An Outbreak of Rabbit Sudden Death in Korea Suspected of a New Viral Hepatitis
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A new sudden death in rabbits appeared in China [7] and Korea [6] in 1984 and 1985, respectively, and was recognized to be an acute infectious disease caused by a virus. The first of the disease was reported as a new viral disease [7], and a tentative name of viral hemorrhagic disease [13], hemorrhagic pneumonia [11] or viral hemorrhagic pneumonia [2] has been described in the case reports. In a previous study, the present authors [6] suggested that the principal lesion of the disease was an acute hepatitis. Thereafter, it has been called rabbit hemorrhagic disease syndrome [10], necrotic hepatitis of rabbits [4, 8], X disease of rabbits [1] and similar names [9, 12] in other countries.

The purpose of this report is to describe the clinical and pathological manifestations of the newly recognized disease suspected of viral etiology in rabbits in Korea.

About 6,200 Angora rabbits from 7 farms were examined. The usual age of the disease onset was 3 to 12 months. The disease was not observed in rabbits more than 1 1/2 years old. Within the affected herds, the deaths were usually sudden, affecting several groups simultaneously. The morbidity and mortality rates were about 20% to 95% with young rabbits most severely affected. Rabbits that recovered were severely emaciated and suffered from loss of hair.

One hundred and twenty six rabbits submitted to our laboratory were necropsied. Lesions were present in the liver of all rabbits examined. There were both diffuse and focal pale foci, with or without hemorrhages of variable size scattered throughout the liver. Liver parenchyma also was paler than normal. The livers of five rabbits that showed recovery had mild cirrhosis. Other lesions often consisted of petechiae in the kidney, and hyperemia and hemorrhages in the spleen and the lung. The urinary bladder was generally enlarged and contained turbid urine.

Microscopically, the liver lesions of affected rabbits revealed periportal necrosis with infiltration of mononuclear cells (Fig. 1), which was characteristic of viral hepatitis [5]. Some focal necroses were also seen scattered in the hepatic lobules. In the five subacute cases that showed emaciation, marked infiltration of lymphocytes and plasma cells, proliferation of fibroblasts, and atrophy of hepatocytes were observed. In about 20% of examined rabbits, perivascular cuffing was found in the brain. Kidneys in some cases presented glomerulonephritis, hemorrhages, tubular necroses and hyaline casts. Proliferation of fibroblasts and infiltration of mononuclear cells were occasionally found in the renal interstitium. There were hemorrhages and edema in the lung, hyperemia and hemorrhages in the tracheal and bronchial mucosae, and focal necrosis in the myocardium.

Fig. 1. Photomicrograph of the liver of a naturally infected case, showing marked peripheral necrosis of the hepatic lobules. Hematoxylin and eosin. ×50.
Hyperemia and hemorrhages were seen in the spleen.

To reproduce the disease, homogenates of livers from the rabbits that died suddenly were used as inoculum after centrifugation at low speed. Prior to inoculation, clarified liver suspensions were passed through a 0.45 μm membrane filter. Fifty four rabbits from 4 to 12 months of age were inoculated intramuscularly. Clinically, the infected rabbits had elevated body temperatures (41°C). All infected rabbits except 6 (48/54) died within 24–72 hr without conspicuous signs. No further deaths occurred until the survivors were necropsied at 30 days postinoculation. Gross and microscopic lesions in the liver were similar to those of the spontaneous cases. Other organs showed no remarkable findings.

The livers of natural cases were chosen for further study by electron microscopy because the lesions in both the naturally occurring and the experimentally induced infections were more consistent in the liver than in other organs. Crystalline arrays of virus-like particles resembling those of the family picornaviridae [3] could be demonstrated in the cytoplasm of the hepatocytes (Fig. 2), sinusoidal endothelial cells including Kupffer’s cells and macrophages in the hepatic tissues.

The size of the particles ranged from 24 to 30 nm and the shape was polygonal. There were also immature virus-like particles associated with dilated rough endoplasmic reticulum and destruction of nuclear membrane in the hepatocytes.

A spontaneous outbreak of sudden death in rabbits occurred in Korea, of which the clinical features, morbidity and mortality rates, lesions, causative agent, and transmissibility were essentially consistent with the viral hemorrhagic disease and the viral hemorrhagic pneumonia in rabbits in China [7]. However, hepatitis is the principal lesion in this outbreak, and the present authors have called this disease “rabbit viral hepatitis”. The suspected agent in necrotic hepatitis of rabbits in Mexico was a parvovirus [4], while a calicivirus in rabbit hemorrhagic disease in West Germany [9].

Further study will be required for isolation and characterization of the agent of this novel entity.

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要 約

韓国のウサギにおける急性致死性疾病の発生；肝炎を主症状とする新しいウイルス性疾病（短報）：李且秀・朴清圭・申台均1)・趙鍾焕2)・鄭宗植2)（慶北大学校獣医科大学，1)忠南大学校医科大学，2)慶尚北道家畜衛生試験所）——韓国において1985年から発生しているウサギの急性致死性疾患について検索した。罹患例の臨床所見、罹病率と致死率病理的所見、原因ウイルスおよび再現試験などから本疾患は1984年度から中国に発生し、ウサギのウイルス出血症あるいはウイルス出血性肺炎と呼ばれている疾患と基本的には同じであった。しかし検索例の病変が急性肝炎であったことから著者らは、本疾患をウサギのウイルス肝炎と命名することを提唱した。