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Some serum biochemical and pathological changes in squabs of domestic pigeons (Columba Livia) infected with Trichomonas

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The present study was carried out to represent a field problem in squabs of domestic pigeons (*columba livia*) at Ismailia Province. Squabs were grossly examined and showed typical lesions including yellowish caseous, fibronecrotic patches in mouth due to infection with *T. gallinae*. Forty squabs were collected and tested individually for the presence of *Trichomonas gallinae (T. gallinae*). Squabs were divided into equal four groups, the 1st was un-infected control group, the 2nd was *T. gallinae* infected untreated group, the 3rd and the 4th groups were *T. gallinae* infected and treated with metronidazole. The obtained results showed that the mortality (%) were 0, 50, 20 and 30 % in 1st, 2nd, 3rd and 4th group, respectively. Body weight was significantly reduced in all groups, although the drugs improved the weight reduction as compared to pre-treatment. Organs' weights were significantly increased after treatment as compared with the control group. Serum biochemical analysis revealed significant elevation in total protein, globulins; β- globulin and γ-globulin but albumin , α-globulin levels and A/G ratio were significantly reduced in infected squabs and increased in treated groups. Serum urea, creatinine and uric acid levels were increased, while, Serum glucose , cholesterol Na, K, Ca, P, Mg and serum iron as well as plasma ChE activity were decreased in both treated and infected groups. Serum AST, ALT, LD, γ -GGT, CK, AP activities were significantly increased in infected groups, Destructive changes in buccal cavity, hyperemia in blood vessels, necrotic changes in the liver with leucocytic infiltration and demylination of brain with preivascular oedema were observed.

Trichomoniasis is commonly known as a sexually transmitted disease of humans (caused by *Trichomonas vaginalis*) or cattle (*Tritrichomonas foetus*), but it is also a ubiquitous disease of pigeons and raptors worldwide and is caused by the flagellate protozoan *(T. gallinae*). It is a pathogenic sarcomastigophoran parasite commonly found in the upper digestive tract of columbids and in certain avian predators that feed on columbids (Conti, 1993). In pigeons, it causes a condition known as canker, the domestic pigeon, *Columba livia* (Anth), is the natural hosts for *T. gallinae*. Transmission is primarily direct, the parasite being passed between adults to their squabs through crop milk which was produced in the crop and the squabs were infected within minutes after hatching (McDougald, 2003) or between adult birds during courtship behaviors, drinking water and food have been identified also as alternative transmission routes (Kocan, 1969).

Trichomonosis has important commercial implications for pigeon breeding, aviculture (McKeon *et al.,* 1997), prevalence of *T. gallinae* infection in pigeons was higher in warmer sites and times besides the lower rainfall (Bunbury *et al.,* 2007). Typical clinical signs of trichomonosis include caseous, proliferative, fibronecrotic lesions in the oropharynx and upper digestive tract which frequently lead to the death of the infected bird by starvation. The high prevalence of *T. gallinae* infection and the low rate of pathological changes in pigeons were the main results of host-parasite relationship (Krone *et al.,* 2005). Pigeons however are more susceptible to secondary organ invasion (liver, air sacs, lungs, and brain) by virulent strains of the parasite. Eiberg strain of the parasite was a virulent hepatotropic flagellate of pigeons, it causes ulcers in the upper digestive tract which allow it to enter the circulatory system, then access the liver where they causes lesions leading to serious losses and high mortality

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especially in young birds(Narcisi *et al.,*1991).In liver, focal necrotic abscesses in all zones of lobules, with an inflammatory reaction characterized by mononuclear cells and heterophils , lesions progressed no intact hepatocytes remained in the center of foci, necrotic lesions develop in these organs leading to the death of the host (Stabler, 1954).

Treatment of *T. gallinae* suggested different antiprotozoal drugs, the effectiveness of a drug (2-amino-5-nitrotiazole), against pigeon trichomoniasis caused by the protozoan *T. gallinae* has been reported (Stabler and Mellentin, 1951). In avian veterinary medicine, several nitroimidazoles including metronidazole, dimetridazole, ronidazole and carnidazole, have been developed as effective drugs against *T. gallinae* (Franssen and Lumeij, 1992). Metronidazole was 100 % effective in naturally infected pigeons with *T. gallinae* when given orally at a dose of 100 mg/pigeon for seven days (Abd El-Motelib and Galal, 1993 and Aydin *et al.,* 2000) and 1 gm/liter for five successive days (Shihata, 1978).

This study was done to estimate mortality %, body and organs weight, some biochemical and pathological changes resulted from infection with *T. gallinae* in squabs of domestic pigeons. In addition, squabs were treated with antiprotozoal drug (metronidazole) at two therapeutic dosage levels to study its efficacy in infected squabs.

Materials and methods

Birds. Squabs were collected from a tower building at Ismailia Province suffered from severe diarrhea, ruffle feather, stop feeding, weakness, head torsion and sudden death was occurred in some squabs. Squabs were examined grossly for lesions of trichomoniasis showed yellowish necrotic patches in mouth cavity. Forty squabs (10-20 days-old and 175-200g) were collected and crop-swabbed individually to test for the presence of *T. gallinae* using sterile disposable cotton swab moistened with lactate Ringer solution. In addition, squabs were also examined to exclude virological, toxicological and bacteriological causes. Faecal examinations were negative for parasite ova, oocysts and salmonellae. Squabs were divided into four equal groups and housed in individual cages; feed and water were offered ad-*libtum*. Sanitation and hygienic measures were applied to avoid subsequent bacterial and parasitological infections.

Experimental groups. Squabs were divided into four equal groups (10 squabs each). The first group was chosen from healthy uninfected birds (control). The second was kept as infected untreated group and the third and the fourth groups were infected and treated with metronidazole at a dose of 10 and 30 mg/kg, respectively. The drug was administered per os for 7 consecutive days.

Drugs. Metronidazole (Flagyl) suspension was obtained from Sanofi Aventis,Egypt (25 mg/ml) in a package of 120 ml bottle.

Blood samples. Blood samples were collected from jugular vein of each squab from all groups at 0 (pre-treatment) and 14 days post treatment. Part of the blood was transferred to heparinized polypropylene microtubes and the other part of blood left to clot then centrifuged at 3000 r.p.m. for 10 min to obtain plasma and serum,respectively, which stored at -20° C for subsequent analysis .

Tissues samples. In addition specimens from buccal cavities, livers, lungs, hearts and brains were sampled from sacrificed bird at the end of the experiment.

Lesions of *T. gallinae are* characteristic but not pathognomonic; those of pox and *Aspergillus sp*. *fungi*, *Candida sp* .yeasts, nematodes of the genus Capillaria and vitamin A deficiency can produce similar lesions. So diagnosis should be confirmed by microscopic examination of smear of mucous or fluid from throat to demonstrate the presence of the parasite. Specimens were taken from sick squabs, or from recently dead squabs that are kept chilled and reach the diagnostic laboratory within 48 hours after death. Samples of tissues with lesions preserved in 10 percent buffered formalin or frozen whole carcasses can be used if fresh carcasses cannot be provided.

Collection of oesophageal swab samples. Mouths of pigeons were examined for the presence of diphtheritic lesions, if any. Two throat swabs were collected from each pigeon. The swabs were kept immediately in tubes containing sterile normal saline and examined immediately.

Collection of crop content. Crop contents were aseptically collected from pigeons by flushing the crop with 1.5 ml of sterile saline using a blunt ended 18G rubber tube. Tubes and syringes were flushed with 70% ethanol after sampling each pigeon.Suspended crop contents were transferred into a sterile 5 ml tube, till examination.

Identification of Trichomonad

Wet smear examination. Oesophageal swabs and crop fluid samples were examined as wet preparation within 2 h of sampling. A drop of swab sample or crop fluid was placed on a clean glass slide covered with cover-slip and examined for motile *Trichomonas* species at 200x and 400x magnifications, under phase-contrast microscope.

Staining method. A smear on clean glass slide was prepared from oesophageal swab and crop fluid samples. It was dried and fixed with osmic acid vapour and stained with Giemsa. The examination was done under 400x and 1000 magnification for trichomonad.

Direct microscopic examination. Diagnosis was established by finding the trichomonads in the abovementioned samples of infected squabs. Direct microscopic examination of wet smears revealed vast numbers of actively motile flagellate protozoa. These were classified as trichomonads because of their elongate ellipsoid shape, the presence of an obvious undulating membrane associated with four free anterior flagella which could be accurately counted only when the trichomonads had slowed down or stopped moving. The body is very plastic, but not particularly ameboid. Most of these morphological features could be recognized in-air-dried smears of throats swabbed, fixed in methanol and stained with Giemsa (Soulsby, 1986)

Serum biochemical changes. Total serum proteins were determined following the Biuret method of Reinhold (Oser, 1976). Serum albumin was determined by the bromocresol green dye-binding technique (Doumas *et al*., 1971). Globulin concentration was obtained by subtracting albumin from total serum proteins (Varely, 1976). The diacetyl monoxime method, as described by Wybenga *et al*. (1971), was followed to estimate the concentration of serum urea. Protein electrophoresis was developed using cellulose acetate as described by Lumeij (1987). Serum creatinine, uric acid and glucose were estimated by the method of Thomas (1992), Caraway (1955) and Quam et al., (1975), respectively. Serum sodium and potassium were measured as described by Oser (1979), total calcium, inorganic phosphorus and magnesium were measured as described by Grindler (1972), Goodwin, (1970) and Gindler (1971), respectively .Iron by Allain and Maurous(1979) and cholesterol was measured as described by Richmond (1973). Serum enzymatic activities were carried out colorimetrically, aspartate aminotransferase (AST) and alanine aminotransferase (ALT) activities were measured as described by Reitman and Frankle (1957). Serum lactic dehydrogenase (LD) was measured according to the method of Howell and Coles (1979), γ-Glutamyltransferase(GGT) as described by Szaz (1969) , Creatinine Kinase(CK) as described by Szaz et al., (1976), alkaline phosphatase (AP) activity according to Haussament, (1977) Plasma Cholinesterse (ChE) activity was determined colorimetrically using the method of Ellman *et al.*, (1961), as modified by Hill and Fleming (1982) for avian brain and plasma ChE.

Body and organs weights. Squabs from all groups were weighed individually to nearest 0.1 g at 0 (pre-treatment) and14 days post treatment using balance, in addition squabs were slaughtered at14 days post treatment and organs (liver, heart, lung and brain) weights were also recorded.

Histopathological examination. Buccal cavities, livers, lungs, hearts and brains were collected from all groups, examined macroscopically and taken for histopathological examination, fixed in formalin 10% and paraffin sections 6 µm thick were prepared and stained with haematoxylin and eosin as described by Bancroft *et al*., (1996).

Statistical analysis. Results were presented as mean ± S.E. from ten replicate samples. Data were analyzed using SPSS 14 (2006) software. Statistical analysis of the obtained results were subjected to ANOVA (Snedecor and Cochran, 1967), means were detected using Duncan′s multiple range test (Duncan, 1955).

Results and Discussion

Avian trichomoniasis is caused by the flagellate protozoan Trichomonas gallinae, and primarily affects Columbiformes, although it has been reported in a number of other avian orders worldwide (Forrester and Foster, 2008), pigeons act as reservoir host or carrier and an important source of infection for other avian host, which share the common parasitic fauna. Prevalence of *T. gallinae* infection in pigeons was higher in warmer sites and times besides the lower rainfall (Bunbury *et al.,* 2007).

The current study represent a field trial problem in squabs in tower building for pigeons at Ismailia Province suffered from severe diarrhea, ruffle feather, stop feeding , weakness , head torsion and sudden death was occurred in some squabs beside squabs were grossly examined showed typical lesions include yellowish caseous, proliferative, fibronecrotic patches in mouth. Further investigations (virological and bacteriological examination) revealed that no bacterial or viral infections were found beside isolation and microscopic examinations of the parasites confirm *T. gallinae* infection.

Results revealed that clinical signs become more evident and persist in the 2nd group, while fade in the 3rd group 2 days post treatment while 4 days post treatment in 4th group. Mortality (Table, 1) was 50, 20 and 30 % for the 2nd, 3rd and 4th group, respectively. No death was observed in the 1st group. Similar resultes were obtained by Abd El-Rhman et al (2008). It was clear that clinical symptoms, morbidity and mortality may be depending upon the protective immunity which may be developed by squabs. In addition, death may be attributed to starvation of birds due to trichomonas lesions, from invasion of the skull and brain by the organism, from starvation following occulsion of the esophagus, or from respiratory failure caused by blockage of the trachea (Kocan and Herman (1971). Moreover, Boal and Mannan (1999) reported a very high prevalence of *T. gallinae* and very high nestling mortality due to trichomoniasis in Cooper's hawks (*Accipiter cooperi*) from urban areas of Arizona. Bunbury (2006) reported that for squab mortality may reach to 77.5% in 1 subpopulation.

Body weight was expressed in grams and calculated at 0 and 14 day from the experimental period as shown in Table (2), results showed that significant reduction ($p<o.o1$)was observed in body weight in infected group and significant increases were observed in treated groups in comparison with infected one , both changes were time-dependant. Organ weight showed that significant ($p<0.01$) increases were observed in liver weight in all groups. Significant reduction was observed in lungs weight in the infected and treated groups. In addition, non significant changes were observed in heart and brains weight in all groups. The reduction in body weight was attributed to the diarrhea, water loss and dehydration in addition the necrotic patches in mouth cavity deprived squabs from eating or drinking to compensate such losses .But the increases in liver weight were attributed to parasitism, but no significant changes in other organs related to non selectivity parasitism for the above-mentioned organs. These results were agreed with the results obtained by Bunbury *et al., (*2007) and Stromberg *et al., (*2008) who observed body weight losses in infected birds.

Squabs in all groups were examined for gross lesions in the bucal cavity (typical lesions include yellowish caseous, proliferative, fibronecrotic patches in mouth cavity), Table (3). Results expressed as (T) for typical lesions and (N) for no lesions. These results were agreed with that obtained by Bunbury *et al., (*2007) who observed yellowish caseous lesions and/or necrotic ulcerations in the upper digestive and respiratory tracts, a foul cheesy smell emanating from the gape, and swelling of parts of the head such as the eyes or nares in infected birds. In addition, Samour (2000) found that the positive birds showed necrotic lesions. In this respect, Krone *et al.(* 2005) concluded that lesions in oropharynx of Northern goshawks (83%) were culture positive for *T. gallinae* and these birds had large oropharyngeal lesions including deformations of the cranio-mandibular apparatus, which probably lead to impaired food intake followed by marked loss of body condition of the nestlings, resulting in their death shortly after examination. Similar results were obtained by Abd El-Rahman *et al.,* (2008).

Effects of trichomoniasis on biochemical parameters were studied. Results of serum proteins (total and electrophoretic pattern) were summarized in Table (4). The obtained results

revealed that significant hyperproteinemia ($p<0.01$) was observed at 0 and 14 days in all experimental groups in comparison with the control group. Meanwhile, significant hypoproteinemia was occurred in the 3rd and 4th groups in comparison with infected group at 14 days. Hypoalbuminemia was noticed in the infected group ($p<0.01$) all over the experimental period, while insignificant hyperalbuminemia was noticed in the 3rd and 4th group at 14 days due to treatment. Serum globulin was significantly increased ($p<0.01$)in the infected group all over the experimental period , while after treatment significant decrease was recorded at 14 days in comparison with the infected group . The combined effect for albumin and globulin was expressed as A/G ratio, it was significantly reduced in all groups all over the experimental period but significant increase ($p<0.01$) was observed as a result of treatment (near to normalization to the control group). Protein electrophoresis for serum globulin revealed that 2nd group showed significant reduction in α- globulin and significant increases ($p<0.01$) in β-and γ-globulins levels. Meanwhile,correction was occurred in serum levels of α , β-& γ- globulins at 14 day in the 3rd and 4th groups when compaered with the control group. Hyperproteinemia may be attributed to severe dehydration cited from the parasitism or increased production of gamma globulin or liver dysfunction which proved by both subsequent enzymatic changes and histopathological changes. Increases in serum globulins levels may be due to parasitic infections and liver dysfunction.The brood increase in serum γ – globulins may be due to an increase in plasma cells production of immunoglobulins as the immune system has been considered to be high priority system during stress and demonstrated by preferential synthesis of immunoglobulins at the expense of other circulating proteins such as fibrinogen (Duncan *et al*., 1994). Decreases in A/G ration in diseased squabs owing to the increased level of globulin with reduced level of albumin. The deteriorated proteins levels were improved by treatment of infected squabs which tend to normalize with abovementioned drug. The results go hand to hand with that obtained by Kocan and Herman (1971) who observed decreases in serum albumin and α- globulins but increases in β- and γ- globulins in pigeon infected with *T. gallinae* and attributed these changes probably to the result of normal physiological equalization of intravascular colloid osmotic pressure. Beside, hypoalbuminemia attributed to malabsorption from the intestine, acceleration in protein catabolism due to stress , infection and fever or could be secondary to increase in globulin concentration(Benjamine,1990) and hepatic lesion (Stewart, 2003) as liver diseases inhibit albumin synthesis (Kaneko *et al.,*1997) because liver is the primary site of protein synthesis (Giralt *et al.,*1997).

Serum urea showed insignificant changes in all groups while creatinine and uric acid levels were increased in the infected groups (as compared with the control) and insignificant increases at 14 day were noticed in 3rd and 4th groups after treatment (Table, 5). Increases in serum creatinine may be owing to renal disease due to parasitism. Hyperuricemia was attributed to renal dysfunction, also, it can be expected due to reduced glomerular filtration as in dehydration , intoxication and some viral and bacterial diseases(Flammer, 1985).The elevated serum creatinine and uric acid may be due to the degenerative changes of the kidneys denoted to renal dysfunction resulted from the degeneration and necrosis of the epithelial lining renal tubules (Stewart, 2003).

Hypoglycemia and significant decrease in cholesterol levels($p<0.01$) were recorded in the infected and treated groups while after treatment there was significant increase as compared with the infected group (Table,5). Hypoglycemia was related to starvation from mouth lesions, the results were agreed with that obtained by Lumeij (1987, b) as starvation of pigeon for 73 hours induced hypoglycemia and Lumeij (1987, a) reported that hypoglycemia in birds associated with aneroxia. In addition, Decreased serum cholesterol levels may be attributed to trichomoniasis which stimulates the β-oxidation of lipid, mostly occurred in the liver by cytochrome P_{450} system (Kaneko *et al*., 1997). Moreover,the results consistent with Wodrpel and Rosskopf (1987) who reported that decreased cholesterol level associated with some cases of liver diseases.

Table (1): Mortality (%) of the control and infected squabs with trichomonas gallinae from Ismailia Province before and after treatment with metronidazole.

G	T. I. (n=10)	T. I. +M1(n=10)	T. I. +M2(n=10)
S-0	0	0	0
G	T. I.(n=6)	T. I. +M1(n=8)	T. I. +M2(n=7)
S-14	50	20	30

G=groups, (S-0 &S-14,Sampling time, days),C. = Control (un-infected group),T. I.= (*T. gallinae* infected untreated group),T. I. +M1= (*T. gallinae* infected group & treated with metronidazole at a dose of 10 mg/kg)& T. I. +M2= (*T. gallinae* infected group & treated with metronidazole at a dose of 30 mg/kg).

Table (2): Body and organs weight (g) of the control and infected squabs with trichomonas gallinae from Ismailia Province before and after treatment with metronidazole (Mean ±S.E.).

G	C.(n=10)	T. I.(n=10)	T. I. +M1(n=10)	T. I. +M2(n=10)
S-0	194.10±1.49^{a}	174.90±2.05^{b}	173.60±1.84^{bc}	173.20±1.96^{bcd}
G	C.(n=10)	T. I.(n=5)	T. I. +M1(n=8)	T. I. +M2(n=7)
S-14	198.30±1.32^{a}	161.60±1.08^{d}	186.50±2.75^{b}	185.40±1.82 ^{bc}
		Organ weight(g)		
	C.(n=10)	T. I.(n=5)	T. I. +M1(n=8)	T. I. +M2(n=7)
Liver	7.49±0.30^{d}	10.30±0.15^{a}	8.55±0.33^{b}	8.43±0.36^{bc}
Heart	2.06±0.05	2.02±0.05	2.03±0.02	2.06±0.05
Lung	1.90±0.03^{a}	1.52±0.09^{d}	1.86±0.02^{b}	1.79±0.03^{c}
Brain	1.24±0.04	1.22±0.15	1.24±0.05	1.24±0.04

G=groups, (S-0&S-14,Sampling time, days), C. = Control (un-infected group),T. I.= (*T. gallinae* infected untreated group),T. I. +M1= (*T. gallinae* infected group & treated with metronidazole at a dose of 10 mg/kg)& T. I. +M2= (*T. gallinae* infected group & treated with metronidazole at a dose of 30 mg/kg).
Means in the same raw with different superscripts are significantly different at ($p< 0.01$).

Results for serum electrolytes were summarized in Table (6). Significant hyponatremia ($p<0.01$) and hypokalemia ($p<0.01$ and $p<0.05$) were noticed in the infected group at 0 and 14 days. In 3rd and 4th groups serum sodium and potassium levels were significantly increased after treatment as compared with the infected group. Hyponatremia resulted from renal dysfunction and its loss in watery diarrhea due to trichomoniais. Similar results were obtained by Gylstorff and Grimm (1987) and Wodrpel and Rosskopf (1987).

Regarding serum calcium, significant hypocalcaemia was noticed in the infected birds at 0 and 14 days ($p <0.01$) while, insignificant elevation were noticed in serum calcium in 3rd and 4th groups at 14 days (Table, 6). Regarding hypocalcaemia may be attributed to calcium loss in diarrhea or may be due to the increase in pH value of small intestine which hinders absorption of calcium, in addition this loss could be explained by Lumeij, (1990) who reported that hypoalbuminemia will reduce the quantity of bound calcium and result in a decrease in total calcium.

Significant hypophosphatemia ($p<0.01$) and hypomagnesaemia ($p<0.05$) at 0 and 14 days were recorded in 2nd group. After treatment, both levels of inorganic phosphorus and magnesium were insignificantly decreased (Table, 6). Serum iron was significantly reduced in infected group all over the experimental period ($p <0.01$) at 0 and 14 days and insignificantly decreased after treatment in 3rd group only. Hypophosphatemia and hypomagnesaemia occurred due to their extreme losses in diarrhea and anorexia. Malabsorption of phosphorus from small intestine due to the change of its pH value cause the decrease in phosphorus level .The decrease in the level of serum iron may be due to loss of appetite with impaired absorption of iron from intestine or may be attributed to involvement of spleen in combating the infection and disturbance of its function in storage and metabolism of iron (Stewart, 2003).

Enzymatic activity results were expressed in Table (7). Concerning serum transferases activities (AST & ALT) in serum, they were significantly elevated ($p<0.01$) all over the experiment period in all groups. After treatment

the levels were significantly reduced when compared with the infected group at 14 days. Serum AST and ALT levels were elevated due to degenerative liver damages supported by histopathological finding of liver. The two enzymes appeared in a direct relation with each other and their determination could be taken as an indicator of both hepatic and intestinal damage (Kaneko *et al*., 1997).

Serum LDH, γ -GT, CK and AP activities were significantly increased ($p<0.01$) all over the experimental period in all groups when compared with the control group. Meanwhile due to treatment the levels were significantly reduced as compared with the infected group at 14 days, Table (7). Elevated serum enzymatic activities may be due to tissue and liver damages. Moreover, the elevated CK activities were related to cell necrosis and convulsions. Also, elevated serum AP related to both liver damage and enteritis (Hochleithner, 1990).

Plasma ChE activities were recorded in Table (7). Results revealed that the infected and treated groups showed significant reduction ($p<0.01$)all over the experimental period in comparing with the control group .While after treatment these levels increased significantly as compared with the infected group at 14 days. This decrease may be supported by pathological changes of the brains and leads to nervous manifestations in diseased squabs.

Concerning the effect on pathological findings for the specimens of buccal cavities, livers, lungs, hearts and brains, Macropathological findings results revealed that, yellowish caseous, proliferative, fibronecrotic patches in mouth cavity were observed in the infected squabs . Also, hemorrhage in outer surface of hepatic lobules and necrotic foci could be determined in 2nd group. Hypermeia in the outer surface of livers were observed in 3rd and 4th groups. Finally, hyperemia on brains of infected and treated groups with necrotic foci in brains of infected group only were noticed.

Micropathological examination revealed that micropathological lesions were noticed in buccal cavities, livers and brains of the infected group only while no histopathogical findings were noticed after treatment. The buccal cavity showed destruction of the lining epithelium replaced by thick layer of fibrinopurulent exudates (Fig. 1) with granulocytes and giant cells infiltration. Such results agreed with Bothaina *et al.*, (2008) who revealed purulent inflammation with caseation and necrosis in addition to inflammatory cells infiltration in both buccal cavity and crop as a result of *Trichomonus gallinae*.

The salivary glands in the lamina propia showed denudation and atrophy of their epithelium (Fig. 1) in addition to hyperaemia of blood vessels.The results also agreed with that obtained by Mesa *et al.,* (1961) who found that lesion in oral mucosa progress into nodules that may then form thick caseous necrotic masses. The earliest phase of infection is characterized histologically by a palisading of tricbomonads on the epithelial surface of the oral mucosa; leucocytic infiltration then occurs, followed by necrosis and an increase in the size of the lesion. In this respect, (Kietzmann, 1993) reported that bell-shaped ameboid form parasite in ring doves has been shown to cause cell damage to avian palatal-esophageal epithelium during disease onset and progression. In addition, Abd El-Rahman (1991) found that an increase in thickening in the wall of crop and oesophagus due to pressure of oedema and hyperkeratinization of mucosa. Similar findings were reported by Abd El-Rahman *et al*., (2008).

Liver exhibited dilatation of the central veins (Fig. 2) with perivascular aggregation of mononuclear cells. Pronounced vaccuolar degeneration (Fig.3) with hepatocellular necrosis especially at the periphery and perivascular. The results were agreed with that reported by Levine (1985), Narcisi *et al.*, (1991) and Abd El-Rahman *et al.,* (2008). Beside, Helmy (1995) reported that the liver showed perivascular aggregation of heterophils and lymphocytes.

Hisopathological findings of the brains revealedcerebellar demylination (Fig.4) and preivascular oedema (Fig.5). The results were agreed with Levine (1985) who reported that T. gallinae causes caseous masses in the roof of the mouth may extend to involve the brain. Patton and Patton (1996) observed that in a juvenile, male mockingbird (*Mimus polyglottos*) died due *Tetratrichomonas gallinarum* infection (which morphologically similar to *Trichomonas gallinae*), left cerebrum had an extensive area of

Table (3): Gross lesion of the control and infected squabs with trichomonas gallinae before and after treatment with metronidazole.

G	C.(n=10)	T. I.(n=10)	T. I.+M1(n=10)	T. I. +M2(n=10)
S-0	N	T.	T.	T.
	N	T.	T.	T.
	N	T.	T.	T.
	N	T.	T.	T.
	N	T.	T.	T.
	N	T.	T.	T.
	N	T.	T.	T.
	N	T.	T.	T.
	N	N.	T.	T.
	N	N.	T.	T.
S-14	**C.(n=10)**	**T. I.(n=5)**	**T. I. +M1(n=8)**	**T. I. +M2(n=7)**
	N	T.	N.	N.
	N	T.	N.	N.
	N	T.	N.	N.
	N	T.	N.	N.
	N	N.	N.	N.
	N	-	N.	N.
	N	-	N.	N.
	N	-	N.	-
	N	-	-	-
	N	-	-	-

G=groups, (S-0 &S-14,Sampling time, days),C. = Control (un-infected group),T. I.= (*T. gallinae* infected untreated group),T. I. +M1= (*T. gallinae* infected group & treated with metronidazole at a dose of 10 mg/kg)& T. I. +M2= (*T. gallinae* infected group & treated with metronidazole at a dose of 30 mg/kg),(T. =typical lesions, N.= no lesions and - = dead animal).

necrosis with massive numbers of uninucleate, round protozoa concentrated around blood vessels. The affected area had an accentuated vasculature due to congestion and perivascular protozoa and inflammatory cells. Much of the left ventricle was obliterated, heterophils and pyknotic round nuclei (spent heterophils) were present among the organisms and vessels had prominent endothelium and many had fibrinoid degeneration. Portions of the meninges had infiltrates of lymphocytes, heterophils, and monocytes and protozoa were found in the cerebrum, inflammed meninges adjacent to the optic nerves on the ventrum of the brain. No macro or micr-pathological changes were observed in the lung and heart.

From the obtained data it was clear that the disease has an important issue on biochemical and pathological parameters in infected pigeons which counteract by treatment by specific antiparasitic drugs. Metronidazole is more effective in treating diseased squabs with more response to alleviate serum biochemical changes. In comparison of the dose effects, no measurable changes were observed on increasing dose, so the effects was not dose related. It is better to control trichomoniasis through its prevention by several means as use several feeding sites to reduce bird numbers at any one site, move the feeding sites regularly to reduce any build-up of debris and infectious agents around the feeders, finally clean and disinfect feeders and feeding stations regularly beside rinse the feeders and allow them to dry before using them again.

Table (4): Total and electrophoretic pattern of proteins in control, infected and treated squabs (Mean ±S.E.).

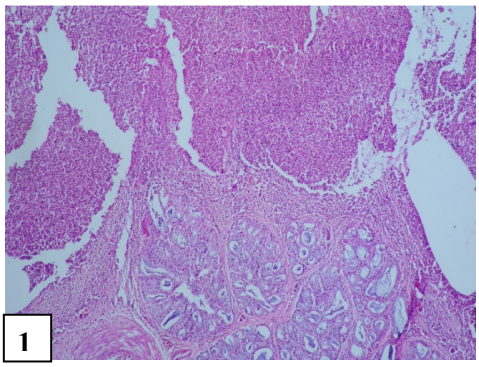
Total Protein (g/L)				
G.	C.(n=10)	T. I.(n=10)	T.I.+M1(n=10)	T. I.+ M2(n=10)
S-0	25.50±0.70^{d}	29.9±0.60^{abc}	30.10±0.60^{ab}	30.30±0.80^{a}
G.	C.(n=10)	T. I.(n=5)	T. I. +M1(n=8)	T. I. +M2(n=7)
S-14	26.50±0.80^{d}	33.2±0.90^{a}	27.10±0.60^{bc}	27.70±0.60^{b}
Albumin (g/L)				
G.	C.(n=10)	T. I.(n=10)	T. I. +M1(n=10)	T. I. +M2(n=10)
S-0	16.70±0.52^{a}	11.5±0.48^{b}	10.60±0.16^{bcd}	11.40±0.54^{bc}
G.	C.(n=10)	T. I.(n=5)	T. I. +M1(n=8)	T. I. +M2(n=7)
S-14	16.80±0.39^{abc}	12.2±1.07^{d}	17.12±0.64^{ab}	17.42±0.97^{a}
Globulin (g/L)				
G.	C.(n=10)	T. I.(n=10)	T. I. +M1(n=10)	T. I.+ M2(n=10)
S-0	8.80±0.49^{d}	18.4±0.62^{abc}	19.50±0.56^{a}	18.90±0.74^{ab}
G.	C.(n=10)	T. I.(n=5)	T. I. +M1(n=8)	T. I. +M2(n=7)
S-14	9.70±0.52^{d}	21±0.71^{a}	10.00±0.46^{bc}	10.28±0.36^{b}
A/G Ratio				
G.	C.(n=10)	T. I.(n=10)	T. I. +M1(n=10)	T. I.+ M2(n=10)
S-0	1.94±0.11^{a}	0.64±0.04^{b}	0.55±0.02^{bcd}	0.61±0.04^{bc}
G.	C.(n=10)	T. I.(n=5)	T. I. +M1(n=8)	T. I. +M2(n=7)
S-14	1.74±0.09^{a}	0.58±0.06^{d}	1.70±0.13^{bc}	1.70±0.14^{b}
α- globulin (g/L)				
G.	C.(n=10)	T. I.(n=10)	T.I.+ M1(n=10)	T. I.+ M2(n=10)
S-0	1.70±0.12^{a}	1.21±0.06^{bcd}	1.21±1.04^{bc}	1.23±0.04^{b}
G.	C.(n=10)	T. I.(n=5)	T. I. +M1(n=8)	T. I. +M2(n=7)
S-14	1.93±0.10^{abc}	1.24±0.02^{d}	2.08±0.15^{a}	2.07±0.20^{ab}
β- globulin (g/L)				
G.	C.(n=10)	T. I.(n=10)	T.I.+ M1(n=10)	T. I. M2(n=10)
S-0	4.43±0.14^{d}	9.30±0.10^{b}	9.96±0.16^{a}	8.89±0.19^{bc}
G.	C.(n=10)	T. I.(n=5)	T. I. +M1(n=8)	T. I. +M2(n=7)
S-14	4.93±0.05^{bcd}	11.16±0.30^{a}	5.25±0.09^{bc}	5.29±0.14^{b}
γ - globulin (g/L)				
G.	C.(n=10)	T. I.(n=10)	T. I. +M1(n=10)	T. I. +M2(n=10)
S-0	2.76±0.07^{d}	7.90±0.12^{abc}	8.20±0.20^{ab}	8.67±0.20^{a}
G.	C.(n=10)	T. I.(n=5)	T. I. +M1(n=8)	T. I. +M2(n=7)
S-14	2.90±0.05^{bc}	8.66±0.30^{a}	2.70 ±0.10^{bcd}	2.90±0.10^{b}

G=groups, (S-0&S-14,Sampling time, days),C. = Control (un-infected group),T. I.= (T. gallinae infected untreated group),T. I. +M= (T. gallinae infected group & treated with metronidazole at a dose of 10 mg/kg)& T. I. +M2= (T. gallinae infected group & treated with metronidazole at a dose of 30 mg/kg).
Means in the same raw with different superscripts are significantly different at ($p< 0.01$).

Table (5): Serum urea, creatinine, uric acid, glucose & cholesterol of the control and infected squabs before and after treatment with metronidazole (Mean ±S.E.).

		Serum urea (mg/L)		
G.	C.(n=10)	T. I.(n=10)	T.I.+M1(n=10)	T.I.+ M2(n=10)
S-0	0.53±0.02	0.62±0.01	0.63±0.01	0.63±0.02
G.	C.(n=10)	T. I.(n=5)	T. I. +M1(n=8)	T. I. +M2(n=7)
S-14	0.55±0.01	0.67±0.02	0.55±0.01	0.58±0.02
		Creatinine (µmol/L)		
G.	C.(n=10)	T. I.(n=10)	T.I.+ M1(n=10)	T. I + M2(n=10)
S-0	23.00±0.60^{d}	27.30±0.50^{abc}	27.70±0.40^{ab}	27.90±0.50^{a}
G.	C.(n=10)	T. I.(n=5)	T. I. +M1(n=8)	T. I. +M2(n=7)
S-14	25.50±0.50^{bcd}	29.60±0.90^{a}	25.88±0.40^{bc}	26.57±0.50^{b}
		Uric acid (µmol/L)		
G.	C.(n=10)	T. I.(n=10)	T. I. +M1(n=10)	T. I.+M2(n=10)
S-0	168.10±4.20^{d}	184.80±4.00^{abc}	189.30±2.90^{ab}	189.80±3.30^{a}
G.	C.(n=10)	T. I.(n=5)	T. I. +M1(n=8)	T. I. +M2(n=7)
S-14	170.80±4.10^{bcd}	191.20±4.20^{a}	176.87±3.90^{bc}	177.14±4.30^{b}
		Glucose (mmol/L)		
G.	C.(n=10)	T. I.(n=10)	T. I. +M1(n=10)	T. I.+ M2(n=10)
S-0	16.30±0.40^{a}	13.70±0.45^{bcd}	14.00±0.56^{b}	13.90±0.64^{bc}
G.	C.(n=10)	T. I.(n=5)	T. I. +M1(n=8)	T. I. +M2(n=7)
S-14	16.80±0.51^{a}	11.60±0.68^{d}	15.50±0.60^{bc}	15.43±0.37^{b}
		Cholesterol (mmol/L)		
G.	C.(n=10)	T. I.(n=10)	T. I. +M1(n=10)	T. I. +M2(n=10)
S-0	3.17±0.08^{a}	2.47±0.09^{bcd}	2.52 ±0.09^{bc}	2.51±0.12^{b}
G.	C.(n=10)	T. I.(n=5)	T. I. +M1(n=8)	T. I. +M2(n=7)
S-14	3.39 ±0.11^{a}	2.16±0.07^{d}	3.23±0.12^{bc}	3.21±0.15^{b}

G=groups, (S-0&S-14,Sampling time, days),C. = Control (un-infected group),T. I.= (*T. gallinae* infected untreated group),T. I.+M1= (*T. gallinae* infected group and treated with metronidazole at a dose of 10 mg/kg) and T. I.+M2= (*T. gallinae* infected group & treated with metronidazole at a dose of 30 mg/kg).
Means in the same raw with different superscripts are significantly different at ($p< 0.01$) except uric acid at 14 days is significant at ($p<0.05$).



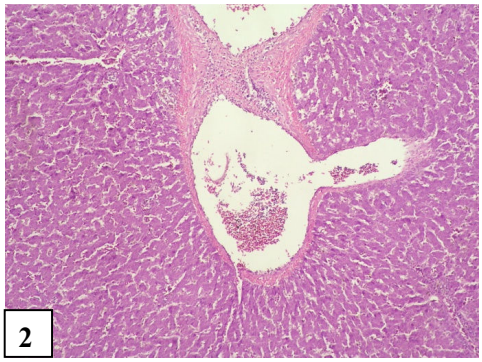


Fig 1: Buccal cavity showed destructed epithelium with thick layer of fibrinopurulent exudates,the salivary glands in the lamina propia showed denudation and atrophy of their epithelium (H&E x 100).
Fig 2: Liver showed dilatation of central veins (H&E x100).

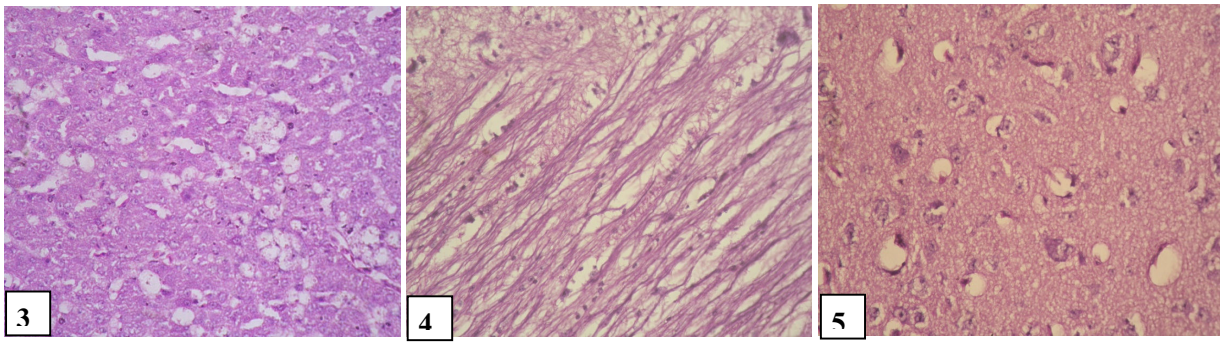


Fig 3: Liver showed vaccoular degeneration and hepatocellular necrosis (H&E x 200)
Fig 4: Cerebellum showed demylination (H&E x 200).
Fig 5: Cerebellum showed perivascular oedema (H&E x 400).

Table (6): Serum sodium, potassium, total calcium, inorganic phosphorus, magnesium and iron of the control and infected squabs before and after treatment with metronidazole (Mean ±S.E.)

Sodium (mmol/L)				
G	C.(n=10)	T. I.(n=10)	T. I.+M1(n=10)	T. I. +M2(n=10)
S-0	149.80±2.30^{a}	136.50±1.60^{b}	134.00±1.20^{cd}	134.40±1.80^{c}
G	C.(n=10)	T. I.(n=5)	T. I. +M1(n=8)	T. I. +M2(n=7)
S-14	152.80±1.80^{a}	126.80 ±1.0^{d}	149.40±2.10^{b}	148.30±1.70^{bc}
Potassium (mmol/L)				
G	C.(n=10)	T. I.(n=10)	T.I.+M1(n=10)	T. I. +M2(n=10)
S-0	4.65±0.18^{a}	4.07±0.03^{b}	4.01 ±0.13^{bcd}	4.03±0.17^{bc}
G	C.(n=10)	T. I.(n=5)	T. I. +M1(n=8)	T.I.+M2(n=7)
S-14	4.69±0.12^{a}	3.68±0.18^{d}	4.68±0.23^{ab}	4.50±0.38^{abc}
Total calcium (mmol/L)				
G	C.(n=10)	T. I.(n=10)	T.I.+M1(n=10)	T. I. +M2(n=10)
S-0	2.28±014^{a}	1.57±0.11^{bcd}	1.59±0.11^{bc}	1.72±0.12^{b}
G	C.(n=10)	T. I.(n=5)	T. I. +M1(n=8)	T. I. +M2(n=7)
S-14	2.60±0.10^{a}	1.90±0.20^{d}	2.44±0.11^{bc}	2.48±0.09^{ab}
Inorganic phosphorus (mmol/L)				
G	C.(n=10)	T. I.(n=10)	T.I.+M1(n=10)	T. I. +M2(n=10)
S-0	0.99±0.07^{a}	0.76±0.06^{b}	0.73±0.05^{bc}	0.72 ±0.05^{bcd}
G	C.(n=10)	T. I.(n=5)	T. I. +M1(n=8)	T. I. +M2(n=7)
S-14	1.31±0.11	0.54 ±0.04	1.20±0.09	1.10 ±0.04
Magnesium (mmol/L)				
G	C.(n=10)	T. I.(n=10)	T.I.+M1(n=10)	T. I. +M2(n=10)
S-0	1.14±0.11^{a}	0.81±0.07^{bcd}	0.84 ±0.08^{b}	0.83±0.09^{bc}
G	C.(n=10)	T. I.(n=5)	T. I. +M1(n=8)	T. I. +M2(n=7)
S-14	0.27±0.090^{a}	0.68±0.07d	1.20±0.09^{ab}	1.16±0.06^{abc}
Iron (µmol/L)				
G	C.(n=10)	T. I.(n=10)	T.I.+M1(n=10)	T. I. +M2(n=10)
S-0	18.40±0.73^{a}	15.50±0.73^{b}	15.40 ±0.82^{bc}	15.00 ±0.64^{bcd}
G	C.(n=10)	T. I.(n=5)	T. I. +M1(n=8)	T. I. +M2(n=7)
S-14	18.50±0.96^{a}	11.80 ±0.37^{d}	17.75 ±0.25^{ab}	17.14 ±0.59^{bc}

G=groups, (S-0 &S-14,Sampling time, days),C. = Control (un-infected group),T. I.= (*T. gallinae* infected untreated group),T. I. +M1= (*T. gallinae* infected group & treated with metronidazole at a dose of 10 mg/kg)& T. I. +M2= (m*T. gallinae* infected group & treated with metronidazole at a dose of 30 mg/kg).
Means in the same raw with different superscripts are significantly different at ($p< 0.01$) except potassium at 14 days and magnesium at 0 and 14 days are significant at ($p<0.05$)

Table (7): Enzymatic activity of the control and infected squabs before and after treatment with metronidazole (Mean ±S.E.).

		AST (IU/L)		
G	C.(n=10)	T. I.(n=10)	T.I. +M1(n=10)	DT. I. +M2(n=10)
S-0	60.90±1.26^{d}	67.40±1.23^{abc}	67.60±1.11^{ab}	68.10 ±0.81^{a}
G	C.(n=10)	T. I.(n=5)	T. I. +M1(n=8)	T. I. +M2(n=7)
S-14	61.00±1.40^{d}	69.60 ±1.30^{a}	63.80±1.00^{bc}	64.30±1.40^{b}
		ALT (IU/L)		
G	C.(n=10)	T. I.(n=10)	T.I.+M1(n=10)	T. I. +M2(n=10)
S-0	24.60±0.70^{d}	28.10 ±0.70^{bc}	28.80±0.60^{ab}	28.90±0.50^{a}
G	C.(n=10)	T. I.(n=5)	T. I. +M1(n=8)	T. I. +M2(n=7)
S-14	25.80±0.70^{d}	35.00±1.50^{a}	27.60±0.70^{bc}	27.70±0.90^{b}
		L DH (IU/L)		
G	C.(n=10)	T. I.(n=10)	T.I. +M1(n=10)	T. I. +M2(n=10)
S-0	85.60±1.90^{d}	127.30±4.40^{a}	127.30±4.80^{ab}	123.70±4.20^{abc}
G	C.(n=10)	T. I.(n=5)	T. I. +M1(n=8)	T. I. +M2(n=7)
S-14	90.70 ±1.50^{d}	141.80±3.70^{a}	95.60±2.10^{b}	94.90±2.50^{bc}
		(γ -GT) (IU/L)		
G	C.(n=10)	T. I.(n=10)	T.I. +M1(n=10)	T. I. +M2(n=10)
S-0	2.30±0.10^{d}	4.40±0.30^{abc}	4.43±0.10^{a}	4.44 ±0.20^{ab}
G	C.(n=10)	T. I.(n=5)	T. I. +M1(n=8)	T. I. +M2(n=7)
S-14	2.60±0.10^{dc}	4.80±0.20^{a}	2.90±0.20^{b}	2.730±0.30^{c}
		CK (IU/L)		
G	C.(n=10)	T. I.(n=10)	T.I. +M1(n=10)	T. I. +M2(n=10)
S-0	166.40 ±6.40^{d}	197.60 ±2.20^{bc}	200.40±2.50^{a}	202.00±1.80^{ab}
G	C.(n=10)	T. I.(n=5)	T. I. +M1(n=8)	T. I. +M2(n=7)
S-14	174.50±3.70^{c}	202.80±1.60^{a}	173.10±5.10^{dc}	181.40±3.10^{b}
		AP(IU/L)		
G	C.(n=10)	T. I.(n=10)	T.I. +M1(n=10)	T. I. +M2(n=10)
S-0	330.20±11.40^{d}	436.00±11.60^{abc}	436.30±15.00^{ab}	437.10±14.70^{a}
G	C.(n=10)	T. I.(n=5)	T. I. +M1(n=8)	T. I. +M2(n=7)
S-14	348.30±6.60^{d}	448.60±10.90^{a}	364.40 ± 1.00^{bc}	365.10±1.70^{b}
		ChE(IU/L)		
G	C.(n=10)	T. I.(n=10)	T.I. +M1(n=10)	T. I. +M2(n=10)
S-0	1075.70±7.40^{a}	964.40±16.20^{b}	952.10±23.70^{bcd}	955.20±15.10^{bc}
G	C.(n=10)	T. I.(n=5)	T. I. +M1(n=8)	T. I. +M2(n=7)
S-14	1068.10±14.00^{a}	947.80±21.90^{d}	999.00±19.00^{b}	975.28 ±19.90^{c}

G=groups, (S-0, &S-14,Sampling time, days),C. = Control (un-infected group),T. I.= (T. gallinae infected untreated group),T. I. +M1= (T. gallinae infected group & treated with metronidazole at a dose of 10 mg/kg)& T. I. +M2= (T. gallinae infected group & treated with metronidazole at a dose of 30 mg/kg),AST(aspartate a inotransferase),ALT(alanine aminotransferase), LDH (lactic dehydrogenase), γ –GT (γ-Glutamyltransferase) , CK (Creatinine Kinase), AP(alkaline phosphatase), ChE(Cholinesterase).
Means in the same raw with different superscripts are significantly different at ($p< 0.01$).

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بعض التغييرات البيوكيمائية والباثولوجية في زغاليل الحمام المصاب بمرض الترايكومونس

أجريت هذه الدراسة والتي كانت تمثل أحد المشاكل الحقلية بزغاليل الحمام بمحافظة الاسماعيلية والذي كان يعاني من اسهال شديد, نفش بالريش, فقدان للشهية , ضعف عام , التواء بالرأس مع وفيات مفاجئة ببعض الزغاليل. وبفحص الزغاليل المصابة وجد بها علامات مميزة بالفم علي شكل بقع صفراء متجبنة ومنتشرة ومتكرزة نتيجة الأصابة بمرض الترايكومونس. وقد أجريت هذه الدراسة علي عدد ٤٠من زغاليل الحمام والتي يتراوح عمرها من ١٠-٢٠ يوماً ووزنها من ١٧٥-٢٠٠ جراماً والتي تم اختبارها علي حدة لتشخيص المرض عن طريق التعرف علي الطفيل وفحصه مجهرياً ، وتم تقسيم الزغاليل الي أربعة مجاميع , المجموعة الأولي هي المجموعة الضابطة , المجموعة الثانية هي المصابة بمرض الترايكومونس و المجموعة الثالثة والرابعة هي المصابة بمرض الترايكومونس والمعالجة بالميترانذازول بجرعات ١٠, ٣٠ مجم/ كجم من الوزن الحي علي التوالى بالفم و لمدة سبعة أيام متتالية. و أسفرت النتائج عن ان نسبة الوفيات كانت٥٠, ٢٠, ٣٠ % في المجموعات الثانية , الثالثة , الرابعة علي التوالي , كما حدث نقصاً معنوياً في وزن الجسم نتيجة الأصابة بمرض الترايكومونس في المجموعات الثانية , الثالثة , الرابعة بالمقارنة مع المجموعة الضابطة علي الرغم من حدوث تحسن بالعلاج وحدث زيادة معنوية في وزن الأحشاء الداخلية بالمقارنة مع المجموعة الضابطةوقد اثبتت الدراسة زيادة معنوية في البروتين الكلي,الجلوبين , البيتا جلوبين و الجاماجلوبين في المجموعات الثانية , الثالثة , الرابعة بالمقارنة مع المجموعة الضابطة وانخفاضاً معنوياً في مستوي الألبومين , الفا جلوبين ونسبة الأبيومين الى الجلوبيولين في الزغاليل المصابة والتي تحسنت نسبتها بالعلاج . و ايضا ظهرت زيادة معنوية في مستوي الكرياتنين وحامض اليوريك بالمصل في كل الزغاليل عدا المجموعة الثالثة بعد ١٤ يوماً من العلاج وذلك بالمقارنة مع المجموعة الضابطة.كما حدث انخفاضاً في مستوي الجلوكوز, الكوليسترول و الصوديوم , البوتاسيوم, الكالسيوم , الفوسفور الغير عضوي والماغنسيوم والحديد بالمصل في المجموعات المصابة والمعالجة. كما اوضحت الدراسة ارتفاع معنوى فى نشاط انزيمات الكبد الترانز امينيزيز (ALT، AST), والاكتيك ديهيدروجيناز (LDH), والجاما جلوتيميك ترانسفيراز- γ (GT)والكرياتينين كينيز(CK) و الفوسفاتيز القاعدي (AP) في المجموعات المصابة مما يدل علي ان هناك تلف تدميري بالأنسجة و الخلايا الكبدية والذي أحدث العلاج بالميتروندازول تحسناً بها كما حدث ايضاً انخفاضاً في مستوي انزيم الكولين أستراز(ChE) بالبلازما . وبدراسة التغيرات الباثولوجية, وجدت تغييرات تدميرية مع تجمع للخلايا المحببة والعملاقة ودمور بالغدد اللعابية وأحمرار بالأوعية الدموية فى التجويف الفموى . وتغييرات تنكرزية بالأكباد مع أنتشار لخلايا كرات الدم البيضاء .كما أحدث فقد للخلايا الميلانية بالمخ مع تورم بالأوعية الدموية فى الطيور المصابة بالترايكومونس .