, hyperemia of visceral blood vessels, inflammation and edema around the navel, discoloration of yolk sac (greenish yellow or dark brown) and in severe cases distension of chick's abdomen, congestion of internal organ and fibrinous airsacculitis was found.

Isolation and identification of bacterial isolates

Bacteriological examination of diseased chicks with omphalitis revealed that 43 out of 45 broiler flocks (95.5%) were bacteriologically positive. Single bacterial isolation was in 35 flocks (77.8 %), but mixed bacterial infection was in 8 flocks (17.8 %). Identification of bacterial isolates revealed that out of 45 examined samples *E. coli* 39 isolates (86.6%), *Salmonella* 7 isolates (15.5%), coagulase-negative *Staphylococcus* 3 isolates (6.6%) and *Pseudomonas aeruginosa* 6 isolates (13.3%). Morphology of growing colonies on selective media (MacConkey, EMB, XLD, Baird parker and Cetrimide agar media) was shown in Table 5, and biochemical test results were shown in Table 6.

Serotyping of E. coli isolates

Serological identification of *E. coli* isolates revealed suspected enteropathogenic *E. coli* serotypes O119: H6 and O146: H21, enterohemorrhagic *E. coli* O55: H7 and O128: H2 as enterotoxigenic *E. coli*.

Serotyping of Salmonella isolates

Serological identification of (O) and (H) antigen of 4 *Salmonella* isolates revealed *S. Enteritidis* O (1, 9, 12) and H (g, m).

Identification of Staphylococcus isolates

Three *Staphylococcus* isolates were identified by using VITEK 2 Compact technique into *Staphylococcus xylosus*, *Staphylococcus saprophyticus* and *Staphylococcus lentus*.

Seasonality

This Study was performed in 6 months from May to October and Its noticed that all *Pseudomonas aeruginosa* isolates recovered during October and November (Fig. 1).

PCR for virulence genes identification of bacterial isolates

E. coli isolates

Four *E. coli* isolates had iroN, hlyF and ompT virulence genes (Fig. 2).

Salmonella isolates

Four *Salmonella* isolates had the invA, stn and fimA virulence genes (Fig. 3 and 4).

Staphylococcus species isolates

Three *Staphylococcus* isolates examined by PCR for virulence genes of sea, tsst and etb. Sample no. (1) (*Staphylococcus lentus*) was positive to sea and tsst genes and negative to etb gene, sample no. (2) (*Staphylococcus saprophyticus*) was positive to sea, tsst and etb genes while sample no. (3) (*Staphylococcus xylosus*) was positive to etb gene and negative to sea and tsst genes (Fig. 5).

P. aeruginosa isolates

The four examined P. aeruginosa isolates had these virulence genes toxA, phzM and oprL (Fig. 6).

Antimicrobial Sensitivity tests

The in-vitro antimicrobial sensitivity test for different bacterial isolates of *E. coli*, *S. Enteritidis*, *Staphylococcus* species and P. aeruginosa are presented in Table 7.

Discussion

The present study was carried out for the isolation of bacteria associated with yolk sac infection (omphalitis) in broiler chicks from 45 flocks of 1 - 8 days old suffered from high mortality, depression, pasty vent, watery chalky diarrhea and decrease in vitality. Postmortem examination of sick chicks showed retained yolk sac, hyperemia of visceral blood vessels, inflammation and edema around the navel, discoloration of yolk sac (greenish yellow or dark brown) and distension of chick's abdomen in all cases. In addition, in severe cases no (1, 3, 8, 13, 15, 22, 27, 34, 36, 37, 39, 42 and 43) there were putrefactive odor of yolk sac, enteritis, severe congestion of internal organs and fibrinous airsacculitis.

Bacteriological examination of chicks with omphalitis from 45 broiler flock revealed that 43 flock (95.5%) of examined samples were positive for bacterial isolation. *E. coli* was the most predominant bacteria causing omphalitis in 86.6%, which was very close to results of Shaheen et al. [27] with a rate of 87.5%. Virulence factors of *E. coli* may be toxins or surface structures that enable the bacterium to adhere to vector cells or to attack the immune system. Detection of the iroN, hlyF and ompT genes using PCR indicated that all the four examined *E. coli* isolates are virulent. The iroN gene was widely prevalent among APEC strains, consistent with findings from previous studies by Pourhossein et al. [28], reporting prevalence rate of 78.69%. Similarly, the hlyF gene was identified in 100% of cases in the study by Sedeek et al. [29]. The prevalence of the ompT gene was reported as 86.9% by Mohamed et al. [30]. The iroN, hlyF, and ompT genes are primarily located on the ColV plasmid, which is known to harbor numerous antimicrobial resistance genes [17]. The presence of these plasmid-associated virulence genes in the isolates examined in this study may explain their resistance to multiple antibiotics, reflecting the co-selection and dissemination of both virulence and resistance determinants through horizontal gene transfer.

Antimicrobial therapy has played a crucial role in controlling bacterial infections in poultry, which can lead to significant health issues and economic losses. However, the widespread use of antimicrobials has also contributed to the emergence of resistant strains, making it challenging to control infections effectively. This resistance can lead to cross-resistance issues, especially concerning human enteric pathogens, as some antimicrobials used in veterinary settings overlap with those used in human medicine.

Antimicrobial sensitivity test was performed on *E. coli*, *Salmonella* and P. aeruginosa isolates which cause severe omphalitis in addition to three recovered coagulase-negative *Staphylococcus* species. Multidrug resistance in *E. coli* has become a worrying issue that is observed worldwide in humans and veterinary medicine. As it has a great ability to gain antibacterial resistance genes, mostly through horizontal gene transmission [31]. In the present *E. coli* isolates were completely resistant to ampicillin, showed also 75% resistance to cefotaxime and gentamycin and 50% to amikacin, ciprofloxacin and trimethoprim-sulfamethoxazole but showed no resistance to doxycycline and imipenem.

Salmonella Enteritidis was the second pathogen responsible for omphalitis with a percentage of 15.5% and this rate of infection was constituted with results obtained by Kaboudi et al. [32] who reported *Salmonella* isolation rate from omphalitis at 10.6%. Also, *Salmonella* infection was the second bacteria causing omphalitis but with high rate of infection as 62.5% as reported by Shaheen et al. [27]. The main infection route with *Salmonella* species was through an unhealed navel, but transmission can also occur via the bloodstream or through yolk contamination, leading to infection in chicks [3]. Based on serological identification of (O) and (H) antigen, 4 tested *Salmonella* isolates were *S. Enteritidis*. The cPCR results indicated the presence of invA, stn and fimA virulence genes in these *S. Enteritidis* isolates. The invA and stn genes are considered main genes and widely spread in *Salmonella* isolates as it was detected with 100 % in the studies of Shen et al. [33].

In addition, the fimA gene was also detected in all isolates in the previous studies of Lozano-Villegas and Rondón-Barragán [34].

Epidemiological studies indicate that multidrug resistant *Salmonella* serotypes are more virulent than others, as reflected by increased severity and more prolonged disease in patients infected by these strains [35]. Antimicrobial sensitivity test of *Salmonella* isolates showed complete resistance to Ampicillin, high resistance (75%) to cefotaxime and doxycycline, 25% resistant to ciprofloxacin, trimethoprim-sulfamethoxazole and cefepime, and *Salmonella* isolates were completely sensitive to gentamycin, amikacin and imipenem.

Staphylococcus infection rate was 6.6%. However, coagulase-positive *Staphylococcus* is more common and more virulent than coagulase-negative *Staphylococcus* species, all 3 recovered *Staphylococcus* isolates were coagulase-negative (*S. lentus*, *S. saprophyticus and S. xylosus*).

Similarly, Nasrin et al. [36] isolated coagulase-negative staphylococci with percentages (24% in 1-3 days old chicks and 28.6% in 4-7 days old chicks).Our data revealed that there is a complete correlation between coagulase-negative *Staphylococcus* (CoNS) infection and *Salmonella* infection as all three recovered CoNS isolates are mixed infection with *Salmonella* isolates, and that may be due to activation of a complex immune response by *Salmonella* infection, which not only combats the invading pathogen but also disrupts the accurate balance of the gut microbiota. In the past, CoNS have been considered as a non-pathogenic microorganism as most of these species can establish a commensal relationship with humans and animals [37]. But CoNS have gained high importance over the past decade, as they have been involved in different infections in humans and animals [38]. Historically, toxic shock syndrome (TSS) has been associated with coagulase-positive *Staphylococcus* species. However, in the present study, the tsst gene was identified in two CoNS isolates (*Staphylococcus lentus* and *Staphylococcus saprophyticus*) but was not detected in *Staphylococcus xylosus*. Similarly, the presence of the tsst gene in CoNS strains has been documented in studies by Nasaj et al. [39] with reported rates of 25%. Enterotoxin genes were widely distributed among CoNS strains. For instance, Nasaj et al. [39] reported that CoNS isolates harbored enterotoxin-encoding genes with rate of 43% respectively. In addition, the present study found that the sea gene was identified in two CoNS isolates (*Staphylococcus lentus* and *Staphylococcus saprophyticus*) but was absent in *Staphylococcus xylosus*. Exfoliative toxins (ETs) play a significant role in cell-to-cell adhesion in the superficial epidermis, leading to the formation of blisters. These toxins contain a critical peptide bond that blocks their active sites, rendering them enzymatically inactive in their native state [40]. Although the etb (exfoliative toxin B) gene is rarely detected in CoNS isolates. In the present study, it was detected in two isolates (*Staphylococcus saprophyticus* and *Staphylococcus xylosus*) but was absent in the *Staphylococcus lentus* isolate. Coagulase-negative staphylococci are an important reservoir of antimicrobial-resistant genes as almost all CoNS had a multidrug-resistant profile [41]. Presence of tsst, sea and etb genes in the genome of CoNS isolates may refer to indirectly increased their possibility of antibiotic resistance, as these virulent genes and antibiotic resistance genes are often found together due to their association with mobile genetic elements. This facilitates co-selection under antibiotic pressure, especially in clinical environments. In the present study CoNS isolates were completely resistant to clindamycin and highly resistant to trimethoprim -sulfamethoxazole, linezolid and erythromycin with 66.6%. No resistance was reported to doxycycline ciprofloxacin and gentamycin.

Pseudomonas aeruginosa causes high mortality in young birds due to omphalitis and yolk sac infections acquired in the hatchery, giving a mortality following yolk sac inoculation reached 100% mortality in experimentally inoculated chickens [42]. In the precent study, the incidence of P. aeruginosa infection was 13.3% and this result was nearly constitute with results obtained by EL-Sawah et al. [43] who recovered P. aeruginosa with a percentage of 11%, Hassan [44] isolated P. aeruginosa with a percentage of 20% from one day old broiler chicks Although the bacterial isolation was carried out during the period from May to October, all P. aeruginosa isolates were recovered only during September and October and that may be due to the high humidity resulting from using cooling pads during summer months in breeder houses [45]. PCR test identified the presence of Tox A, phzM and oprL virulence genes in the four examined P. aeruginosa isolates recovered from diseased chicks. The toxA gene is recognized as a prevalent marker in P. aeruginosa, with studies by Algammal et al. [46] reporting that all tested isolates were positive for this gene. The phzM gene was detected in P. aeruginosa isolates from various clinical sources, with rates of 53.7% as reported by Ali et al. [47]. Additionally, the oprL gene is widely distributed in the P. aeruginosa genome, as it was reported in 100% of isolates in the studies of Algammal et al. [46].

Pseudomonas is naturally resistant bacteria to most antibiotics and able to quickly develop resistance to the commonly used ones [48], The multidrug resistant P. aeruginosa developed by several mechanisms such as multi-drug resistance efflux pumps, production of β- lactamase enzymes, biofilm formation and aminoglycoside modifying enzymes [49]. In the present study antimicrobial sensitivity test of P. aeruginosa isolates showed complete resistance towards imipenem, 75% resistant to ceftazidime, 25% resistance to gentamycin, and complete sensitive to amikacin, cefepime and ciprofloxacin.

Finally, we can conclude that the most common bacterial causes of omphalitis in chicks cause high mortality during the first 8 days of age are *E. coli*, *Salmonella*, staphylococci and P. aeruginosa. All these isolates have different virulence genes and showed multidrug resistant (MDA) to different antimicrobial drugs.

Conclusion

The current study highlighted the role of virulent and multidrug resistant (MDR) *E. coli*, *Salmonella Enteritidis*, *Pseudomonas aeruginosa*, and coagulase-negative *Staphylococcus* spp., in omphalitis and early mortality in broiler chicks in the Delta region of Egypt. Ongoing monitoring of bacterial profiles and resistance patterns is crucial for developing appropriate treatment strategies and reducing economic losses in poultry production.

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Disclosure statement

No potential conflict of interest was reported by the author(s).

TABLE 1. Oligonucleotide primer sequences (Metabion, Germany).

Bacteria	Target gene	Sequence	Amplified product	Reference
E. coli	*iroN*	ATC CTC TGG TCG CTA ACT G	847 bp	**[16]**
		CTG CAC TGG AAG AAC TGT TCT		
	ompT	TCATCCCGGAAGCCTCCCTCACTACTAT	496 bp	**[17]**
		TAGCGTTTGCTGCACTGGCTTCTGATAC		
	hlyF	GGCCACAGTCGTTTAGGGTGCTTACC	450 bp	
		GGCGGTTTAGGCATTCCGATACTCAG		
Salmonella	*fimA*	CCT TTC TCC ATC GTC CTG AA	85 bp	**[18]**
		TGG TGT TAT CTG CCT GAC CA		
	invA	GTGAAATTATCGCCACGTTCGGGCAA	284 bp	**[19]**
		TCATCGCACCGTCAAAGGAACC		
	Stn	TTG TGT CGC TAT CAC TGG CAA CC	617 bp	**[20]**
		ATT CGT AAC CCG CTC TCG TCC		
Pseudomonas	*toxA*	GACAACGCCCTCAGCATCACCAGC	396 bp	**[21]**
		CGCTGGCCCATTCGCTCCAGCGCT		
	oprL	ATG GAA ATG CTG AAA TTC GGC	504 bp	**[22]**
		CTT CTT CAG CTC GAC GCG ACG		
	phzM	ATGGAGAGCGGGATCGACAG	875 bp	**[23]**
		ATGCGGGTTTCCATCGGCAG		
Staphylococcu	*sea*	GGTTATCAATGTGCGGGTGG	102 bp	**[24]**
		CGGCACTTTTTTCTCTTCGG		
	etb	ACAAGCAAAAGAATACAGCG	226 bp	
		GTTTTTGGCTGCTTCTCTTG		
	tsst	ACCCCTGTTCCCTTATCATC	326 bp	
		TTTTCAGTATTTGTAACGCC		

TABLE *2*. Zone diameter interpretative standards for *E. coli* and *Salmonella* species

Antimicrobial drug	Susceptible	Susceptible dose dependent	Intermediate	Resistant
Ampicillin	≥ 17	_	14-16	≤ 13
Gentamycin	≥ 15	_	13-14	≤ 12
Amikacin	≥ 17	_	15-16	≤ 14
Cefepime	≥ 25	19-24	_	≤ 18
Cefotaxime	≥ 26	_	23-25	≤ 22
Doxycycline	≥ 14	_	11-13	≤ 10
Ciprofloxacin	≥ 31	_	21-30	≤ 20
Imipenem	≥ 23	_	20-22	≤ 19
Trimethoprim-Sulfamethoxazole	≥ 16	_	11-15	≤ 10

TABLE 3. Zone diameter interpretative standards for coagulase-negative Staphylococcus

Antimicrobial drug	Susceptible	Intermediate	Resistant
Erythromycin	≥ 23	14-22	≤ 13
Trimethoprim -Sulfamethoxazole	≥ 16	11-15	≤ 10
Linezolid	≥ 21	_	≤ 20
Clindamycin	≥ 21	15-20	≤ 14
Doxycycline	≥ 16	13-15	≤ 12
Ciprofloxacin	≥ 21	16-20	≤ 15
Gentamycin	≥ 15	3-14	≤ 12

TABLE 4. Zone diameter interpretative standards for Pseudomonas aeruginosa

Antimicrobial drug	Susceptible	Intermediate	Resistant
Ceftazidime	≥ 18	15-17	≤ 14
Ciprofloxacin	≥ 25	19-24	≤ 18
Cefepime	≥ 18	15-17	≤ 14
Amikacin	≥ 17	15-16	≤ 14
Gentamycin	≥ 15	13-14	≤ 12
Imipenem	≥ 19	16-18	≤ 15

TABLE 5. Morphology of colonies growth on specific media.

Bacteria Species	MacConkey agar	EMB Agar	XLD agar	Baird parker agar	Cetrimide agar
E. coli	Dry Pink colonies	Convex black colonies with green sheen	Large, flat, yellow colonies	Large brown colonies	No growth
Salmonella Species	Colorless colonies	-	Pink colonies with black center	No growth	No growth
Pseudomonas aeruginosa	Colorless colonies	-	Red flat colonies	No growth	Blue-green colonies
Staphylococcus species	No growth	-	No growth	Black colonies without opaque zone around	No growth

EMB agar: eosin methylene blue, XLD: xylose lysine deoxycholate agar

TABLE 6. biochemical tests results of suspected isolates recovered from specific media

Biochemical test	*E- coli*	*Salmonella species*	*Pseudomonas aeruginosa*	*Staphylococcus species*
Gram stain	-	-	-	+
Indole	+	-	-	NT
Methyl red	+	+	-	NT
Voges Proskauer	-	-	-	NT
Citrate utilization	-	-	+	NT
Urease	-	-	-	NT
Oxidase	-	-	+	-
Catalase	+	+	+	+
Coagulase	NT	NT	NT	-
TSI	A/A, gas +, H_2S -	K/A, gas +, H_2S +	K/K, gas -, H_2S -	NT

NT: Not Tested; A/A: Acid slant/acid butt; K/A: Alkaline slant/acid butt; K/K: Alkaline slant/alkaline butt; "+" positive reaction, "–" negative reaction

TABLE 7. Antimicrobial sensitivity test.

Bacteria isolates	*E. coli*				*S.* Enteritidis				*Staphylococcus spp.*			*P. aeruginosa*			
Sample no.	8	15	42	43	1	3	13	27	27	22	13	34	36	37	39
Ampicillin	R	R	R	R	R	R	R	R	-	-	-	-	-	-	-
Gentamycin	S	R	R	R	S	S	S	S	S	S	S	-	-	-	-
Amikacin	S	S	R	R	S	S	S	S	-	-	-	S	S	S	S
Cefepime	S	SDD	S	SDD	SDD	R	S	SDD	-	-	-	S	S	S	S
Cefotaxime	I	R	R	R	I	R	R	R	-	-	-	-	-	-	-
Ciprofloxacin	S	R	R	S	R	S	S	S	S	I	S	I	I	I	S
Imipenem	S	S	S	S	S	S	S	S	-	-	-	R	R	R	R
Trimethoprime-Sulphamethoxazole	S	R	R	S	R	S	S	S	R	R	S	-	-	-	-
Doxycycline	S	S	S	S	R	R	R	S	S	S	S	-	-	-	-
Ceftazidime	-	-	-	-	-	-	-	-	-	-	-	R	R	R	S
Erythromycin	-	-	-	-	-	-	-	-	I	R	I	-	-	-	-
Linezolid	-	-	-	-	-	-	-	-	R	R	S	-	-	-	-
Clindamycin	-	-	-	-	-	-	-	-	R	R	R	-	-	-	-

R, resistant; S, sensitive; I, intermediate; SDD, sensitive-dose dependent; (-) not tested

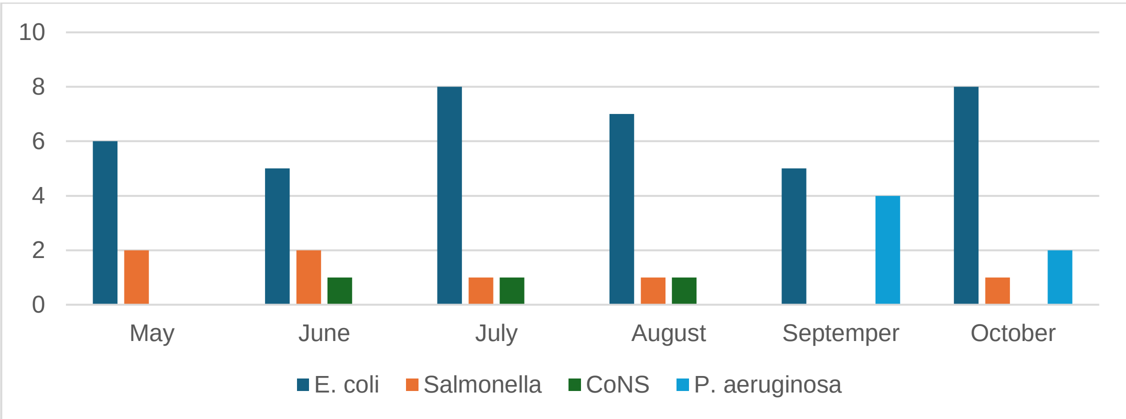

Fig. 1. E. coli, Salmonella, coagulase-negative Staphylococcus and Pseudomonas aeruginosa isolates number recovered in months from May to October

Fig. 2. Agarose gel electrophoresis (1%) stained with ethidium bromide showing PCR amplification of hlyF, ompT, and iroN genes in *E. coli* isolates. L: 10 band DNA ladder from100 to 1000bp; Lanes N: negative control; Lanes P: positive control; Lanes E1-E4: examined *E. coli* isolates showing positive amplification for the tested genes. Amplified products appeared at 450 bp for hlyF, 496 bp for ompT, and 847 bp for iroN gene

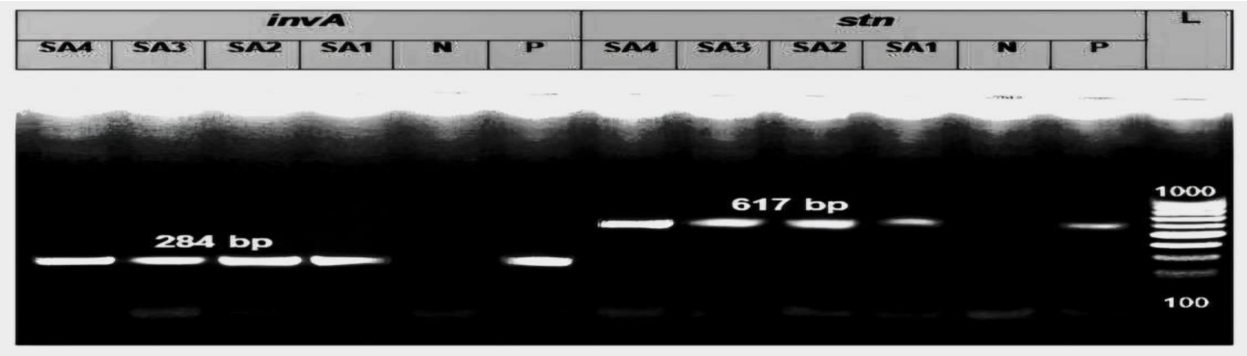

Fig. 3. Agarose gel electrophoresis (1%) stained with ethidium bromide showing PCR amplification of invA and stn genes in Salmonella isolates L: 10 band DNA ladder from100 to 1000bp; Lanes N: negative control; Lanes P: positive control; Lanes SA1-SA4: examined Salmonella isolates showing positive amplification for the tested genes. Amplified products appeared at 284 bp for invA, 617 bp for stn

Fig. 4. Agarose gel electrophoresis (1%) stained with ethidium bromide showing PCR amplification of fimA gene in Salmonella isolates L: 17 band DNA ladder from 50 to 1500 bp; Lanes N: negative control; Lanes P: positive control; Lanes SA1-SA4: examined Salmonella isolates showing positive amplification for the tested gene. Amplified products appeared at 85 bp for fimA gene.

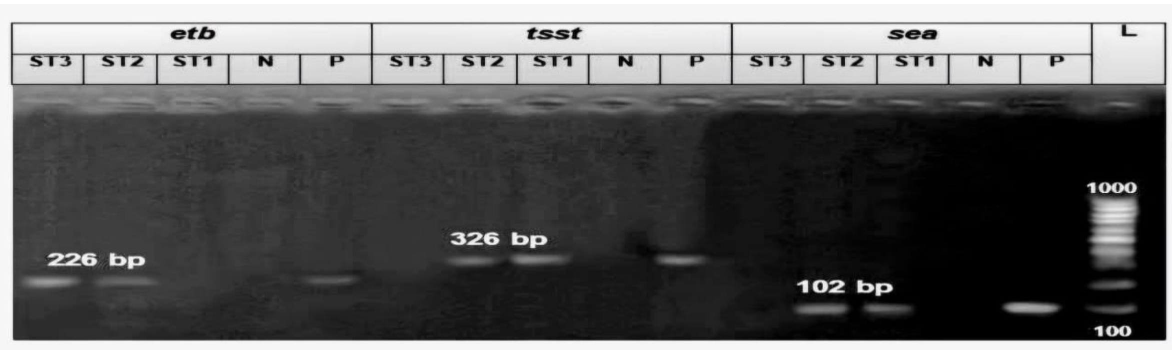

Fig. 5. Agarose gel electrophoresis (1%) stained with ethidium bromide showing PCR amplification of etb, tsst and sea genes in *Staphylococcus* isolates. L: 10 band DNA ladder from100 to 1000bp; Lanes N: negative control; Lanes P: positive control; Lanes ST1; ST2 and ST3: examined *Staphylococcus* isolates showing positive amplification for the tested genes. Amplified products appeared at 226 bp for etb, 326 bp for tsst, and 102 bp for sea gene

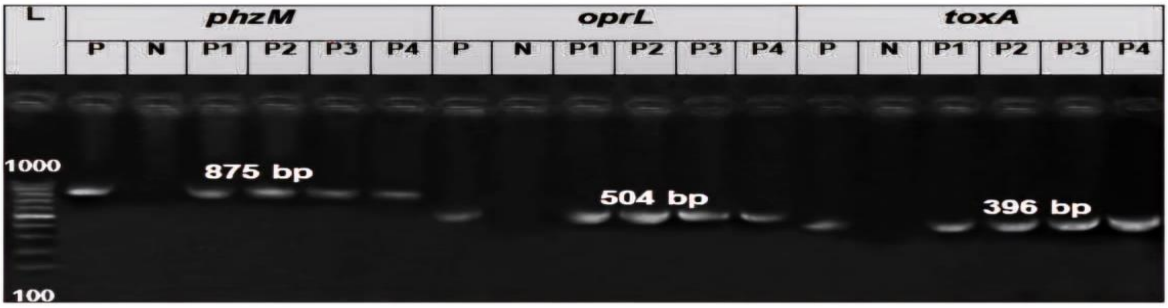

Fig 6. Agarose gel electrophoresis (1%) stained with ethidium bromide showing PCR amplification of phzM, oprL and toxA genes in Pseudomonas aeruginosa isolates. L: 10 band DNA ladder from100 to 1000bp; Lanes N: negative control; Lanes P: positive control; Lanes P1-P4: examined Pseudomonas aeruginosa isolates showing positive amplification for the tested genes. Amplified products appeared at 875 bp for phzM, 504 bp for oprL, and 396 bp for toxA gene

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الكشف عن بعض المسببات البكتيرية شديدة الضراوة لالتهاب كيس المح في دجاج التسمين

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الملخص

يُعد التهاب كيس المح من الأسباب الرئيسية للنفوق وضعف الأداء في كتاكيت التسمين. هدفت هذه الدراسة إلى الكشف عن بعض الأسباب البكتيرية المرتبطة بعدوى كيس المح في الكتاكيت. شملت الدراسة فحص 45 قطيعًا من دواجن التسمين في منطقة الدلتا بمصر، تراوحت أعمارها بين 1 إلى 8 أيام، وكانت تعاني من معدلات نفوق مرتفعة، وخمول، وإسهال، وضعف في الحيوية. أسفر العزل البكتيري عن تحديد 39 معزولة من الإشريكية القولونية (86.6%)، و7 معزولات من السالمونيلا (15.5%)، و6 معزولات من السودوموناس (13.3%)، و3 معزولات من المكورات العنقودية سالبة التخثر (6.6%). أظهرت نتائج اختبار تفاعل البلمرة المتسلسل (PCR) للكشف عن الجينات الفاتكة أن جميع معزولات الإشريكية القولونية المختبرة احتوت على جينات **iroN** و **hlyF** و**ompT** ، في حين احتوت معزولات **السالمونيلا** على جينات **invA** و **stn** و **fimA** كما أظهرت معزولات المكورات العنقودية نتائج متفاوتة من حيث وجود الجينات **sea** و**tsst** و**etb**. وتم الكشف عن جينات **toxA** و**phzM** و **oprL** في معزولات السودوموناس كما أظهرت اختبارات الحساسية للمضادات الحيوية مقاومة تامة لمعزولات الإشريكية القولونية تجاه الأمبيسيلين، بينما كانت حساسة تمامًا للدُوكسي سيكلين والإيميبينيم. كما أظهرت معزولات **السالمونيلا** مقاومة تامة للأمبيسيلين، لكنها كانت حساسة للجنتاميسين والأميكاسين والإيميبينيم. أما معزولات المكورات العنقودية سالبة التخثر فكانت مقاومة تمامًا للكليندامايسين، لكنها حساسة للدُوكسي سيكلين، السيبروفلوكساسين، والجنتاميسين. وبيّنت معزولات السودوموناس مقاومة تامة للإيميبينيم، وحساسية كاملة تجاه الأميكاسين والسيفيبيم والسيبروفلوكساسين.

الكلمات الدالة: التهاب كيس المح، جينات الضراوة، بكتريا متعددة المقاومة للمضادات الحيوية.