Clinico-histopathological Studies on the Correlation Between Some Parasitic Infestation on Liver and Ovarian Efficiency in Small Ruminants

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Abstract: Seventy-five liver samples of (55 adult sheep and 20 adult goats) were collected from abattoirs at Giza governorate Egypt during the period from September 2011-May 2012. Liver samples were visually examined for some parasitic infestation as well as pathological changes. From each animal, liver, heart muscle, ovary, uterus and serum samples were taken. 26.7% of them showed parasitic infestation. With a rate, 11.8% fasciolasis (4 sheep, 5 goat), as well as 10.6 % C.tenuicollis (5 sheep, 3 goat). Histopathologically, liver tissues with fascioliasis showed 2.6 % acute hepatitis, 9% chronic catarrhal cholangio-hepatitis with hyperplasic biliary epithelium including granulome formation in 4 % of them. Biliary epithelium was greatly hyperplastic forming papillomatous projections with goblet cell hyperplasia. While liver Sections infested with C. tenuicollis revealed presence of 2.6% acute hepatitis with cyst formation, 8% chronic cholangio-hepatitis. Out of 21(26.7%) parasitically infested animals, 17(22.6%) Ovaries and their uteri showed morphological and pathological changes, including smooth inactive ovaries with atrophied uterine mucosa at five animals, as well as cystic ovaries with cystic endometritis at twelve animals. Concerning with species smooth inactive and cystic ovaries were noticed in eight (40%) of twenty goats and nine (16.3%) of 55 sheep. With Parasitized animals revealed higher values (P<?0.05) for AST, ALP, GGT and ALT activity as well as cholesterol when compared to control non infested animals. While showed a significant decrease inserum glucose level and total protiens.The present study concluded that a tight relationship is existed between hepatic and ovarian efficiency. Also the parasitic infestations in small ruminants. C. tenuicollis may be affected on the animals fertility as fascioliasis as its damage effect on liver.

Key words: Fasciolasis %C. tenuicollis %Histopathology %Serum Biochemistry %Cystic Ovary %Endometritis

INTRODUCTION

Reproductive disorders and parasitic infestation are the main problems that affecting productivity and cause great economic losses in farm animals [1,2]. Parasitic liver affections in meat-producing animals are one of major factors that reduce our national income and cause economic losses, either directly through condemnation of the pathologically affected livers, or indirectly by their effect on the animal growth and so its meat production, [3,4]. From those parasitic liver affections, fascioliasis and cysticeriosis which recently have been shown to be with widespread zoonosis throughout the world [5, 6]. Fascioliasis is an important helminthes disease caused by two trematodes, Fasciola hepatica (the common liver fluke) and Fasciola gigantica. The infective metacercariae usually migrate the liver capsule and hepatic tissue this migration usually cause direct trauma with hemorrhages, necrosis and subsequent granulation tissue end by liver cirrhosis [7]. Cestodes of the family Taeniidae that infect the dog (definitive host) and transmitting to a range of intermediate host species where they cause cysticeriosis [8]. Cysticercus tenuicollis (C. tenuicollis) is the larval stage of the canine tape warm Tania hydatigena. The cysticercoids are developed as fluid-filled cysts and commonly found attached to the omentum, mesentery, liver and peritoneum. Massive invasion and migration of the cysticercoids through the liver tissue and encysts on the peritoneal membranes of ruminants, results in acute severe traumatic hepatitis with hemorrhagic and fibrotic tracts known as hepatitis cysticeriosis [6,9, 10]. Ahmed et al. [11], Simsek et al. [12] and Khadrawy et al. [1] were studied the relationship between ovarian activity and Fascioliasis in farm animals.
They found a tight relationship between Fascioliasis in buffalo-cows and cessation of ovarian activity or repeat breeding. In the same time, Lopez et al. [13] were suggested that liver flukes somehow alter normal metabolism and balance of sex hormones in infected heifers.

Serum biochemistry of infected animals is very sensitive indicators for the degree of hepatic damage and the parasitic infestation severity, in which liver damage upsets the vital metabolic processes for normal health and optimum productivity of the animal [14].

The purpose of this study was to overview the Correlation between liver histopathology due to some parasitic infestation and its effect on hepatic and ovarian efficiency in small ruminants.

MATERIALS AND METHODS

Collection of Samples: Seventy-five liver samples (55 adult sheep and 20 adult goats) were collected randomly among liver samples of slaughtered sheep and goat in two slaughterhouses (El-Warrak and El-Monib abattoirs) at Giza governorate, Egypt during the period extended from September 2011-May 2012. In addition to, serum of twenty(20) noninfested animals taken as control for hepatic enzyme activities and Biochemical variables. From each animal samples included (liver, heart muscle, ovary, uteri and serum) were be collected.

Tissue Preparation for Histopathological Examination: Specimens from collected liver, heart muscle, ovary and uteri were immediately taken from the slaughter does and ewes and immersed in 10% formalin. The fixed specimens were trimmed, washed, dehydrated in ascending grades of alcohol, cleared in xylene and embedded in paraffin. The embedded samples were sectioned at 3-5 μm thickness, stained with H and E stain and Masson trichrome stain as special stain to collagen fibrous tissue according to Bancroft et al. [15].

Serum Analysis

Blood Samples Collection: Whole blood samples were collected from slaughter sheep, goat and the control animals. Serum was obtained by centrifugation at 3000 rpm for 20 minutes and stored in 1 ml aliquots at-20°C according to Coles [16]. With conventional laboratory techniques, sera were analyzed for estimation of hepatic enzymes levels including serum activities of Aspartate aminotransferase (AST), Alanine aminotransferase (ALT), Gama glutamyl transferase (GGT), Alkaline phosphatase (ALP), serum total protein, albumin, glucose and cholesterol using commercial test kits supplied by Spectrum Diagnostics (Cairo, Egypt) and by means of Digital VIS/Ultraviolet Spectrophotometer (Cecil instruments, Cambridge, England, Series No. 52.232).

Statistical Analysis: Statistical analysis was conducted using SPSS 16.0 for windows (SPSS, Chicago, USA) and was carried out using one-way ANOVA. Data were expressed as Mean ± SD. according to Petrie and Watson [17]

RESULTS AND DISCUSSION

Visual inspection, conventional histopathology, as well as serum levels of hepatic enzymes have assessed evaluation of hepatic damage in this study visual hepatic detection for some parasitic infestation:

In the present work, visual examination of 75 slaughtered small ruminants liver tissues revealed, the presence of 11.8% fasciolasis(4 sheep, 5 goat), as well as 10.6 % C.tenuicollis (5 sheep, 3 goat) as single infestation as shown in table (1). This rate, more than that recorded by Mohsen et al. [18], who recovered 6.28% Fasciola infestation and 6.86%. C.tenuicollis in slaughtered goats at Egypt. However, less than that recorded by Fathi and Abdel Haseeb [19] and Ahmedullah et al. [20], who recorded Fasciola in 19% and 22.5% in goats and buffaloes at Egypt and Brazil respectively. While nearly approach to that recorded by Radfar et al. [21]. Who recovered 12.87% of sheep infested with C.tenuicollis in Iran. In addition, less than that recorded by Nimbalkar et al. [22] who recorded 18.75 %, 15.17% in goats and sheep were infested with C.tenuicollis respectively in India. High incidence of fascioliasis among the totally parasitic infested goat may be referred to the grazing behaviors of animals [23].

Histopathological Examination

liver

Fasciola Infestation: Histopathological examination in the present work revealed two types of hepatitis as shown in table (2)

Acute Hepatitis: Represented in 2 Cases (2.6 %): Grossly, affected liver was enlarged with thick capsule. Hemorrhagic patches and white necrotic foci of abcessiation. On cut section numerous yellowish-white migratory tracks and the markedly dilated, thickened wall bile ducts were noticed as well as different sizes of liver flukes can be seen.
Table 1: Incidence of some parasitic infestations in visually examined small ruminants liver samples:

<table>
<thead>
<tr>
<th>Animal</th>
<th>Fasciola spp.</th>
<th>C. tenuicollis</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>NO</td>
<td>%</td>
</tr>
<tr>
<td>Sheep</td>
<td>4</td>
<td>5.3</td>
</tr>
<tr>
<td>Goat</td>
<td>5</td>
<td>6.6</td>
</tr>
<tr>
<td>Total (n=75)</td>
<td>9</td>
<td>11.8</td>
</tr>
</tbody>
</table>

*Incidence of some parasitic infestations in visually examined small ruminants (n=75) liver samples*

Table 2: Hepatic lesions due to fascioliasis in small ruminants:

<table>
<thead>
<tr>
<th>Animal</th>
<th>Acute hepatitis with abscesses</th>
<th>Chronic catarrhal cholangio-hepatitis with hyperplasic biliary epithelium</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Animal</td>
<td>(%)</td>
</tr>
<tr>
<td>Sheep</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Goat</td>
<td>0</td>
<td>5</td>
</tr>
<tr>
<td>Total (n=75)</td>
<td>2</td>
<td>7</td>
</tr>
<tr>
<td>(%)</td>
<td>2.6</td>
<td>9</td>
</tr>
</tbody>
</table>

*Hepatic lesions due to fascioliasis in small ruminants (n=75) liver samples*

Fig. 1: Liver of small ruminants showing hepatocytes ballooning degeneration and necrosis with dilated hepatic sinusoids. (H and E X 40).

Fig. 2: Liver of small ruminants showing hepatocytes ballooning degeneration with periportal vein cellular infiltration, mainly eosinophiles, macrophages and lymphocyte (H and E X 40).

Fig. 3 Liver of small ruminants showing, parenchymal destruction and cirrhosis with mononuclear cellular infiltrations (H and E X 10)

Fig. 4: Liver of small ruminants showing, hyperplasic bile glandular epithelium with goblet cell hyperplasia and mononeuclear cellular infiltrations (H and E X 40).

Fig. 5: Liver of small ruminants showing, papillomatous projections of newly formed bile ductules surrounded by thick fibrous tissue, focal and diffuse cellular infiltrations mainly lymphocytes, histocytes and eosinophiles (H and E X 10).

Fig. 6: Liver of small ruminants showing, desquamated bile epithelium, associated with beneath granulome formation (blue arrow) and diffuse cellular infiltration, mainly eosinophiles, lymphocytes, macrophages and plasma cells, (H and E X 10).

Microscopically, hepatic cords were disorganized, swollen hepatocytes with acidophilic cytoplasm and pyknotic nuclei while other hepatocytes showed coagulative necrosis, as well as dilated hepatic sinusoids that engorged with blood (Fig. 1). Also haemorrhagic migratory tracts were noticed within the necrotic hepatic cords. Portal veins were surrounded with cellular infiltration mainly eosinophiles; macrophages and lymphocyte with kupffer cells activation, in addition to hepatocytic ballooning degeneration, (Fig. 2). Bile duct in some parts showed periductal cellular infiltrations, mainly neutrophiles, lymphocyte and eosinophiles.
Fig. 7: High power of the previous figure demonstrate the inflammatory cells forming granulome mainly, eosinophiles, lymphocytes, macrophages and plasma cells, (H and E X 40).

Fig. 8a: Liver parenchyma showing fatty necrosis accompanied with cellular infiltration, mainly easinophilis, macrophages, lymphocyte and multinucleated giant cell formation (H and E X 10).

Fig. 8b: multinucleated giant cell formation (blue arrow) (H and E X 40)

Fig. 9: liver of small ruminants showing several hemorrhagic migratory tract some of them containing sections of the parasites (blue arrow) with hepatocytic necrosis, periductal fibrosis and cellular infiltrations(H and E X 10).

Chronic Catarrhal Cholangio-Hepatitis with Hyperplasic Biliary Epithelium Represented in 7 Cases (9%), Three of Them Showed Granulomatous Reaction: Gross appearance of the affected lobe was hard, pale, reduced in size with thickened capsule and in three cases (4%) of the seven cases, there were circumbried small granulomes with patchy cirrhosis. On cut section, few flukes appeared within the thickened dilated bile ducts.

Microscopically, it characterized by hepatic parenchymal destruction with increased fibrous connective tissues proliferation resulting in hepatic cirrhosis as well as mononuclear cellular infiltrations (Fig. 3). Bile ducts glandular epithelium showed hyperplastic papillomatous projections thrown into the lumen forming newly bile ductules with goblet cell hyperplasia and mononuclear cellular infiltrations. Both bile ducts and newly formed bile ductules surrounded by thick fibrous tissue, associated with focal and diffuse cellular infiltrations mainly lymphocytes, histocytes and eosinophiles (Fig. 4 and 5). In three cases of the seven, necrosis and desquamation of bile duct epithelial lining associated with eosinophilic granulome formation beneath it were noticed. This granulome characterized by central necrosis with cellular infiltration, mainly lymphocytes, macrophages, plasma cells, eosinophiles (Fig 6 and 7). In some cases it accompanied by multinucleated giant cell formation (Fig. 8a and b). Proliferation of fibroblasts that replacing the hepatic parenchyma and parts of mature fluke could be seen within the migratory tracts (Fig. 9). Collagen stained blue with Masson trichrome stain in hepatic fibrous septa and periductal fibrosis.

The gross and histopathological alterations of both types observed in this study are agreement with those described by Ghazani et al. [24], Zafra et al. [25], Sohair Badr and Eman Nasr [8] and Sanaa El-Shamy and Mariem bekhit [26]in sheep, goat and cattle. Those alterations were attributed by Gajewska et al. [27], Ruoss et al. [28], Mbaya1 et al. [29] as host tissue response against parasitic infestation and continuous mechanical irritation by the migrating fluke. As they, cause extensive parenchymatous destruction accompanied with intensive haemorrhagic lesions and immunological reactions through the mechanical invasion. On the other hand, it chemically digest hepatic tissue by enzymes and toxins that produced by the fluke as proteases, which can be responsible for negative effect on liver parenchyma and biliary epithelium. Granulomes observed in this work illustrated by Kaya et al. [30] and Mendes et al. [31] as host immune response that resulted in Fasciola eggs or dead larvae trapped in the liver parenchyma that causing granulomatous reaction.

Cysticercus Tenuicollis (C. Tenuicollis): In the present study two types of hepatitis were distinct histopathologically in hepatic tissue infested with C. Tenuicollis as shown in table (3)

Acute hepatitis with cyst formation: was found in two cases (2.6%) of C. tenuicollis infested ruminant’s livers, Grossly, multiple cysts attached to the omentum, liver and peritoneum., associated with numerous tortuous burrowing migratory tracts of C. tenuicollis larvae were seen on liver surface (Fig. 10).
Table 3: Histopathological hepatic lesions in examined small ruminant’s liver infested with *C. Tenuicollis*

<table>
<thead>
<tr>
<th>Animal</th>
<th>Acute hepatitis with cyst formation</th>
<th>Chronic Parasitic cholangio hepatitis</th>
</tr>
</thead>
<tbody>
<tr>
<td>sheep</td>
<td>1</td>
<td>4</td>
</tr>
<tr>
<td>goat</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Total</td>
<td>2</td>
<td>6</td>
</tr>
<tr>
<td>(%)</td>
<td>2.6</td>
<td>8</td>
</tr>
</tbody>
</table>

*hepatic lesions due to *C. Tenuicollis* in small ruminants (n=75) liver samples*

Fig. 10: liver of small ruminants showing multiple *C. tenuicollis* cyst (red arrow) associated with hepatic cellular necrosis and fibrosis around it (blue arrow).

Fig. 11: liver of small ruminants showing *C. tenuicollis* cyst associated with hepatic cellular necrosis, fibrosis and cellular infiltration zone around it (H and E X 4)

Fig. 12: liver of small ruminants showing several hemorrhagic migratory tracts with hepatocytic necrosis, fibrosis and cellular infiltrations (H and E X 10)

Fig. 13: Cardiac muscle showing the multiple *C. tenuicollis* cysts surrounded with mononuclear inflammatory cellular infiltrations and eosinophiles and the cardiac muscle showing degeneration and edema (H and E X 10)

Fig. 14: Uterus of small ruminants showing gland cystic dilatation (H and E X 4)

Microscopic examination revealed that one or more hepatic cysts of *C. tenuicollis* larvae, that surrounded by a thick inflammatory demarcation zone. This zone formed mainly of Kupffer cells, scanty lymphocytes and fibroblasts(Fig. 11). Hepatic cords showed disrupted with hepatocellular degeneration and necrosis especially around cysts. Hepatic veins and sinusoids were dilated engorged with blood. The migratory tracts were noticed, that filled with, serofibrinous exudates and sections of *C. tenuicollis* larvae as well as cellular inflammatory infiltrations, mainly neutrophiles, eosinophiles, macrophages and few lymphocytes, plasma cells (Fig. 12).
Chronic Cholangiohepatitis: In this work, this type was noticed in 6 cases (8%) of examined small ruminants livers.

Grossly, multiple calcified cysts attached to pale hard liver with thickened capsule and fibrosed tortuous burrowing migratory tracts of *C. tenuicollis* larvae. Microscopically, dilated hepatic veins surrounded with fibrosed migratory tracts and others contain *C. tenuicollis* larvae. Hepatocytes adjacent to those tracts appeared necrosed with patches of cirrhosis, as well as mononuclear cellular infiltrations mainly lymphocytes and macrophages. Collagen stained blue in periductal fibrosis areas with trichrome Masson stain.

Similar gross and microscopic findings were described in sheep and goat by Mohsen et al. [18], Senlik, [4], Nath et al. [32], Al-Jashamy [33], Nourani, et al [12] and Mellau et al.[5], who are referred those changes as a response to the cysticerci migration in liver parenchyma.

Cardiac Muscle: Gross, one or more whitish nodules or foci scattered on cardiac muscle. The cardiac muscle in some cases showed congestion or pale streaks. Microscopically, Cardiac muscle showed degeneration, focal necrosis and infiltration of mixed inflammatory cells, including lymphocytes, plasma cells and macrophages were associated with the presence of *C. tenuicollis* cyst (Fig. 13).

Morphological and Histopathological Changes in Ovaries and Their Uteri: In this work, as showed in (table 4), out of 21 (26.7%) parasitically infested animals, 17 (22.6%) ovaries showed morphological and pathological changes. The latter included five (2 sheep and 3 goat) showed smooth inactive ovaries and twelve (7 sheep and 5 goat) showed cystic ovaries. Concerning with species smooth inactive and cystic ovaries were noticed in eight (40%) of twenty goats and nine (16.3%) of 55 sheep from examined animal ovaries.

In this work the animals that showed bilateral smooth inactive ovaries were, emaciated exhibiting signs of ill-health associated with helminthiasis and with small size genital organs. Their uteri were atrophic with atrophic mucosa. As well as ovaries lack mature follicles and corpora lutea or its degenerative derivatives.

Noticed ovarian inactivity in which, affected is explained by Arthur et al., [34], Morrow, [35] and Tanaka et al. [36] as Parasitism induces nutritional deficiencies which result in body weight loss. Also as consequence to folliculogenesis failure due to absence or suboptimal release of gonadotrophins resulted in ovarian reduced production of ovarian steroids. Lack of cyclicity is associated with malnutrition and chronic debilitating diseases.

In the present study, animals that showed Cystic ovaries characterized, by unilateral or bilateral cyst filled with yellowish to milky white exudates. In cross section, follicles appeared with thin walls. Microscopically, Cystic follicles wall noticed formed of degenerative granulosa cells and a partially luteinised theca cell layer. While uteri of those animals showed cystic glandular dilatation (Fig. 14). Cystic ovarian disease is attributed by Arthur et al. [34], Smith, [37], Jones et al., [38], Tanaka et al. [39] as resulted of insufficient or mistimining of the luitenising hormone release as a main cause. This condition has been reported among dairy goats that graze on estrogenic pastures, as well as that showed heredity and phosphorous deficiency. While follicular cysts development were associated with progesterone and oestradiol treatment among goats. Those ovarian alterations (smooth inactive or cystic ovaries) were discussed by Kaplan, [40] as liver fluke reduces animal fertility due to their effect on hormonal balance that alter the normal metabolism of sex hormones.

Hepatic Efficiency: In the present work, The degree of hepatic dysfunction was assessed basing on measuring serum hepatic enzyme activities and Biochemical variables in, parasitically infested animals that showed ovarian pathology (n=17) comparing with control non infested sheep and goat as (n=20). Parasitized animals revealed higher values (P<0.05) for AST, ALP, GGT and ALT activity as well as cholesterol when compared to control non infested animals. While showed a significant decrease in serum glucose level and total proteins as shown in table (5).

<table>
<thead>
<tr>
<th>Table 4: Morphological and histopathological changes in examined small ruminant’s ovaries that showed parasitic infestation</th>
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<tbody>
<tr>
<td>Smooth inactive ovaries</td>
</tr>
<tr>
<td>------------------------</td>
</tr>
<tr>
<td>Animal (n=75)</td>
</tr>
<tr>
<td>Sheep (n=55)</td>
</tr>
<tr>
<td>Goat (n=20)</td>
</tr>
<tr>
<td>Total (n=75)</td>
</tr>
<tr>
<td>(%)</td>
</tr>
</tbody>
</table>

Group I (A): Control non infested sheep, n =10
Group I (B): Control non infested goat, n =10
Group II: Animals with Smooth inactive ovaries, n =5
Group III: Animals with Cystic ovaries, n=12
Table 5: Hepatic enzyme activities and Biochemical variables (Mean ± S.E) in examined small ruminant’s serum that showed parasitic infestation and ovarian pathology

<table>
<thead>
<tr>
<th>Parameter</th>
<th>AST (IU/ L)</th>
<th>ALT (IU/ L)</th>
<th>ALP (IU/ L)</th>
<th>GGT (IU/ L)</th>
<th>Glucose (mg/ dl)</th>
<th>Total protein (g/ dl)</th>
<th>Albumin (g/ dl)</th>
<th>Globulin (g/ d)</th>
<th>Cholesterol(mg/dl)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group I (A)</td>
<td>46.40 ±2.98</td>
<td>34.25 ±2.52</td>
<td>61.45 ±3.82</td>
<td>33.92 ±3.19</td>
<td>84.72 ±3.26</td>
<td>7.18 ±0.14</td>
<td>4.5x ±0.24</td>
<td>2.64 ±0.19</td>
<td>63.46 ±3.48</td>
</tr>
<tr>
<td>Group I (B)</td>
<td>32.29 ±4.57</td>
<td>26.40 ±2.53</td>
<td>56.42 ±2.68</td>
<td>33.62 ±2.98</td>
<td>31.62 ±1.09</td>
<td>6.61 ±0.75</td>
<td>2.87 ±0.30</td>
<td>3.17 ±0.59</td>
<td>63.46 ±3.48</td>
</tr>
<tr>
<td>Group II</td>
<td>86.45 ±3.22</td>
<td>43.86 ±5.42</td>
<td>89.87 ±4.16</td>
<td>49.61 ±2.97</td>
<td>67.64 ±4.14</td>
<td>6.08 ±0.36</td>
<td>2.84 ±0.10</td>
<td>3.24 ±0.12</td>
<td>90.64 ±4.53</td>
</tr>
<tr>
<td>Group III</td>
<td>65.33 ±2.50</td>
<td>38.75 ±1.98</td>
<td>72.33 ±4.36</td>
<td>56.68 ±2.93</td>
<td>73.76 ±3.48</td>
<td>6.43 ±0.16</td>
<td>3.77 ±0.15</td>
<td>2.66 ±0.09</td>
<td>71.00 ±2.84</td>
</tr>
</tbody>
</table>

Our result showed that, the activities of AST, ALT, ALP and, GGT in the serum of animals that showed parasitic infestation and ovarian pathology, a noticed significantly increases in animals of Group II (that showed smooth in active ovaries) as following, 86.45 ±3.22**, 43.86 ± 5.42*, 89.87 ± 4.16** and 49.61 ± 2.97* U/L respectively. While in in animals of Group III (that showed cystic ovaries) showed anon significant decrease comparing to animals of Group I A and B (control non infested) as following 65.33 ± 2.50* 38.75 ± 1.98*, 72.33 ± 4.36* and 56.68 ± 2.93** U/L respectively. Aspartate transaminase (AST) is a cytoplasmic and mitochondrial enzyme that catalyses the transamination of L-aspartate to oxaloacetate and glutamate and AST activity is found in almost all cells. Alanine Transaminase (ALT) is a cytoplasmic enzyme that catalyses the reversible transamination of L-alanine and 2-oxoglutarate to pyruvate and glutamate[41]. Similar result recorded by Ellah et al. [42] and Nazifi et al. [6] in sheep and goat. Those results attributed to liver damage and cirrhosis at chronic hepatitis with liver trauma from parasitic infestation.

Also in this study the concentration of glucose in infested animals with ovarian pathology were 67.64 ± 4.14** and 73.76 ± 3.48 mmol/L, respectively. Obtained values of serum glucose level revealed a significant decrease in animals of Group II and non significant decrease in animals of Group III comparing to animals of Group I A and B, this observed result may be attributed to the depression of glucogenesis pathways as arsult of liver damage and / or to the glucose consumption by the parasite [42, 6].

The concentration of total protein in the serum of animals of Group II and III was 6.08 ± 0.36* and 6.43 ± 0.16 g/L, this indicates significant hypoproteinemia, hypoalbuminemia and hypergloblinemia comparing to animals of Group I A and B. Similar results were observed by Abd Ellah, [43] and Abd-El-Salam et al. [44] who attributed this hypoproteinemia, to alteration in protein catabolism, decreased synthesis, or losses, which seen in chronic liver disease. Values of serum albumin were significantly decreased in both group II and III comparing to animals of Group I A and B. This finding was supported by histopathological examination, which revealed degeneration and necrosis of hepatocytes due to extensive fibrosis which replaced the hepatic parychyma. As, the liver is the only site of albumin synthesis, so there is a direct relation between the decrease of albumin level and the degree of liver damage. Similar results obtained in Fasciola infestation in goat and sheep respectively by Mbwaye, [45], and Ellah et al. [42].

The concentrations of cholesterol was 90.64 ± 4.53** and 71.00 ± 2.84 mmol/L, respectively. This valuss indicating a high significant increase in animals of group II and with non significant change in animals of group III comparing to animals of group I A and B. Similar results recorded by Ellah et al. [42], who reported a high significant increase in cholesterol level in sheep suffering from hepatic damage. This finding discussed by Latimer et al. [46], as Lipoproteins are synthesized in the liver; the presence of hepatic dysfunction may results in disturbance in their serum levels.

The Relation Between Liver Parasites, Hepatic and Ovarian Efficiency: In the current study, hepatic enzyme analysis for GGT and AST concentrations were significantly higher in infected especially in animals showed smooth inactive ovaries or cystic ovaries. This explained by Shaaban et al. [47]. López-Díaz et al. [15], and Melissa Paczkowski [48] as metabolic clearance rate of cyclic hormones in the liver might be impaired due to liver damage and cirrhosis. More over Picardi et al [49], suggesting that chronic hepatitis and liver trauma from parasitic infection could damage the GH and GH-receptor interactions, resulting in increased circulatory concentrations of GH and decreased concentrations of IGF-1. In addition, Spicer et al [50] and Melissa Paczkowski [48] stated that Leptin is synthesized and secreted by adipose tissue and evidence indicates that it may play a role in the hypothalamo-pituitary-gonadal axis. If adipose tissue deposition is delayed by parasitic infection, the level of leptin may not be increased to trigger the hypothalamus to stimulate the pituitary to secrete gonadotropins.
CONCLUSIONS

We conclude that:

C. A tight relationship is existed between hepatic and ovarian efficiency and parasitic infestations in small ruminants.

C. C. tenuicollis may be have the same effect on the animal fertility as fascioliasis as its damage effect on liver.

REFERENCES


