



Therapeutic Effect of *Balanites Aegyptiaca* Fruit's Ethanol Extract in Egyptian Baladi Goats Experimentally Infected with *Haemonchus Contortus*: Blood Serum Biochemical, Oxidative Stress Markers and Pathological Studies



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THE THERAPEUTIC effect of *Balanites aegyptiaca* fruit's ethanolic extract (BAF-EE) in goats experimentally infected with *Haemonchus contortus* (*H. contortus*) was evaluated. Serum biochemical parameters, tissue oxidative stress status and pathological changes were investigated. Twelve goats were divided into 4 equal groups, G1 (infected-untreated), G2 (infected-BAF-EE treated), G3 (infected-albendazole treated) and G4 (uninfected-BAF-EE treated). Infection was conducted at 0-time, whereas treatment with BAF-EE and albendazole was given in the 5th week post infection (PI). Blood samples were drawn on 0-time, 3rd week PI and then biweekly till the 9th week for serum biochemical analyses. At the end of experiment, all animals were slaughtered to estimate oxidative stress markers and histopathological alterations in the abomasal and hepatic tissues. Serum biochemistry of infected animals showed significant depression of total serum proteins, albumin, albumin/globulin ratio, total antioxidant capacity and calcium levels. The activity of AST, ALT and γ GT and the cholesterol, urea and creatinine levels were significantly elevated in the serum of infected animals. Glutathione peroxidase values decreased significantly in tissue homogenate of G1 and G3 goats than those in G2 and G4 goats. Malondialdehyde followed a reverse course. Abomasa of infected animals in G1 revealed inflammation, necrosis, petechial hemorrhages and cellular infiltration. Liver showed bile duct hyperplasia, degeneration of hepatocytes and cell infiltration. Intensity of alterations were lower in infected-treated animals. In conclusion, BAF-EE has hepatoprotective effect and can improve serum biochemical alterations and oxidative stress status in goats infected with *H. contortus*, justifying its use as a safe natural anthelmintic agent.

Keywords: *Haemonchus contortus*, Goats, Blood serum biochemistry, Pathology, *Balanites aegyptiaca*.

Introduction

Infection by gastrointestinal nematode parasites, especially *Haemonchus contortus* (*H. contortus*) in small ruminants causes great danger to sheep and goat production all over the world. In Egypt,

the native breed of goats that is located in the Nile Delta and along the Nile Valley is known as Baladi goats. Amongst other breeds of goats, the Baladi goats have a great economic value [1] and play an essential role in the economy of small, marginal farmers and landless labourers in Egypt [2].

Infection of goats with *H. contortus* parasite has deleterious effect on their health, causing anaemia, loss of body weight and productivity [3] and may cause death due to haemorrhages in the gastrointestinal tract of the animals [4]. The parasite induces decreased appetite and digestibility of food as well as diversion of nutrients from production sites toward the repair of tissue-damage, which lead to disturbances in the hematological, blood biochemical and antioxidant parameters of the host [5-9].

When inhabiting the abomasum of sheep and goats, the parasite mainly affects the abomasal mucosa, consuming blood of the host and causing pathological alterations such as hyperplasia of the abomasal epithelium, edema of the mucosa and submucosa, hemorrhages, elevated abomasal pH and necrosis of different parts of the abomasum [10,11].

On the other hand, development of resistance to most of the commercially available anthelmintic drugs, besides the unavailability of these drugs to the poor farmers of developing countries opened way for using herbal remedies as alternative anthelmintics [12]. Among these, *Balanites aegyptiaca* (*B. aegyptiaca*) -a well-known medicinal plant- is present in tropical and subtropical regions of the world [13].

B. aegyptiaca (L.) *Delile* is a species of a tree, belonging to the family *Balanitaceae*, which has compound leaves, greenish yellow flowers, and brown or pale brown fruit known as desert date [13]. Its traditional roles and values are

Total number of larvae

$$= \frac{\text{No. of larvae counted in petri dish} \times \text{Total volume of solution}}{\text{Amount of solution transferred to petri dish}}$$

well known for thousands of years as fruits were found in cemeteries of the 12th Egyptian dynasty. Recently, numerous studies reported that *B. aegyptiaca* contains wide variety of compounds, which show a wide range of biological and pharmacological properties such as antioxidant, anti-inflammatory, anti-parasitic, antimicrobial, antidiabetic and cytotoxic activities [9,13,14].

The objective of the present study was to evaluate the anthelmintic suppressant effects of the medicinal plant *B. aegyptiaca* fruit's mesocarp ethanolic extract (BAF-EE) in Egyptian Baladi goats experimentally infected with *H. contortus* in comparison with the effects of albendazole as a

broad spectrum anthelmintic. Evaluation of blood serum biochemical parameters, tissues oxidant-antioxidant status, gross and histopathological alterations in abomasum and liver were considered in this study. Assessment of the BAF-EE's therapeutic anthelmintic effect in Egyptian Baladi goats experimentally infected with *H. contortus* has been identified elsewhere through the study of parasitological and hematological parameters [9].

Material and Methods

Procurement of fruit and preparation of BAF-EE

B. aegyptiaca fruit has been collected from a local market in Upper Egypt and has been scientifically verified by the Medicinal Plants Department, NRC, Egypt. The BAF-EE was prepared following the method described by Wang and Weller [15]. The mesocarp of the fruits was macerated several times with 70% ethyl alcohol at room temperature for 1 week then filtered. Rotary evaporator drained the solvent under vacuum at 40 °C and the extract was stored at -4 °C.

Genesis of the dose of infective larva of H. contortus parasite (L₃)

Based on methods described by Roberts and O'Sullivan [16] and Hunter and Mackenzie [17], the female worms were used to obtain the infective third larval stage (L₃) of *H. contortus* worms in a suitable culture. The L₃ were obtained by means of Baerman Wetzal funnel technique and the obtained larvae were counted and their number in the total amount of the solution was calculated using the formula described by Soulsby [18].

A goat kid that has been shown to be free from internal parasites was inoculated orally with a dose of 10,000 L₃ in 10 ml physiological saline solution as recommended by Jaheed *et al.* [9] and Howlader *et al.* [19] and kept as a donor animal. The donor animal was subsequently used as a source for monospecific L₃ to cause infection in the experimental animals.

Experimental animals and design

A total of 12 male, six to nine months old Egyptian Baladi goats (*Capra hircus*), weighing 15-20 Kg., apparently healthy and free of internal parasites were used. The animals were housed indoor in a barn at the Faculty of Veterinary

Medicine, Cairo University and fed on a balanced diet, given fresh tap water, and kept under close observation for 30 days until experimentation to acclimatize together. They were randomly divided into four experimental groups, each of three animals, as group 1 (G1), group 2 (G2), group 3 (G3) and group 4 (G4). Animals in G1 were infected orally with 10,000 infective third larval stage (L_3) of *H. contortus* and served as control positive group. Animals of G2 were infected with L_3 as mentioned above and administered BAF-EE at an oral dose of 9 g / Kg body weight. Animals of G3 were infected as mentioned above and treated with a single oral dose of albendazole at the rate of 5 mg/kg body weight, in the form of 2.5% oral suspension as recommended by the manufacturer company (the Veterinary Division of EVA Pharma, Cairo, Egypt). Animals of G4 were kept as normal control group (uninfected-BAF-EE treated group). During the experimental period, which lasted for 9 weeks, each group of experimental animals was kept isolated in a separate ban. The infection was confirmed on the 3rd week post infection (PI) by fecal analysis as reported in the first part of this study [9].

Assessment of treatment efficacy

Blood serum biochemical assays

Samples for serum biochemical analyses were obtained from whole blood collected from the jugular vein of each animal into plain vacutainer tubes at zero-time of the experiment, and at the 3rd week PI, then every other week PI until the end of the experiment. Sera were kept frozen at (-20 °C) until used. Blood serum biochemical parameters were determined utilizing T80 UV/VIS spectrophotometer (England) and standard commercial test kits for each component; total protein (TP) was determined according to the Biuret method [20], albumin was assayed by the Bromocresol green method [21] and value of total globulins was calculated by subtracting albumin concentration from total proteins [22]. Activities of aspartate aminotransferase (AST), and alanine aminotransferase (ALT) were evaluated according to the method of Reitman and Frankel [23], while that of gamma-glutamyl transferase (γ GT) was measured according to Szasz [24]. Levels of serum total cholesterol and triglycerides were determined according to Allain et al. [24] and Fossati and Prencipe [26], respectively. Total bilirubin was measured as described by Kazmierczak [27]. Serum creatinine value was obtained using method of Houot [28] and urea level was determined according to Patton

and Crouch [29]. Serum total antioxidant capacity (TAC) was estimated after Koracevic et al. [30].

Calcium (Ca) and inorganic phosphorus (P) values were obtained according to Gindler and King [31] and Goodwin [32], respectively. Sodium (Na), potassium (K) and chloride (Cl) values were estimated according to Trinder [33], Henry [34] and Schoenfeld and Lewellen [35], respectively. Total iron binding capacity (TIBC) was determined according to Fairbanks and Klee [36].

Evaluation of oxidant-antioxidant status in tissue samples

All research animals were slaughtered at the end of the experiment, and a collection of tissue samples were taken from the abomasum and liver, placed in labeled plastic bags and kept at -80 °C until processing. For measuring oxidant-antioxidant status, the samples were fully homogenized by means of glassy mortar using liquid nitrogen. From each homogenized sample one gram was mixed thoroughly with 10 ml of physiological saline and centrifuged at 4000 rpm for 10 minutes. The activity of the antioxidant enzyme; glutathione peroxidase (GPx) and the level of malondialdehyde (MDA) -a marker of lipid peroxidation- were estimated in the supernatant following the methods of Paglia and Valentine [37] and Ohkawa et al. [38], respectively.

Pathological observations

Gross lesions

All research animals have been necropsied and gross pathological alterations were recorded.

Assessment of histopathological lesions

Tissue specimens from abomasa and livers were sliced at a size of 0.5-1 cm and preserved into 10% neutral buffered formalin. Specimens then were processed for routine histopathological examination as described by Prophet et al. [39]. Microtomy was performed by rotatory microtome (Baird and Totlock, England), and sections of 5 microns thickness were prepared and stained with haematoxylin and eosin (H&E).

Statistical analysis

All data were subjected to statistical analysis according to Snedecor and Cochran [40] using SPSS (version 17) computer program. Statistical analysis included calculation of the mean and standard error of the mean (mean \pm SE). Differences between control and treated groups were tested for significance using one-way

analysis of variance (ANOVA), and Duncan's multiple range test to detect the significance among means in between different experimental groups and weeks. Differences were considered significant at $P \leq 0.05$ level of probability.

Results

Blood serum biochemical analysis

Mean values of blood serum constituents of normal control animals (at 0-time) were fluctuated within the normal range throughout the experimental period. Blood serum constituents of experimental animals are shown in tables 1 through 4 below:

Serum protein profile

Significant ($P \leq 0.05$) decline in TP levels was observed in G1 goats throughout the experiment

compared with normal animals in G4 at 0-time. Goats of groups G2 and G3 showed significant ($P \leq 0.05$) decrease in TP levels during the 3rd and 5th weeks PI, values then were elevated at the 7th and 9th weeks to be within the normal range. Values of TP of G4 goats were comparable to normal. Decreases of total proteins values were attributable to variations in albumin values as globulin levels of all groups did not record significant differences. A significant ($P \leq 0.05$) depression was observed in A/G ratio in G1 goats on week 3 PI and thereafter, on weeks 3, 5 and 7 PI in G2 and on the 5th week PI in G3 goats compared with comparable 0-time ratios. A/G ratios of experimental goats at other times of the experiment were comparable to ratios of 0-time (Table 1).

TABLE 1. Serum protein profile in different groups of goats during the experimental period (Mean \pm SE)

Parameters	Groups	Weeks post infection (PI)				
		0	3 rd	5 th	7 th	9 th
Total proteins (g/dl)	G1	7.32 \pm 0.12 ^b	6.01 \pm 0.30 ^{ABa}	6.04 \pm 0.20 ^{Aa}	6.23 \pm 0.14 ^{Aa}	6.30 \pm 0.16 ^{Aa}
	G2	7.45 \pm 0.24 ^b	6.03 \pm 0.12 ^{Ba}	6.04 \pm 0.20 ^{Aa}	6.63 \pm 0.21 ^{ABab}	6.94 \pm 0.61 ^{ABab}
	G3	7.25 \pm 0.36 ^b	6.48 \pm 0.23 ^{ABbc}	6.10 \pm 0.18 ^{acA}	6.52 \pm 0.55 ^{ABab}	6.79 \pm 0.52 ^{ABab}
	G4	7.50 \pm 0.39	7.23 \pm 0.34 ^A	7.44 \pm 0.10 ^B	7.56 \pm 0.00 ^B	7.55 \pm 0.11 ^B
Albumin (g/dl)	G1	4.07 \pm 0.05 ^b	2.70 \pm 0.20 ^a	2.72 \pm 0.19 ^{Aa}	2.85 \pm 0.09 ^{Aa}	2.88 \pm 0.05 ^{Aa}
	G2	4.29 \pm 0.47 ^b	2.60 \pm 0.25 ^a	2.54 \pm 0.20 ^{Aa}	3.16 \pm 0.21 ^{ABab}	3.15 \pm 0.63 ^{ABab}
	G3	3.78 \pm 0.31 ^b	2.90 \pm 0.22 ^a	2.70 \pm 0.01 ^{Aa}	2.89 \pm 0.02 ^{Aa}	3.08 \pm 0.37 ^{ABab}
	G4	4.06 \pm 0.45	3.80 \pm 0.42	3.66 \pm 0.07 ^B	3.88 \pm 0.17 ^B	3.85 \pm 0.38 ^B
Total globulins (g/dl)	G1	3.25 \pm 0.14	3.32 \pm 0.38	3.33 \pm 0.19	3.38 \pm 0.33	3.32 \pm 0.23
	G2	3.20 \pm 0.21	3.43 \pm 0.34	3.50 \pm 0.35	3.47 \pm 0.08	3.39 \pm 0.29
	G3	3.47 \pm 0.18	3.56 \pm 0.44	4.20 \pm 0.47	3.62 \pm 0.57	3.62 \pm 0.51
	G4	3.45 \pm 0.13	3.44 \pm 0.11	3.47 \pm 0.19	3.67 \pm 0.17	3.51 \pm 0.18
A/G ratio	G1	1.25 \pm 0.02 ^b	0.82 \pm 0.08 ^a	0.81 \pm 0.03 ^{Aa}	0.87 \pm 0.12 ^a	0.86 \pm 0.16 ^a
	G2	1.36 \pm 0.16 ^b	0.78 \pm 0.11 ^a	0.72 \pm 0.08 ^{ACa}	0.91 \pm 0.17 ^{ab}	0.95 \pm 0.23 ^{ab}
	G3	1.19 \pm 0.07 ^b	0.86 \pm 0.17 ^b	0.66 \pm 0.07 ^{Ca}	0.84 \pm 0.14 ^b	0.86 \pm 0.17 ^b
	G4	1.19 \pm 0.15	1.11 \pm 0.16	1.07 \pm 0.05 ^B	1.07 \pm 0.09	1.11 \pm 0.15

Values bearing superscripts A, B, C, D in a column and a, b, c, d in a row differ significantly at $P \leq 0.05$.

G1: Goats infected- untreated and expressed as control positive.

G2: Goats infected and treated with BAF-EE.

G3: Goats infected and treated with albendazole.

G4: Goats uninfected and dosed with BAF-E E and expressed as control negative.

A/G: Albumin/Globulins

Serum enzymes activity

Activities of ALT, AST and γ GT elevated significantly ($P \leq 0.05$) in all infected groups (G1, G2 and G3) at the 3rd and 5th weeks PI. At the 7th and 9th weeks PI, activities were still elevated in G1 goats. Activities of γ GT were lowered in G2 and G3 goats though still higher than 0-time values. Activities of ALT and AST in G2 goats were comparable to normal in G4 at the 7th and 9th week PI, respectively. Activities of the tested serum enzymes in G4 goats were within normal limits throughout the experiment (Table 2).

Serum lipid, total Bilirubin, urea, creatinine and TAC Profiles

The relevant profiles for the above parameters in all experimental groups are shown in Table 3. Serum cholesterol values in infected-untreated (G1) and infected –BAF EE treated goats (G2) showed significant ($P \leq 0.05$) increase from the 3rd week PI and thereafter compared to infected – albendazole treated (G3) and uninfected

control goats (G4). At the end of the experiment (9th week), cholesterol values in groups 2, 3 and 4 were within normal levels. No significant difference was observed in triglycerides and total bilirubin levels within and among groups of the experimental animals. Values of serum urea in G1 and G3 goats recorded significant ($P \leq 0.05$) elevation than similar values in groups G2 and G4 as of the third week PI and onwards till the end of the experiment. Mean levels of creatinine of infected goats of groups G1, G2 and G3 showed significant ($P \leq 0.05$) increase starting of the 3rd week PI and thereafter to the end of the experiment comparing to 0-time values. It was observed that values of creatinine at the 3rd and 5th weeks were higher than those of the 7th and 9th weeks. Values of creatinine of G4 group were within normal levels throughout the experiment. Serum TAC levels were significantly ($P \leq 0.05$) depressed in infected animals of G1, G2 and G3 groups at the 5th, 7th and 9th weeks PI, while those of G4 animals were elevated from the 3rd week

TABLE 2. Serum enzymes activity in different groups of goats during the experimental period (Mean \pm SE)

Parameters	Groups	Weeks post infection (PI)				
		0	3 rd	5 th	7 th	9 th
ALT (U/L)	G1	13.43 \pm 0.19 ^a	16.55 \pm 1.67 ^{Bb}	15.04 \pm 0.82 ^{Bb}	15.27 \pm 0.28 ^{Bb}	16.82 \pm 0.11 ^{Bb}
	G2	11.45 \pm 0.63 ^a	17.02 \pm 0.65 ^{Bb}	16.80 \pm 2.32 ^{Bb}	14.76 \pm 2.94 ^{ABab}	14.11 \pm 2.28 ^{Aab}
	G3	12.77 \pm 0.49 ^a	17.68 \pm 1.43 ^{Bb}	18.63 \pm 1.32 ^{Bb}	17.49 \pm 1.12 ^{Bb}	16.64 \pm 1.22 ^{ABb}
	G4	14.15 \pm 2.69 ^b	12.83 \pm 1.30 ^A	11.19 \pm 0.22 ^A	12.85 \pm 0.16 ^A	13.45 \pm 2.00 ^A
AST (U/L)	G1	69.09 \pm 0.60 ^a	107.81 \pm 3.00 ^{Bb}	102.14 \pm 2.15 ^{Bb}	98.88 \pm 1.01 ^{Bb}	102.48 \pm 2.29 ^{Bb}
	G2	78.89 \pm 0.23 ^a	102.37 \pm 2.03 ^{Bb}	113.61 \pm 2.02 ^{Bb}	95.09 \pm 3.48 ^{Bb}	84.98 \pm 3.90 ^{ABab}
	G3	65.98 \pm 0.34 ^a	111.91 \pm 0.27 ^{Bb}	103.42 \pm 3.69 ^{Bb}	102.37 \pm 0.74 ^{Bb}	103.41 \pm 2.70 ^{Bb}
	G4	81.47 \pm 4.50	73.61 \pm 0.47 ^A	64.82 \pm 1.14 ^A	76.18 \pm 2.83 ^A	79.51 \pm 1.96 ^A
γ GT (U/L)	G1	20.33 \pm 0.39 ^a	30.20 \pm 2.13 ^{Bb}	30.05 \pm 0.20 ^{Bb}	31.86 \pm 0.14 ^{Bb}	29.11 \pm 1.55 ^{Bb}
	G2	17.59 \pm 1.20 ^a	26.96 \pm 2.57 ^{ABb}	27.48 \pm 2.04 ^{Bb}	22.35 \pm 2.37 ^{Aab}	19.34 \pm 2.54 ^{Aa}
	G3	19.50 \pm 0.73 ^a	28.52 \pm 0.032 ^{Bb}	27.37 \pm 0.99 ^{Bb}	25.15 \pm 1.55 ^{Ab}	24.88 \pm 2.84 ^{ABb}
	G4	21.36 \pm 0.85	22.93 \pm 0.89 ^A	23.75 \pm 0.54 ^A	23.45 \pm 0.70 ^A	22.87 \pm 1.05 ^A

Values bearing superscripts A, B, C, D in a column and a, b, c, d in a row differ significantly at $P \leq 0.05$.

G1: Goats infected- untreated and expressed as control positive.

G2: Goats infected and treated with BAF-EE.

G3: Goats infected and treated with albendazole.

G4: Goats uninfected and dosed with BAF-EE and expressed as control negative.

ALT: Alanine aminotransferase. AST: Aspartate aminotransferase. γ GT: gamma-glutamyl transferase.

through the 9th week of the experiment compared to 0-time values.

Serum minerals /electrolytes levels

Values of blood serum minerals/electrolytes of the experimental groups are shown in Table 4. Mean Ca concentration declined significantly in all infected goats on the 3rd week PI and continued thereafter to the 9th week compared with their 0-time values and values of animals of group (4). Calcium concentration in BAF-EE treated animals (G4)

remained within normal levels throughout the time of the experiment. No significant differences were observed in serum P, Na, K, Cl and TIBC levels of all experimental groups, either within different times of the experiment or among the groups.

Oxidant-antioxidant status in tissue samples

Oxidant-antioxidant (GPx and MDA) status in the abomasum and liver tissues of different experimental groups is shown in Table 5. Statistical analysis of GPx value revealed

TABLE 3. Blood serum lipid, total bilirubin, urea, creatinine and total antioxidant capacity (TAC) profiles in different groups of goats during the experimental period (Mean ± SE).

Parameters	Groups	Weeks Post Infection (PI)				
		0	3 rd	5 th	7 th	9 th
Cholesterol (mg/dl)	G1	127.93 ± 2.61 ^a	147.75 ± 4.68 ^{Bb}	136.93 ± 5.35 ^{Bb}	146.63 ± 7.79 ^{Bb}	141.81 ± 4.39 ^{Ba}
	G2	125.92 ± 5.13 ^{ac}	135.01 ± 6.06 ^{BCa}	153.97 ± 2.44 ^{Bb}	99.16 ± 8.99 ^{Ac}	110.02 ± 9.53 ^{Aac}
	G3	115.32 ± 5.72 ^a	125.23 ± 6.24 ^{ACa}	146.22 ± 4.28 ^{Bb}	137.99 ± 3.86 ^{Bb}	128.69 ± 8.81 ^{ABab}
	G4	111.46 ± 11.07	108.11 ± 7.81 ^A	110.81 ± 5.26 ^A	102.71 ± 8.84 ^A	108.27 ± 7.60 ^A
Triglycerides (mg/dl)	G1	117.65 ± 9.59	124.82 ± 11.91	93.96 ± 7.93	140.54 ± 12.00	131.74 ± 13.84
	G2	128.46 ± 14.81	134.39 ± 13.11	129.81 ± 14.12	76.33 ± 11.84	89.75 ± 10.10
	G3	106.04 ± 6.03	129.62 ± 14.96	100.55 ± 11.10	96.71 ± 1.90	108.23 ± 13.96
	G4	105.49 ± 5.71	118.17 ± 11.17	95.00 ± 14.39	80.22 ± 12.69	90.72 ± 16.33
Total bilirubin (mg/dl)	G1	0.08 ± 0.04	0.24 ± 0.03	0.34 ± 0.14	0.47 ± 0.16	0.28 ± 0.15
	G2	0.04 ± 0.02	0.31 ± 0.13	0.05 ± 0.01	0.05 ± 0.03	0.12 ± 0.11
	G3	0.06 ± 0.01	0.23 ± 0.14	0.13 ± 0.04	0.42 ± 0.20	0.21 ± 0.16
	G4	0.05 ± 0.01	0.15 ± 0.08	0.21 ± 0.06	0.04 ± 0.02	0.11 ± 0.08
Urea (mg/dl)	G1	27.90 ± 1.56 ^a	46.35 ± 3.45 ^{Ab}	51.60 ± 3.88 ^{Bbc}	60.30 ± 3.64 ^{Bb}	50.54 ± 2.93 ^{BCb}
	G2	30.00 ± 2.10 ^a	31.23 ± 1.28 ^{Ca}	49.60 ± 1.28 ^{Bb}	42.86 ± 3.01 ^{ACbc}	36.43 ± 4.48 ^{ACac}
	G3	34.65 ± 2.86 ^a	45.83 ± 0.18 ^{Ab}	51.30 ± 2.19 ^{Bb}	52.65 ± 3.57 ^{BCb}	45.60 ± 1.46 ^{BCb}
	G4	38.36 ± 3.34	39.05 ± 4.07 ^{AC}	40.84 ± 1.68 ^A	36.25 ± 1.22 ^A	36.62 ± 1.51 ^A
Creatinine (mg/dl)	G1	0.26 ± 0.07 ^{BCa}	0.46 ± 0.04 ^{Ab}	0.53 ± 0.13 ^{Bbc}	0.61 ± 0.05 ^{Bc}	0.54 ± 0.01 ^{Bc}
	G2	0.37 ± 0.13 ^{Aa}	0.71 ± 0.02 ^{Bb}	0.66 ± 0.15 ^{Bb}	0.57 ± 0.05 ^{Bb}	0.45 ± 0.13 ^{Aac}
	G3	0.36 ± 0.05 ^{Aa}	0.66 ± 0.07 ^{Bb}	0.53 ± 0.06 ^{Bb}	0.46 ± 0.04 ^{Cb}	0.40 ± 0.08 ^{Ab}
	G4	0.33 ± 0.10 ^{AC}	0.40 ± 0.14 ^A	0.35 ± 0.02 ^A	0.33 ± 0.08 ^A	0.40 ± 0.10 ^A
TAC (mmol/L)	G1	0.39 ± 0.17 ^a	0.30 ± 0.04 ^{Ca}	0.14 ± 0.12 ^{Ab}	0.22 ± 0.04 ^{Ab}	0.19 ± 0.10 ^{Cb}
	G2	0.55 ± 0.09 ^a	0.20 ± 0.04 ^{Bb}	0.20 ± 0.14 ^{Ab}	0.24 ± 0.02 ^{Ab}	0.39 ± 0.03 ^{Ac}
	G3	0.48 ± 0.12 ^a	0.21 ± 0.13 ^{Bc}	0.25 ± 0.10 ^{Abc}	0.28 ± 0.09 ^{bcA}	0.34 ± 0.02 ^{Ab}
	G4	0.45 ± 0.04 ^a	0.47 ± 0.11 ^{Aa}	0.51 ± 0.02 ^{Ba}	0.71 ± 0.09 ^{Bb}	0.66 ± 0.01 ^{Bb}

Values bearing superscripts A, B, C, D in a column and a, b, c, d in a row differ significantly at $P \leq 0.05$.

G1: Goats infected- untreated and expressed as control positive.

G2: Goats infected and treated with BAF-EE.

G3: Goats infected and treated with albendazole.

G4: Goats uninfected and dosed with BAF-EE and expressed as control negative.

TAC: total antioxidant capacity.

significant difference ($P \leq 0.05$) between groups; G1 and G3 compared with G2 and G4 groups. The highest value was recorded in abomasa and livers of G4 followed by G2 animals, while values of G3 and G1 were lower. Value of MDA recorded significant ($P \leq 0.05$) elevation in tissues of G1 and G3 goats compared to values of G4 and G2 goats which were lower.

Pathological

Necropsy Findings

Infected-untreated G1 goat carcasses were emaciated and their mucous membranes were

pale. The abomasal contents were fluidy, stained dark brown and tinged with bloody flakes with large number of viable actively moving adult *H. contortus* parasite. The abomasal serosa showed focal ecchymotic hemorrhagic areas (Fig. 1A) and the mucosa showed hyperaemic mucosal folds with focal ulceration (Fig. 1B & 1C). These changes were more severe in fundic than in cardiac and pyloric areas. The associated lymph nodes were edematous and enlarged in all infected goats. The liver was pale in color and showed focal pale areas of coagulative necrosis (Fig. 1D).

TABLE 4. Serum minerals /electrolytes levels in different groups of goats during the experimental period (Mean \pm SE)

Parameters	Groups	Weeks Post Infection (PI)				
		0	3 rd	5 th	7 th	9 th
Calcium (mg/dl)	G1	10.13 \pm 0.06 ^b	7.99 \pm 0.39 ^{aA}	8.44 \pm 0.29 ^{aA}	8.84 \pm 0.12 ^a	8.35 \pm 0.24 ^{ABa}
	G2	10.66 \pm 0.16 ^b	8.63 \pm 0.23 ^{aA}	8.13 \pm 0.08 ^{aA}	9.31 \pm 0.82 ^{ab}	9.18 \pm 0.76 ^{ABab}
	G3	11.07 \pm 0.27 ^b	9.01 \pm 0.01 ^{ABa}	8.82 \pm 0.02 ^{aA}	8.78 \pm 0.40 ^a	9.14 \pm 0.44 ^{ABa}
	G4	9.91 \pm 0.70	9.92 \pm 0.09 ^B	9.92 \pm 0.09 ^B	9.91 \pm 0.06	10.72 \pm 0.33 ^B
Inorganic Phosphorus (mg/dl)	G1	6.05 \pm 0.62	5.07 \pm 0.41	5.25 \pm 0.48	5.20 \pm 0.50	5.19 \pm 0.60
	G2	5.44 \pm 0.29	4.49 \pm 0.32	4.29 \pm 0.17	5.75 \pm 0.62	5.00 \pm 0.61
	G3	5.86 \pm 0.27	4.75 \pm 0.53	5.08 \pm 0.38	5.34 \pm 0.39	4.93 \pm 0.46
	G4	5.07 \pm 0.42	4.83 \pm 0.23	5.44 \pm 0.47	5.64 \pm 0.35	5.25 \pm 0.46
Sodium (mmol/L)	G1	126.90 \pm 3.47	105.38 \pm 6.39	133.54 \pm 11.33	130.07 \pm 3.83	123.97 \pm 10.92
	G2	122.99 \pm 6.18	115.40 \pm 6.89	124.64 \pm 8.72	133.04 \pm 10.88	121.51 \pm 12.68
	G3	136.39 \pm 6.39	121.39 \pm 6.21	130.85 \pm 4.29	134.18 \pm 5.85	120.45 \pm 12.86
	G4	125.32 \pm 2.78	123.42 \pm 2.92	129.43 \pm 4.93	121.52 \pm 10.96	129.92 \pm 9.34
Potassium (mmol/L)	G1	4.23 \pm 0.13	4.44 \pm 0.26	5.40 \pm 0.40 ^A	4.33 \pm 0.48	4.35 \pm 0.62
	G2	4.32 \pm 0.46	5.51 \pm 0.74	4.95 \pm 0.18 ^A	4.19 \pm 0.28	4.06 \pm 0.53
	G3	3.77 \pm 0.61	3.96 \pm 0.76	4.93 \pm 0.13 ^A	3.92 \pm 0.55	3.89 \pm 0.53
	G4	3.83 \pm 0.70	3.95 \pm 0.82	3.65 \pm 0.16 ^B	3.81 \pm 0.18	3.51 \pm 0.25
Chloride (mmol/L)	G1	100.74 \pm 3.14	95.90 \pm 2.21	105.63 \pm 3.33	108.16 \pm 10.22	101.86 \pm 4.45
	G2	104.35 \pm 2.08	100.33 \pm 2.53	103.80 \pm 3.98	107.12 \pm 1.50	103.91 \pm 3.32
	G3	106.93 \pm 0.89	110.28 \pm 4.28	108.73 \pm 0.14	109.29 \pm 1.98	108.81 \pm 1.46
	G4	102.69 \pm 6.17	102.28 \pm 3.01	103.92 \pm 0.47	99.59 \pm 5.71	102.12 \pm 3.79
TIBC (μ g/dl)	G1	295.29 \pm 17.4	277.57 \pm 20.75	280.29 \pm 17.11	272.34 \pm 20.36	263.87 \pm 37.02
	G2	260.38 \pm 22.9	245.68 \pm 15.29	258.29 \pm 22.95	256.04 \pm 20.29	232.59 \pm 24.11
	G3	272.13 \pm 26.8	264.33 \pm 19.66	282.97 \pm 19.66	310.27 \pm 21.84	274.92 \pm 42.59
	G4	293.26 \pm 11.1	280.00 \pm 13.11	275.58 \pm 18.13	274.33 \pm 9.83	281.79 \pm 30.89

Values bearing superscripts A, B, C, D in a column and a, b, c, d in a row differ significantly at $P \leq 0.05$.

G1: Goats infected- untreated and expressed as control positive.

G2: Goats infected and treated with BAF-EE.

G3: Goats infected and treated with albendazole.

G4: Goats uninfected and dosed with BAF-EE and expressed as control negative.

TIBC: Total iron binding capacity.

TABLE 5. Oxidant-antioxidant status in tissue samples from abomasum and livers of different experimental goat groups (Mean \pm SE).

Organ	Groups	GPx	MDA
		(U/g tissue)	(nmol/g tissue)
Abomasum	G1	20.53 \pm 2.79 ^a	964.59 \pm 138.68 ^b
	G2	65.07 \pm 10.11 ^b	555.15 \pm 42.92 ^a
	G3	38.42 \pm 8.68 ^a	683.57 \pm 65.54 ^a
	G4	78.45 \pm 5.86 ^b	430.77 \pm 40.53 ^a
Liver	G1	11.42 \pm 0.86 ^a	2433.33 \pm 698.41
	G2	29.18 \pm 5.62 ^b	1099.03 \pm 71.12
	G3	16.21 \pm 1.87 ^a	2497.58 \pm 725.17
	G4	36.11 \pm 3.39 ^b	815.83 \pm 93.27

Values bearing superscripts a, b, in a column (among groups) differ significantly at ($P \leq 0.05$).

G1: Goats infected- untreated and expressed as control positive.

G2: Goats infected and treated with BAF-EE.

G3: Goats infected and treated with albendazole.

G4: Goats uninfected and dosed with BAF-EE and expressed as control negative.

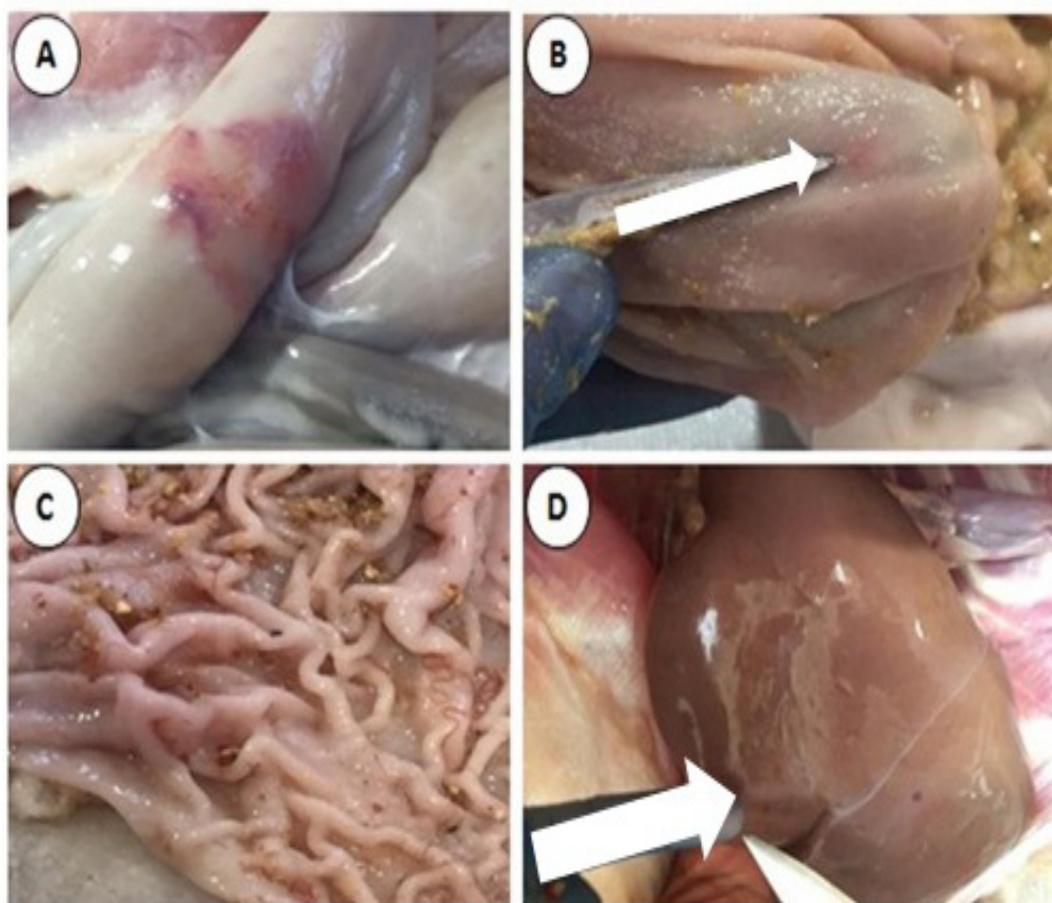


Fig. 1. Gross lesions in the abomasum and liver of infected goats: (A & B) abomasum, (C) abomasal mucosa and (D) liver.

In all Infected-treated animals, the general carcass condition was good. No salient gross lesions were seen in internal organs except the abomasa showed fluid content that stained with lighter brown in colour. The mucosa was slightly hyperemic and showed petechial hemorrhages with very few number of adult *H. contortus* worms. Goats of G4 showed normal macroscopic feature of the carcass and internal organs.

Microscopic Findings

Histopathological alterations in abomasa

Abomasa of infected - untreated goats (G1) showed inflammatory reaction involving different parts of the abomasum. Mucosa of the cardiac region of the abomasum revealed focal ulcerations with inflammatory cells' infiltration into the underlying submucosa (Fig. 2a). The fundic region showed marked mucosal

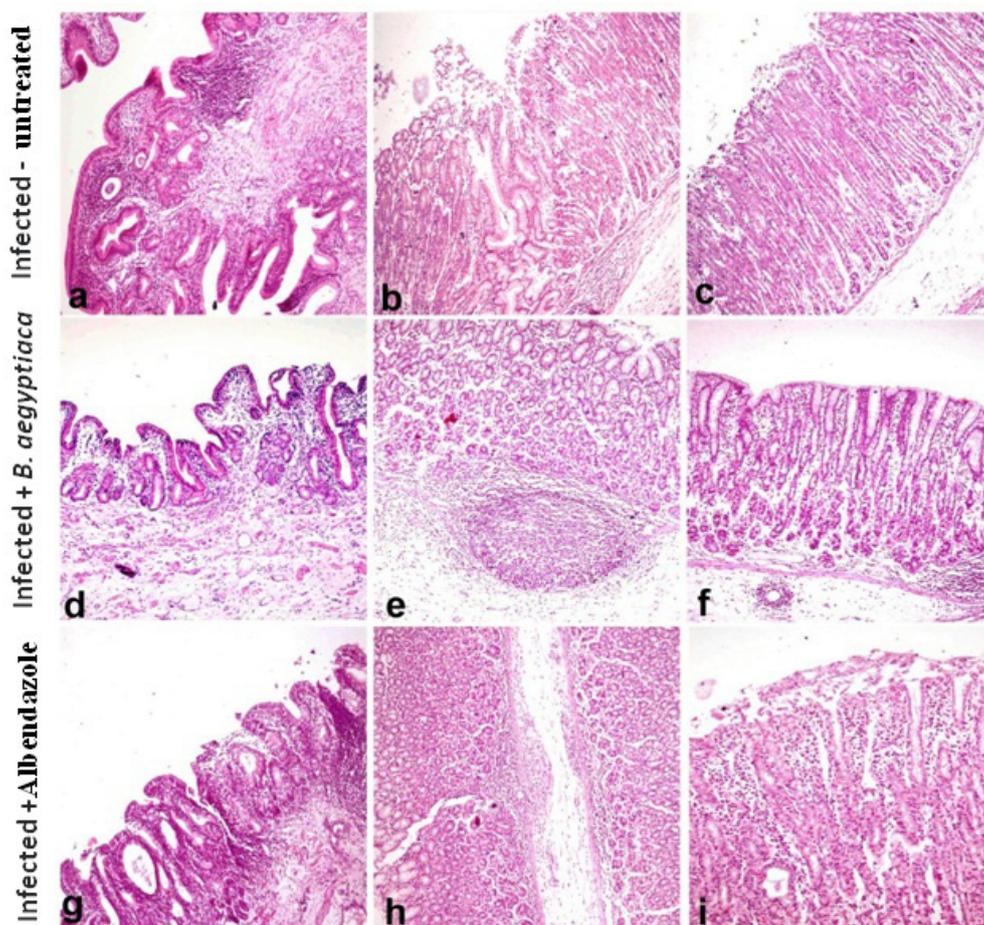


Fig. 2. Histological sections of abomasum from experimental goat groups; a) Abomasum (cardiac region) showing mucosal inflammatory cells infiltration associated with hyperplastic proliferation of abomasal glands and dilatation of individual ones (H&E, $\times 100$). b) Abomasum (fundic region) showing hypertrophy of abomasal mucosa with increased depth of gastric pits and apical minute hemorrhage and marked goblet cells hyperplasia (H&E, $\times 100$). c) Abomasum (fundic region) showing denuded gastric mucosa with inflammatory cell infiltration in the lamina propria and marked submucosal edema (H&E, $\times 100$). d) Abomasum (cardiac region) showing a mild inflammatory reaction in mucosa with normal epithelial mucosal lining (H&E, $\times 100$). e) Abomasum (fundic region) showing submucosal lymphoid aggregation (H&E, $\times 200$). f) Abomasum (pyloric region) showing inflammatory reaction with mucosal and submucosal edema with the presence of perivascular lymphocytic aggregation (H&E, $\times 100$). g) abomasum (cardiac region) showing mucosal inflammatory cell infiltration with dilatation of abomasal glands (H&E, $\times 100$). h) Abomasum (fundic region) showing mucosal hypertrophy with inflammatory cell infiltration at mucosal base with submucosal edema and inflammatory cells infiltration (H&E, $\times 40$). i) Abomasum (fundic region) showing denuded epithelium with eosinophils infiltration associated with dilated abomasal gland (H&E, $\times 200$).

hypertrophy associated with hyperplastic proliferation of the glands extending into underlying tissue (Fig. 2b). Minute hemorrhages with increase in the depth of gastric pits denoting sites of worms attachment were observed. There was an inflammatory reaction of the mucosa extending into submucosa. The inflammatory cells consisted mainly of eosinophiles mixed with neutrophils, lymphocytes and macrophages laden with hemosiderine pigment. Marked goblet cell hyperplasia was noted in the abomasal glandular epithelium. Lesions seen in the pyloric region of the abomasum were marked ulcerations and mucosal inflammation infiltrated mainly with eosinophiles and neutrophils (Fig. 2c).

Histopathological picture of abomasa of infected - BAF-EE treated goats (G2) was similar to that of G1 goats, though less severe. Mucosa of the cardiac region of the abomasum showed normal columnar epithelium and there were mild inflammatory cells' infiltration in the submucosa (Fig. 2d). The severity of histopathological alterations in the fundic region of this group was the same as G1, but the depth of gastric pits was normal. Mucosal hypertrophy and glandular goblet cells hyperplasia were less than G1 goats. Degenerated larvae surrounded by focal lymphocytic aggregations were detected in the deep fundic region. The inflammatory reaction in the fundic region consisted of mononuclear cells mixed with few neutrophils and there was necrosis of the abomasal glandular epithelium. There was eosinophils' infiltration in the deeper

mucosa extended into underlying submucosa. The pyloric part of the abomasum showed moderate inflammation associated with submucosal edema and perivascular lymphocytic aggregation (Fig. 2f).

Goats of G3 (infected and albendazole-treated) revealed dilated abomasal glands and the cardiac region showed an inflammatory reaction less than that of the infected group (G1) and more than that of the BAF-EE treated group (G2) (Fig. 2g). The fundic area showed hypertrophy, inflammation of mucosa and edema of the submucosa (Fig. 2h). Degenerated larvae were observed in the deeper abomasal mucosa with a normal depth of mucosal pits compared with those of G1 goats. The inflammatory cells mainly consisted of eosinophils and macrophages and there was individual necrosis of abomasal glandular epithelium. The pyloric region showed denuded mucosa with the presence of degenerated larvae at the mucosal surface and few eosinophils infiltration in the mucosa (Fig. 2i). Abomasa of (G4) goats appeared normal.

Histological alterations in liver

Liver of G1 goats revealed diffuse macrovesicular steatosis and cholangiohepatitis (Fig. 3a). Lesions were characterized by bile duct hyperplasia, portal fibrosis and portal mononuclear cells infiltration and necrosis of hepatocytes associated with oval cell proliferation. Liver of G2 goats revealed vacuolization of hepatocytes and histologically normal portal triad (Fig. 3c).

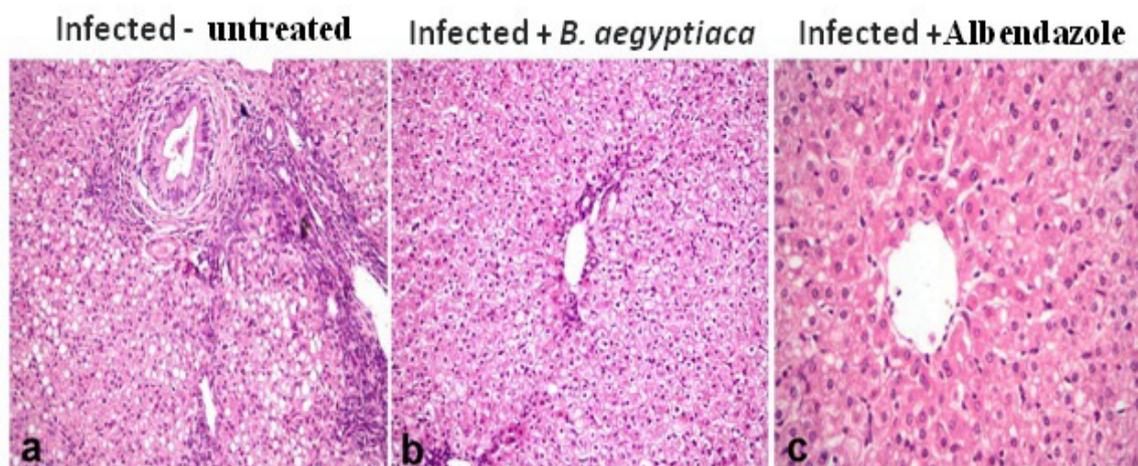


Fig.3. Histological sections of liver from the experimental goat groups; a) Liver showing portal fibrosis, bile duct hyperplasia with newly formed bile ductules and macrovesicular steatosis of hepatocytes (H&E,x200). b) Liver showing vacuolization of hepatocytes with normal histological structure of portal area (H&E, x200). c) Liver showing apoptosis of hepatocytes (H&E, x400).

Liver of G3 goats showed apoptosis of individual hepatocytes (Fig. 3e) with oval cell proliferation. Goats of G4 showed normal histological structure of the portal area.

Discussion

Gut parasites cause many physiological disturbances in the host body including metabolic changes, weight loss, hematological alterations and increased susceptibility to oxidative stress [6,41].

Alteration of an infected animal's serum biochemistry is correlated with the degree of damage and severity of infection [42,43].

The purpose of this experiment was to determine the therapeutic effects of a medicinal plant, *B. aegyptiaca* fruit's mesocarp ethanolic extract (BAF-EE) compared to a common commercial drug, albendazole in haemonchosis experimentally induced in goats. Alterations in blood serum biochemical parameters, tissue oxidant-antioxidant markers and pathological changes in abomasa and livers were studied in four groups of goats, G1 infected-untreated, G2 and G3 infected-BAF-EE/ or albendazole treated, respectively, and G4 uninfected-BAF-EE treated.

Results of serum biochemistry revealed decrease in total proteins and albumin values in all infected groups. The decrease continued throughout the experiment in G1 animals and was normalized in G2 and G3 animals towards the end of the experiment. Similar results were obtained by several authors worked on haemonchosis in sheep and goat; Qamar and Maqbool⁴, Albadawi⁵, Bordoloi et al. [7], Rashid [8], Mir et al. [44] and Hosseini et al. [45]. The observed hypoproteinemia may be attributed to various factors including; poor digestion and absorption of food, increased permeability of the abomasal mucosa leading to plasma leakage, direct blood loss caused by the parasite's sucking activity, bleeding caused by abomasal mucosal damage or ulcerations, stimulation of the proliferation of epithelial cell and replacement of cells producing abomasum acid by immature cells resulting in loss of serum protein in the gut, and liver damage [46,47]. The drop in serum albumin resulted in a decline in A/G ratio which in line with Qamar and Maqbool [4] and Hosseini et al. [45] findings. Protein values were increased progressively in treated animals two weeks after treatment which indicate improvement by treatment and reduction

of worm burden. Our results are similar to those reported by Albadawi [5], Koko et al. [48] and Adam [49]. Pathological studies performed on the present experimental goats support the observed serum protein changes as there were moving adult *H. contortus* parasite in the abomasa of infected animals and blood tinged fluidal contents indicating hemorrhages from the abomasal mucosa due the sucking habits of *H. contortus*. Histopathological changes of abomasa showed mucosal and submucosal hemorrhages especially in the untreated goats of G1 followed by both infected-treated groups G2 and G3 which are related directly to the migration of larvae into the pits of gastric glands and also to the physical injury caused by the attachment of adult and pre adult stages of *H. contortus* [50].

Activities of serum enzymes AST, ALT, and γ GT were elevated in infected groups (G1, G2, and G3) from the 3rd week PI, and continued to be elevated to the end of the experiment in G1 and G3 animals while returned to normal level at the end of the experiment in G2 goats treated with BAF-EE. Similar results were reported by a group of researchers; Bordoloi et al. [7], Rashid [8] and Sharma et al. [51] in sheep and goat infected with *H. contortus*, Manga-Gonzalez et al. [52] in lambs infected with *D. dentriticum* and Kolodziejczyk et al. [53] in rat infected with *F. hepatica*. Elevation of serum enzymes could be attributed to altered membrane permeability of liver cells which was proven by histopathological examination. Administration of BAF-EE to G2 goats normalized serum enzyme activities which may be attributed to membrane stabilizing effect of BAF-EE due to its content of total phenolics or flavonoids that reduce lipid peroxidation and/or steroids and triterpenoids which have an antioxidant activity [13]. The improvement noticed by the use of *B. aegyptiaca* fruits' mesocarp was attributed to its content of a wide range of biological constituents such as phytoconstituents, crude proteins, carbohydrates, organic acids and vitamin C as reported by Jaheed et al. [9] and Chothani and Vaghasiya [13].

Serum cholesterol values in (G1) and G2 goats showed significant increase from the 3rd week PI and thereafter compared to (G3) and (G4) animals. At the end of the experiment, cholesterol values in groups 2, 3 and 4 were within normal levels, but that of G1 was still high. Elevation of cholesterol in infected animals may be attributed to the parasitic stress resulting in increase of

epinephrine and corticosteroids output [47]. Also, it could reflect negative energy balance created by heavy load of parasitic burden leading to enhanced lipolysis [54]. The elevation of serum cholesterol has been reported in buffaloes infected with *Toxocara vitulum* [55]. The higher levels of urea and creatinine in infested animals suggest impaired regulation of renal tubular transport [56]. It has been reported [57] that the aqueous and ethanolic extracts of *B. aegyptiaca* fruit induce a significant reduction in the levels of serum glucose, total lipids, total cholesterol, triglycerides, transaminases (AST, ALT) and γ GT activities in diabetic rats.

The significant depression of TAC level observed in sera of infected animals (G1, G2, and G3) may be attributed to the oxidative stress developed by *H. contortus*. Pivoto *et al.* [58] determined the total oxidation status and antioxidant capacity of lambs infected with *H. contortus* and reported increased oxidative stress in lambs' sera. On the other hand, increase in levels of Ferric Reducing Ability of Plasma (FRAP), an indicator of TAC in sera of lambs, was observed during infection with *H. contortus* [59]. The authors suggested that TAC increased to minimize the effect of reactive oxygen species (ROS) produced during infection with *H. contortus*. TAC was increased in G4 animals from the 3rd week to the end of the experiment indicating improvement by BAF-EE.

Serum minerals are involved in various fundamental physiological processes. In the present experiment, the decrease in serum Ca level of infected goats may be related to indigestion of Ca in the abomasa. Radostits *et al.* [50] stated that presence of *H. contortus* in the abomasum seems to interfere with digestibility of Ca. As most serum Ca is bound to plasma protein; the resulted hypocalcaemia may also be related to the reported hypoproteinemia. Albadawi [5], Rashid [8] and Hosseini *et al.* [45] also reported a significant decline in Ca concentration during *H. contortus* infection in sheep. Other examined serum electrolytes didn't show significant changes during the experiment. Level of TIBC showed no significant variations among or within experimental groups, a result that is in agreement with Kozat *et al.* [60] in ewes infected with gastrointestinal parasites and suffering from anemia.

Host reacts to parasites by a number of ways including ROS production, which contributes in killing or expelling parasites from their host

[61,62]. The host may be exposed to oxidative stress during this defense mechanism, which is induced by imbalance between the production and removal of ROS within the organism causing damage to biomolecules. Such damage may either be due to depletion of antioxidant defense or increase in ROS production, or both [63]. In the present experiment, the activity of GPx in the abomasal and hepatic tissues of infected- non-treated (G1) and infected- Albendazole-treated (G3) goats was lowered which is in accordance with the findings of Rashid⁸ in lambs infected with *H. contortus*. Similar findings have also been reported by Kolodziejczyk *et al.* [53] and Heidarpour *et al.* [64] in rat infected with *F. hepatica* and camel infected with *E. granulosus*, respectively. In contrast to this, some researchers reported increased activity of GPx in cattle infected with *E. granulosus* [65] and *T. saginata* [66], and in sheep infected with *F. hepatica* [67] and distomatosis [68]. On the other hand, no significant differences were observed in sheep infected with *E. granulosus* [69] and rat infected with *F. hepatica* [70]. The above differences in GPx activity could be related to the species and pathogenicity of the parasite and species of the animal. On the contrary to G1 and G3 animals, G2 (infected- BAF-EE-treated) and G4 (BAF-EE-treated only) goats revealed significant elevation in GPx activity which could be attributed to the BAF-EE antioxidant effect [9,71]. Antioxidant enzymes superoxide dismutase and catalase were elevated in mice treated with different parts of *B. aegyptiaca* extracts [72]. Phenolic and flavonoid contents of *B. aegyptiaca* were found to be responsible for its antioxidant effect [9,73].

The oxidation products are being used as biomarkers to monitor the irreversible consequences of oxidative stress in animals. Determination of MDA allows detection of the degree of lipid peroxidation and the level of free oxygen radicals indirectly [74]. In the present experiment, MDA followed a reverse course to that of GPx (high in tissues of G1 and G3 goats and low in G2 and G4 goats). Similarly, it has been reported that lipid peroxidation is increased during helminth infections in a number of studies [8,64,69,70,75]. According to Ezzat *et al.* [14], ethyl acetate extract from *B. aegyptiaca* has a defensive effect against oxidative stress induced by streptozocine with reduction in MDA levels. It also prevents lipid oxidation in food thus inhibiting many diseases as cancer and atherosclerosis [76].

In the current study, the presence of blood tinged, dark red fluid contents in the abomasa of infected animals indicating abomasal mucosal hemorrhages due the sucking habits of *H. contortus* parasite. These findings were fully agreed with the findings reported earlier by Pérez et al. [77], Tehrani et al. [78] and Saminathan et al. [79].

Histopathological changes noticed in the abomasa of the present goats included mucosal and submucosal hemorrhages and cellular infiltration with inflammatory cells. These findings were obvious in the infected-untreated group, followed by both infected-treated groups. Differences may be due to difference in the number of adult worms found in the abomasa of different groups. The present observations are in line with those of Tehrani et al.⁷⁸ in sheep and, Al-Malki [11] and Dutta et al. [80] in goats. Histopathological changes seen in the liver were similar to the findings of Dutta et al. [80] and Darzi et al. [81]. The reaction of the liver in the infected-BAF-EE treated goats was restricted to vacuolization of hepatocytes with histologically normal portal triad which may be due to the hepatoprotective effect of *B. aegyptiaca* extract as described by Chothani and Vaghasiya [13], El-Masry et al. [82] and Yadav and Panghal [83].

The observed eosinophilic infiltration is supported with the observation of Tehrani et al. [78]. Eosinophils are responsible for parasitic infection pathogenesis, and are considered as the host body's first line of defense against the parasite [10]. In addition, ovine gastrointestinal nematode infections produce a series of factors that encourage the migration of eosinophils [84].

Conclusion

Results of the present study did not show any abnormal behavioral changes or evidence of toxicity during or after treatment with BAF-EE. Also, BAF-EE has hepatoprotective effect and relieves serum biochemical alterations, oxidative stress in tissue and pathological changes induced by *H. contortus* in goats due to its wide range of biological constituents. The present findings justify the use of this plant as a novel safe natural anthelmintic agent.

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Compliance with ethical standards

The required ethical approval certificate revealing registration number of 16229 was obtained before conduction of the experiment from the Medical Research Ethics Committee of National Research Centre, Egypt.

Conflict of interest

The authors declare no conflict of interests.

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التأثير العلاجي للمستخلص الكحولي الإيثيلي لفاكهة بلح الصحراء في الماعز المصري البلدي المصاب تجريبياً بالهيمونكس كونتوريس: دراسات كيميائية حيوية لمصل الدم، علامات الاجهاد التأكسدي والتغيرات الباثولوجية

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تهدف الدراسة الى تقييم الأثر العلاجي للمستخلص الكحولي لبلح الصحراء في الماعز المصابة تجريبياً بالهيمونكس كونتوريس فيما يتعلق بالدراسات الكيميائية الحيوية بمصل الدم والاجهاد التأكسدي بالأنسجة والتغيرات الباثولوجية . تم تقسيم عدد ١٢ ماعز الى ٤ مجموعات متساوية حيث تم اجراء العدوى على ثلاث مجموعات تجريبياً: **المجموعة ١** معدية - غير معالجة، **المجموعة ٢** معدية ومُعالجة بالمستخلص، **المجموعة ٣** معدية وعولجت بالبيندازول و **المجموعة ٤** غير معدية ومعالجة بالمستخلص. تم سحب عينات دم في بداية التجربة (قبل العدوى)، الأسبوع الثالث للعدوى، ثم كل أسبوعين لمدة ٩ أسابيع. في نهاية التجربة تم ذبح جميع الحيوانات لقياس الاجهاد التأكسدي ودراسة التغيرات الباثولوجية في أنسجة الأنفحة والكبد. أظهرت نتائج التحليل البيوكيميائية لمصل الدم في الحيوانات المصابة انخفاضاً معنوياً في مستويات البروتين الكلي، الألبومين، نسبة الألبومين / الجلوبيولين، مستويات السعة الكلية لمضادات الأوكسدة والكالسيوم مقارنةً بالماعز غير المصابة. لوحظ أيضاً ارتفاعاً معنوياً في نشاط انزيمات أسبرتيت أمينو ترانسفيريز، ألانين أمينو ترانسفيريز، جاما جلوتاميل ترانسفيريز وكذلك مستويات الكوليسترول، اليوريا، والكرياتينين للحيوانات المصابة. في حين لم تسجل أي اختلافات كبيرة في مستويات الفسفور غير العضوي، الصوديوم، البوتاسيوم، الكلوريد والسعة الكلية لربط الحديد. أما فيما يتعلق بتقييم الاجهاد التأكسدي بأنسجة أنفحة وكبد المجموعات التجريبية المختلفة فقد لوحظ انخفاضاً كبيراً في نشاط انزيم الجلوتاثيون بيروكسيديز في الماعز بالمجموعتين الأولى والثالثة مقارنة مع تلك في المجموعتين الثانية والرابعة، بينما اتبع المألونداهدايد مساراً عكسياً. أظهرت الدراسات الهستوباثولوجية التي أجريت على أنفحة الحيوانات المصابة غير المعالجة التهاب، تنخر، نزيف وتسلل خلوي حاد. أظهر الكبد تضخم في القناة الصفراوية، انحطاط في الخلايا الكبدية وتسلل الخلايا ولقد كانت التغيرات الهستوباثولوجية في أنسجة الحيوانات المعالجة أقل كثافة من تلك المصابة ولم تتلقى علاج. أظهرت نتائج التجربة الحالية أن المستخلص الكحولي الإيثيلي لبلح الصحراء له قدرة على حماية الكبد فضلاً على أنه يحسن التغيرات الكيميائية الحيوية في مصل الدم والاجهاد التأكسدي بالأنسجة في الماعز المصابة بالهيمونكس كونتوريس. ولذلك ترشح هذه الدراسة استخدام هذا النبات كمضاد طفيلي طبيعي وأمن.