Electron Microscopy of Renal Arterial Lesion in a Pup Infected with Canine Herpes Virus

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Disseminated hemorrhage and necrosis in various organs have generally been regarded as the most prominent feature of the lesions in canine herpes virus (CHV) infection [1, 2]. The authors described, as the results obtained from pathological study on pups infected with CHV, that fibrinoid necrosis of the interlobular arteries in the kidney has an important role in the pathogenesis of typical wedgy-shaped lesions [4]. The present note describes the fine structure of renal vascular lesion in a pup with CHV infection.

Tissue blocks for electron microscopy were prepared as usual from the formol-fixed kidney specimen of a pup (pup No. 1) described in the previous paper [4]. The pup exhibited typical change of fibrinoid necrosis in the interlobular arteries and their branches.

Figure 1 illustrates the representative view of a portion of a branch of the interlobular artery having undergone fibrinoid necrosis. Only the intima and media were distinguishable and the fine structure of tissue components was obscure owing to marked degenerative changes and fixation artifact. All the endothelia and media cells contained vacuoles and phagosomes, and were completely

devastated by degrading process. Collections of fibrin occurred as irregular shaped electron opaque aggregates, which were seen lying in the cytoplasm of necrotic medial cells and to the lesser extent in the endothelia. In higher magnification picture shown in Fig. 2, a banded pattern with a periodicity of 20-30 nm, a typical fine structural characteristic of fibrin, was clear. This deposit of fibrin was a prominent finding in the necrotic vascular wall, and thus the histopathological diagnosis as "fibrinoid necrosis" was confirmed.

A noticeable finding in Fig. 3, a high-power magnification of a part of the cell shown in the upper left of Fig. 1, was the occurrence of a cluster of particles ca. 80 nm in diameter, which were considered to be virions of CHV, in the mass of cellular debris. Most of the particles were single-membrane bounded, however, some of these had dense nucleoid.

Concerning the relationship between vascular system and CHV, Hashimoto *et al.* [3] detected CHV antigen in the wall of blood vessels of the fetal liver parenchyma and maternal and fetal placental tissue in experimentally infected pregnant dogs. In addition, they found intranuclear inclusion bodies in the cells of maternal, fetal and allantoic blood vessels. The present study demonstrated

Fig. 1. Portion of a branch of interlobular artery, showing thickening of the intima (I) and media (M). Vacuolation and occurrence of electron opaque, irregularly shaped masses of fibrin (F) are conspicuous. L; Lumen $\times 3,900$.

Fig. 2. Higher magnification of fibrin deposits. Disorderly occurred masses exhibit banded pattern with regular periodicity. $\times 17,500$.

Fig. 3. High power magnification of part of a necrosed cell shown upper left of Fig. 1. Clusters of virions (V) are seen to be intermingled in cellular debris and fibrin. $\times 17,500$.

that the CHV may have played an essential role in the development of vascular lesion. Consequently, the vascular system was considered to be important as one of the possible sites of CHV replication.

It may be concluded that the muscle cells of the media of branches of the interlobular artery underwent degeneration because of CHV infection and the infiltration of serum components into the degenerated vessel wall resulted in fibrinoid necrosis.

REFERENCES

1. Carmichael, L. E., Squire, R. A., and Krook, L. 1965. *Am. J. Vet. Res.* 26: 803-814.
2. Hashimoto, A., Banba, H., Hirai, K., and Shimakura, S. 1979. *Res. Bull. Fac. Agric. Gifu Univ.* 42: 177-187.
3. Hashimoto, A., Hirai, K., Yamaguchi, T., and Fujimoto, Y. 1982. *Am. J. Vet. Res.* 43: 844-850.
4. Kojima, A., Fujinami, F., Takeshita, M., Minato, Y., Yamamura, T., Imaizumi, K., and Okaniwa, A. 1990. *Jpn. J. Vet. Sci.* 52: 145-154.