

Clinical, Haematological and Biochemical Study of Experimental Salmonellosis in Puppies

***Rahman K. Muhsen , Saadi ,A. G. Al-Sammarrae and Abul-Munaf H. Al- judi**

***Department of Veterinary Internal and Preventive Medicine, University of Basrah
Department of Veterinary Internal and Preventive Medicine, University of Baghdad**

Summary

This study was carried out to study the clinical, haematological and biochemical changes in dogs experimentally infected with *Salmonella typhimurium*. Ten (10) puppies were included in this study which divided into two groups. The first group inoculated orally with 10 ml of sterile trypticase soya broth (control group) while the second group were inoculated orally with 10 mL of trypticase soya broth which containing 4.8×10^9 CFU /mL of *Salmonella typhimurium* (infected group). All animals were observed daily until the death of infected group to investigate the clinical, haematological and biochemical parameters and the results revealed that, the dogs experimentally infected with *Salmonella typhimurium* showed both septicemic and gastrointestinal forms of the disease accompanied with isolation of *Salmonella typhimurium* from blood and stool throughout the study. The results also revealed that, there were severe haematological and biochemical changes in dogs experimentally infected with *Salmonella typhimurium*

Introduction

Salmonellosis : An infectious disease of man and animals caused by *Salmonella species* (Murdoch , 1979, Radostitis *et al .*, 2000) Salmonellosis is a worldwide problem and considered as one of the most important zoonotic disease in developing countries . It is an economically important disease in farm animals (Williams , 1980).

Clinical Salmonellosis is uncommon in adult dogs although a some of serotype may be carried in normal animals , while the disease is more severe in

young animals and in those subjected to stress condition (Kallow and Hasso, 2001). The clinical feature of salmonellosis in dogs varies from asymptomatic carriers; gastrointestinal to septicemic forms (Nation , 1984).

The disease has public health importance because it is one of commonest disease in man and its principle reservoir are domestic animals including dogs (Rubin and Weinstein 1987).

Material and Methods

Ten puppies from local breed aged between 2-4 months and weighted between 3-4.5 K.g were used in this study. All animals were prepared to experiment by treatment with ciprofloxacin (20mg/kg B.W daily for six day); Ivermectin (0.2 mg/kg B.W) s/c single dose and Niclosamind (50mg/kg B.W). The animals included in this study were divided into two groups.

- a) **Control group:** the animals of this group inoculated orally with 10 ml of sterile trypticase Soya broth.
- b) **Infected group:** The animals of this group were inoculated orally with 10 ml of trypticase Soya broth containing 4.8×10^8 CFU of *Salmonella typhimurim* per ml.

All animals were observed daily pre and post inoculation until the death of infected animals as the following.

- 1- Clinical examination which include temperature, appetite, general condition, respiration, presence of diarrhea, dehydration and examination of skin.
- 2- Culture of faeces and blood according to Baron *et al.* (1994).
- 3- Haematological examination including PCV, Hb, RbCs, total and differential leukocyte counts, was estimated according to Coles (1986).

- 4- Total serum protein and albumin were estimated by using kits from Randox company , while plasma fibrinogen were estimated using referctometer according to Schalm *et al.*, (1975).
- 5- Liver enzymes (AST, ALT and ALP) were estimated using kits from Randox Company.
- 6- Estimation the concentration of various element including sodium , potassium , chloride, calcium and phosphorus were done using atomic absorption and kits from Randox company.

Result

All infected puppies showed two forms of the disease which were septicemic form and gastrointestinal form. The bacteria were isolated from blood and stool from the first day of inoculation up to death of the animals of the infected group. The clinical signs that appeared on infected puppies include ; vomiting , diarrhea , dehydration , fever , tenesmus , dysentery, anorexia , abdominal pain , reluctant to move, dullness , congestion of conjunctiva, engorgement of scleral capillaries , tachycardia and tachypnea in the first stage of the disease.

The first clinical signs appeared was vomiting, which appeared 6 hours post inoculation followed by diarrhea after a day. Diarrhea increased in severity with progression of the disease. Fever began to appear during the second day and continued for two days and then fall until the death of animals.

The last stage of the disease (fifth and sixth days post inoculation) characterized by dehydration; sunken eyes and loss of skin elasticity. All animals of infected group were dead on the sixth day post inoculation.

The means of temperature were elevated significantly ($P < 0.0.1$) in the second and third days post inoculation in the second group compared with the first group and then fall until to reach to subnormal temperature at the end of experiment (Table 1).

The statistical differences between infected and control groups of total leukocyte counts , Neutrophils , lymphocytes and monocytes were similar to those of temperature , while there were no statistical differences in the means of eosinophils between infected and control groups throughout the study ($P>0.05$) (Table 1).

The means of PCV, Hb, and RBCs count were elevated significantly ($P<0.01$) in the second day post inoculation in the infected compared with control groups and similar pictures were reported daily throughout the study. While there was no statistical difference in the means of RBCs indices (MCV, MCH, and MCHC) between infected and control group throughout the study ($P>0.05$) (Table).

The statistical differences in the means of total serum protein , fibrinogen and globulins between the infected and control groups were similar to those of RBCs parameters , while ; there was no statistical difference in the means of serum albumin concentration between infected and control groups throughout the study (Table 3).

The statistical differences between infected and control groups in the means of liver enzymes (AST, Alt and ALP) were similar to those of total serum protein (Table 4).

The means of serum sodium , potassium and chloride concentrations were decreased significantly ($P<0.01$) during the second day of inoculation in the infected group compared with control group and similar picture were reported daily throughout the study ; while there was no statistical difference in the means of serum calcium and phosphorus concentration between infected and control groups throughout the study (Table 5).

Discussion

All experimentally infected puppies showed two forms of disease, septicemic form and gastrointestinal form supported with isolation of bacteria from blood and stool throughout the study, this result was in agreement with Nation (1984). The septicemic form of disease can be explained by the inability of the mesenteric lymph nodes to limit the invasion of bacteria together with high virulence activity of bacteria (Futton *et al.*, 1975; Radostitis, 2000).

In gastrointestinal form; the most important clinical signs was diarrhea, which mainly due to the invasion of bacteria to the intestinal epithelium and elaboration of endotoxins which cause the local injury lead to release of prostaglandins and activation of adenyle cyclase receptors that leading to increase cyclic AMP in the enterocytes result in increase of the secretion of water and electrolytes into intestinal lumen (Waller, 1973, Radostitis *et al.*, 2000) accompanied with acute inflammatory reaction lead to diarrhea (McCracken and Lorenz; 2001).

The temperature were elevated at first and then decreased and this result was in agreement with Clarvet (1985). The evaluation of temperature is due to release of endogenous pyrogens from macrophages in response to endotoxin. These endogenous pyrogens stimulate the thermoregulatory center in the hypothalamus that causes fever (Waller, 1978, Radostitis *et al.*, 2000 and Clark *et al.*, 2001). While the decrease in temperature may be due to the circulatory disturbance together with continuous diarrhea which cause loss of fluids (Clark *et al.*, 2001).

The result of total and differential leukocyte count can be divided into two stages. The first stage characterized by increase in the total and differential leukocyte count during the second and third days post inoculation, while the second stages characterized by the decrease of total and differential leukocyte

count compare with first stage. These results were in agreement with Yass (1990) and Lawson *et al.*, (2000). The increase of total leukocyte count was attribute to increase of neutophils and lymphocytes , the increase of neutophils may due to production of specific granular protein called colony stimulating factor from macrophages in response to bacteria products (Kramer *et al.*, 2001 ; Balk , 2002). The increase of monocyte may be due to production of chemotactic factor by bacteria (Dekkers *et al.*, 2000) while the increase of lymphocytes is mainly due to the hyperplasia of the mesenteric lymph nodes (Abraham *et al.*, 2000). The decrease of total leukocyte count on the fourth day post inoculation was attributed to decrease of neutrophils , lymphocytes and monocyte . The decrease in the number of neutrophils is due to overwhelming infection and inhibition effect of endotoxin on the production of WBCs from bone marrow (Kramer *et al.*, 2001; Balk. 2002). The decrease of monocyte may be due to redistribution of these cells during massive bacterial infection (Dekkers *et al.*, 2000) while the decrease of lymphocyte mainly due to the lysis and depletion of lymphocyte from lymph nodes and spleen (Abraham *et al.*, 2000).

The gradual increase of (PCV, Hb and RBCs) in experimentally infected puppies attributed to dehydration resulting from the loss of fluids due to vomiting and diarrhea that lead to haemoconcentration together with decrease of circulatory blood volume (Schalm *et al.*, 1975; Coles, 1986 ; Feldman *et al.*, 2002). There were no changes in (MCV, MCH and MCHC) throughout the experiment this can be explained by that , there is no production of immature red blood cells from the bone marrow and the increase of RBCs parameters mainly due to the plasma loss and haemoconcentration (Dekkers *et al.*, 2000).

The total serum proteins was elevated in experimentally infected puppies due to the plasma loss and this result was in agreement with (Tennat

et al., 1975 ; Smith *et al.*, 1979, Bayram, 1995). The increase of globulin levels and this result was in agreement with Maassen *et al.* (1998).

The evaluation of serum levels of liver enzymes in infected puppies is due to the direct effect of bacterial endotoxin on hepatocytes lead to necrosis and elaboration of liver enzymes into blood (Helms *et al.*, 2002, 2003, 2005).

The gradual decrease in the concentration of sodium , potassium and chloride in the serum of infected animals may be attributed to the loss of these minerals and electrolytes resulting from the loss of water and salts during a short period due to vomiting and diarrhea (Show and Ihle , 1997).

Table (1): The means of temperature and WBCs parameters between infected and control groups.

Parameter Days	Temperature			Total WBC count			Neutrophils		
	1 st group	2 nd group	P value	1 st group	2 nd group	P value	1 st group	2 nd group	P value
1 st day pre	37.8± 0.158	37.8± 0.126	P>0.05	11970± 52.7±494	11760± 255.9	P>0.05	7828± 462.24	7506± 429.6	P>0.05
Day of Ino	37.9± 0.158	37.9± 0.127	P>0.05	12460± 272.5	12620± 230.8	P>0.05	7694± 698.5	7438± 184.3	P>0.05
1 st day post	37.8± 0.175	37.82 ±0.192	P>0.05	13650± 416.8	13210± 263.15	P>0.05	7520± 393.2	7622± 259.2	P>0.05
2 nd day post	37.9± 0.25	39.2± 0.158	P<0.01	14192± 239.5	20630± 774.27	P<0.01	8054± 540.9	16392± 400.8	P<0.01
3ed day post	37.9± 0.25	39.2± 0.185	P<0.01	13390± 373.162	28450± 469.041	P<0.01	7677± 723.27	18580± 570.07	P<0.01
4 th day post	37.9± 0.25	40.22± 0.13	P<0.01	12550± 340.954	13370± 330.088	P>0.05	7358± 150.1	8290± 269.9	P>0.05
5day post	37.94± 0.207	38.6± 0.65	P<0.01	12080± 213.892	8550± 951.38	P<0.01	7406 435.4	4581± 232.3	P<0.01
Sixth day post	37.94± 0.178	36.32± 0.238	P<0.01	13090± 236.952	6380± 450.952	P<0.01	7772± 505.4	3662± 385.19	P<0.01

Continue

Parameter Days	Lymphocytes			Monocytes			Esonophils		
	1 st group	2 nd group	P value	1 st group	2 nd group	P value	1 st group	2 nd group	P value
1 st day pre	2924± 113.9	2832± 35.8	P>0.05	627.4± 310.7	727.4± 66.7	P>0.05	152± 25.64	187± 48.166	P>0.0°
Day of Ino	2874± 736.8	2852± 33.28	P>0.05	635± 46.6	735.4± 50.01	P>0.0°	153± 19.23	183± 45.63	P>0.0°
1 st day post	1448± 55.5	2722± 37.7	P>0.05	643.8± 58.11	757± 51.67	P>0.0°	157± 20.186	167± 41.92	P>0.0°
2 nd day post	2701± 55.94	3278± 29.03	P<0.01	646.2± 30.36	1135± 80.137	P<0.0\	155± 36.33	203± 64.32	P>0.0°
3ed day post	2708± 92.94	4494± 380.88	P<0.0\	620.8± 42.199	1834± 99.81	P<0.0\	146± 36.124	183± 62.9	P>0.0°
4 th day post	2709± 71.8	3848± 297.1	P<0.0\	674.2± 32.67	843.2± 60.67	P<0.0\	136± 43.93	153± 42.36	P>0.0°
5day post	2723± 75.1	3343± 33.87	P<0.0\	649.2± 78.16	658± 55.4	P>0.0°	151± 36.98	187± 87.98	P>0.0°
Sixth day post	2685± 40.3	3060± 34.5	P<0.0\	646.4± 38.559	600.4± 67.68	P>0.0°	148± 53.455	196± 55.49	P>0.0°

Table (2): The means of RBCs paramereters between infected and control groups.

Parameter Days	PCV L/L			Hb gr/L			RBCs × 10 ¹² cell/L		
	1 st group	2 nd group	P value	1 st group	2 nd group	P value	1 st group	2 nd group	P value
-1	40.2± 2.387	38.0± 1.851	P>0.05	142.5± 9.35	144± 5.47	P>0.05	6.67± 0.389	6.774± 0.508	P>0.05
0	38.6± 2.4	39.8± 1.3	P>0.05	136± 8.21	146± 7.41	P>0.05	6.95± 0.555	7.12± 0.443	P>0.05
1	38.9± 2.28	41.6± 2.07	P>0.05	143± 5.7	143± 10.03	P>0.05	6.536± 0.625	7.206± 0.531	P>0.05
2	39.4± 2.08	50.2± 1.3	P<0.01	135± 1.6	163± 10.68	P<0.01	6.412± 0.189	8.182± 0.526	P<0.01
3	38.8± 2.38	53.6± 2.5	P<0.01	136± 10.88	189± 11.38	P<0.01	6.596± 0.909	9.036± 0.554	P<0.01
4	39.6± 1.18	55± 1.1	P<0.01	145± 10.27	192± 11.204	P<0.01	6.636± 0.668	9.966± 0.520	P<0.01
5	38.2± 28.6	57.6± 2.07	P<0.01	140.0± 10.0	204± 11.1	P<0.01	6.623± 0.552	10.488± 0.464	P<0.01
6	40.2± 3.193	61.2± 1.3	P<0.01	134± 10.34	210.8± 10.24	P<0.01	6.806± 0.42	10.57± 0.228	P<0.01

Continue

Parameter Days	MCV FL			MCH picogram			MCHC gr/L		
	1 st group	2 nd group	P value	1 st Group	2 nd group	P value	1 st group	2 nd group	P value
-1	59.89± 5.672	57.95± 3.119	P>0.05	23.39± 0.83	23.32± 0.96	P>0.05	349.4± 9.39	348± 9.73	P>0.05
0	56.21± 5.715	56.02± 2.793	P>0.05	23.14± 0.820	23.46± 0.882	P>0.05	349± 10.04	349.8± 9.37	P>0.05
1	63.1± 6.567	57.99± 4.571	P>0.05	23.64± 0.782	23.54± 0.884	P>0.05	349.8± 9.88	351.4± 9.23	P>0.05
2	61.49± 2.158	61.56± 5.072	P>0.05	23.5± 0.758	23.5± 0.927	P>0.05	348.4± 11.97	346.8± 12.63	P>0.05
3	59.6± 7.391	59.47± 3.829	P>0.05	23.62± 0.673	23.3± 0.731	P>0.05	350± 10.19	350.6± 9.71	P>0.05
4	60.04± 4.838	58.3± 2.694	P>0.05	23.24± 0.808	23.44± 0.870	P>0.05	351± 10.07	349.8± 10.25	P>0.05
5	57.47± 3.825	55.74± 0.709	P>0.05	23.02± 0.887	23.23± 0.826	P>0.05	350.4± 8.84	352± 9.51	P>0.05
6	59.13± 3.067	57.9± 0.699	P>0.05	23.08± 0.63	23.12± 0.944	P>0.05	351± 9.38	349.6± 10.76	P>0.05

Table (4) : The means of liver enzymes activities between infected and control groups.

Parameter Days	AST Iu/L			ALT Iu/L			ALP Iu/L		
	1 st group	2 nd group	P value	1 st group	2 nd group	P value	1 st group	2 nd group	P value
1 st day pre	21.2± 6.3	19.4± 2.19	P>0.05	17.2± 4.833	12.4± 3.334	P>0.05	26.8± 6.22	24.4± 4.722	P>0.05
Day of Ino	18.0± 6.363	17.0± 4.847	P>0.05	19.2± 3.148	15.4± 3.268	P>0.05	26.8± 2.588	27.8± 2.588	P>0.05
1 st day post	18.2± 7.224	19.8± 3.271	P>0.05	17.6± 3.820	16.4± 3.58	P>0.05	22.4± 7.073	27.0± 5.567	P>0.05
2 nd day post	21.2± 2.726	43.2± 2.387	P<0.01	17.8± 3.903	135± 1.22	P<0.05	23.4± 2.073	164.4± 15.836	P<0.05
3ed day post	21.8± 3.077	57.8± 1.788	P<0.05	19.2± 4.438	146.4± 1.959	P<0.05	23.6± 3.874	266.4± 17.1	P<0.05
4 th day post	18.8± 4.984	66.6± 2.988	P<0.05	13.6± 5.711	158.2± 1.833	P<0.05	25.6± 4.827	338.2± 32.345	P<0.05
5day post	21.2± 7.563	76.4± 3.209	P<0.05	20.2± 4.445	165.2± 1.72	P<0.05	24.6± 3.209	392.2± 8.167	P<0.05
6 th day post	17.8± 8.167	90.4± 3.642	P<0.05	18.6± 4.317	188.0± 13.623	P<0.05	25.0± 3.674	445.6± 27.446	P<0.05

Table (5): The means of some minerals and electrolytes between infected and control groups.

Parameter Days	Sodium mmol/L			Potassium mmol/L			Chloride mmol/L		
	1 st group	2 nd group	P value	1 st group	2 nd group	P value	1 st group	2 nd group	P value
-1	150.8± 2.2	150.4± 1.14	P>0.05	4.56± 0.568	4.52± 0.08	P>0.05	111.4± 6.96	111.2± 6.48	P>0.05
0	150.8± 2.378	150.4± 2.297	P>0.05	4.52± 0.618	4.44± 0.55	P>0.05	113.4± 5.319	113.8± 4.969	P>0.05
1	151.6± 4.615	151.2± 3.033	P>0.05	4.3± 0.62	4.33± 0.664	P>0.05	111.0± 6.442	110.8± 5.805	P>0.05
2	149.8± 4.147	118.6± 6.268	P<0.01	4.24± 0.585	2.19± 0.204	P<0.01	1112.2± 8.467	96.0± 3.872	P<0.01
3	152.2± 3.349	94.9± 4.335	P<0.01	4.26± 0.568	1.776± 0.114	P<0.01	112.6± 7.924	81.2± 2.387	P<0.01
4	151.2± 1.923	80.4± 3.03	P<0.01	4.52± 0.580	1.048± 0.056	P<0.01	112.8± 7.155	72.6± 2.701	P<0.01
5	150.2± 2.863	68.8± 3.114	P<0.01	4.52± 0.804	1.26± 0.03	P<0.01	112.8± 14.38	61.8± 5.941	P<0.01
6	150.2± 2.288	56.4± 8.502	P<0.01	4.44± 0.841	0.82± 0.286	P<0.01	112.8± 14.808	56.4± 3.633	P<0.01

Continue

Parameter Days	Calcium mmol/L			Phosphorus mmol/L		
	1 st group	2 nd group	P value	1 st group	2 nd group	P value
-1	2.56± 0.201	2.65± 0.120	P>0.05	1.12± 0.079	1.06± 0.123	P>0.05
0	2.6± 0.174	2.56± 0.238	P>0.05	1.16± 0.123	0.98± 0.14	P>0.05
1	2.58± 0.278	2.54± 0.219	P>0.05	1.12± 0.139	1.16± 0.008	P>0.05
2	2.52± 0.149	2.58± 0.285	P>0.05	1.14± 0.166	1.2± 0.051	P>0.05
3	2.52± 0.105	2.6± 0.138	P>0.05	1.02± 0.109	1.18± 0.176	P>0.05
4	2.28± 0.101	2.68± 0.278	P<0.05	1.22± 0.155	1.28± 0.116	P>0.05
5	2.42± 0.037	2.6± 0.134	P<0.05	1.26± 0.098	1.3± 0.048	P>0.05
6	2.62± 0.156	2.4± 0.046	P<0.05	1.2± 0.096	1.24± 0.095	P>0.05

References

- 1- Abraham, E ; Carmody, A ; Shenkar , R and Arcaroli , J (2000). Neutrophils as early immunologic effectors in haemorrhage or endotoxemia induced acute injury. Am.J. Physiol. Lung cell Mol. Physiol ; 279 (6) : 1133-1145.
- 2- Balk, R.A (2002). Endotoxemia in critically ill patients . Why a reliable test could be beneficial . Crit. Care , 6 : 289-290.
- 3- Baron, E.J ; Peterson , L.R and Fingold , S.M (1994). Baily and Scotts diagnostic Microbiology 9th edition . Mosby . St Louis.
- 4- Bayram, M.S (1995). Isolation of Salmonella. Study the pathogenicity of *Salmonella give* in dogs in Baghdad province . Msc . Thesis , College of Veterinary of Baghdad.
- 5- Clark, C; Cunningham , J ; Ahmed , R; Woodwar , D; Issacs , S; Ellis, A ; Anand , C ; Ziebella , K ; Sockett , A and Rodres, F (2001). Characterization of Salmonella associated with ear of dogs in Canada . J. Clinic . Micorbiol .; 39 (11) : 3962-3968.
- 6-Clarvet C.A (1985). Salmonella infection in hospitalized dogs Epizootiology , diagnosis and Prognosis . J.Am. An. Hosp. Assoc , 21: 499-503.
- 7- Coles, E.H (1986) Veterinary Clinical Pathology . 4th edition W.B Saunders Co. USA. 486.
- 8- Dekkers, P.E.P ; Hove , T ; Velde , A.A ; Daventer, S.J.H and Poll , T (2000). Up regulation of Monocyte urokinase Plasminogen activator receptor during human endotoxemia. Infection and Immunity , 68 (4) : 2156 – 2160.

- 9- Feldman, B.F ; Zinkl , J.G and Jain , N.C (2002). Schalm's Veterinary Haematology. 5th edition. Lippicott Williams and Wilkins. London. 1859 P.
- 10- Futton , M ; Bladel, B and Lesko , M (1975). Salmonella in dogs and cats of Medical School. Colony control Vet , 45: 265-267.
- 11- Helms , M; Ethelberg , S and Molbak , K (2005). International *Salmonella typhimurium* DT 104 infections 1992-2000 . Emerg . Infect . Dis ; 625-632.
- 12- Helms , M; Vastrup, P; Gerner- Smidt , P and Molbak. K (2002). Excess mortality associated with antimicrobial drug resistance *Salmonella typhimurium*. Emerg. Infec. Diseases ; 8 ; 409-495.
- 13- Helms , M; Vastrup, P and Molbak , K (2003). Short term mortality associated with food borne bacterial gastrointestinal infection registry based study. B.M.J :320-357.
- 14- Kallow , O.J and Hosso , S.A (2001). Prevalence of Salmonella Serotypes in dogs and their sensitivity to antimicrobial agents. J. Vet. Sci ; 14(1).
- 15- Kramer, B.W; Moss, T.J; willet , K.E; Newnham, J.P; Sly , P.D ; Kallapure , S.G ; Ikegami , M and Job , A.H (2001). Dose and time response after intra amniotic endotoxin in preterm lambs. Am. J. Respir . Crit. Car Med , 164 (6) : 289-988.
- 16- Lawson , J.A ; Burns , A .R ; Farhood , A ; Lynn- Bajt , M ; Collins , R.G ; Smith , C.W ; Jaeschke, H. (2000). Pathophysiologic importance of E and L selection for Neutrophil induced liver injury during endotoxemia in mice. Hepatology , 32: 990-998.
- 17- Maassen, C.B; Van Holt, J.C ; Heijne denbak- Glashouwer, M.J ; Leer , R; Laman , J.D ; Boersmo, W.J and Glaassen , E (1998). Orally administered Lactobacillus strains differentially affect the direction and efficacy of the immune response. Veterinary

Quarterly. Belhaven. The Netherlands, 20 (Supplement 3) : 581-583.

- 18- McCracken, V.J and Lorenz , R.G (2001). The gastrointestinal ecosystem : a precarious alliance among epithelium immunity and microbiota. *Cell. Microbiol* , 3: 1-11.
- 19- Murdoch, D.B (1979). Alimentary tract and associated. In : Chandler , E.A ; Evans , J.M ; Singlton , W.B ; Tartup ; F.G ; Sutton , J.B and Taverner , W.D (editors). *Canine Medicine and therapeutics* .Blackwell . Sc. Puble. UK P: 293-303.
- 20- Nation , P.N (1984). *Salmonella dubline* septicemia in two puppies. *Cand . Vet. J*, 25:324-326.
- 21- Radostitis, O.M ; Blood , D.C, Gay , C.C and Hinchlff, K.W (2000). *Veterinary Medicine* 9th edition . W.B sounders. Co.
- 22- Schalm, O ; Jain , S and Camol , E. (1975). *Veterinary Haematology*. Philadelphia. Lea and Fibger.
- 23- Show , D.H and Ihle , S.L (1997). *Small animal internal Medicine* . William and Wilkins Co. Baltimore.
- 24- Smith , B ; Habasha , F ; Reina . Guera , M and Hardy , A (1979). Bovine Salmonellosis. Experimental production and characterization of disease in calves using oral challenge with *Salmonella typhimurium* . *Am. J. Vet . Res* , 40 :1510 – 1515.
- 25- Tennat , B ; Harrold, H and Reina – Guero , M. (1975). Haematology of neonatal calves. H. Response associated with acute enteric infection. Gram negative septicemia and experimental endotoxemia. *Cornell. Vet* ; 65: 457-475.
- 26- Waller, S(1973). Prostaglandins and gastrointestinal tract. *Gastroenterology*; 14:402 -417.

27- Williams , L. P (1980). Salmonellosis. In steel , J.H. (editor). Handbook series in zoonotic section A. Vol. 11 Florida . CRC Press.

28- Yass, A.A (1990). Experimental study on pathogenesis of *Salmonella typhimurium* infection in calves. PhD thesis. College of veterinary medicine , University of Baghdad.

دراسة سريرية، دمبة وكيموحيوية لخمج السالمونيلا التجريبي في الجراء

*رحمن كاظم محسن ،سعدي احمد غناوي السامرائي و عبد المناف حمزة الجودي
*فرع الطب الباطني والوقائي البيطري – كلية الطب البيطري – جامعة البصرة
*فرع الطب الباطني والوقائي البيطري – كلية الطب البيطري – جامعة بغداد

الخلاصة

اجريت هذه الدراسة لمعرفة التغيرات السريرية ، الدمية والكيموحيوية للكلاب المخمجة تجريبيا بجراثيم سالمونيلا تايفيموريم. استخدمت في هذه الدراسة عشرة جراء وقد قسمت الى مجموعتين حيث جرعت حيوانات المجموعة الاولى 10 مل من مرق trypticase soya الخالي من الجراثيم واعتبرت كمجموعة سيطرة في حين جرعت المجموعة الثانية 10 مل من المرق اعلاه بحيث يحتوي على 4.8×10^8 جرثومة سالمونيلا 1 مل. تم مراقبة وفحص جميع الحيوانات يوميا لحين هلاك حيوانات مجموعة الاصابة لدراسة التغيرات السريرية ، الدمية والكيموحيوية وظهرت النتائج ان الحيوانات المخمجة تجريبيا بجراثيم السالمونيلا اظهرت اعراض كلا الشكلين الانتاني والمعي المعوي مع عزل جراثيم السالمونيلا من دم وبراز الحيوانات المخمجة طول مدة التجربة . كما اظهرت النتائج ايضا وجود تغيرات دموية وكيموحيوية شديدة في الحيوانات المخمجة تجريبيا بجراثيم السالمونيلا .