Rift Valley Fever antibody in human sera collected after an outbreak in domestic animals in Kenya

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Rift Valley Fever (RVF) virus is a member of the family Bunyaviridae, genus Phlebovirus (Bishop et al., 1980). As first described by Daubney et al. (1931), the disease was a highly fatal epizootic of sheep at a farm north of Lake Naivasha, Kenya. Since that time, periodic epizootics of RVF have been recorded in Kenya. They principally affected domestic sheep and cattle populations, particularly those imported into the country. Human cases have occurred in laboratory workers and people associated with the affected animals, but are comparatively rare (Davies, 1975; Davies et al., 1985). In June 1989 an outbreak of RVF occurred in domestic cattle and sheep on farms bordering Lake Naivasha, less than 20 km from the site of the 1931 outbreak (Logan et al., 1991).

Rift Valley fever was recognized when virus was isolated from a bovine foetus brought to the Veterinary Research Laboratory at Kabete on 15 June 1989 (Davies et al., 1991). Further investigations were made at the farm where the foetus was aborted and at 2 adjacent farms. Sera from RVF-virus positive animals on the same farm were tested for sheep and cattle during the outbreak and a high percentage of ruminants were RVF antibody-positive in a virus serum neutralization test (Davies et al., 1991). RVF virus was also isolated from 3 pools of cattle, 2 pools of wildlife (Culex and Mansonia) and 1 pool of Manossia australis. The results were transmitted to the managers of the farms visited during this study.

This report describes the prevalence of RVF virus-specific immunoglobulin (Ig) G and IgM antibody in herdsmen working on the 3 affected farms. Blood samples were taken by finger stick on to filter paper (WHATMAN 1) and sera from 26 herdsmen at a farm where an abortion in one cow was thought to have been associated with the epizootic. The samples were allowed to dry on the filter paper by soaking in the test diluent, and then stored in a container at 20°C. They were tested by an enzyme-linked immunosorbent assay (MEEGAN et al., 1987). The serum was eluted from the filter paper by soaking in the test diluent, and then further diluted to 1/400. The IgM test was performed with a mouse-anti-human IgG conjugate. Samples that were RVF antibody-positive for IgG were tested for RVF IgM antibody with an IgM capture technique (Ksiazek et al., 1991). Samples were considered positive if they had standard optical density values greater than 3 standard deviations above the mean of a group of RVF antibody-negative human sera.

Four samples were collected from farm 1, where the index case occurred (altitude 1920 m; 0°43'S, 36°18'E); 18 from farm 2, where much clinical disease was seen in sheep and cattle (1920 m; 0°43'S, 36°18'E); and 8 from farm 3, where an abortion in one cow was thought to have been associated with the epizootic (1920 m; 0°48'S, 36°24'E). Twelve (40%) of the 30 herdsmen tested had detectable RVF IgG antibody. Five (42%) of these 12 also had RVF virus-specific IgM antibodies. At farm 1, 2 of 4 herdsmen were IgG positive; at farm 2, 26 of 18 were IgG-positive and 2 of these were IgM-positive; at farm 2, 6 of 18 were IgG-positive and 2 of these were IgM-positive; at farm 3, 4 of 8 were IgG-positive and 2 of these were IgM-positive. No clinical disease had been apparent during the outbreak that might have been RVF, nor could the herdsmen recall experiencing any illness during that period. This sample was clearly heavily biased to detect positive cases, for all men had been closely associated with diseased cattle during the epizootic.

The widespread epizootics of RVF in ruminants in Kenya over the last 50 years were not accompanied by similar epidemics of disease in the human population. Of serum samples taken from 26 herdsmen, only 80–90% of cattle were affected during an earlier RVF epizootic, only 2 were positive to RVF antigen in an indirect fluorescent antibody test (F. G. Davies, unpublished data). Other cases of RVF have been reported in East Africa (Metselaar et al., 1974; Smithburn et al., 1949; Daubney et al., 1931) and RVF antibody has been shown to occur at a low level in human populations (Johnson et al., 1983). RVF viral isolates from East Africa are considered to be as pathogenic for humans as, for example, the Egyptian strain ZH501 (Battles & Dalrymple, 1988).

There has been extensive human involvement, with mortality, in RVF epizootics in other African countries, most recently Madagascar (Morvan et al., 1991), Mauritania (Jouan et al., 1989), Egypt (Meegan, 1979) and South Africa (Van Velden et al., 1977). It is possible that the low human to animal contact ratio throughout the enzootic areas may be significant. Often 1 or 2 herdsmen will manage between 400 and 1000 animals in each farm. In Egypt and Mauritania, family groups generally have far fewer cattle, sheep or goats and live much more closely associated with them. Vector biology may be a further and important determinant.

The samples were ethically obtained from all subjects after informed oral consent to the purpose and procedure of the serology; the data were recorded anonymously using a number system. The results have been transmitted to the managers of the farms visited during this study.

References


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