VERVET MONKEY DISEASE, A NEW ZOONOSIS

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SUMMARY

The literature on Vervet monkey disease has been reviewed to draw the attention of tropical veterinarians to this new zoonosis.

INTRODUCTION


Definition.—Vervet monkey disease is an acute, often fatal, infection of man derived through contact with monkey tissues and characterized by fever, severe headache, muscular pains, vomiting, diarrhoea and a maculo-papular rash (Martini et al., 1968).

History.—Vervet monkey disease first came to light in August, 1967 when 17 animal handlers, veterinarians and laboratory workers in Marburg, and 6 in Frankfurt developed acute illnesses after handling monkey tissues. There were 3 secondary cases at Marburg and several secondary cases at Frankfurt. Five of the Marburg patients and 2 of the Frankfurt patients died (Martini et al., 1968; Stille, Bohle, Helm, van Rey & Siede, 1968; Martini, 1969).

From the beginning Vervet monkeys (Cercopithecus aethiops) were incriminated as the source of the infection (Martini et al., 1968). Dick (1969) untangled their trail. Vervet monkeys were trapped in Uganda and batches of monkeys were shipped weekly to Czechoslovakia, Italy, Japan, Sweden, Switzerland, U.S.S.R. and Yugoslavia. One consignment of 100 monkeys was directed to London because of the Middle East war and it arrived on the night of 28th July 1967. The consignment was forwarded to Dusseldorf where it was split. Twenty monkeys were sent on to Frankfurt, 73 on to Marburg and 4 on to Biberach. The Uganda exporters claimed that the infection must have been acquired in transit but their claim was nullified when 2 new human cases occurred in Belgrade through contact with Vervet monkeys consigned on 1st August 1967. A retrospective analysis of the incubation periods of some of the Marburg patients indicated that they were infected by a third consignment of monkeys which had arrived on the 21st July 1967. The number of infected monkeys is unknown; there were at least 8 primary infections of monkeys in German laboratories (Hennessen, Borin & Mauler, 1968). According to Hennessen et al. (1968) the monkeys were all apparently healthy although Dick (1969) reported that 2 of the 73 monkeys received at Marburg were dead on arrival. Others died soon after arrival (Simpson, Bowen & Bright, 1968a).

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AETIOLOGY

Because German laboratories were, at first, reluctant to handle the agent, the Microbiological Research Establishment at Porton in England was asked to investigate its nature. Guinea pigs proved susceptible and dramatic histological changes were observed in the liver after two serial passages (Gordon Smith et al., 1967). There was necrosis of parenchymatous liver cells and many of the cells contained basophilic inclusion bodies reminiscent of those produced by rickettsiae and bedsoniae. The agent, however, was not sensitive to antibiotics. Complement-fixing antigens and antisera were prepared and were tested with known arboviruses, bedsoniae, rickettsiae and toxoplasma organisms and their antisera with negative results.

Later in 1967 Siegert, Shu, Slenczka, Peters & Müller drew attention to the clinical similarities between Vervet monkey disease and simian haemorrhagic fever. Investigations failed to identify the agent and they concluded that it was previously unknown to man. Siegert and his colleagues demonstrated the presence of the agent in liver and testis cells by immuno-fluorescence and by electron microscopy. They suggested that the agent was a virus bearing a strong resemblance to vesicular stomatitis and rabies viruses, the so-called bullet-shaped viruses.

May & Knothe (1968), Kissling, Robinson, Murphy & Whitfield (1968) and Zlotnik, Simpson & Howard (1968b) independently confirmed the viral nature of the agent. It had helical symmetry, contained RNA and essential lipid. The particle shapes were bizarre; they were structurally cylindrical with a uniform diameter that varied from 70 to 90 nanometers and a length that ranged from 130 to more than 2,600 nanometers. The core diameter was about 45 nanometers. The rounded ends of the particles were often bulbous. Many particles were bent into loops, some were coiled and a few were branched.

Exposure to 60°C for one hour destroyed infectivity (Bowen, Simpson, Bright, Zlotnik & Howard, 1969). Exposure to 56°C revealed two components; a major component that was inactivated within 20 minutes and a small residual component that was relatively heat-stable. There was no loss of activity after storage at -70°C for 12 months and little loss after storage at 4°C for 5 weeks. Ultraviolet light inactivated the agent completely in 2 minutes. Both infectivity and immunogenicity were destroyed by one hour's exposure to acetone, chloros, formalin, methyl alcohol and Tego MHG (Bowen et al., 1969). Infectivity but not immunogenicity was abolished by 24 hours' treatment with 1:2000 beta-propiolactone (Kissling et al., 1968). On the other hand, phenol, cetramide and trypsin merely reduced the titre of the agent (Bowen et al., 1969).

Despite the evidence, Bowen and his colleagues (1969) at Porton regarded the characterization and classification of the Vervet monkey disease agent as premature. They emphasised that if the agent was a virus it was the first of its type known to form agglomerations visible under the light microscope. Recently Almeida, Waterson, Berry & Turner (1969) examined cultures of various leptospires in the electron microscope and found tubular structures arising from the bodies of the leptospires that were morphologically similar to the Vervet monkey disease agent. The controversy is not yet resolved.

HUMAN INFECTIONS

Incubation period.—The first reports from Germany cited incubation periods