The Iraqi J. Vet. Med. 19 & 20 , No.(1), 1995-1996.

ARTERIOLOSCLEROSIS IN BOVINE HEPATIC TISSUE INFESTED CHRONICALLY BY FASCIOLIASIS

RAJEHA A. ABDUL RAHMAN

Dept of Pathology . , College of Vet. Med. , University of Baghdad Iraq

SUMMARY

This investigation aimed at studying the arteriolar lesions in the hepatic infestation with fascioliasis. Specimens from slaughterhouse were collected, and studied by histopathology. Sclerotic lesion was revealed in the arteriolar walls of chronic type infestation, while mild arteriolitid was only found in the acute migratory phase.

INTRODUCTION

Endarterial prpliferative reaction has been reported accompanying perivascular inflammation (Gore, 1970). Also, thickening of intimal tissue has been described in the vicinity of many chronic inflammatory processes (Smith, et. al. 1972). Several vascular lesions have been revealed in hepatic tissue infested naturally (Rahko, T. 1969) and experimentally (Urquhart, G.M. 1956) by Fasciola hepatica.

The present communication elucidates the arteriolar wall reaction in the hepatic tissue to chronic fascioliasis in cattle.

MATERIALS AND METHODS

Twenty liver specimens of chronically infested cattle and ten specimens of acute phase were collected from Shua'lla abattoir. Specimens were preserved in 10% formalin solution and routinely processed and embedded tissues were stained with hematoxylin and eosin.

RESULTS

The walls of the arterioles close to the healed tracks inflicted by flukes migration in the hepatic tissue, in addition to those to the hyperplastic bile ducts which harbour the mature flukes, showed several changes in all tunics. tunica intima revealed disrupted endothelium, while there was a remarkable increase in the number of the smooth muscle layersin media. The smooth muscle cells were hypertrophied too (Fig. 1&2). Other lesions presented projection of the proliferating subintimal tissue into the lumen of arterioles (Fig.3).. tunica adventitia which was surrounded by mononuclear infiltration showed eminent thickening due to fibroblastic hyperplasia (Fig. 1 & 2). However, inflammatory cells did not have a significant access to any of the tunics. On the other hand, the arteriolar lesion described above was not evident in the tissues subjected to acute migratory phase, rather there was mild infiltration of eosinphils mainly and lymphocytes in the tunica adventitia and tunica media (Fig. 4).

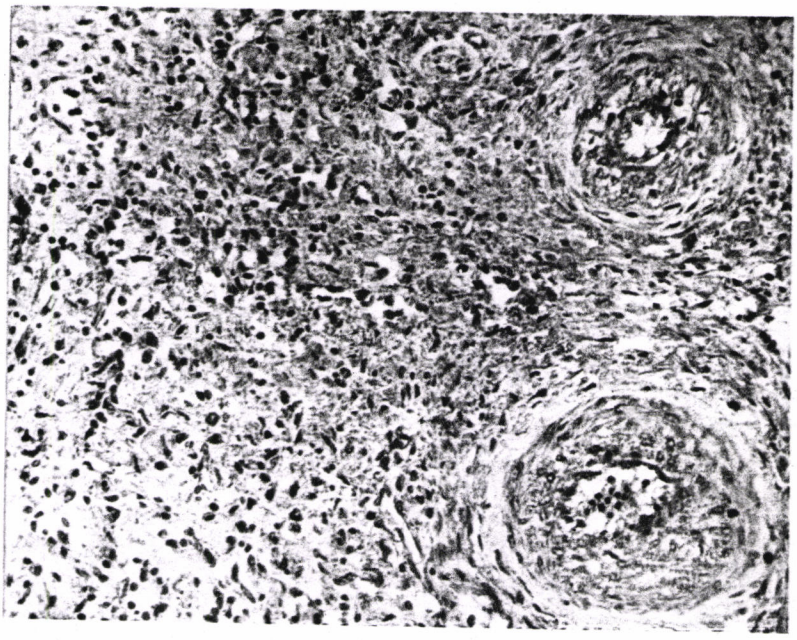

Fig.1 Illustrating two blood vessels of chronically infested livers showed narrowing lumen, disrupted endothelium, hypertrophied smooth muscle cells and thickened adventitia, surrounded by infiltration of mononuclear cells, (H & E.x200)

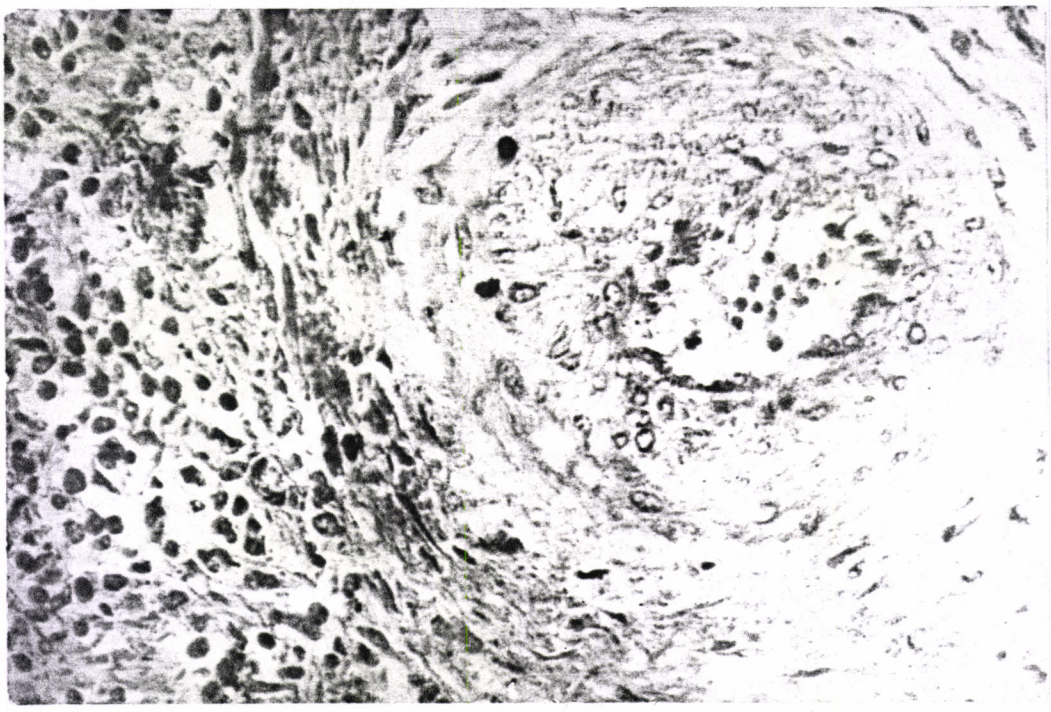

Fig. 2 Illustrating a blood vessel of chronically infested livers showed same findings as seen in the previous section. (H & E.x400).

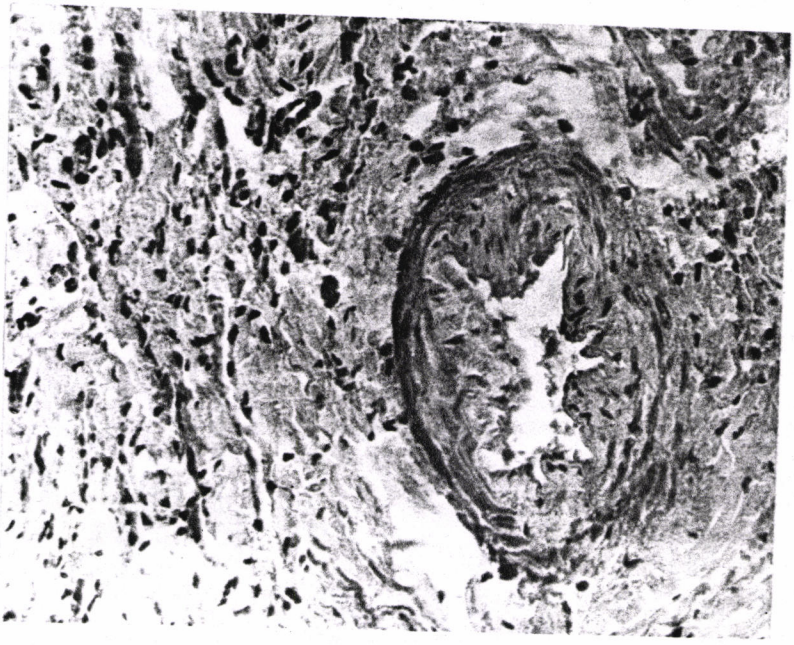

Fig. 3 Section of a blood vessel of chronically infested livers showed projection of proliferated subintimal tissue into the lumen of arteriole with narrowing of the lumen. (H & E.x400).

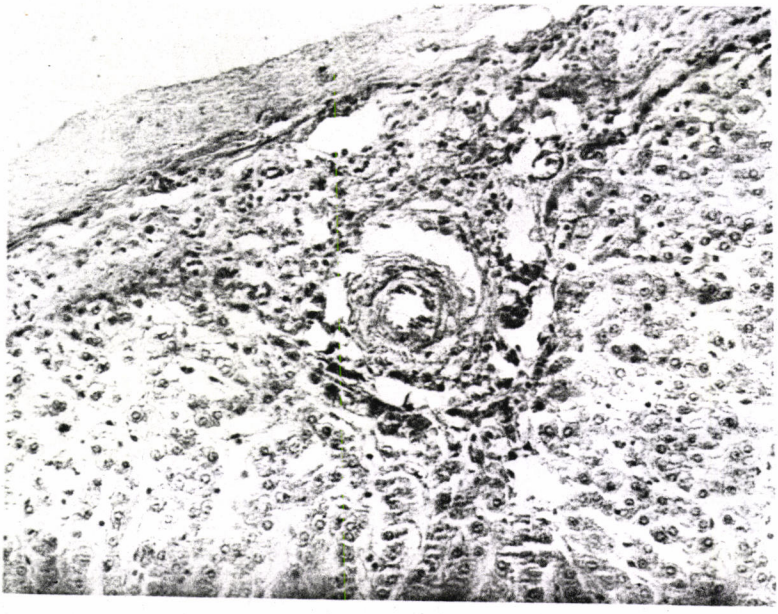

Fig. 4 Section of acutely infested liver illustrating the infiltration of tunica adventitia of subcapsular arteriole with eosinophils and lymphocytes. (H & E.x400).

DISCUSSION

Endarteritis obliterans accompanying chronic parasitic gastritis reported previously (Al-Shebeb, et. al. 1981) can not be employed in the present data. Instead, other than sclerotic changes, were not detected in the arteriolar wall of the chronically infested hepatic tissue by Fasciola flukes. Yet, in acute migratory phase arteriolilis was shown, a finding that is in agreement with those described by (Rahko, T. 1969). Endothelial disruption of the sclerotic arteriole found in this study might be brought about an endogenous mediator affecting the smooth muscle cells and/or endothelium. Supporting this explanation, is the study conducted by (Cuendoud, et. al. 1987). Together with this proposed mediator is the growth factor which could be produced by macrophage and act as an autocrine or paracrine on the proliferated smooth muscle and fibrobalstic cells (Lovette, et. al. 1986).

Proliferation of these cells could be enhanced too, by interleukin-2 produced by peripheral lymphocytes (Lovette, et. al. 1983). Macrophages and lymphocytes were intimately surrounding the sclerotic arterioles in this investigation.

thus, it might be tempting to speculate that the arteriolar proliferative changes were triggered by the mononuclear cells found in chronic lesion of fascioliasis.

REFERENCES

1- Al-Shebeb,Abdul Rahman,R.A.,and Makkawi, T. A., (1981). Pathological study of a vasculopathy accompanying chronic parasitic gastritis in equine. The Iraqi J. Vet. Med. 5: 56-62.

2- Cuenoud, H. F., Joris, I., Langer, R. S., and Majno, G. (1987). Focal arteriolar insudation. A response of arterioles to chronic non-specific irritation.Am.J. Pathol. 127: 592-406.

3- Gore, I. (1971). Inflammatory lesions of arteries In: Anderson, W. A. D. (ed.), Pathology, pp. 734. 6th edition, the C. V. Mosby Company.

4- Lovette, D. H., J. L., and Sterzel, R. B. (1983). Athymocyte-activating factor derived from glomerular mesangial cells. J. Immunol. 130: 1969-1801.

5- Lovette,D.H.,Szamel,M.Rayan,J.L.,andSterzel,R.B.,Gemsa, D.and Resch,K.(1986).Interleukin I and the glomerular mesangium.I.Purification and characterization of a mesangial cell-derived growth factor. J. Immunol. 136: 3700-3705.

6- Rahko, T. (1969). The pathology of natural Fasciola hepatica infection in cattle. Path Vet. 6: 244-256.

7- Smith, H. A. , Jones, T. C. and Hunt, R. D. (1972). Veterinary Pathology. 4th edition, Lea and Fabiger, Philadelphia.

8- Urquhart, G. M. (1956). The pathology of experimental fascioliasis in the rabbit. J. Path. Bact. 71: 301-310.

الخـــــلاصة

لقد تضمنت هذه الدراسة التركيز على التغيرات المرضية للشـريانات الموجودة في اكباد الابقار المصابة بداء ديدان الكبد، حيث تم جمع (30) عينة مرضية من مجزرة الشعلة، وقد وجد ان آفات التصلب الشرياني واضحة في حالة الاصابة المزمنة، بينما أوضحت الآفات الحادة للمرض التهاب الشريانات الصغيرة.