Clinico-Pathological Studies on Fascioliasis among Slaughtered Sheep in Kaliobia Governorate Abattoirs

Amira, M.M. Metwally; Enas, A.H. Farag; Zeinab, M. Labib and El-Bealawy, M.A.
Benha Provincial Laboratory, Animal Health Research Institute, Agriculture Research Center.

Abstract

The present study was conducted on a total number of 298 native bread sheep of different age and sex which slaughtered in different Kaliobia Governorate abattoirs during period from September 2007 to March 2008 to study the prevalence of fascioliasis in sheep. Haematological and biochemical changes in blood serum of infected sheep with histopathological changes of the liver were studied. The prevalence rate of fascioliasis in Kaliobia Governorate was 9.39% with high incedence in Benha 14.5% then Shibin El Kanater 11.5% while the lowest level was found in Kaleub 5.35%. Clinically, the infested sheep showed diarrhea, emaciation dehydration, anemia and pale mucus membrane, together with submandibular edema and shedding of wool. The hematological examination revealed significant decreases of red blood cell counts (RBCs), hemoglobin concentration (Hb) and packed cell volume (PCV). Leukocytosis associated with eosinophilia and neutrophilia were recorded. Biochemical changes showed significant increases in enzymatic activities of serum Aspartate aminotransferase (AST), Alanine aminotransferase (ALT), serum alkaline phosphatase (AP), gamma glutamyl transferase (GGT), total bilirubin, urea and creatinine. In contrast, there was significant hypoglycemia, hypoproteinemia, hypoalbuminemea. However a significant increases in α and β globuline were observed. The gross findings of the liver showed grayish white foci together with reddish irregular patches on the hepatic surface. Great thickening of the wall of bile ducts was found in some examined liver. The microscopic examination of the liver demonstrated moderate congestion of hepatic blood vessels together with vacuolar and hydropic degeneration of the hepatic cells. Necrotic areas surrounded by inflammatory reaction were also observed. In addition, metaplasia, hyperplasia and desquamation of the bile duct epithelium with extensive fibrous connective tissue proliferation and monolobular cirrhosis were also seen.

Therefore, the role of veterinary extension service is very important to advise the farmers how to maintain their animals free from fasciolia infection and to be a ware about the disease and the size of the problem.

Introduction

Liver fluke disease or fascioliasis is an important worldwide parasitic disease that affects cattle, sheep, goats, buffaloes, wild animals and man **(32)**. Fascioliasis exists in all countries where the suitable environments for the intermediate snails prevail. This disease caused significant economic losses, great expenses with antihelmintics, condemination of parasitized liver, production loss due to mortality, lower production of meat, milk and wool, reduced weight gain and impaired fertility, also impeding the selection of animals **(11, 32, 33 and 36)**.

According to Egyptian Academy of Scientific Research and Technology (1990), losses due to fascioliasis were estimated at L.E.190 million per year in Egypt. Fascioliasis in Egypt cause not easily observed indirect economic losses among ruminants, as the infestation runs in subclinical long stating course with severe degree of morbidity.

Chronic fascioliasis is the more common form, in which adult flukes were found in the bile ducts of the liver of cattle and sheep **(30 and 36)**.The clinical signs of the chronic form are manifested by shedding of the wool, bottle jaw, pale mucous membrane, emaciation and watery diarrhea **(34)**. Animal infested with liver flukes -suffered from bile duct fibrosis, thickening, calcification and liver cirrhosis. Estimation of blood parameters in fascioliasis characterized by anemia, decreases in red blood cells counts and haemoglobin concentration with increases in the number of white blood cells especially eosinophils. Infected animal with Liver fluke was suffered from impaired renal function causing chronic renal failure with hematouria and mild proteinuria **(32)**.

The present study was aimed to evaluate the incidence of fascioliasis in Kalicbia Governorate abattoirs and determine both clinico-pathological and pathological changes among naturally infected sheep.

Materials and Methods

1-Animals:-

A total 298 native breed sheep of different age and sex were examined from different localities in Kaleubia Governorate abattoirs during the period from September 2007 to March 2008.

2- Samples:-

A-Faecal Samples:-

Faecal samples were collected directly from the rectum of all examined diseased and healtny animal for identification of fasciola eggs. The faecal samples were examined traditionally by concentration floatation technique according to **(47)** and the free animals were kept as control.

B-Blood samples:-

Three blood samples were collected; the first one was placed in clean dry tubes containing EDTA for determination of erythrocytic count, haemoglobin concentration and packed cell volume according to **(25)**. The second one was collected on sodium floride to determine glucose level according to **(49)**. While the third was collected in plane tube for serum separation to determine Alanine aminotransferase and Aspartate aminotransferase according to **(43)** and alkaline phosphatase was determined according to **(5)**. In addition gamma glutamyl transpeptidase was estimated by using Calorimetric method according to **(42)** and total proteins were estimated by biuret according to **(13)**. Electrophoretic pattern of serum electrophoresis (SPE) kits and was quantitated by a densitometer **(12)**. Albumin/globulin (A/G ratio) was calculated. Serum bilirubin was estimated according to **(46)**. Moreover blood urea nitrogen was estimated according to **(23)** and serum creatinine was determined according to **(4)**.

C-Tissue Samples:-

The liver of the slaughtered sheep animals were examined macroscopically and recorded all abnormalities. Tissues specimens from liver were fixed in 10% neutral buffered formalin. The paraffin sections were 3-5 µm thickens were prepared and then stained by Hematoxylin and Eosin stain and examined microscopically for histopathological changes according to **(2)**.

Statistical analysis:-

Statistical analysis of the obtained data was done by means of Soft Ware Computer Program (48).

Results

The results of slaughtered sheep examination (Table, 1) showed that the incidence of fascioliasis at Kaliobia Governorate was 9.39% with the highest incidence in Benha region (14.5%) then Shibin El Kanatar (11.5%), Tokh (10.8%), Shubra (10%), El Kanatar (6.o6%) and Kaleub (5.35%) (Table, 1). The infected sheep clinically showed diarrhea, emaciation, dehydration, anemia with pale mucus membrane, together with submandibular edema (bottle jaw) and shedding of wool.

The hematological examination revealed that there were a significant decreases in erythrocytic count, hemoglobin concentration (Hb) and PCV while there was leukocytosis associated with eosinophilia and neutrophilia (Table, 2).

The results of seum biochemical analysis showed a significant increases in enzymatic activities of Aspartate aminotransferase (AST), Alanine aminotransferase (ALT), alkaline phosphatase (AP),gama glutamyl transferase (GGT) with a significant increase in total bilirubin ,urea and creatinine, with significant decrease of glucose (Table,3). Moreover a significant decreases in total protein, albumin with a significant increase in globulin fraction, α and β globulin fractions were also recorded (Table, 4).

Regarding to histopathological finding of Fascioliasis, the macroscopic examination of the liver revealed multiple areas of migrating tracts in the form of grayish white foci and streak scattered on the hepatic surfaces. Some cases showed reddish irregular patches which oozed blood freely on cut section. In other cases the examined liver were pale in color , firm in consistency, smaller in size and showed thickened capsule with great thickening of the main bile ducts which appeared as twisted cords on the hepatic surfaces (Fig. 1).The cut sections of these bile ducts revealed flukes mixed with inspissated bile and debris. The microscopic examination of the liver in some cases showed moderate congestion of central veins, hepatic sinusoids and portal blood vessels. In other cases single and multiple migrating tracts formed from central necrotic areas infiltrated and surrounded by inflammatory reaction, consisted of eosinophils,

lymphocytes, macrophages and multinucleated giant cells were prevalent (Fig.2 and 3). Some of these migrating tracts contained immature flocks and other were filled with extravasated erythrocytes and golden brown granules of hemosiderin and enclosed with connective tissue capsule (Fig.4). In addition to the forementioned lesions, the hepatocytes were suffered from degenerative changes represented by hydropic and vacuolar degeneration. Moreover, some bile ducts showed mild hyperplasia of their lining epithelium (Fig.5). The histopathological examination of the bile ducts revealed longitudinal section of the worm in the form of gut structure in the center and cuticle with irregularly spaced hooks indicating fasciola species (Fig.6 and 7). The presence of such fluckes was accompanied with chronic cholangitis and cholangiohepatitis evidenced by metaplasia, hyperplasia and desquamation of the bile ductular epithelium with extensive fibrous connective tissues proliferation and inflammatory cellular aggregation around these bile ducts (Fig.8). In addition, other bile ducts showed hyperplasia of their lining epithelium with periductular fibrosis that gave positive reaction with Crossman's trichrome stains (Fig. 9 and 10). This chronic inflammatory reaction was extended to the adjacent hepatic parenchyma causing pressure atrophy with loss of lobular architecture (Fig.11). Monolobular cirrhosis represented by fibrous connective tissues proliferation infiltrated with mononuclear inflammatory cells enclosed separated compressed hepatocytes with irregular sinusoids and absence of central veins was predominant (Fig.12). Multiple areas of inflammatory cellular infiltration of the hepatic tissues mainly eosinophils, lymphocytes, macrophages and plasma cells were detected. Formation of non functional bile ductules with severe inflammatory cellular infiltration of the portal areas mainly mononuclear cells were also observed (Fig.13). Focal areas of necrosis enclosed with chronic inflammatory cells and connective tissue capsules were also observed in some examined liver (Fig.14).

Table (1): Incidence of fascioliasis in slaughtered sheep at different abattoris at Kaliobia Governorate.

Abattoirs at Kaliobia Governorate	No.of examined animals	No. of infested animals	% of infestation
Benha	48	7	14.5
Tokh	46	5	10.8
Kaleub	56	3	5.35
Shibin elkanater	52	6	11.5
Elkanater	66	4	6.06
Shubra	30	3	10
Total	298	28	9.39

Table (2): Haematological analysis of blood of slaughtered sheepnon infected and infected with fascioliasis in different abattoirs of Kaliobia Governorate.

Parameters	*Control*	*Benha*	*Tokh*	*kaleub*	*Shibin*	*Elkanater*	*Shoubra*	*LSD*at P≤0.05
RBCs (cell.x10^6)	9.53^a ±0.09	6.63^e ±0.09	7.03^d ±0.09	8.03^b ±0.09	7.53^c ±0.09	7.83^b ±0.09	7.19^d ±0.08	0.00*
Hb g/dl	8.63^a ±0.09	5.53^f ±0.09	6.06^e ±0.10	7.14^{bc} ±0.09	6.63^d ±0.09	7.23^b ±0.09	6.93^c ±0.09	0.03*
PCV %	31.5^a ±0.96	23.5^c ±0.96	24.5^c ±0.96	28.5^b ±0.96	25.5^c ±0.96	28.5^b ±0.96	24.9^c ±0.87	3.00*
Total WBCs (cell.x10^3)	8.69^f ±0.07	11.80^a ±0.08	11.50^b ±0.08	8.90^{ef} ±0.10	10.55^d ±0.10	9.05^e ±0.10	11.15^c ±0.11	0.30*
Neutrophils 10^3 / ul	3.65^f ±0.04	8.05^a ±0.10	7.65^b ±0.10	4.93^e ±0.09	6.34^d ±0.10	4.71^e ±0.10	7.09^c ±0.11	0.40*
Lymphocytes 10^3 / ul	4.36^a ±0.11	2.50^e ±0.02	2.79^{de} ±0.01	3.40^b ±0.10	2.91^{cd} ±0.01	3.30^b ±0.08	2.95^c ±0.10	0.26*
Eosinophils 10^3 / ul	0.25^f ±0.01	0.54^a ±0.01	0.51^b ±0.01	0.27^e ±0.01	0.32^d ±0.01	0.36^c ±0.01	0.29^e ±0/01	0.03*
Monocytes 10^3 / ul	0.43^f ±0.01	0.73^c ±0.01	0.67^d ±0.01	0.54^e ±0.01	0.89^a ±0.01	0.68^d ±0.01	0.82^b ±0.01	0.04*

(*): Significance at P≤0.05

LSD: Least significance difference among means at P≤0.05

Means with different alphabetical superscripts in the same row are significantly different at P≤0.05

Table (3): Serum biochemical analysis of sheep non infected and infected with fascioliasis in different abatoris of Kaliobia Governorate.

Parameters	Control	Benha	Tokh	Kaleub	Shibin	El kanatar	Shoubra	LSD at P≤0.05
ALT ug /dl	$33.2^{a}\pm 0.50$	$55.35^{e}\pm 0.58$	$50.55^{d}\pm 0.44$	$46.40^{c}\pm 0.36$	$50.25^{d}\pm 0.50$	$40.95^{b}\pm 0.64$	$49.95^{d}\pm 0.52$	2.25*
AST ug / dl	$61.41^{f}\pm 0.42$	$187.05^{a}\pm 0.91$	$125.35^{c}\pm 0.67$	$85.05^{e}\pm 0.50$	$123.3^{c}\pm 0.73$	$87.65^{d}\pm 1.09$	$127.8^{b}\pm 0.88$	2.45*
AP ug / dl	$45.70^{f}\pm 0.87$	$104.0^{a}\pm 0.82$	$92.1^{b}\pm 0.74$	$70.3^{d}\pm 0.70$	$86.5^{c}\pm 0.96$	$59.5^{e}\pm 0.96$	$87.9^{c}\pm 0.80$	4.20*
GGT ug / dl	$33.19^{f}\pm 0.83$	$71.50^{a}\pm 0.96$	$55.5^{b}\pm 0.96$	$38.7^{e}\pm 1.06$	$45.5^{d}\pm 0.96$	$40.5^{e}\pm 0.96$	$50.4^{c}\pm 1.01$	4.90*
Creatinine mg/dl	$0.55^{g}\pm 0.01$	$1.01^{a}\pm 0.01$	$0.70^{d}\pm 0.01$	$0.06^{f}\pm 0.01$	$0.80^{c}\pm 0.00$	$0.64^{e}\pm 0.01$	$0.85^{b}\pm 0.01$	0.03*
B.U.N mg/dl	$10.30^{d}\pm 0.14$	$14.23^{a}\pm 0.35$	$10.89^{c}\pm 0.11$	$10.85^{c}\pm 0.09$	$12.23^{b}\pm 0.09$	$10.74^{c}\pm 0.05$	$10.15^{d}\pm 0.10$	0.55*
Glucose mg/dl	$83.38^{a}\pm 0.49$	$51.97^{f}\pm 0.40$	$55.14^{e}\pm 0.82$	$70.64^{b}\pm 0.65$	$59.76^{d}\pm 0.86$	$71.02^{b}\pm 0.58$	$62.7^{c}\pm 0.65$	2.94*
Total bilirubin mg/dl	$0.42^{f}\pm 0.01$	$0.94^{a}\pm 0.01$	$0.67^{d}\pm 0.01$	$0.48^{e}\pm 0.01$	$0.80^{b}\pm 0.01$	$0.50^{e}\pm 0.01$	$0.71^{c}\pm 0.01$	0.047*

(*): Significance at P≤0.05

LSD: Least significance difference among means at P≤0.05.

Means with different alphabetical superscripts in the same row are significantly different at P≤0.05.

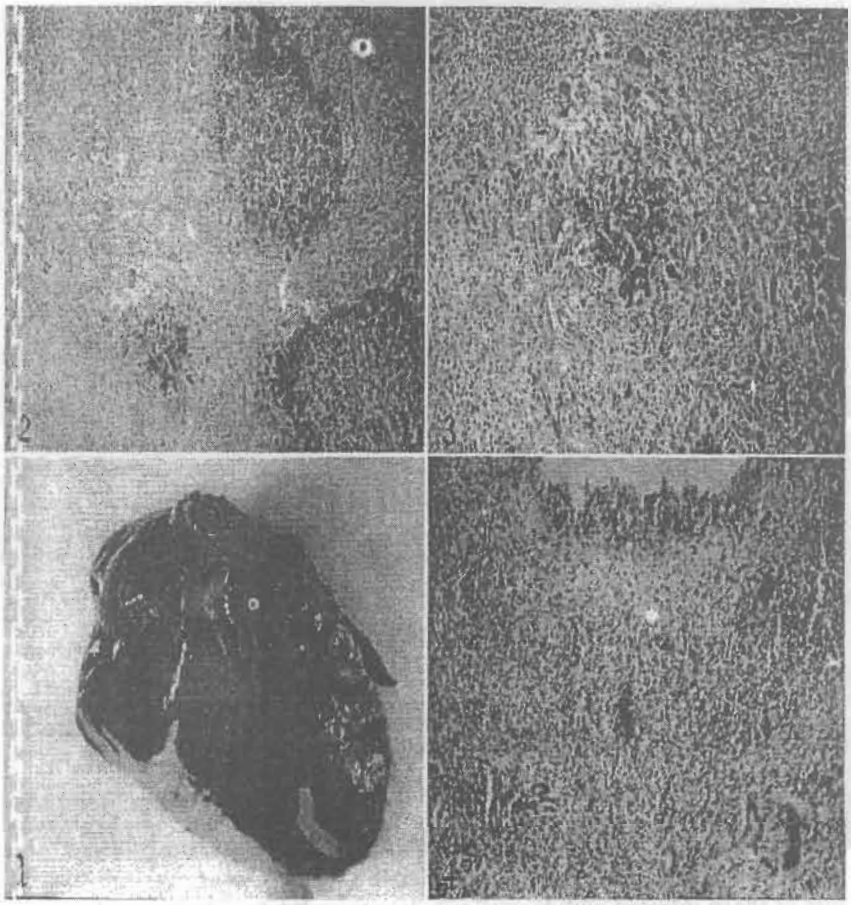
Table (4): Total protein and protein fractions in sheep non infected and infected with fascioliasis in different abatoirs of Kaliobia Governorate.

parameters	*Contrl*	*Benha*	*Tokh*	*Kaleub*	*Shibin*	*Elkanatar*	*Shoubra*	*LSD*
Total protein mg/dl	$7.08^{a} \pm 0.07$	$5.54^{e} \pm 0.09$	$5.96^{d} \pm 0.07$	$6.66^{b} \pm 0.07$	$6.32^{c} \pm 0.07$	$6.56^{b} \pm 0.07$	$6.19^{c} \pm 0.02$	0.24*
Albumin mg/dl	$4.43^{a} \pm 0.07$	$2.45^{d} \pm 0.10$	$2.99^{c} \pm 0.08$	$3.06^{b} \pm 0.10$	$3.11^{c} \pm 0.09$	$4.20^{a} \pm 0.08$	$3.11^{c} \pm 0.08$	0.244*
α globulin mg /dl	$0.91^{e} \pm 0.01$	$1.23^{a} \pm 0.01$	$1.18^{b} \pm 0.01$	$1.06^{d} \pm 0.01$	$1.12^{c} \pm 0.01$	$1.05^{d} \pm 0.01$	$1.13^{c} \pm 0.01$	0.05*
β globulin mg / dl	$0.68^{d} \pm 0.01$	$1.41^{a} \pm 0.01$	$1.32^{a} \pm 0.01$	$0.82^{c} \pm 0.01$	$1.33^{a} \pm 0.10$	$0.73^{cd} \pm 0.04$	$1.1^{b} \pm 0.03$	0.14*
γ globulin mg / dl	$0.94^{c} \pm 0.01$	$1.20^{ab} \pm 0.01$	$1.28^{a} \pm 0.10$	$1.05^{bc} \pm 0.10$	$1.13^{abc} \pm 0.11$	$0.96^{c} \pm 0.04$	$1.02^{bc} \pm 0.05$	0.23*

(*): Significance at $P \leq 0.05$

LSD: Least significance difference among means at $P \leq 0.05$

Means with different alphabetical superscripts in the same row are significantly different at $P \leq 0.05$.

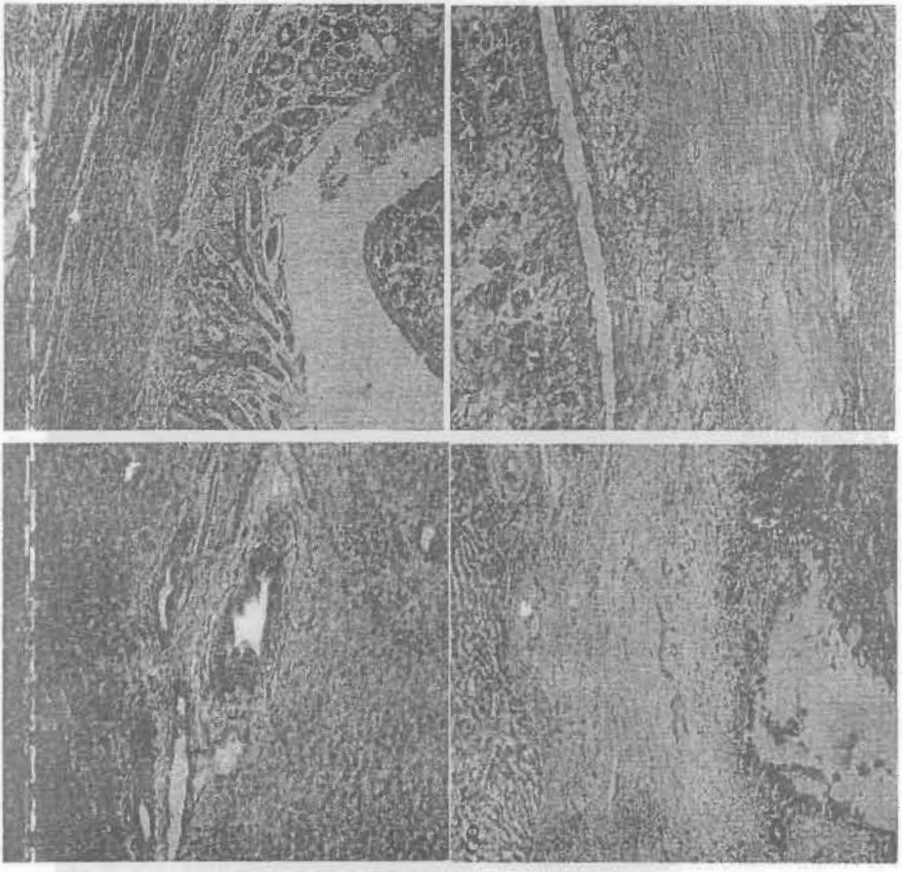


Fig(1): Liver of sheep infected with fascioliasis showing thickened capsule with twisted cords represented thickening bile ducts.

Fig(2): Liver of sheep infected with fascioliasis showing multiple parasitic tracts scattered among its parenchyma. H&E stain. X100.

Fig(3): High power of the previous figure showing parasitc tract formed from central necrotic areas enclosed with inflammatory reaction and C.T. capsules. Notice numerous gaint cells at the periphery. H&E stain. X200.

Fig(4): Liver of sheep infected with fascioliasis showing golden brown hemosiderin pigments within the migrating tract. H&E stain. X200.

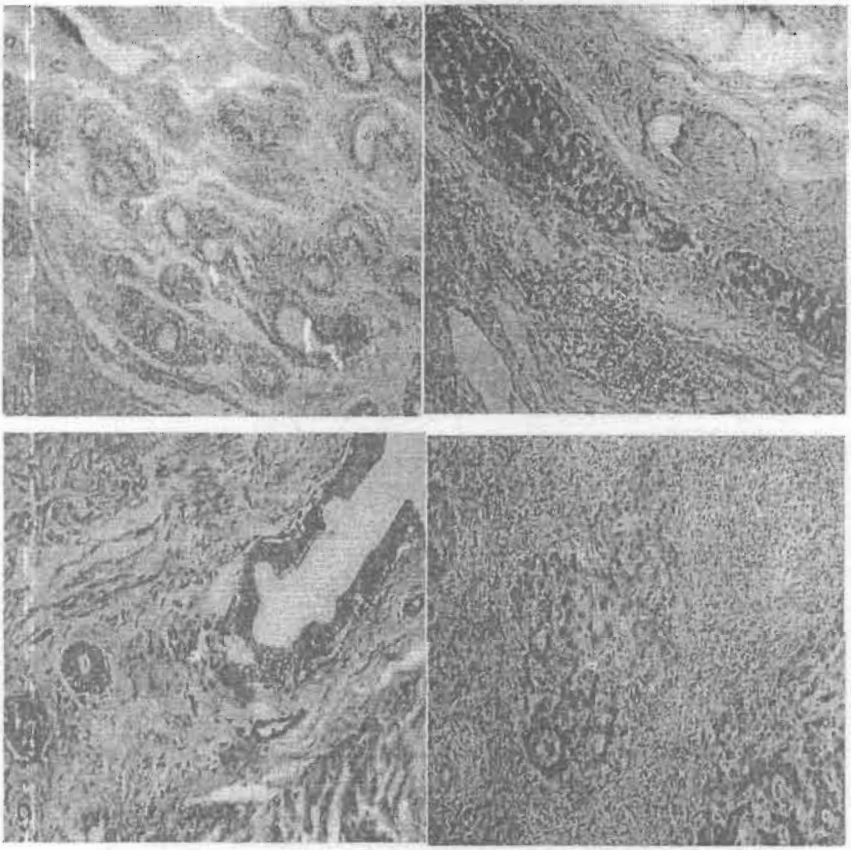


Fig(5): Liver of sheep infected with fascioliasis showing mild ductular hyperplasia. H&E stain. X200.

Fig.(6): Liver of sheep infected with fascioliasis showing longitudinal section of *Fasciola* within lumen of bile duct with chronic cholongitis. H&E stain. X100.

Fig(7): Liver of sheep infected with fascioliasis showing longitudinal section of adult *Fasciola* covered with cuticle and irregularly spaced hook. H&E stain. X100.

Fig(8): Liver of sheep infected with fascioliasis showing extensive desquamation of the bile ductular epithelium with extensive periductular fibrosis. H&E stain. X200.



Fig(9): Liver of sheep infected with fascioliasis showing hyperplasia of bile ductular epithelium with periductular fibrosis H&E stain. X400.

Fig(10): Liver of sheep infected with fascioliasis showing hyperplasia of bile duct with greenish discoloration of periductular fibrous connective tissue proliferation. Crossman's trichrome stain.X200.

Fig(11): Liver of sheep infected with fascioliasis showing loss of lobular architecture due to extensive fibrosis and mononuclear cellular infiltration H&E stain. X200.

Fig(12): Liver of sheep infected with fascioliasis showing monolobular cirrhosis with fibrous connective tissue proliferation enclosed separated hepatic lobule. Notice compression of hepatocytes with absence of central vein. H&E stain. X 200.

Fig(13): Liver of sheep infected with fasciolasis showing severe inflammatory cellular infiltration with non functional bile ductules.H&E stain.X200.

Fig(14): Liver of sheep infected with fascioliasis showing focal necrosis enclosed with chronic inflammatory cells and connective tissue capsule.H&E stain.X200.

Discussion

Fascioliasis or liver fluke disease is worldwide parasitic disease that affects cattle, sheep, goat and buffaloes, wild animals and man **(32)**.

In the present study the incidence of fascioliasis at Kaliobia Governorate abattoirs was (9.39%) with the hightest incidence in Benha (14.5%) then Shibin El kanatar (11.5%), Tokh (10.8%), Shubra (10%), El Kanatar (6.06%) and Kaleub (5.35 %).Our results are lower than that recorded by **(22) and** (45) who cited that the incidence of fascioliasis was 11.6 % and 11.3% respectively. The difference in these results may be due to snail control in some localities, difference in rearing system and method of feeding and grazing. Also the relatively lower rates of infection in our study were probably attributed to the efforts of the Veterinary authorities at Kaliobia in mass treatment of infected animals .In this investigation most of infected sheep showed clinical sings of diarrhea ,emaciation, dehydration, shedding of the wool and anemic pale mucus membrane . Few of these sheep showed icteric mucus membrane with submandibular edema (bottle jaw) .These signs came in accordance with that reported by **(1, 20, 42 and 45)**.

As shown in Table (2), the haematological examination revealed a significant decreases in erythrocytic count, haemoglobin concentration (Hb) and PCV.These results agreed with **(1, 35 and 38).**These results may be attributed to the sever anemic condition of the diseased sheep which may be due to either inability to assimilate the haemopiotic principles or the effect of toxins produced by fasciola species **(10 and 31).** In addition, leukocytosis associated with eosinophilia and neutrophilia were recorded in diseased animale when compared with normal control ones. Similar results were obtained by **(35 and 44).** Leukocytosiss may be attributed to the defence of body against fasciola destructive effects or due to toxin mediated lesions of bone marrow **(15 and 39).**

The results of serum biochemical analysis (Table,3) revealed a significant increases in enzymatic activities of serum Aspartate aminotransferase (AST),serum Alanine aminotransferase (ALT),serum alkaline phosphatase

(AP),gama glutamyl transferase (GGT) in diseased animals when compared with normal control ones. These results agreed with **(14, 19, 21, 29, 35, and 38).** These results may be due to degenerative changes and cirrhosis of liver tissues as well as obstruction of bile duct and damage to the epithelial lining bile ducts **(50)** or may be due to irritation of the liver cells by toxins or metabolic products of worms.

Also, there were significant increases in total bilirubin .These results were in agreement with those reported by **(1, 17, 37 and 44).** These may be due to hepatic and extra hepatic damage as well as disturbance in the efficiency of bile excretion. In the present study the level of serum blood urea nitrogen and creatinine were significantly increased. This finding agreed with (3 and 45) .This may be due to toxic by- product of liver fluke on kidney. Dealing with glucose levels a significant decrease in its level was found in diseased animals when compared with normal control ones .These results were agreed with **(40 and 45).** These may be attributed to the depression of gluconeogenic pathway as a result of liver damage **(22 and 40).**

Hypoproteinemia, hypoalbuminemia and hyperglobulinemia were reported in diseased sheep when compared with normal control ones (Table, 4) .The reduction of total protein were agreed with those determined by **(16, 21, 35 and 42).** The reduction of serum albumin may be attributed to damaged liver tissues or to its loss into gastrointestinal tract **(28).** Also, there were a significant increase in α and β globulins. Similar results were obtained by **(26 and 28).** These may be attributed to stimulation of reticulo-endothelial system of infected sheep **(28).**

Our haematological and biochemical results came in a harmony with the histopathological findings of examined liver .Concerning with the macroscopical findings of the liver, peticheial hemorrhage appeared as reddish irregular patches which oozed blood in cut surface in addition to multiple area of grayish white foci scattered on the hepatic surfaces were observed. These gross lesions were in agreement with those described by **(6, 7, 18 and 34).** The peticheal hemorrhage in our results may be due to the entrance of the immature worm into the liver structure and the grayish white foci which scattered on the hepatic surface in the present study may be attributed to the parasitic tacts which formed during the migration of the

larvae.Our macroscopical findings of the liver in other cases of fascioliasis showed pale in color , firm smaller in sized with thickened hepatic capsule the hepatic together with thickening of the main bile ducts .These results came in agreement with that recorded by **(14 and 30).**

The microscopical pictures of the liver in the present study showed in some cases moderate congestion of central veins, hepatic blood vessels and sinusoids together with hyropic and vacular degeneration .These results were in agreement with those obtained by **(9 and 18).**

In addition to the forementioned lesions our microscopical findings were characterized by the presence of multiple migrating tracts formed from central necrotic areas infiltrated and surrounded by inflammatory reaction. The migrating tracts in some cases were filled with erythrocytes and golden brown granules of hemosiderin and enclosed with connective tissues capsules .Our results were in a harmony with the results obtained by **(8, 18 and 30).**

These microscopical changes may be attributed to the effect of toxic by-products of migrating larvae.On the same line **(21)** explained the mechanisms of damaging of host liver during the invasion of ***Fasciola hepatica*** ,the authors mentioned that the parasites digest the hepatic tissue and cause extensive parenchyma destruction with intensive hemorrhagic lesions and immunological reaction. This mechanical liver damage could be attributed to migrating Juvenile fluke and the macerated hepatic cells were observed inside the oral sucker and pharynx of the parasites **(21).**

The microscopic finding of the examined liver of other sheep infected with fasciolisis revealed longitudinal sections of the adult worm accompanied by chronic cholangitis with extensive fibrous connective tissue proliferation causing hepatic cirrhosis. Our findings came in a complete accordance with that results maintained by **(14, 18, 30 and 36).**These lesions may be attributed to the chronic irritating effect of the adult worm occupied the lumen of the bile duct.

From our study we concluded that, the role of veterinary extension service is very important to advise the farmers how to maintain their animals free from fasciolia infection and to be a ware about the disease and the size of the problem.

References

1. **Anwar, M.A. (2000):** Some studies on the haematological and serum biochemical changes on sheep affected with liver fluke disease in Sharkia Governorate.Benha Vet. Med. J.Vol. , 11, No (1) Jun.1-9.
2. **Bancroft, J.D. and Stevens, A. (1996):** Theory and Practice of histopathological technique. 4th edition, Churchill, Livingston, Edinburgh, London Melbourne and New York.
3. **Banting,A.D.;Bouchard,C.;Barr,H.L.;Martinez,P. and Rose,F. (1975):**Changes in blood values in sheep and rabbits following experimental infection with ***Fasciolia hepatica***.Revue De Med.Vet.126(2):249-270.
4. **Barterls, S.A. (1971):** A coloremtic method for creatinine estimation. J. Clin. Chem. Acta 32, 81.
5. **Belfied, A. and Goldberg, D.M. (1971):** A Calorimetric method for determination of alkaline phosphatase activity .Enzyme 12, 561-573.
6. **Bhashara Rao, P. and Madhubala, K. (1998):** Outbreaks of fascioliasis in sheep with pulmonary involvement.Indian Vet.J.Vol.75, 183-184.
7. **Blood, D.C.; Radostits, O.M. and Handerson, J.A. (1983):** Veterinary Medicine.6th Ed .Bailliere Tindal, London.
8. **Bostelmann, S.C.W.; Luz, E.; Soccol, V.T. and Cirio, S.M. (2000):** Hepatopathological of infection by ***Fasciola hepatica*** in cattle, buffaloes and sheep.Archives of Veterinary Science 5:95-100.
9. **Clark, A.M.; Stephen, F.B. and Paterson, M.A. (1996):** Acute fascioliasis in sheep .Vet .Rec. 21, 139 (12):299.
10. **Coles, E.M. (1986):** Veterinary ClinicalPathology. 4th Ed W.B. Sounders Company .Philadelphia London Toronto.
11. **Dalton, J.P. (1999):** Epidemiology and control. In: Dalton, J. P. Fascioliasis. CABI. Puolishing, Cambridge, P.P 113-149.
12. **Danials, J.C.; Cobb, E.K. and Jones, J. (1979):** Serum electrophoretic patterns of imunoglobulins and transport proteins. J. Trauma, 14:137.
13. **Doumas, B.T.; Bayse, D.D.; Corter, R.J.; Peters, T. and Schaffer, R. (1981):** Determination of total protein.Clin.Chem.27:1942.
14. **Duff, J.P.; Maxwell, A.J. and Claxton, J.R. (1999):** Chronic and fetal fasciolasis in lambs in U.K.Veterinary Recored.145-(11):315-316.
15. **Eissa, J.M. (1998):** Concurrent fascoiliasis and samonellosis in cattle and buffaloes .4th Vet.Zagazig Congress (26-28 August) Hurghoda.
16. **El-Magdoub, A.; El-Sayed, A.; Samak, M.A.; Hassan, A.A. and El-Sawy, M.F. (1993):** Effect of ration and fasciola infection on some blood parameters of ewes.J.Egypt Soc. Parsitol. 23(3)741-749.
17. **Entessar, A,; Effat Assem El-Sheshtawy and Sahlab, A.A. (1999):** Studies on hepatic internal parasites.Beni-Sueif, Vet.Med.J. 9(2):71-77.

18. **Farha, M.A. (1993):** Pathological studies on ***Cysticericus tenuicallis*** and ***Fasciola gigantica*** in some farm animals in Sharkia Governorate. M.V.Sc.Thesis Fac.of Vet.Med.Zagazig University.
19. **Ferri,I.;Barrio,J.P.;Gonzalez-Gallego,J.andRojo-Vazquez,F.A.(1994):**Appetite depression in sheep experimentally infected with ***Fasciola hepatica*** .Vet.Parasitology,55,71-79.
20. **Fikry,H.Y.;Wafaa,M.;A;Abdel-Razek;El-Sadawy,H.A.andMenazi,A.M.(1988):**The relationship between biochemical changes and clinical signs in sheep affected with liver fluke disease .Zagazig Vet.J.Vol.Xv1,.113-122.
21. **Gajewska, A.; Smaga–Kozlowska, K. and Wisniewski, M. (2005):** Pathological changes of liver infection of ***Fasciola hepatica*** .Wiad Parazytol, 51(2):115-23.
22. **Ghazy, A.A. (1987):** Some studies on fascioliasis in sheep in Egypt. M.V.Sc.Thesis Fac .of Vet. Med. Cairo University.
23. **Henery, R. J. (1974):** Calorimetric method for determination of total protein. Clinical chemistry Harp and Row P.181.
24. **Jacobs, W.L.W. (1971):** A colorimetric assay for G glutamyl tanspeptidase. Clin. Chem. Acta, 31:175-179.
25. **Jain, N.C. (1986):** Shalm's Veterinary Haematology. 4th Ed . PP . 55-69, Lee and Fibiger Philadelphia, U.S.A.
26. **Kadhim, J.K. (1989):** Changes in serum protein values of sheep infected with ***Fasciola gigantica*** .Am. J.Vet. Res., 37(2):229-31.
27. **Kaneko, J.J. (1989):** Clinical Biochemistry of Domestic Animals. Academic Press 1 Nc Santago. New York, London, Tokyo, Toronto.
28. **Kaneko, J.J. (1997):** Clinical Biochemistry of Domestic Animals .5th Ed Academic Press, San Diego,New York,London,Tokyo.
29. **Kumar, M.; Masu, A. and Pachauzi,S.P. (1983) :** Changes in blood cellular components ,serum protein and serum enzymes activities in buffaloes and sheep infested with ***Fasciola gigantica***.Vet.Bull.53:384.
30. **Lotfy, H.S. Samira, M.Mohamed and M.A.Abdel-Gawad (2003):** Some studies on fascioliasis in Mid- Egypt. Animal Health Research Institute Agricultural Research Center, Ministry of Agriculture, Dokki, Giza, Egypt.
31. **Marcel, K.S. (1993):** Dynamics of elimination of ***Fasciola hepatica*** in a closed dairy farms.Vet.Med.J.Giza Vol.41, No 1, 43-45.
32. **Marques, S.T., Scroferneker, M.L. and Edelweiss, M.I.(2005):** Kidney pathology in cattle naturally infected by ***Fasciola hepatica***:Isreal J.Vet.Med.Vol.60(1)p1:5.
33. **Marques, S.M.T. and Scroferneker, M.L. (2003):*Fasciola hepatica*** infection in cattle and buffaloes in the state of Rio Grande DO SUL ,Prasitol.Latinoan,58:169-172.
34. **Martin, W.B. (1983):** Diseases of sheep.Black well Scientific Publication.
35. **Matanovic,K.;Severin,K.;Martinkovic,K.;Simprga,M.;Janicki,Z.andBarisic,J.(2007):** Hematological and biochemical changes in organically farmed sheep naturally infected with ***Fasciola hepatica*** .Parasitol Res.Nov.,101(6):1627-61.

36. **Mohammed,H.MovassaghGhazani;Mohammed,R.Valilou;Ali,R.;AhmedZadeh;Amir,R.and Karmi Khadijeh Zirak(2008):**The prevelance of sheep liver trematodes in the North West region Iron.Turk.J.Vet.Anim.Sci.32(4):305-307.
37. **Nabila, S.; Degheidy, M.M.and Ghazy, A.A. (1990):** Effect of fascioliasis on liver function before and after treatment in sheep. Egypt. Comp. Path. Clin.Path.Vol.3 No.1.
38. **Omran, H.H.Anwaar, M.; Abd El-Azim and El-Khalony, K. (2000):** Studies on internal parasites in farm animals in Sharkia Governorate .Egypt.J.Comp.Path.and Clin.Path.Vol.13 No1.
39. **Penny,C.D.;Low,J.E.;Scott,P.R.andHoneyman,P.C.(1996):**Bovine fascioliasis in Nigeria topical animals .Health and Production 14 (2) 121-124.
40. **Phiri, I.K.; Phiri, A.M. and Harrisonl, J. (2007):** The serum glucose and betahydroxy butyrate levels in sheep with experimental ***Fasciola hepatica*** and ***Fasciola gigantica*** infection .Vet.Parasitol.Feb.28. 143(3-4)287-93.
41. **Roadsma,H.W.;Kingsford,N.M.;Suharyanta;Spithill,T.W.andPiedrafita,D.(2006):** Host responses during experimental infection with ***Fasciola gigantica*** or ***Fasciola hepatica*** in Marino sheep 1 .Comparative immunological and plasma biochemical changes during early infection .Vet. Parasitol. Feb., 28 143(3-4):275-86.
42. **Radostitis, O.M.; Blood, D.C.and Gray, C.C. (1995):** Veterinary Medicine Atext book of diseases of cattle, sheep, pig, goat and horses.The English Language Book Society and Baillere Tindall 8th Ed 1223-1268.
43. **Reitman, S. and Frankel,S.(1957):** A Colorimetric determination of GOT and GPT activities. Am. J.Clin.Path., 28-56.
44. **Saiem, A.A.; Shawkat, M.E. and El Seify, M.A. (1990):** Effect of some drugs on egg count and blood chemistry of fasciola infested animals in Mid. Egypt .Assuit Vet.Med.J.Vol.22.No 44.
45. **Sanaa, A.A.A. (2002):** Clinicopathological studies on fasciolasis in sheep in Kalubia Province.Thesis (Ph.D.V.Sc.) Fac.of Vet.Med.Zagazig University.Benha branch.
46. **Sherlock, S. (1951):** Liver Disease .Churchill.London. p.204.
47. **Soulsby, E.J.L. (1982):** Helminthes, Arthropods and protozoa of domesticated animals.7th Ed Bailliere, Tindall and Cassell L td, London.
48. **SPSS for windows, Version: 11 (19 September, 2001)** .Copyright SPSS Inc. 1989 - 2001. All rights reserved.
49. **Tinder, P. (1969):** A Colorimetric determination of glucose. Amm. Clin. Biochem. 6:24.
50. **Urguhart, G.M.; Armaur, J.; Dunn, A.M. and Jennings, F.W. (1987):** Veterinary Parasitology. English Language Book Society / Longma.

دراسات اكلينيكية وباثولوجية في الأغنام المصابة بالدودة الكبدية في مجازر محافظة القليوبية

أميرة محمد محمود متولي - ايناس عبد الرحمن حسن فراج - زينب محمد لبيب ومعروف عابدين محمد البعلاوي

الملخص العربي

استهدفت هذه الدراسة فحص ٢٨٩ من الأغنام البلدية المحلية من مختلف الأعمار في مجازر مختلفة من محافظة القليوبية في الفترة من سبتمبر ٢٠٠٧ حتى مارس ٢٠٠٨من حيث نسبة الإصابة و التغيرات الهيماتولوجية والبيوكيميائية والهستوباثولوجية لكبد الحيوانات المصابة.

وقد اوضحت النتائج أن نسبة الإصابة في مجازر محافظة القليوبية كانت 9.39% مع نسبة عالية بمركز بنها ١٤,٥% ثم شبين القناطر ١١,٥% وأن اقل نسبة اصابة كانت في قليوب ٥,٣٥%. وكانت الأغنام تبدو هزيلة ضعيفة باهتة الأغشية المخاطية تعانى من اسهال وسقوط الصوف واستسقاء تحت الفك. بالنسبة للتغيرات الهيماتولوجية كان هناك نقص في عدد كرات الدم الحمراء وتركيز الهيموجلوبين بينما ذادت خلايا الدم البيضاء والحمضية والمتعادلة. أما التغيرات البيوكيميائية اوضحت زيادة معنوية في نشاط الإنزيمات الاسبارتيت امينوترانس فيريز والفوسفاتيز القلوي وجاما جلوتاميل ترانس فيراز بالإضافة إلي زيادة في مستوي الأصباغ المرارية واليوريا والكرياتينين . وقد كان هناك نقص معنوي في مستوي الجلوكوز والبروتينات الكلية والألبيومين مع ملاحظة زيادة معنوية في البيتا والجاما جلوبيولين. كما أوضحت الدراسة الباثولوجية وجود احتقان في الأوعية الدموية الكبدية مع وجود تنكسات مختلفة للخلايا الكبدية ومناطق بها نخر تخثري محاطة بخلايا التهابية ، كما يوجد زياده في عدد الأنسجة الطلائية المبطنة للقنوات المرارية مع وجود تليف شمل معظم القنوات المرارية مما ادى الي سمك جدارها أيضا تليف خلاياه .

ويتضح لنا مما سبق أن الجهود الطبية البيطرية لها دور هام في إعطاء النصائح والإرشادات والكشف الدوري الشامل اللازم لجمهور المربيين لوقاية حيواناتهم من الإصابة بمرض الفاشيولا.