LETTER TO THE EDITOR

ULTRASTRUCTURAL CHANGES IN THE LIVER OF BIRDS EXPERIMENTALLY INFECTED WITH THE AGENT OF HYDROPERICARDIUM SYNDROME

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Hydropericardium syndrome (HPS), a hazard to poultry industry, is characterized by the accumulation of straw-coloured clear fluid in the pericardial sac, discolouration of the liver with large necrotic patches, spongy lungs and enlarged kidneys with distended tubules (*1*). The disease was first reported in Pakistan followed by India and some countries of Latin America (*1*). Recently, it has also been identified in Japan (*2*). Fowl adenovirus 4, a member of the *Fowl adenovirus C* species in the *Aviadenovirus* genus, has been identified as the causative agent of this disease. Transmission electron microscopy (TEM) of liver sections from birds experimentally infected with resuspended pellet obtained after ultracentrifugation of liver homogenate extract showed presence of virus particles resembling adenovirus in morphological details (*3, 4*). However, detailed ultrastructural changes in the liver were not reported. The present paper describes the details of virus particles and virus-induced changes in the liver, which is the target organ in HPS.

The experimental infection of birds, collection of livers from affected birds in 2.5% glutaraldehyde solution in PBS and its processing up to the level of block formation has already been described (*4*). The tissue samples were embedded in araldite CY 212, ultrathin sections were prepared and stained with uranyl acetate and lead citrate.

TEM of the liver showed margination of chromatin and enlargement of nuclei of hepatocytes. The viral replication sites in the nucleus were characterized by the presence of virus particles resembling adenoviruses (Fig. A, bar = 1 μm). The hexagonal virus particles of 70–75 nm in diameter with an electron-dense nucleoid (mature particles) or with adielectronic nucleoid (immature particles) were seen in the nucleus of infected hepatocytes (Fig. B, bar = 225 nm). The virions were generally scattered throughout the nucleus, although they were also found in paracrystalline arrangements associated with electron dense material in both cases. The membranous structures associated with viral replication sites were also observed. The enlarged nuclei occupied by homogenous and granular electron-dense material and apoptosis of some hepatocytes were regularly seen.

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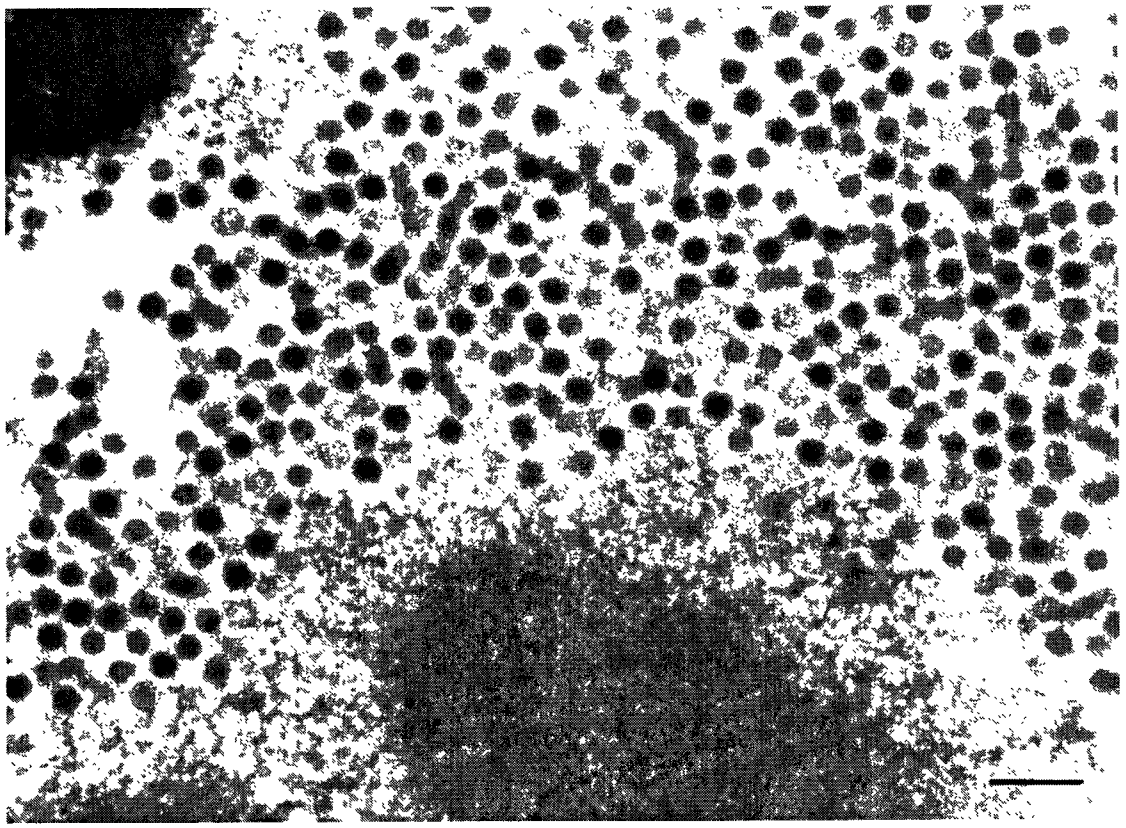
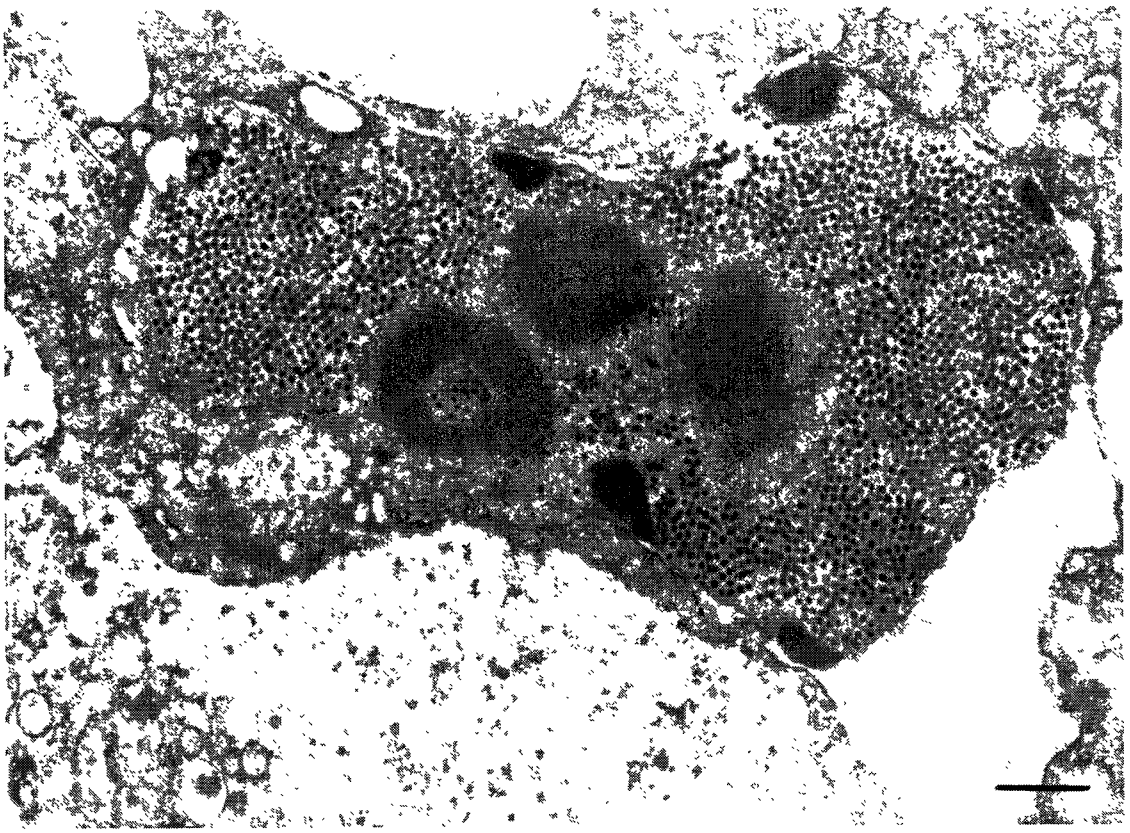
Abbreviations: HPS = hydropericardium syndrome; TEM = transmission electron microscopy



A



B

Hexagonal viral particles measuring 70–75 nm in diameter resembled adenovirus particles and confirmed our earlier observations that an adenovirus is involved in the causation of disease (*4*). However, the size of virus particles, which has been found earlier of approximately 90–92 nm in diameter, was now estimated at 70–75 nm. The isolation and characterization of the etiological agent in embryonic liver cell culture (*5*) and histopathological changes in the liver (*6, 7*) support the present observations. The apoptosis of hepatocytes, which was a constant feature, appears to be the cytopathic effect produced by the virus, as no viral particles could be demonstrated in the cells undergoing apoptosis. However, this needs further investigation to confirm whether it is an artifact or a direct effect of disease.

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