MORPHO-PATHOGENESIS OF ENCEPHALOMALACIA IN FOXES

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There was an outbreak of the neurological disease, by which about 260 silver foxes died, at a fox farm stocking about 900 silver foxes in central Hokkaido in spring 1983, 7 affected silver foxes (less than 1 week old–3 years old) were examined histopathologically.

Findings revealed scattered or disseminated ischemic nerve cell necrobioses and bilateral-symmetrical ischemic focal necroses or malacias (bilateral-symmetrical polioencephalomalacia) in the prosencephalic cortices, mesencephalic nuclei, nuclei of the posterior brain stem and cerebellar cortices.

The small and minute arterioles in the meninges overlying the necrotic or malacic foci frequently showed edematous swelling and loosening (microvascular alterations). The nerve bundles, including the vascular nerves, in the meninges of the cerebral basis and gyri only sometimes showed degeneration of axons.

Mesothelial proliferation was observed frequently in the cerebral meninges and optic nerve sheaths, and less frequently in the spinal dural sheaths ; occasional multinuclear giant cell formation of mesothelial cells was also found.

Generally, the specimens revealed slight leukomyelodegeneration, white matter degeneration in the posterior brain stem and optic nerve degeneration.

Feebly eosinophilic inclusion bodies were seen in the nerve cells of the posterior brain stem from one fox of less than one week old. Electron microscopic examination revealed the inclusions to be "fine-particle aggregating structures" (including particles of 13–14 nm in diameter).

Morpho-pathogenesis : The ischemic changes in the brain were conjectured to be due to neurogenic local functional disturbances in the blood circulation.

Causal pathogenesis : Giant cell formation of the meningeal mesothelium, neuronal cytoplasmic inclusions ("fine-particle aggregating structures") in the posterior brain stem and basophilic intranuclear and cytoplasmic inclusions in the hair matrices should not be ignored in investigating the causal genesis of the disease.