Zagazig Veterinary Journal
Volume 43, Number 3, p. 126-134, 2015

DOI: 10.21608/zvjz.2015.28449

Clinicopathological Studies on the Effect of Filariasis in Egyptian Buffaloes

Nasr A. M. Nasr Eldeen
Clinical Pathology Department, Faculty of Veterinary Medicine, Zagazig University, 44511, Egypt

Article History: Received: 14/6/2015 Received in revised form: 5/7/2015 Accepted: 14/7/2015

Abstract

Filariasis is a vector-transmitted parasitic disease and has a worldwide spread. This work aimed to study the effect of filariasis on hematology, blood chemistry and antioxidant status of adult buffaloes as well as efficacy of ivermectin in disease treatment. The present study was done on fifteen male buffaloes, 2-4 years old, (7 apparently health and 8 suffered from filariasis). The diagnosis was based on the clinical signs and presence of Microfilariae in wet smear. One animal died during treatment. Hematological results revealed hemolytic anemia associated with a significant decrease in erythrocytic count, hemoglobin concentration and packed cell volume. A significant increase in the total and differential leukocytic counts was recorded compared with the normal control. Evaluation of blood chemistry of infested animals showed significant changes in liver and cardiac function, as well as antioxidant parameters. These parameters returned to the normal levels 21days post treatment with ivermectin. It could be concluded that filariasis induced hemolytic anemia, with alteration in antioxidant enzymes and liver and cardiac functions. Also ivermectin is effective in treatment of filariasis.

Keywords: Filariasis, Buffalo, Antioxidant enzyme, Liver, Blood

Introduction

Filariasis is a vector-transmitted parasitic disease caused by Filarioidea nematode with world wide spread especially throughout the developing countries [1, 2]. The parasites develop inside the vector from microfilariae to infective larvae then migrate to the mouth parts of arthropod and transmitted to the mammalian host during feeding [3]. Filariasis is associated with considerable economic losses to beef and dairy buffaloes as they caused high morbidity and mortality [4]. Filarial parasites succeed to escape from host immunity through the releasing of immunosuppressive materials [5]. Lymphatic filariasis is endemic disease in tropical and subtropical areas [6]. For many years ago, microscopical examination of blood film has been the only tool available for the detection of parasites [7]. Presence of microfilaria in blood smear is a good diagnostic method [8], in addition to the presence of dermatological or lymphatic manifestations. Nowadays ivermectin administration is the major tool in the control of filariasis [4]. Although many investigators studied filariasis in different animals there is a shortage in studying the disease in buffaloes. Also there are many aspects that remain unclear, so this work was aimed to study the effect of filariasis on hematology, blood chemistry and antioxidant status of adult infested buffaloes.

Material and Methods

Animals

Fifteen adult male buffaloes, 2-4 years old, were used in the present study. The animals were sporadic cases admitted to a private clinic and the Clinic of Teaching Animal Hospital, Faculty of Veterinary Medicine, Zagazig University, Shakia, Egypt. Control animals were selected from the same farms of the diseased animals and nearly within the same age range. One animal died during the treatment. The other fourteen animals were divided into two equal groups, apparently healthy and naturally infested male buffaloes (n=7/each) The diagnosis was based on clinical examination of animals and identification of microfilariae by late night direct smear [8].

***Corresponding author e-mail: (nanasreldin@zu.edu.eg), Clinical Pathology Department, Faculty of Veterinary Medicine, Zagazig University, 44511, Egypt**

Drugs

Ivermectin (Merial celebrates, Lyon Porte-des-Alpes, France): bottle of 50 ml injected s/c at a dose of 1ml/50kg BW -3 times with 5 days interval [4]. It is used for treatment of filariasis.

Norocillin LA (Norbrook Lab lemitis, station works NEWRY, N. Ireland): bottle of 50 ml used by deep I/M injection at a dose of 1ml/25kg BW twice with 4 days interval [9]. It is used to prevent secondary bacterial infection to the opened nodules.

Blood samples

Blood samples were taken at 0,7,14 and 21 days post treatment from the jugular vein of each animal. The first set of blood was 1 ml received in a clean tube containing dipotassium salt of EDTA and used for hematological examination. The second blood sample (10ml) was taken without anticoagulant in chemical free test tubes, allowed to clot then centrifuged at 3000 rpm for ten minutes for separation of clear serum.

Hematological studies

Erythrocytic count and leukogram were performed [10]. The packed cell volume (PCV) was estimated by the microhematocrit centrifuge [10] and hemoglobin (Hb) concentration was determined using the cyanmethemoglobin colorimetric method [11]. Mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH) and mean corpuscular hemoglobin concentration (MCHC) were calculated.

Serum biochemical studies

Serum total proteins (TP), albumin and globulins were estimated according to Grant et al.,Webster and Doumas& Biggs [12, 13, 14].Aaminotransferases (AST & ALTbilirubin malondialdehyde and cardiac troponinI (cTnI) concentration) were determined following to [15,16, 17and 18]. Serum activity of glutathione peroxidase and superoxide dismutase (SOD) was measured after [19, 20]. Lactate dehyrogenase (LDH) and creatine phosphokinase (CK-MB) were also estimated [21, 22].

Statistical analysis

Data obtained from this investigation were statistically analyzed using the one way analysis of variance ; ANOVA [23]. Means at the same row followed by different letters were significantly different and the highest value was represented with the letter (a).

Results

Clinical signs

The main clinical manifestations recorded in affected animals were fever (40-40.6ºC), anorexia, enlargement of one leg or more (Figure 1A) and presence of cutaneous nodules (Figure 1B). These nodules opened spontaneously and produced hemorrhagic exudates (Figure 1C). One animal died during treatment.

Hematological changes

Evaluation of erythrogram revealed that filarial - infested group had a significant decrease in erythrocytic count, packed cell volume and hemoglobin concentration at Zero and 7 days post treatment (PT) compared with the healthy control. The estimation of MCV and MCHC proved normocytic normochromic anemia before treatment and macrocytic hypochromic 7 days post treatment. The MCH revealed a significant increase before treatment only in the infested group compared with the non infested control. At 14 and 21 days PT, the erythrogram returned to the normal values (Table1).

Regarding the leukogram the results revealed a significant leukocytosis at 0, 7 and 14 days post treatment compared with the normal control. Also a significant eosinophilia, basophilia and lymphocytosis were recorded at 0 and 7 days post treatment. Moreover a significant neutrophilia (day Zero PT) and monocytosis (7 days PT) were recorded (Table 1).

Table 1: Erythrogram and Leukogram of filaria-infested buffaloes compared to normal control (mean± SE)

Parameters	Groups				
	Normal group (control)	Diseased group (days post treatment) 0	7	14	21
RBCs (x 10^6/µl)	8.14 ^{a}±0.15	6.76 ^{c}±0.09	7.22 ^{b}±0.06	7.81 ^{a}±0.15	8.15 ^{a}±0.13
PCV(%)	40.33 ^{a}±0.69	34.57^{c}±0.53	38.00 ^{b}±0.31	39.43 ^{ab}±0.78	40.00 ^{a}±0.62
Hb (gm/dl)	13.26 ^{a}±0.15	11.43^{c}±0.19	11.89 ^{b}±0.06	12.91 ^{a}±0.16	13.10 ^{a}±0.19
MCV (Fl)	49.83 ^{bc}±0.41	51.60^{b}±0.55	52.61 ^{a}±0.42	50.49 ^{bc}±0.61	49.29 ^{c}±0.40
MCHC (%)	32.63^{a}±0.20	33.07^{a}±0.41	31.29 ^{b}±0.30	32.78 ^{a}±0.38	32.79 ^{a}±0.37
MCH (pg)	16.29 ^{b}±0.15	16.91^{a}±0.19	16.45 ^{ab} ±0.11	16.55 ^{ab}±0.20	16.08 ^{b}±0.11
TLC (x 10^6/µl)	8.88 ^{d}±0.27	12.59 ±0.29	10.95 ^{b}±0.49	9.96^{c}±0.22	9.36 ^{cd}±0.16
Neutrophils (x 10^6/µl)	2.15 ^{b}±0.15	2.58 ^{a}±0.19	2.31 ^{ab}±0.13	2.08 ^{b}±0.18	2.14 ^{b}±0.17
Eosinophils (x 10^6/µl)	0.64^{c}±0.12	2.31^{a}±0.16	1.09 ^{b}±0.15	0.83 ^{bc}±0.11	0.71 ^{bc}±0.13
Basophiles (x 10^6/µl)	0.23 ^{b}±0.09	0.73 ^{a}±0.08	0.52 ^{ab}±0.09	0.35 ^{b}±0.08	0.38 ^{b}±0.12
Lymphocytes (x 10^6/µl)	4.25 ^{c}±0.22	5.38 ^{a}±0.10	4.87 ^{ab}±0.18	4.76 ^{bc}±0.25	4.25^{c}±0.15
Monocytes (x 10^6/µl)	1.61 ^{b}±0.09	1.59 ^{b}±0.05	2.02 ^{a}±0.11	1.94 ^{ab}±0.23	1.87 ^{ab}±0.11

Means with different superscript letters within the same row are significantly different at p≤0.05.

Biochemical results

Serum proteins profile investigation nrevealed a significant hypoproteinemia and hypoalbuminemia in infested group at 0, 7 and 14 days post treatment. The serum globulins level was significantly increased at 14 and 21 days post treatment .Albumin /globulin ratio showed a significant decrease in infested group at 0, 7, 14 and 21 days post treatment compared with the normal control (Table 2).

Table 2: Serum protein profile, aminotransferases activity and bilirubin level of filaria-infested buffaloes compared to normal control (mean ± SE)

Parameters	Groups				
	Normal group (control)	Diseased group (days post treatment) 0	7	14	21
Total protein(g/dl)	7.04 ^{a}±0.09	6.10 ^{c}±0.11	6.38 ^{c}±0.14	6.69^{b}±0.06	7.31^{a}±0.08
Albumin (g/dl)	3.83 ^{a}±0.12	3.00 ^{c}±0.08	3.16 ^{bc}±0.09	3.31^{b}±0.06	3.66 ^{a}±0.05
Globulin (g/dl)	3.20 ^{c}±0.06	3.10 ^{c}±0.05	3.22 ^{bc}±0.07	3.38^{b}±0.06	3.65 ^{a}±0.04
A/G ratio	1.20 ^{a}±0.05	0.96 ^{b}±0.02	0.98 ^{b}±0.03	0.98 ^{b}±0.02	1.00 ^{b}±0.01
AST (U/L)	41.81^{d}±0.63	85.79^{a}±1.23	74.33 ^{b}±1.59	56.71^{c}±1.51	44.27^{d}±1.40
ALT (U/L)	17.25 ^{d}±0.80	57.24 ^{a}±1.04	47.14 ^{b}±1.96	29.25^{c}±1.31	18.77 ^{d}±1.66
Total bilirubin (mg/dl)	0.35 ^{d}±0.05	1.09 ^{a}±0.06	0.85 ^{b}±0.08	0.60^{c}±0.10	0.41 ^{cd}±0.04
Direct bilirubin (mg/dl)	0.13^{c} ±0.01	0.38 ^{a}±0.03	0.29 ^{b}±0.02	0.22^{b}±0.03	0.12^{c}±0.01
Indirect bilirubin (mg/dl)	0.22 ^{d}±0.04	0.71^{a}±0.05	0.56 ^{b}±0.05	0.38 ^{c}±0.07	0.28 ^{cd}±0.03

Means with different superscript letters within the same row are significantly different at p≤0.05.

The disturbances in the liver functions in filaria infested group manifested by a significant increase in the serum activities of aspartate aminotransferase (AST), alanine aminotransferase (ALT) and serum levels of total bilirubin, unconjugated bilirubin (UB) and conjugated bilirubin(CB) at 0, 7 and 14 days post treatment. This elevation was returned to the normal level 21 days post treatment compared to the normal control values (Table 2).

Significant decrease in the activities of glutathione peroxidase (GPX), superoxide dismutase (SOD) and catalase (CAT) with a significant increase in malondialdehyde (MDA) level in the affected animals at 0, 7 and 14 days post treatment compared to the normal control (Table 3). The obtained data revealed that these parameters became non significant with the normal control at 21 days post treatment.

Table 3: Antioxidant enzymes and malondialdehyde of filaria-infested buffaloes compared to normal control (mean± SE)

Parameters	Normal group (control)	Groups Diseased group (days post treatment)			
		0	7	14	21
Glutathione peroxidase (U/L)	26.14 ^{a}±1.01	17.26^{d}±0.60	21.03 ^{c}±0.68	23.24 ^{b}±0.43	25.43 ^{a}±0.78
Superoxide dismutase (U/L)	11.07 ^{a}±0.29	7.59 ^{d}±0.16	8.58 ^{c}±0.15	9.61^{b}±0.30	10.83 ^{a}±0.25
Catalase (U/L)	2.53 ^{a}±0.08	1.57 ^{d}±0.14	1.88 ^{c}±0.05	2.21 ^{b}±0.06	2.49 ^{a}±0.08
Malondialdehyde (nmol/ml)	0.83 ^{d}±0.05	1.40 ^{a}±0.06	1.24 ^{b}±0.03	1.04 ^{c}±0.06	0.88 ^{d}±0.05

Means with different superscript letters within the same row are significantly different at p≤0.05.

Alterations in the cardiac markers in filarial-infested animals were manifested by a significant elevation in the serum activities of LDH, CK-MB and serum level of cTnI at 0, 7 and 14 days post treatment (Table 4). This elevation was returned to the normal level 21 days post treatment compared to the normal control values.

Table 4: Cardiac markers in serum of filaria-infested buffaloes compared to normal control (mean± SE)

Parameters	Normal group (control)	Groups Diseased group (days post treatment)			
		0	7	14	21
cTnI (ng/ml)	0.10 ^{d}±0.01	0.44 ^{a}±0.03	0.35 ^{b}±0.03	0.25 ^{c}±0.02	0.13 ^{d}±0.02
LDH (U/L)	518.43 ^{d}±3.15	612.00 ^{a}±7.15	580.91 ^{b}±5.74	555.89 ^{c}±3.69	525.69 ^{d}±5.23
CK–MB (U/L)	281.66 ^{d}±2.95	348.07 ^{a}±6.32	323.13 ^{b}±2.07	308.90 ^{c}±2.99	289.86 ^{d}±5.39

Means with different superscript letters within the same row are significantly different at p≤0.05.

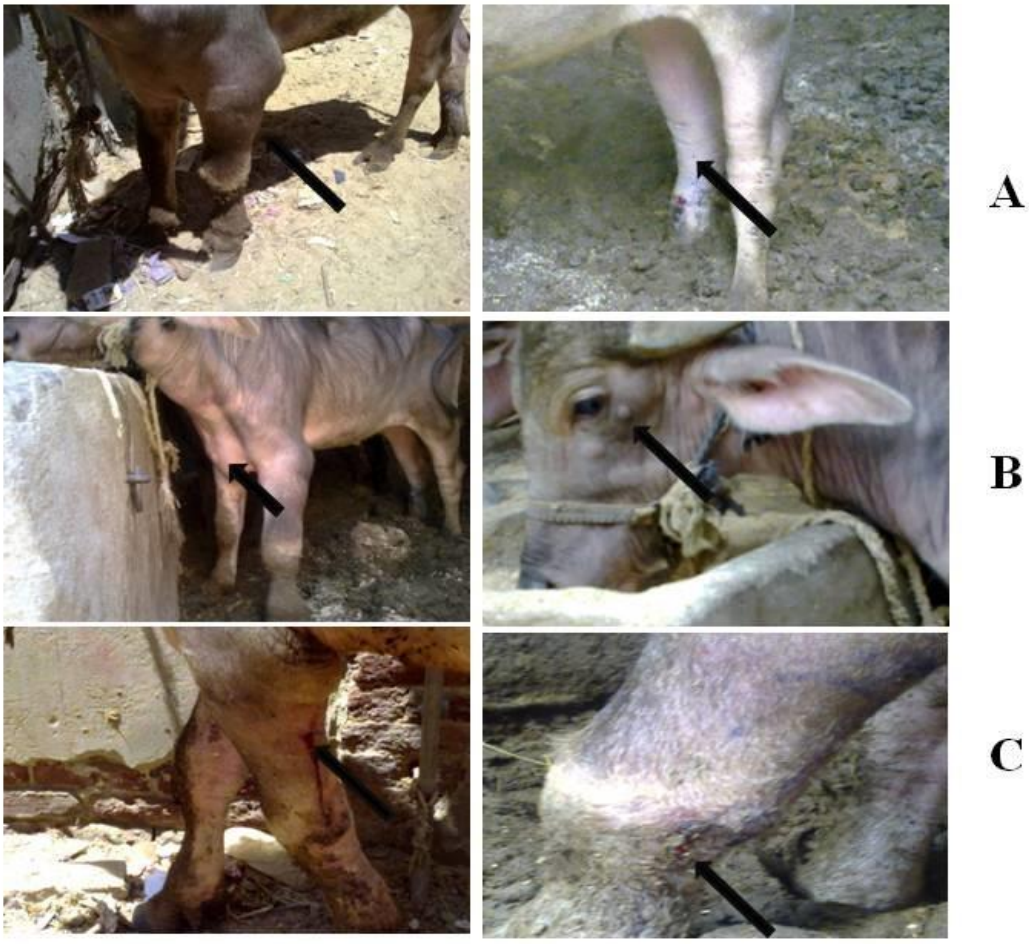

Figure 1: A. An enlargement of left forelimb and right hind limb in the infested male buffaloes aged 2-4 years. B. Cutaneous nodules in filaria - infested buffaloes. C. Hemorrhagic exudates in filaria - infested buffaloes.

Discussion

The clinical signs observed in the diseased animals were comparable to those previously mentioned by several investigators as suggestive to bovine flarasis [4,24]. The picture of the erythron mass in the infested group was normocytic normochromic anemia before treatment and macrocytic hypochromic 7 days post treatment. The aforementioned results could be correlated with hemolysis. The significant increase of the mean corpuscular hemoglobin that recorded before treatment supported the presence of hemolysis. As supported by the previously obtained results of Sharma& Joshi and Sarojini, & Senthilkumaar [24,25] who mentioned that microfilariae caused erythrocyte degeneration .

The leukocytosis recorded at 0 and7 days post treatment is due to neutrophilia, eosinophilia, basophilia, lymphocytosis and monocytosis. The lymphocytosis recorded in infested animals may be due to an early antigenic stimulation with an increased demand for transformation of lymphocytes to plasma cells. Such increase in phagocytic cells (neutrophils & monocytes) could be due to tissue destruction [10]. The observed eosinophilia was due to the hypersensitivity resulted from the migration and the foreign protein of the parasite [10]. Nearly similar

results were recorded previously by Hashem, and Badawy [26].

The significant hypoproteinemia and hypoalbuminemia in infested group at 0, 7 and 14 days PT could be attributed to hypoalbuminemiawhich in turn might be due to the decreased feed intake and disturbed metabolism of the liver. The hyperglobulinemia may be due to the reaction to the antigenic stimuli. Albumin /globulin ratio showed a significant decrease in infested group at 0, 7, 14 and 21 days PT. This is due to the hypoalbuminemia and hyperglobulinemia The obtained results were in harmony with earlier findings of Brzezińska-Slebodzińska [31].

The present study showed a highly significant increase in the serum AST and ALT activities in the infested group. The increased serum activities of aminotrasnferases (AST and ALT) were associated with hepatocellular damage [27]. This increase in serum aminotransferases activity in infested animals suggested liver and/or myocardial cells degeneration caused by migratory microfilaria [24] or secondary to circulatory disturbance [26]. The hyperbilirubinemia was due to the increased serum levels of conjugated and unconjugated bilirubin. The increased serum conjugated bilirubin level suggested liver dysfunction. While, the increased serum unconjugated bilirubin level may be attributed to the presence of hemolytic jaundice resulted from excessive hemolysis [27].

The antioxidants defense systems play an important role in protection of living organisms from the deleterious effects of reactive oxygen metabolites.Superoxide dismutase; glutathione peroxidase and catalase play an important role. The field of oxidative stress in buffalo's medicine is still in the early juncture. The evaluations of free radicals damage and body's defenses have become increasingly important in clinical medicine as complementary tool in the evaluation of metabolic status of the animals [28]. Host immune effector cells such as macrophages, neutrophils and eosinophils release free radicals such as superoxide radical, hydroxyl radicals, singlet oxygen and hydrogen peroxide as defense mechanisms to kill the invading parasites [29]. All living tissues protect themselves from the damaging effects of free radicals by antioxidants and antioxidant enzymes.

The present study showed that serum superoxide dismutase, glutathione peroxidase and catalase enzyme activities were decreased significantly in the infested group. Parasitic infestation causes the release of reactive oxygen species [30]. In this study oxidative stress following the infestation and inflammation caused increased use and weakened protective effect of the antioxidant enzymatic system in the infested animals. Moreover, fever induced oxidative stress [31]. No available data dealing the effect of filariasis on the antioxidants enzymes in buffaloe could be found. Lipid peroxidation is used as an indicator of oxidative stress. Serum malondialdehyde level significantly increased in infested animals compared with the normal health control. This supports the previously obtained result by Doumas and Biggs [4]who recorded a significant increase in serum malondialdehyde level post filarial infestation in buffaloes.

Several biochemical parameters can be used as markers for cardiac function. Among them the myocardial bound creatine kinase (CK-MB) and lactate dehydrogenase (LDH) are important in the diagnosis of heart disease [32]. Troponin I (cTnI) is gold -standard, blood biomarkers with high sensitivity and specificity for myocardial degeneration [33]. Troponin I, lactate dehydrogenase and creatine kinase were increased significantly in the infested buffaloes. So this indicated the presence of myocardial injury. This may be due to necrotic degeneration of myocardial cells caused by migratory microfilaria [24]. The most characteristic finding recorded in the dead animal was severe increase in serum level of troponin I (1.50 ng/ml).

Conclusion

It could be concluded that filariasis induced hemolytic anemia, with alteration in antioxidant enzymes and liver and cardiac functions. The ivermectin treatment succeeded to ameliorate the negative impact of parasites on hemato-

biochemical parameters towards the normal control values. These findings support the efficacy of ivermectin for treatment of filariasis in buffaloes. However increased serum troponinI level to 1. 50 ng/ml or more indicates bad prognosis.

Conflict of interest

The authors declare no conflict of interest.

References

[1] Wilke, A.B. and Marrelli, M.T. (2015): Paratransgenesis: a promising new strategy for mosquito vector control. Parasit Vectors, 8(1):342.

[2] Li, M.C.; Mei, H.G.; Tang, J.P.; Xu, A.Q.; Hu, Y.H. and Cao, H.J. (2015): Effect of comprehensive care for chronic filariasis patients with lymphedema. Zhongguo Xue Xi Chong Bing Fang Zhi Za Zhi., 27(1):73-75.

[3] Goodridge, H.S.; Stepek, G.;, Harnett, W. and Harnett, M.M. (2005): Signalling mechanisms underlying subversion of the immune response by the filarial nematode secreted product ES-62. Immunol, 115(3): 296–304.

[4] Radwan, M.E. and Talkhan, O.F. (2009): Some Studies on Filariasis and associated biochemical alteration in Egyptian buffaloes. Report and Opinion, 1 (1):35-44.

[5] Muthian, G.; Pradeep, C.G.; Sargapradeep, K.; Kaleysaraj, R. and Bright, J.J. (2006): Setaria digitata secreted filarial lipids modulate IL-12 signaling through JAK-STAT pathway leading to the development of Th1 response. Exp Parasitol.114 (3):193-203.

[6] Ben-Chetrit, E. and Schwartz, E. (2015): Vector-borne diseases in Haiti: a review. Travel Med Infect Dis., 13 (2):150-158.

[7] Melrose, W.D.; Durrheim, D.D. and Burgess, G.W. (2004): Update on immunological tests for lymphatic filariasis. Trends Parasitol. 20 (6):255-257.

[8] Kaufmann, J. (1996): Parasitic Infections of Domestic Animals :A Diagnostic Manual. Basel; Boston; Berlin: Springer Basel AG.

[9] DeDonder, K.D.; Gehring, R.; Baynes, R.E.; Tell, L.A.; Vickroy, T.W.; Apley, M.D. and Riviere, J.E. (2013): Effects of new sampling protocols on procaine penicillin G withdrawal intervals for cattle. J Am Vet Med Assoc. 243(10):1408-1412.

[10] Feldman, B.F.; Zinki, J.G. and Jain, V.C. (2000): Schalm's Veterinary Hematology .5th ed. Lippincott Williams and Wilkins. Canda; PP: 1145-1146.

[11] zijlstra WG, van Kampen E. (1960): Standardization of hemoglobinometry. I. The extinction coefficient of hemiglobincyanide. Clin. Chem. Acta, 5: 719-726.

[12] Grant, G.H.; Sliverman, L.M. and Christenson, R.H. (1987): Amino Acids and proteins; Fundamentals of Clinical Chemistry, Tietz N. W. Editor, 3rd Edition, WB Saunders company Philadelphia USA, 328-329

[13] Webster, D. (1977): Determination of serum albumin. Clin. Chem., 23 (4): 663-665.

[14] Doumas, B.T. and Biggs, H.G. (1971): Determination of serum albumin. In: Standard methods of clinical chemistry. G.R. Cooper (ed). Academy press, New York vol. 7 p. 175

[15] Reitman, S. and Frankel, S. (1957): A colorimetric method for determination of serum glutami coxaloacetic transaminase and serum glutamic pyruvic transaminase. Am J Clin Pathol., 28:56-58.

[16] Water, M. and Gerade, H. (1970): A colorimetric method for determination bilirubin in serum and plasma. Micro Chem J. 15:231–236

[17] Mihara, M. and Uchiyama, M. (1978): Determination of malondialdehyde

precursor in tissues by thiobarbituric acid test. Anal. Biochem. 86 (1):271-278.

[18] Collinson, P.O.; Boa, F.G. and Gase, D.C. (2001): Measurement of cardiac troponins. Ann Clin Biochem. 38(5):423-449.

[19] Jacobson, B.; Quigley, G. and Lockitch, G. (1988): Adaptation of glutathione peroxidase assay to the Technicon RA-1000. Clin Chem. 34:2164-2165.

[20] Minami, M. and Yoshikawa, H.A. (1979): Simplified assay method of superoxide dismutase activity forclinical use. Clin Chim Acta, 92 (3): 337-342.

[21] The committee on Enzymes of the Scandinavian Society for Clinical Chemistry and Clinical Physiology (1974): Recommended Methods for the determination of four enzymes in blood. .Scand J. Clin. Lab. Invest. 33(4):291-306.

[22] Hørder, M.; Elser, R.C.; Gerhardt, W.; Mathieu, M. and Sampson, E.J.(1991): International Federation of Clinical Chemistry (IFCC). Approved recommendation on IFCC methods for the measurement of catalytic concentration of enzymes. Part 7. IFCC methods for creatine kinase. Eur J Clin Chem Clin Biochem; 29 : 435-456.

[23] Tamhane, A. and Dunlop D. (1999): Statistic and data analysis from elementary to intermediate. New Jersey.USA: Prentice Hall, Upper Saddle River.

[24] Sharma, M.C. and Joshi, C. (2002): Serum mineral and haematobiochemical profile of microfilariae infected cattle in india: its effects on production and therapy. Asian-Australas J Anim Sci, 15(3): 357-365.

[25] Sarojini, S. and Senthilkumaar, P. (2013): Haematological studies of lymphatic filariae, Wuchereria bancrofti affected patients in Arakkonam area, Tamil Nadu, India. Euro. J. Exp. Bio., 3(2):194-200.

[26] Hashem, M. and Badawy, A. (2007): Hematological and biochemical studies on filariasis of dogs.The Internet Journal of Veterinary Medicine, 4 (2):1-7

[27] Kaneko,J. J.; Harvey, J. W. and Bruss, M. L. (1997): Clinical Biochemistry of Domestic Animal. 5th ed., Academic Press Sandiego, California, USA, pp: 857-883.

[28] Castillo, C.; Hernandez, J.; Bravo, A.; Lopez-Alonso, M.; Pereira, V. and Benedito, J.L. (2005): Oxidative status during late pregnancy and early lactation in dairy cows. Vet. J. 169 (2): 286-292.

[29]Bannister, J.V. and Bannister, W.H. (1985): Production of oxygen- centered radicals by neutrophils and macrophages as studied by electron spin resonance (ESR).Environ. Health Perspect. 64:37-43.

[30] Bal, B.K.; Kulkarni, S.; Bhandari, Y.; Ganesh, B.B.; Goswami, k. and Reddy, M.V.(2006):Lymphatic Filariasis : Possible pathophysiological Nexus with oxidative stress. Trans R Soc Trop Med Hyg. 100 (7) : 650-655.

[31] Brzezińska-Slebodzińska, E. (2001): Fever induced oxidative stress: the effect on thyroid status and the 5'-monodeiodinase activity, protective role of selenium and vitamin E. J Physiol Pharmacol. 52(2):275-284.

[32] Reef, V.B. and McGuirk, S.M. (2008): Diseases of the cardiovascular system. In: Smith BP, editor. Large Animal Internal Medicine. 4th ed. St. Louis, Missouri: Elsevier-Saunders; pp. 453–489.

[33]O'Brien, P.J.; Smith, D.E.C.;, Knechtel, T.J.; Marchak, M.A.; Pruimboom-Brees, I.; Brees, D.J.; Spratt, D.P.; Archer, F.J.; Butler, P.; Potter, A.N.; Provost, J.P.; Richard, J.; Snyder, P.A. and Reagan, W.J. (2006): Cardiac troponin I is a sensitive, specific biomarker of cardiac injury in laboratory animals. Lab Anim., 40 (2): 153-171.

الملخص العربى

دراسات باثولوجية إكلينيكية على تأثير مرض الفلاريا فى الجاموس المصرى

نصر عبد الوهاب محمد نصر الدين
قسم الباثولوجيا الإكلينيكية – كلية الطب البيطرى – جامعة الزقازيق

مرض الفلاريا هو مرض طفيلي ينتقل بالنواقل وله انتشار في جميع أنحاء العالم. ويهدف هذا العمل إلى دراسة تأثير الفلاريا على خلايا ، كيمياء الدم والأنزيمات المضادة للأكسدة فى الجاموس. تم إجراء الدراسة على خمسة عشرمن ذكور الجاموس ، عمر ٢-٤ سنوات ، (٧ اصحاء و ٨ تعانى من مرض الفلاريا). وقد توفي حيوان واحد خلال فترة العلاج. واعتمد التشخيص على الإعراض المرضية ووجود الميكروفلاريا في الدم. وكشفت النتائج عن جود فقر دم انحلالي مرتبط بانخفاض عدد الكريات الحمراء وتركيز الهيموجلوبين و حجم كرات الدم المضغوطه . كما وجدت زيادة معنوية في العدد الكلى والنوعى لخلايا الدم البيضاء .كما وجدت تغيرات معنوية فى كيمياء الدم ووظائف الكبد والقلب مع اضطراب فى الانزيمات المضادة للاكسدة. وعادت هذه القياسات إلى المستويات الطبيعية بعد ٢١يوم من العلاج. ويمكن أن نستخلص من ذلك إلى أن اصابة الجاموس بالفلاريا ينتج عنه فقر دم من النوع الانحلالى ، مع اضطراب في الانزيمات المضادة للاكسدة ووظائف الكبد و القلب. وايضا وجد ان الايفرمكتين علاج مؤثر ضد مرض الافلاريا . وعلاوة على ذلك فإن زيادة التروبونين فى السيرم إلى ١.٥ نجم / ملى أو أكثر يدل على عدم جدوى العلاج .