# SOME NUTRITIONAL, BIOCHEMICAL AND CLINICOPATHOLOGICAL STUDIES ON THE EFFECT OF ESCHERICHIA COLI 0157:H7 IN QUAILS 

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#### Abstract

Fourty quails 4 weeks old and $80-100 \mathrm{~g}$ average body weight were used. They kept in a balanced diet to study some nutritional, and clinicopathological changes under E. coli O157: H7 infection. Ten quails kept as control and 40 were infected with E-coli $0157: H 7$ by dose 0.05 ml inoculated intramuscularly. Body weight were recorded, blood samples were collected at 3, 7, 21 days post infection, serum was separated for determination of AST, ALT, Total protein. Albumin, urea, creatinine, calcium phosphorous, sodium, potassium and cortisol hormone. The biochemical analysis showed an ircrease in AST and ALT and a significant change in protein. Hypoalbuminemia was observed, increase of serum urea, creatinine, hypocalcaemia, hyperphosphatemia, and increase in level of potassium, sodium and cortisol hormone in the serum.


## INTRODUCTION

Escherichia coli comprises a group of bacteria found in the intestines of humans, animals and birds, E. coli O157: H 7 strain produces potent toxins and can cause food born poisons to person transmitted disease after ingestion of very low numbers of microorganism E. coli O157: H7 was first identified as a human pathogen in 1982 (Riley et al., 1983).

Griffin and Tauxe (1992), reported that strain of E. coli infection is more often reported in the young, illness signs are bloody diarrhea, severe abdominal pain, low grade of fever and vomiting. The major source of food born E. coli O157: H7 ciated disease is undercooked grand beef. Roast beef, roast chicken, raw milk and water an outbreak of the
disease in persons, who had eaten fast food in these restaurant chain. Marks and Robert (1993), reported that the cytotoxins of $E$. coli O157: H7 production seems to be important factors in the pathogenesis of disease. These cytotoxins are among the most potent bacterial toxins. These toxins in active host cell ribosomes disrupting protein synthesis and causing cell death (Obrien et al., 1992).

Prevention of illness is especially critical in addition to strategies designed to prevent food born illness. Controlled production of live animal, meat processing relatively little information is available on clinicopathological changes in experimental animals with this disease. The present work designed to study some serum biochemical changes after experimental infections of quails with $E$. coli O 157 : H 7 .

## MATERIAL AND METHODS

## E. coli strain:

E. coli O 157 : H 7 strain was used for the experimental infection for 30 quails 4 weeks old as well as $80-100 \mathrm{~g}$ average body weight. Bird proved to be free from pathogenic bacteria and parasitic infection. Quails were infected i.m with 0.05 ml of viable organisms of E. coli O157:H7.

Blood samples were collected from wing vein and serum was separated and used for the determination of asparatate aminotransferase (AST) and alanine aminotransferase (ALT) according to Reitman and Frankel (1957), total protein and albumin according to Doumas and Biggs (1972), urea according to Saunders (1980), creatinine was estimated according to Bartels (1971), calcium was determined according to Sarkar and Chankan (1967), phosphorous was measured according to Goodwin (1970), sodium and potassium were determined by atomic absorption and serum cortisol was analyzed by means of a gammacoat 125 I cortisal vadiaimminassay kit according to the method described by Campbell and Coles (1986). Also at the time of scarification feaces from these birds are cultivated on surbital Maconkey agar medium for bacteriological examination according to Ratnam and March, (1986). Statistical analysis according to Snedecor and Cochran (1967).

## RESULTS

Bacteriological results recorded fail to ferment sorbitol and can be recognized as colorless colonies. Further confirmation, we made by agglutination test with anti-serum against the flagella antigen H 7 it gives positive results.

In Table (1) there is a significant decrease on body weight at 7 and 21 days and mortality rate increases in the $1^{\text {st }}$ week after infection. Signs of infection appear in the form of depression, loss of body weight, bloody diarrhea and ascites.

In Table (4) there is a significant increase in ALT and AST if compared with control $\mathrm{P}<0.05$. Total protein and Albumin showed highly significant decrease if compared with control group. Concerning cortisol the result showed highly significant increase if compared with control group.

As show in Table (3) Renal function test in infected squail compared with control group, showed a highly significant increase in urea, creatinine, calcium, and sodium at $\mathrm{P}<$ 0.01 . Meanwhile, phosphorus and potassium are significantly decrease at $\mathrm{P}<0.01$.

## DISCUSSION

The increase in serum AST levels in this work could be due to liver damage produced by the infected bacteria. Campell and Coles (1986) mentioned that the increased the activity of AST has been associated with hepatocellular damage in birds.

Concerning ALT in Quail some studies reported elevation of ALT in birds infected with bacteria. (Bokori and karasi, 1969). Our result agrees with Omaima (1987), who observed a significant increase in (AST \& ALT) in chicken infected with E. coli. The significant change in total protein and albumin in the present work could be due to liver and kidney damage which could be associated with bacterial infection.

Similar findings were previously mentioned by Campbell and Coles (1986), Pai, (1984), Ostroff et al., (1989) and Riley $e_{\imath}$ cil., (1983).

The increase in urea and creatinine could be due to the effect of the microorganisms or its toxin on the kidneys. Our results completely agree with Pai et al., (1986), Tzipori et al., (1987), and Obrig et al., (1987) who reported increased creatinine and urea levels in case of renal disease.

Hypocalcaemia and Hyperphosphataemia could be due to decrease calcium resorption by damaged renal tubules and associated with hypoalbuminemia as reported by Campell and Coles (1986), Beery et al., (1985) and Marks and Robert (1993).

The increase of potassium and sodium levels in serum could be due to renal disease as reported by Campbell and Coles (1986). Also the metabolism of calcium and ${ }^{-}$ phosphorus is closely linked in the body and hypocalcaemia always accompanied with hyperphosphataemia. Concerning serum cortisol level, the significant increase of serum cortisol level may be attributed to the activation of Hypothalamus pituitary axis due to stress. Our result agrees with Ghanem (1986) and Campbell and Coles (1986).

In conclusion infection of quails with E . coli O 157 : H 7 injured liver and Kidneys. The changes in liver and kidney function were more severe in 21 days of infection.

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Table (1) : Ration Composition

| Soy bean meal | 20 |
| :--- | :---: |
| Wheat middlings | 6 |
| Yellow corn | 15 |
| Cellulose | 10 |
| Vitamin premix | 2 |
| Mineral premix | 1 |
| Selenium premix | 1 |

Analysis

| Protein | 36,4 |
| :--- | :---: |
| Lipid | 13,6 |
| Ash | 7,6 |
| Glucose | 0,8 |
| Moisture | 4,2 |

Vitamin premix supplied in milligrams per/ kg of diet vit. A 4000 IU , vit $\mathrm{D}_{3} 1000 \mathrm{G} / \mathrm{U}$., vit. l. 300 mg , vit $\mathrm{k}_{3} 27$, thiamin 50 riboflavin, 61 Calcium pontathenate 150 , biotin 0.5 folic acid 10 , Sourbic acid 900 , cyanocobalamin 30 , Mineral premix in milligram per $/ \mathrm{kg}$ feed diet. Sodium chitoride, 5 gm potassium iodide 6 mg , zinc sulfate 30 , selenium 10 mg as sodium selenite.

Table (1 A): Changes in body weight in infected squail with E . coli 0157 :
H7 (mean $\pm$ S.E.)

|  | 3 days | 7days | 21 days |
| :---: | :---: | :---: | :---: |
| Control | $80 \pm 0.72$ | $89 \pm 0.13$ | $105 \pm 0.72$ |
| Infected | $82 \pm 0.42$ | $74 \pm 0.16^{*}$ | $70 \pm 0.89^{* *}$ |
| $\quad{ }^{*} \mathrm{P}<0.05$ |  |  |  |

Table (1 B): The mortality of squail per/day infected with E. coli O157: H7 (mean $\pm$ S.E.)

| No. of birds | Days | No. of dead quails |
| :---: | :---: | :---: |
| 30 | 0 | 0 |
| 30 | 3 | 6 died within 3 days |
| 24 | 7 days | 4 died |
| 20 | 21 days | 2 died |

Table (3): Renal function in quail infected with E. coli O157: H 4 (mean $\pm$ S.E)

| Parametcrs | Day of Infection | Urea $\mathrm{mg} / \mathrm{dl}$ | Creatinine $\mathrm{mg} / \mathrm{dl}$ | Calcium $\mathrm{mg} / \mathrm{dl}$ | Phosphorus $\mathrm{mg} / \mathrm{dl}$ | Sodium $\mathrm{mg} / \mathrm{dl}$ | Potassium mg/dl |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
| Control | 3 days | $2.83 \pm 0.72$ | $1.24 \pm 0.23$ | $8.74 \pm 0.62$ | $5.32 \pm 0.75$ | $136 \pm 0.72$ | $7.7 \pm 0.23$ |
| Infected |  | $3.02 \pm 0.72$ | $2.24 \pm 0.16$ | $7.82 \pm 0.42$ | $4.36 \pm 0.24$ | $140 \pm 0.62$ | $6.74 \pm 0.23$ * |
| Control | 7 days | $2.52 \pm 0.24$ | $1,34 \pm 0.62$ | $8.8 \pm 0.23$ | $5.4 \pm 0.72$ | $130 \pm 0.55$ | $7.3 \pm 0.62$ |
| Infected |  | $3.9 \pm 0.96^{*}$ | $2.72 \pm 0.24$ * | $6.7 \pm 0.16^{*}$ | $4.0 \pm 0.13$ * | $160 \pm 0.63$ * | $6.1 \pm 0.23 *$ |
| Control | 21 days | $2.62 \pm 0.44$ | $1.39 \pm 0.44$ | $8.9 \pm 0.72$ | $5.9 \pm 0.74$ | $129 \pm 0.26$ | $7.3 \pm 0.1$ |
| Infected |  | $4.8 \pm 0.84$ * | $3.24 \pm 0.52$ * | $6.1 \pm 0.24$ * | $4.4 \pm 0.13 *$ | $164 \pm 0.63$ * | $5.4 \pm 0.26^{*}$ |

Table (4): Change of liver function test \& cortical germane hormone in quail infected with E. coli O157: H7 (mean $\pm$ S.E)

| Parameters | Days of infection | AST U/ML | ADT U/ML | Total protein gm/dl | Albumin $\mathrm{mg} / \mathrm{dl}$ | Cortisol mg/dl |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: |
| Control | 3 days | $20 \pm 404$ | $12 \pm 0.23$ | $4.45 \pm 0.27$ | $2.84 \pm 0.04$ | $0.08 \pm 0.72$ |
| Infected |  | $32 \pm 0.52$ * | $79 \pm 0.23$ * | $3.94 \pm 0.14 *$ | $2.27 \pm 0.12 *$ | $0.14 \pm 0.05^{*}$ |
| Control | 7 days | $19 \pm 0.13$ | $13 \pm 0.17$ | $4.50 \pm 0.12$ | $2.72 \pm 0.02$ | $0.09 \pm 0.62$ |
| Infected |  | $36 \pm 0.48 *$ | $22 \pm 0.64 *$ | $3.00 \pm 0.24 *$ | 1.85 $\pm 0.23$ * | $0.19 \pm 0.08^{*}$ |
| Control | 21 days | $21 \pm 0.24$ | $11 \pm 0.63$ | $4.2 \pm 0.23$ | $2.72 \pm 0.03$ | $0.08 \pm 0.60$ |
| Infected |  | $54.8 \pm 0.42$ * | 19士0.74* | $2.25 \pm 0.62^{*}$ | $1.24 \pm 0.42^{*}$ | $0.28 \pm 0.07 *$ |

* $P<0.0]$

الملمص العربـبـ

## بعص الدراسات على التغذية والتغيرات الإكلينيكية الباثولوجحية <br> التى تحدث نتيجة لعدوى السـمان (E. coli O157: H7)

$$
\begin{aligned}
& \text { منى سعد على زكى * أحمد تهامى ** محمد نبيل شلبىى *** } \\
& \text { المركز القومي للبحوث قسم الطفيليات وأمراض الحّيوان } \\
& \text { المركز القومي للبحوث قسم تغذية الحيوان و الاو اجن. } \\
& \text { قسم الميكروبيولوجى معهد بحوث صحة الحيوان بالدقى. }
\end{aligned}
$$

E. coli) أجريت هذه التجربة على طائر سمان عمره ؛ أسابيع وڤد تمت العدوى بميكروب
(dose 0.05 ml) (O157: H7 الإكلينيكية البـتولوجية للكبد و الكلى فى اليوم الثثالث و السابع و الواحد وعشرون.

وقد أُثبتت النتائنج أن هذا الميكروب يؤثر كاملاً على وظائف انكلى و الكبد لهذه الطيور ويجلّها هزيلة ومصابـة بحالات إسهال مدمم بما يؤثر على إنتاجية هذه الطيور.

