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Brief Communication

CEREBROSPINAL OBLITERATING ENDARTERITIS IN A PIG

Obliterating endarteritis is characterized pathomorphologically by overgrowth of cellular fibrous tissue in the intima causing concentric and symmetric intimal thickening (*Anderson & Kissane* 1977). The process in small vessels may proceed to total obliteration of the lumen. Obliterating endarteritis is observed in gastric ulcers in swine (*Nafstad* 1967) and is a feature of chronic stages of polyarteritis nodosa in several species (*Easley* 1979). Proliferative endarteritis is a frequent finding in dirofilariasis in the dog (*Smith et al.* 1972). The purpose of this communication is to report observations on a cerebrospinal angiopathy in a pig characterized by obliterating endarteritis and occasional necrotizing vasculitis.

Three out of a group of 6 fattening pigs, 3—4 months old, exhibited anorexia. One animal had mild incoordination and another animal showed tilting of the head and tended to circle in 1 direction. A third animal became recumbent and was sacrificed in a moribund state after a course of 4—5 days, while the other 2 pigs recovered. One month prior to the onset of disease symptoms, polyvalent Escherichia coli antiserum of equine origin had been administered. The following observations are based on necropsy of 1 pig.

Gross examination showed an adequate nutritional state. The lungs were congested. In the liver multiple scars due to ascarid larvae were observed.

Vascular lesions were found in medium-sized and small arteries and, less frequently, veins. The lesions, mostly concentric and cellular, consisted of proliferations from the vascular intima of fibrous tissue and endothelial cells. The lumina of the vessels were reduced in diameter, and often a total obliteration of the vessel was seen (Fig. 1). In vessels with less prominent changes, fibrinoid substance could be demonstrated subintimally and in the media. Sometimes the lumina were occluded by fibrin thrombi. In some of the arteries adventitial infiltrations of mononuclear cells, mostly lymphocytes, were observed (Fig. 2). Fibrosis of the adventitia and of the perivascular tissues was not present. Necrosis of the vessel wall with sparse mononuclear cell infil-

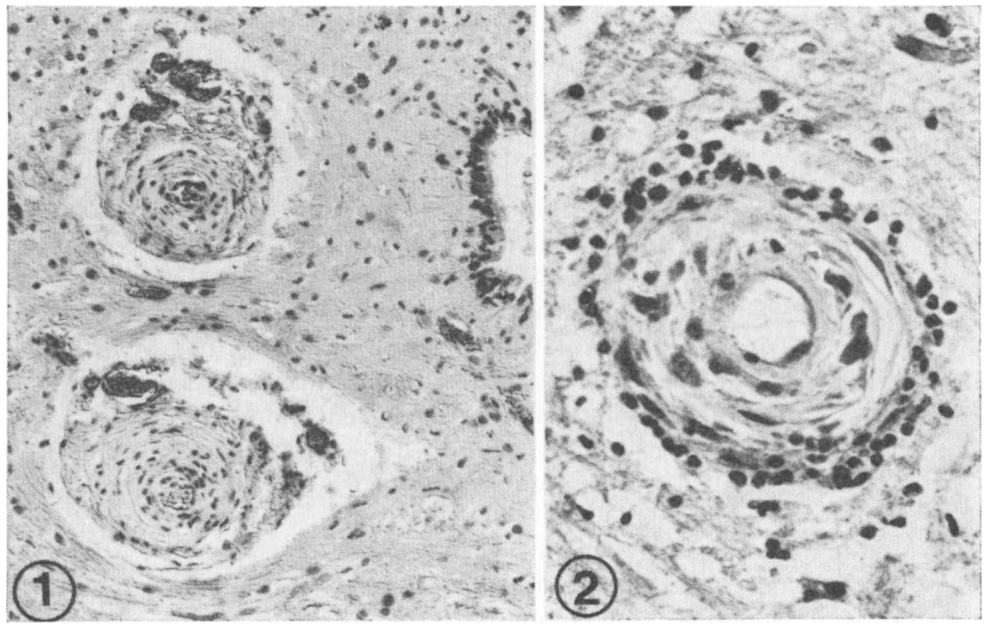

Figure 1. Two arteries exhibiting concentric thickening of the intima by fibrous tissue. The vessels are almost totally obliterated. Spinal cord. HE × 750.

Figure 2. An artery with intimal thickening of fibrous tissue, some subintimal fibrinoid substance, and adventitial infiltration of mononuclear cells. Midbrain. HE × 475.

trations, and sometimes with haemorrhage in the perivascular space, was observed in some vessels. Affected vessels were found in various organs. The mesencephalon, the pons and the medulla oblongata were most severely affected. Infrequently, vascular changes were found in the cerebral hemispheres and cerebellum. In the spinal cord vascular changes were found in the gray substance. In the interlobular and the arcuate arteries in the kidneys endothelial proliferations were seen. PAS-positive material was not demonstrated in the glomerular capillaries. Endothelial cell proliferations were also noted in the lungs.

Advanced extravascular lesions were observed in the brain. Malacic foci and infiltration of large fat-laden macrophages were demonstrated in the midbrain. Focal accumulations of mononuclear cells were seen in the cerebrum. In the spinal cord neuronal degeneration and demyelinization were found. In the liver broad perilobular strands of fibrous tissue were formed and

infiltrated with a few eosinophils. In the myocardium focal mononuclear cell infiltrations were observed. Parasites were not observed in any organ.

The lesions described in the present communication are in some aspects compatible with the necrotizing vasculitis reported by *Corner & Jericho* (1964). Vascular changes reported by *Harding* (1966) demonstrate similar distribution in the central nervous system, but different morphology, with fibrinoid degeneration as the dominant feature. The lesions also differ both in distribution and in character from acute necrotizing vasculitis resembling malignant catarrhal fever recently described (*Bratberg* 1980). The character of the observed lesions differs from both immune complex diseases and cell-mediated hypersensitivity reactions, as defined by *Robbins & Angell* (1976). Although the salient feature with respect to vascular lesions in the present case was obliterating endarteritis, the changes resemble, and might possibly be classified as, polyarteritis nodosa. Whether endotoxins may contribute to lesions of the type described remains unknown.

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