THE PATHOLOGY OBSERVED IN EXPERIMENTAL *FASCIOLA GIGANTICA* INFECTED YANKASA SHEEP IN ZARIA, NIGERIA

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Abstract

A study was conducted to determine the pathogenic effects of Fasciola gigantica infection on Yankasa sheep for a period of four months (April-July ,2010). Pathological lesions were observed in four Fasciola gigantica infected Yankasa sheep that died at the 10th, 11th and 12th week post-infection in an experimental infection at the Reproduction unit of the National Animal production Research institute, Shika-Zaria, Nigeria. The experiment involved twelve Yankasa sheep that were divided into two groups of infected and controls. The six animals in the infected group were each orally inoculated with 1200 Fasciola gigantica metacercariae and monitored for a period of 16 weeks. The pathogenic effects of the Fasciola gigantica infection began to manifest through death of four sheep among the infected group; with one death observed on the 10th week, two on the 11th and one on the 12th week post-infections respectively. The gross pathological lesions observed were hepatomegally, appearance of migratory tracts on the liver surface, appearance of puncture wounds through which protruded the anterior ends of the flukes to the liver surface as well as a fluid filled cavity with each having a fluid content of not less than 2.5 litres within the abdominal cavity. Other features observed grossly were marked distension of the gall bladder in which numerous flukes were present. The histopathological lesions were presented inform of intense hemorrhage both in the parenchyma and in the parasite tracts. There was fibrosis and distortion of the normal architecture of the hepatic cells. Observed clinical signs were inappetance, progressive anaemia and emaciation. There was a marked reduction in albumin and total plasma protein levels in the blood of the infected sheep compared to their controls. The findings of this study revealed that Fasciola gigantica is highly pathogenic to Yankasa sheep, therefore strategic control of the parasite and its intermediate host in the study area is recommended for improved sheep production.

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INTRODUCTION

Animal fasciolosis is distributed in countries with high cattle and sheep production but human and animal fasciolosis occur worldwide (Hillyer, 1999). Also, fasciolosis occurs only in areas where suitable conditions for intermediate hosts exist (Robert, 1996).

Studies on the prevalence of fasciolosis have been extensively carried out in Northern Nigeria (Schillhorn Van Veen, 1979., Ogunrinade *et. al*., 1984; Ulanyi *et. al*., 2005). Most of these studies were based on data gathered from slaughtered house records which gave a positive score citing the presence of adult worms in the bile ducts and eggs in the gall bladder. According to Schillhorn Van Veen (1979), fasciolosis can be observed in the chronic form, either in young animals during the raining season due to recently acquired infections, or in the dry season in older animals that are in a poor condition and unable to resist the effect of relatively small number of flukes.

In Nigeria, acute liver fluke infections are rarely seen in cattle but have been reported in the small ruminants (Ogunrinade *et. al*., 1984). Similarly, acute infections result from the immature flukes tunneling through the liver parenchyma with extensive tissue damage and hemorrhage that culminate in severe clinical disease with high mortality in grazing sheep in Africa (Demelash *et. al*., 2006). Therefore, this current study seeks to undertake the pathological challenges posed by this parasite on an experimental infection basis in Yankasa sheep.

MATERIALS AND METHODS

Experimental animals

Twelve (12) Yankasa ewes obtained from the Reproduction unit of the National Animal Production Research Institute, Shika-Zaria, Nigeria, between 10-12 months old were used. The animals each received concentrate feed at 300g per ewe per day (Akinbamijo *et. al*.,1993). Hay, water and salt licks were given *ad libitum*. Baseline pre-infection data were collected and the ewes were ranked on the basis of live weight and body condition scores (Ahmed *et. al*., 2003) and randomly assigned to two treatment groups.

Isolation and preservation of infective materials

Fasciola gigantica metacercariae were obtained from naturally infected *Lymanaea natalensis* snails collected at Ahmadu Bello

University Zaria dam and other small streams in Zaria environs.

Collected snails were taken to the laboratory in The Department of Parasitology, Faculty of Veterinary Medicine, Ahmadu Bello University, Zaria where they were crushed in water using petri-dishes and snail tissues removed. The swimming cercariae were viewed under a microscope and left to attach to the petri-dish; after which they were left in water in petri-dish for 3days at room temperature to become infective (Ajanusi, 1987). Just before infections, metacercariae were examined under stereomicroscope to ascertain their viability.

Animal infection

The 12 animals were randomly divided into two groups of six animals each representing infected and control group. Each of the animals in the infected group were inoculated orally with 1200 *Fasciola gigantica* metacercariae as described by (Ajanusi, 1987).

Post infection monitoring

3mls. of blood was collected in 12 bijou bottles containing EDTA from all the sheep two weeks pre-infection and at weekly intervals following infection. The packed cell volume (PCV) was determined using microhaemacrotic centrifuge technique. Total plasma protein and albumin were evaluated using autoanalyzer (Bayer clinical chemistry Analyzer, Germany). Haematological parameters were analysed using a statistical package SAS (2002).

Histopathological examination

Liver damage was assessed grossly and pictorially. Studies on the sections of the histopathological lesions of liver tissues from dead animals were determined using H & E stain in order to assess the extent of liver damage.

RESULTS

Plates I – III show livers with pin-point areas of hemorrhages on their surfaces and the presence of fistulous tracts within the liver parenchyma.

Plate I: Photograph of liver from Fasciola gigantica infected Yankasa sheep at 10th week Pi. Note a distended gall bladder (G) and areas of hemorrhages (arrows) and evidence of fistulous tracts. Ventral surface.

Plate II: Photograph of liver from Fasciola gigantica infected Yankasa sheep at 11th week Pi. Note that the liver is pale with areas of ecchymotic and pin-point hemorrhages (capillaries oozing out blood). Showing surface evidence of fistulous tracts (caused by parasite migration).

Plate III: Photograph of liver from *Fasciola gigantica* infected Yankasa sheep at 11th week Pi. Showing marked distension of the gall bladder.

The gall bladders were distended and flukes were present (Plates III and IV). The fluid content obtained from the abdominal cavity as a consequence of fluke damage was between 2.2 – 2.5 litres in each of the four animals. Mean packed cell volume (PCV) among the infected sheep dropped significantly ($P < 0.05$) from 5th week post infection (Table 1). Similarly, the mean total plasma protein had a significant ($P < 0.05$) drop in the infected group from the 2nd week post infection to the end of the experiment (Table 1). From the 7th week post infection, plasma albumin levels began to drop significantly ($P < 0.05$) (Table 1).

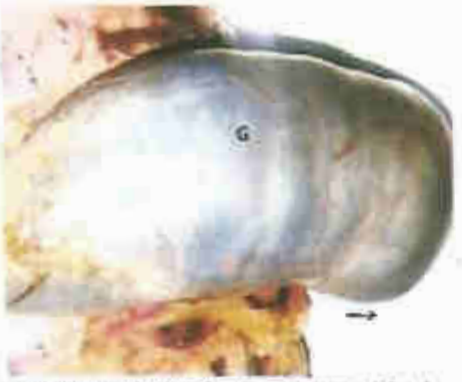

Plate IV: Photograph of liver from *Fasciola gigantica* infected Yankasa sheep at 12th week Pi. Showing enlarged hepatic lymphnode (arrow) and gall bladder (G).

PLATE V: Photomicrograph of a liver from *Fasciola gigantic* infected Yankasa sheep at 10th week Pi. Note the areas of fibrosis (F) and dead spaces (arrow). H & E stain × 400

Table 1. Packed Cell Volume (PCV), Total Protein and Albumin Levels obtained from *Fasciola gigantica* infected Yankasa sheep and their controls

	PCV		T.P		ALBUMIN	
	Infected	Control	Infected	Control	Infected	Control
P_0	35±1.88	34.7±1.76	6.25±0.13	6.56±0.30	3.17±0.27	2.98±0.18
1	34±0.70	36.7±2.05	6.27±0.14	6.68±0.26	3.10±0.41	2.93±0.24
2	32.8±0.70	32±1.52	6.0±0.21*	6.7±0.32*	3.23±0.14	3.01±0.14
3	31.6±0.71	34±1.81	5.88±0.27**	7.25±0.49**	3.20±0.18*	3.38±0.11*
4	32.5±1.20	35.5±1.6	5.6±0.26**	7.6±0.38**	2.95±0.07	2.62±0.54
5	29±0.66**	34±1.62**	5.7±0.35**	7.5±0.29**	2.37±0.05*	3.15±0.14*
6	27.8±0.94**	34±1.34**	5.58±0.22**	7.53±0.49**	2.40±0.05	3.10±0.13
7	26.6±0.76**	34.7±1.28**	5.31±0.24**	7.55±0.49**	2.52±0.10**	3.13v+.12**
8	25±0.61**	34.5±1.08**	4.91±0.39**	7.5±0.24**	2.47±0.15**	3.38±0.13**
9	22±2.46**	34.8±1.49**	4.45±0.55*	6.61±0.40*	2.30±0.04**	3.33±0.15**
10	12.8±4.00**	34.8±1.47**	4.1±0.50**	6.8±0.39**	2.17±0.06**	3.02±0.12**

Where P_0 - 2 weeks pre-infection data* level of significance ($p<0.05$)** Highly significant ($p<0.01$)

Histopathological lesions observed in *Fasciola gigantica* infected Yankasa sheep that died on the 10th, 11th and 12th week post infections are shown on Plates V-VIII. Areas of pin-point hemorrhages, fibrosis and the distortion of normal hepatic cells were seen in (Plates V and VI). It also shows cellular reactions through the infiltration of inflammatory cells. There was evidence of hemorrhage due to parasite migration and intrahepatic location of *Fasciola gigantica* as well as evidence of the distortion of the normal architecture of the hepatic cells (Plate VII). Hemorrhage and massive fibrosis were seen in (Plate VIII).

PLATE VI: Photomicrograph of liver from *Fasciola gigantic* infected Yankasa sheep at 11th week Pi. Note a generalized fibrosis of the entire liver surface (arrow). H & E stain × 400

PLATE VII: Photomicrograph of liver from *Fasciola gigantica* infected Yankasa sheep at 11th week Pi. Note the intrahepatic *Fasciola gigantica* (arrow) and hemorrhage (H). H & E stain × 400

PLATE VIII: Photomicrograph of liver from *Fasciola gigantica* infected Yankasa sheep at 12th week Pi. Note the massive hepatic fibrosis (arrow). H & E stain × 400

DISCUSSION

The gross pathological lesions observed in the infected dead animals at 10th, 11th and 12th week post-infection respectively, depicts numerous pinpoint areas of hemorrhage that

were distributed on liver surfaces and presence of fistulous tracts within the liver parenchyma. This also revealed hepatomegally and friability of the infected liver. The impact of the flukes also resulted into hemorrhage in which the fluid content obtained from the abdominal cavity as a consequence of fluke damage in the four of the dead animals was 2.2 – 3.5 litres. The fluid content obtained compares with the findings of Ajanusi (1987) where fluid content obtained was 2-3 litres of blood tinged fluid.

This current study indicates that the migration of immature flukes is responsible for traumatic hepatitis, hemorrhage and inflammatory cellular responses observed, which is in conformity with earlier reports (Ajanusi, 1987; Eguale and Abie, 2003) that the cellular response by the host contributes immensely to the destruction of the liver and the disruption of its architecture. Formation of parasite tracts made up of infiltrating inflammatory cells observed, agrees with earlier works (Sanghster, 1999; Murray, 2002; Behm). Generalised fibrosis observed is in line with earlier reports (Soulsby *et. al.*, 1982; Chauvin *et. al.*, 2001). Marked distension of the gall bladder evident in this study confirmed earlier findings (Ogunrinade *et. al.*, 1984; Eguale and Abie 2003) that chronic fasciolosis practically results from adult flukes often in pairs, lodging within bile duct causing duct wall hyperplasia, progressive occlusion and ultimate calcification of the duct wall with characteristic chronic wasting syndromes and various hypertrophies. Therefore, this study demonstrates that Yankasa sheep showed low resistance to *Fasciola gigantica* infection; as such, strategic preventive and control programmes against this parasite and its intermediate hosts in the study area are recommended for improved sheep production.

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