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Rift Valley Fever in Rhodesia

REPORT OF A CASE IN A LABORATORY WORKER

BY

LIONEL STERN, M.B., CH.B., D.P.H.
Salisbury.

DEFINITION

Rift Valley fever or enzootic hepatitis is an acute virus infection transmitted by mosquitoes and is pathogenic under natural conditions for sheep and cattle. Man may be infected by direct transmission through contact with meat or tissue of diseased animals or accidental contamination, as in the case of veterinary and laboratory research workers, and suffers from a short febrile illness which is almost never fatal. Epidemics of the disease occur in sheep and cattle, when it is associated with a high rate of mortality amongst lambs and a high incidence of abortion amongst pregnant ewes.

HISTORY AND DISTRIBUTION

The disease was first discovered in 1931 as an epidemic occurring amongst sheep and cattle in the Rift Valley, Kenya, where a high mortality was recorded (Daubney *et al.*, 1931). During the course of the investigation several human workers developed a short febrile illness and a Native volunteer developed a similar illness on being inoculated with the filtered virus. The disease has since been described throughout Africa and has been reported in Kenya, Uganda, Sudan, French Equatorial Africa and South Africa. A mouse protection test has been shown to demonstrate the presence of immune bodies to the virus in the sera of inhabitants of these areas. An epidemic of the disease in cattle and sheep was described in the Union of South Africa, when several cases of acute febrile illnesses occurred in humans in close contact with infected animals or their tissues. Several housewives too were infected, presumably from the handling of infected meat. Direct spread of the disease from patient to contact did not occur (Schrire and Gear, 1956).

PATHOLOGICAL CHANGES

Focal degeneration of liver cells is the main pathological change in Rift Valley fever in susceptible animals. The cells show characteristic hyaline degeneration of the cytoplasm and bear a striking resemblance to the liver changes in monkeys and humans dying of yellow fever (Councilman lesion). In animals there is a tendency to rapid decomposition of carcases related to more or less complete failure of liver function before death. The liver is rich in the virus, as also the spleen. The disease falls into the natural group with yellow fever and dengue fever in man.

THE VIRUS

The virus causing Rift Valley fever measures 23-35 μ and has been shown to be distinct from that causing yellow and dengue fevers. Neutralising and complement fixing antibodies develop in man and animals following infection. Infection confers a long-lasting immunity, possibly lifelong. Different strains of the virus have been discovered, including a neurotrophic strain which can be used for immunisation of sheep. Mice were shown to be highly susceptible to the disease. The virus can be isolated from blood of patients during the acute stage of the illness, and intraperitoneal inoculation of mice produces characteristic liver changes which are of diagnostic significance.

THE DISEASE IN MAN

The incubation period in man is from three to six days, and infection is usually through direct contact with infected tissue. Mosquito-transmitted infection appears to be rare.

The disease begins suddenly with a sharp rise in temperature, 101-103° F., rigors, severe headache, nausea and vomiting and general

malaise. Occasionally epistaxis occurs and pains in the back, photophobia and tenderness of the eyeballs have been noted. The face is flushed, conjunctivae congested and the throat injected. Symptoms and signs of coryza are notably absent. The temperature may reach 104-105° F., lasting one to three days, falling to normal for a day or so and not infrequently rising again to a slightly lower level—the diphasic or saddleback remission. A third rise of temperature may sometimes occur with decreasing severity of symptoms. There are no abnormal findings in the chest, no rash is seen, the spleen is not enlarged, though the liver may be tender to palpation. The urine is dark in colour and bowels constipated. The blood changes are a primary leucocytosis followed by a leucopenia. Convalescence, though rapid, is associated with general malaise which may last for several weeks. The disease is generally non-fatal, though one fatal case has been reported in which a pulmonary thrombosis developed during convalescence. Cases of accidental laboratory infection have been reported.

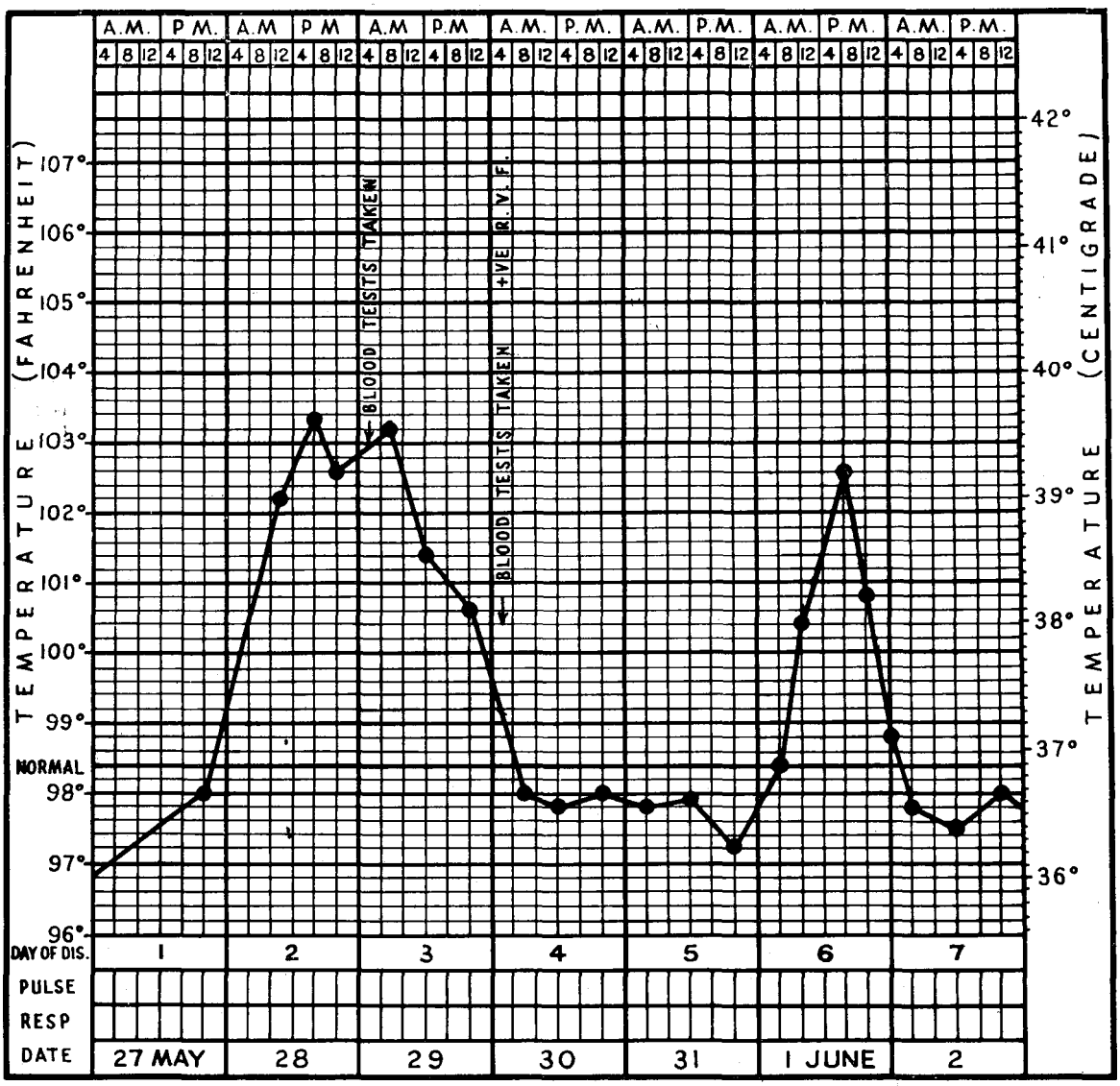

Fig. 1—Temperature chart showing rate of fever.

The disease is generally free of complications. Ocular complications are described and disturbances of vision varying from blurring to almost complete blindness. The changes are those of a central serous retinopathy, characterised by macular swelling and accompanied occasionally by small haemorrhages. The loss of central vision in a case of Rift Valley fever has been reported. These visual disturbances occur a few days to a few weeks after the pyrexial attacks, vision acuity generally returning to normal.

DIAGNOSIS

The diagnosis of Rift Valley fever should be suspected when human beings suffer from an acute febrile illness, generally diphasic, where an epizootic with a high mortality has occurred amongst sheep. It should also be suspected in people handling sheep and cattle or their tissues as well as veterinary and allied workers. During the febrile period the isolation of the virus is possible through intraperitoneal injection of blood into mice, causing characteristic liver necrosis. A mouse protection test demonstrates the presence of antibodies in the blood of a convalescent patient and is of diagnostic value—these neutralising antibodies may persist for many years. One attack of Rift Valley fever confers lifelong immunity. Treatment of the disease is symptomatic and the prognosis is good.

CASE HISTORY

European male (J.B.), aged 18 years. Occupation: technical assistant at the Veterinary Research Laboratory, Salisbury. On the 23rd May his duties involved inoculating young mice with harvested mouse-brain material from known positive Rift Valley fever. Four days later he became ill with what he thought to be a common cold. The next day he had a rigor, complained of a very severe frontal headache, photophobia and had a mild epistaxis. His main complaint at this stage was pain behind the eyes and a feeling as though the eyes were too large for the socket. His temperature remained high (103° F.) for 48 hours and he looked ill; his face was flushed, his eyes red and the throat dry and parched. During this stage he appeared irritable and resented any examination. Backache, nausea and occasional attacks of vomiting were present. The expected symptoms of coryza did not appear, his chest remained clear and clinically, apart from his temperature and general malaise, there was very little of note. His temperature subsided over the next 24 hours and he felt fine though listless, remaining so for a further 24 hours. On 1st June he again became ill, developed a rigor and a rapid rise of temperature, with a recurrence of his symptoms, though to a lesser degree. The fever was, however, of short duration, settled within the next 24 hours, did not recur and the patient passed into the convalescent stage of his illness (Fig. 1).

At no stage was the spleen palpable, there was no enlargement of lymph glands and no rash was in evidence. Prostration was notable throughout. During his convalescence he complained of easily becoming tired and listless, with no energy or inclination to do anything.

Laboratory Investigations.—On the 28th May, at the height of his first attack of fever, a blood count revealed haemoglobin 110 per cent., red cell count 5.54 m./c.mm., colour index 1.0, white cell count 6,800 c.mm., neutrophils 50 per cent., eosinophils 2 per cent., lymphocytes 42 per cent., and monocytes 6 per cent.

Red cells, white cells and platelets were normal and no parasites were seen in thick or thin films. A sample of blood was submitted to the Veterinary Research Laboratory, Salisbury (Mr. D. K. Shone) and the isolation of the virus of Rift Valley fever was reported, confirming the diagnosis. No evidence of liver damage was found. During the first week of convalescence a report on his urine and blood was as follows: Bilirubin negative, urobilin positive. Blood: total protein, 6.52 gram; albumen, 3.1; globulin, 3.42; A/G ratio, 1: 1; thymol turbidity, 1 unit.

No complications were encountered. The fundi, discs and pupillary reactions were normal. No treatment except symptomatic treatment was given, and his recovery was complete.

DIAGNOSIS

The diagnosis on clinical grounds was suspected when the patient's history was elicited, particularly on learning the nature of his occupation, and more so on eliciting the exact nature of the work upon which he had been engaged a few days before he took ill. The Rift Valley fever brain material which he had been handling is highly infectious, and it only required the actual isolation of the virus from his blood at the height of his fever to confirm the diagnosis. This was done, and his progress through the disease was watched with interest.

Other causes of short febrile illnesses, notably coryza, influenza or protozoan diseases such as malaria, were considered early on in the illness. Sandfly fever, dengue fever and yellow fever have also to be considered in the differential diagnosis, as also other virus conditions with a saddle back or diphasic temperature chart, including poliomyelitis, measles, mumps, louping ill and the virus encephalitides.

SUMMARY

A case of Rift Valley fever in a young European laboratory worker is presented, the case history being followed from the time of accidental infection through to convalescence.

A brief summary of the history and distribution of the disease, as well as a few details on the virus itself and the pathological changes in man, are given.

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