Congo-Crimean haemorrhagic fever in South Africa

Report of a fatal case in the Transvaal

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Summary

A 13-year-old boy, after having spent a week at a camp in a nature reserve in the western Transvaal, developed an acute illness of sudden onset, characterized by chills, severe headache, muscle pains and high fever. On the 3rd day he developed a haemorrhagic state with profuse bleeding from the gastro-intestinal tract and other mucous membranes and petechial haemorrhages into the skin, from which he died on the 6th day after onset of the illness.

A tick, identified as a species of Hyalomma, was found attached to his scalp. The provisional clinical diagnosis of Congo virus fever was confirmed in the laboratory by the isolation of the virus in newborn mice inoculated with the patient's blood.

This is the first incrimination of Congo virus as the cause of a fatal case of haemorrhagic fever in South Africa, although it is known to occur in several countries in the tropical region of Africa and in south-eastern Europe and Asia.

Case report

The patient, a 13-year-old boy, attended a veld school camp in the Bloemhof district of the Transvaal on 5–13 February 1981. The site of this camp was in a nature reserve which consists mostly of rolling grasslands and a few sparsely wooded areas adjacent to pans on its southern boundary. After the recent rains the grass had grown luxuriantly, providing excellent grazing for the numerous wild animals, which include wildebees, hartebees, impala, springbok, gemsbok, steenbok and eland, as well as a few head of cattle. Smaller animals, including Cape hares, ground squirrels and yellow mongoose, are common in the area. While on camp the boys were instructed in veld craft, which involved long walks through the veld and canoeing on the pans. For several nights they slept on the ground outdoors, either in the open or in small tents.

The patient returned to his home in Edenvale near Johannesburg on Friday 13 February. On 14 February he told his mother that he was feeling ill, complained of severe headache, muscle pains and high fever. On examination the doctor found a bite mark where the arthropod had been attached. He made a diagnosis of tick-bite fever and prescribed the appropriate treatment. He handed the tick and then discarded it.

The next day, Sunday 15 February, there had been no improvement in the patient’s condition and his temperature rose to 40°C. A physician was called in and arranged for the patient’s admission to a private hospital and initially prescribed erythromycin and thiamphenicol, but with deterioration of the patient’s condition over the next 2 days the treatment was changed to gentamicin and an ampicillin-cloxacillin combination. With further deterioration, evidenced by a
continued swinging temperature, lowering of blood pressure, oozing from needle puncture sites and haematemesis and melaena during the afternoon of 17 February, hydrocortisone, cimetidine, aluminium hydroxide, fresh-frozen plasma and a single dose of 2000 units of heparin were given. Later that night he was transferred to the paediatric intensive care unit of the Johannesburg Hospital.

On admission he was pale, shocked and confused. He had a tachycardia with impalpable pulses, but his blood pressure could initially be recorded at 120 mmHg with a Doppler, although it could not be heard with an ordinary stethoscope. There was no neck stiffness. He was able to move all his limbs, had no localizing signs and his pupils were equal and reacting to light. Fundoscopy was negative. Although he was oozing from needle puncture sites, there were initially no petechiae. These became apparent the next day and were mainly on the right side of the abdomen. There was no rash. The chest was clear but respiration was acidotic in nature. No abnormality of the heart was found, nor was there hepatosplenomegaly. Lymphadenopathy that had been noticed in the neck previously was not evident at this stage. Central venous pressure was found to be nil.

Laboratory studies at the private hospital on 16 February had shown a haemoglobin value of 12.9 g/dl, a white cell count of 19000/μl with a shift to the left, and a platelet count of 44000/μl. The prothrombin index was 67%. The serum bilirubin level was normal. Investigations at the Johannesburg Hospital showed a falling haemoglobin value (7.9 g/dl, falling to a minimum of 4.4 g/dl) which later stabilized at 8-10 g/dl. A leucocyte count dropped from 9400 to 4000/μl with an initial differential count of 84% neutrophils, 4% monocytes and 12% lymphocytes; this later changed to only 14% neutrophils with 29% band forms and 50% lymphocytes. The platelet count dropped from 20000/μl before heparin administration to 13000/μl on admission; soon platelets could not be detected. With platelet infusions the count rose to 69000/μl. The prothrombin index was 21% on admission and this rose to and remained above 40%. The partial thromboplastin time was 2-3 times normal. The fibrin degradation product value was above 160 U/ml, dropping to 10-40 U/ml on treatment. Malarial and other protozoal parasites were not detected in the blood smears. Rickettsial complement fixation tests were negative, as were blood cultures. A lumbar puncture was not done because of the picture of disseminated intravascular coagulation. The patient was passing urine, and the serum creatinine level was only mildly raised at 130 μmol/l. The blood urea level was 15 mmol/l, sodium 127 mmol/l, potassium 4.6 mmol/l, chloride 95 mmol/l, CO₂ 10 mmol/l and calcium 2.31 mmol/l. The clinical acidosis was confirmed by the blood gas values: pH 7.09, PaO₂ 32 mmHg, PaCO₂ 42 mmHg (venous), bicarbonate 9.1 mmol/l (a deficit of –19). The blood lactate level was 19 μmol/l (normal 0.63 - 0.44 μmol/l) and the ammonia level was 70 μmol/l (normal 17 - 47 μmol/l). Other results of note were a total protein level of 52 g/l, albumin 29 g/l, serum phosphate 2.17 mmol/l, uric acid 0.57 mmol/l, total bilirubin 8 μmol/l and indirect bilirubin 2 μmol/l. Serum glutamic pyruvic transaminase was raised at 177 U/l and serum glutamic oxaloacetic transaminase at 345 U/l. The erythrocyte sedimentation rate was 7 mm/h (Westergren). The initial chest radiograph was clear except for small pleural effusions. The abdomen showed dilated loops of bowel with a few fluid levels.

In consultation with a number of haematologists it was thought safer not to continue with heparin therapy. The patient was therefore given vigorous resuscitative therapy, including transfusions of blood, fresh-frozen plasma and several large volumes of platelet suspension. Sodium bicarbonate 8.5% (100 ml) was given to partially correct the severe metabolic acidosis. By the next morning his blood gas status had improved: pH 7.40, PaO₂ 29 mmHg and bicarbonate 18 mmol/l. Cimetidine therapy was continued, but the antibiotic combination was changed to cloxacinil, chloramphenicol and tobramycin.

With the aggressive volume replacement therapy, the blood pressure was maintained the following day, with good perfusion, but the next night he had another massive haematemesis and the blood pressure and perfusion again fell. The haematemesis was treated with stomach wash-outs with cold saline. Because of the possibility of stress ulcers that could be treated surgically, the surgeons performed a gastroscopy but found that there were only multiple fine acute gastric erosions. The melaena and haematemesis continued, despite adequate replacement therapy. In the early morning of 19 February he became hypoxaemic with a PaO₂ of 53 mmHg in 40% oxygen. On being given 60% oxygen by Ventimask the PaO₂ rose to 80 mmHg. The pH was 7.42, PaCO₂ 32 mmHg and bicarbonate 20 mmol/l. The serum creatinine level was 136 μmol/l but he was still passing adequate urine. The serum sodium level was 135 mmol/l, potassium 3.7 mmol/l, chloride 101 mmol/l, haemoglobin 9.7 g/dl and the white cell count 5600/μl. The last platelet count had been 69000/μl; however, the partial thromboplastin time remained prolonged. The blood pressure was 110/80 mmHg and despite the maintenance of an adequate central venous pressure the patient suddenly had a cardiac arrest and could not be resuscitated. This occurred on the 6th day of his illness.

Discussion

Diagnostic considerations

When seen on the Wednesday, the patient was semi-comatose with profuse haematemesis and melaena and it was clearly essential to establish a diagnosis as soon as possible. Not only was this important in the patient’s interests, but also for the protection of the medical and nursing staff caring for him, in view of the possibility of a dangerous infection. While the diagnosis remained in doubt, medical and nursing staff were instructed to wear protective clothing consisting of a gown, mask, gloves, goggles and cap.

Possible causes were systematically considered, special attention being given to conditions of importance in Africa. It was noted that, broadly speaking, the haemorrhagic state might result from blood disorders, from exposure to poisons, chemicals and drugs and from infections, including viral, rickettsial, bacterial and protozoal infections. Of particular importance among the blood disorders were those resulting in platelet abnormalities, either qualitative or quantitative, and those resulting in coagulation defects, but a constitutional disorder seemed unlikely in an acutely ill and previously healthy schoolboy.

At the time it was not clear whether the ‘8-legged insect’ was a spider or a tick. Several spiders are known to inject powerful neurotoxins with their bite. Notable among these is Latrodectus mactans, the black widow spider, but its bite is characterized by a relative lack of local reaction, let alone a haemorrhagic reaction. However, it has become apparent from studies carried out in the Entomology Department of the SAIMR that the ‘assassin’ spider or self-burying spider of the genus Niceratius produces a powerful haemorrhagin which, in experimental rabbits, results in a fatal haemorrhagic state. Although the susceptibility of human beings to this spider venom is not known, the possibility that the patient’s condition was due to a spider bite was seriously considered. However, there were several features of the case which made a spider bite unlikely, in particular the fact that a spider does not remain attached after biting.

The possibility of snake bite was also considered. Among the snakes whose bite results in a haemorrhagic state are the puff-adder (Bitis arietans) and the boomslang (Dispolildus typus). The latter is not found in the open veld of the western Transvaal, but the puff-adder is one of the most frequently encountered snakes in this area. Its venom causes marked interference with the clotting mechanisms and destruction of the endothelial lining of the blood vessels, resulting in a characteristic haemorrhagic state.

The possibility of a tick bite was also considered. The latter is not found in the open veld of the western Transvaal, but the puff-adder is one of the most frequently encountered snakes in this area. Its venom causes marked interference with the clotting mechanisms and destruction of the endothelial lining of the blood vessels, resulting in a characteristic haemorrhagic state.

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state. However, the very marked local reaction and haemorrhagic necrosis of the tissues at the site of the bite characteristic of viperine bites were not evident in the patient. Furthermore, it was doubtful whether a snake could inflict a bite on a healthy schoolboy (even if he was asleep at the time) without him becoming aware of the fact.

### TABLE I. HAEMORRHAGIC FEVERS IN AFRICA

<table>
<thead>
<tr>
<th>Viral infections</th>
<th>Hepatitis virus A, B, non-A, non-B</th>
<th>Enterovirus</th>
<th>Coxsackie B</th>
<th>Paramyxovirus</th>
<th>Togavirus</th>
<th>Arbovirus</th>
<th>Alphavirus</th>
<th>Flavivirus</th>
<th>Bunyavirus</th>
<th>Rubella virus</th>
<th>Arenavirus</th>
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<tbody>
<tr>
<td></td>
<td>Hepatitis</td>
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<td></td>
<td>Measles</td>
<td></td>
<td></td>
<td>HIV</td>
<td>Yellow fever, dengue, West Nile</td>
<td>Rift Valley fever</td>
<td>Rubella, congenital infection</td>
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<tr>
<td>Associated with rodents</td>
<td>Lassa fever, West Africa</td>
<td>Marburg virus disease, Argentina</td>
<td>Uganda</td>
<td>Marburg virus disease, Zimbabwe</td>
<td>Ebola virus disease, Sudan, Zaire</td>
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<tr>
<td>Marburg virus group</td>
<td>Herpesvirus hominis</td>
<td>Herpesvirus varicellae</td>
<td>Epstein-Barr virus</td>
<td>Chlamydial infections</td>
<td>Rickettsial infections</td>
<td>Rickettsia conorii var. pyperi</td>
<td>Rickettsia burnetii</td>
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<tr>
<td>Associated with monkeys</td>
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<td></td>
<td></td>
<td>Chlamydia psitacci</td>
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<td>Source unknown</td>
<td>? ecological cycle</td>
<td>Source unknown</td>
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<td>Psittacosis</td>
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<tr>
<td>Source unknown</td>
<td>Marburg virus disease, India</td>
<td>Ebola virus disease, Sudan, Zaire</td>
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In view of the sudden onset and the rapid evolution of the illness in a previously healthy boy, it seemed more likely that he was suffering from one of the virulent infections, many of which result in the development of a haemorrhagic state. A list was made of the possible infective causes (Table I) and these were assessed systematically on clinical grounds. The appropriate laboratory tests were requested and undertaken. On the clinical findings most of these infections could be excluded, but a viral infection was suspected; among those receiving special attention were viruses of the herpesvirus group and arbovirus group and Lassa fever and Marburg virus disease.

In considering the infections due to the viruses of the herpesvirus group it was noted that infants suffering from kwashiorkor sometimes develop hepatic hepatitis, which often proves fatal. Members of the herpesvirus group are notorious for causing severe, often fatal, infections in patients suffering from immunodeficiency states or patients on immunosuppressive treatment, but the occurrence of severe herpes hepatitis in previously healthy adolescent or young adult males culminating in a haemorrhagic state is most unusual. However, in such cases, caused by *Herpesvirus hominis*, were recently seen in studies carried out in the National Institute for Virology. The possibility that this virus was responsible for our patient’s illness could not be excluded until the results of the laboratory tests were known. Similarly, the tests to exclude infectious mononucleosis, although unlikely to cause such a severe illness, were requested.

Of special importance were the arbovirus infections, of which over 30 have been identified in South Africa and 10 are known to infect man. Among the mosquito-borne infections these include in the alphavirus group, chikungunya and Sindbis viruses; in the flavivirus group, West Nile, Wesselsbron and yellow fever viruses (although the latter is not known to occur in Africa south of the Zambesi valley) and, in the bunyavirus group, the virus of Rift Valley fever.

Chikungunya virus has been incriminated as the cause of some cases of haemorrhagic fever in the Far East, especially in India. In South Africa this virus has been identified in the lowveld of the eastern Transvaal and northern Natal. Clinically, the outstanding signs and symptoms have been fever, often biphasic, associated with severe joint pains and a maculopapular rash, the individual papules sometimes showing petechial haemorrhages. Patients with a fully developed haemorrhagic state, as described in the Far East, have not been seen in South Africa. Since the clinical picture did not fit our patient, and since chikungunya virus is absent from the south-western Transvaal, it was not likely to be the cause of the illness.

Sindbis virus infections, on the other hand, frequently occur on the highveld. However, they are usually associated with a milder course, during which a characteristic papular rash erupts; this rarely shows a tendency to become haemorrhagic, and fatal cases have not yet been seen. Absence of a rash and the severity of our patient’s illness suggested that it was not due to Sindbis virus.

West Nile virus has a similar geographical distribution to Sindbis virus and the infection gives rise to a similar clinical picture with a somewhat similar rash, but the illness is rarely associated with the development of a haemorrhagic state and is rarely fatal, especially in childhood and adolescence. Wesselsbron virus in sheep causes a disease not unlike Rift Valley fever, associated with severe hepatitis, which, especially in lambs, often proves fatal. In man the infection tends to cause a more severe illness than either Sindbis or West Nile virus and it may be complicated by meningo-encephalitis and hepatitis. Fatal infections in man have not been reported in South Africa, but as the infection is known to cause severe illness this possibility could not be excluded. Even more serious consideration was given to Rift Valley fever virus which, like Wesselsbron virus, has periodically given rise to extensive epizootics affecting cattle and sheep on the highveld. They have been associated with epidemics in the human population,
affected in particular farmers, farm labourers and veterinary surgeons handling the carcasses of animals which had died of Rift Valley fever. In 1975, for the first time in South Africa, 7 fatal cases of Rift Valley fever terminating in a haemorrhagic state were identified, in 3 of them by the isolation of the virus from autopsy specimens of the liver. In 1977 in lower Egypt an extensive epizootic was associated with a severe epidemic in the human population, with thousands affected, of whom several hundred died in a haemorrhagic state. This emphasized the importance of Rift Valley fever as a cause of a haemorrhagic state.

Rift Valley fever was known to have been prevalent during the epizootics in 1975 in Bloemhof and the surrounding districts. The virus is transmitted by pan-breeding mosquitoes and in man causes an illness characterized by a biphasic fever and signs and symptoms suggestive of hepatitis, but a rash is not a feature. In the severe and fatal cases the haemorrhagic state developed towards the end of the second bout of fever. Although it was unlikely that this infection would give rise to a severe haemorrhagic state in a previously healthy schoolboy, it remained important to exclude it as the cause of his illness.

In view of the rapid evolution of the patient's illness into a severe haemorrhagic state, Lassa fever and Marburg virus disease were also seriously considered as possible causes of his condition. However, the evolution of his illness was more rapid than is usual with Lassa fever. When seen on the 5th day, the patient had not developed a maculopapular rash, which is typical in patients with Marburg virus disease. Nevertheless, arrangements were made for the tests to exclude both these infections.

As it was likely that the 8-legged arthropod found attached to the patient's scalp was a tick, its identity and the possibility of its being the transmitter of the patient's illness assumed great importance. Among the tick-borne viruses known to occur in South Africa are African swine fever virus and Quaranfil virus. African swine fever virus is not known to cause illness in man and Quaranfil virus infection apparently usually gives rise to only a mild illness. Although Congo-Crimean haemorrhagic fever had not yet been identified in South Africa, the virus causing it had been isolated in several countries of central tropical Africa and this infection was placed high on the list of the possible causes of the patient's illness.

While an attempt was being made to put a telephone call through to the Entomology Department of the SAIMR from the ward of the hospital to discuss the identity of the '8-legged insect', by a fortunate coincidence a call was received from Dr Ledger to say that the doctor's wife had come to the Institute, had been shown the collection of ticks and spiders in the department and had definitely identified the tick removed from the patient's scalp as a species of *Hyalomma*. Dr Ledger noted that this tick was known to be the most frequent transmitter of Congo-Crimean haemorrhagic fever. As the patient's signs and symptoms were similar to those of this infection, it was concluded that his illness had probably been caused by the Congo virus.

The need for protection of the staff in attendance on the patient was re-emphasized. Professor O. W. Prozesky, Director of the National Institute for Virology, was informed of our suspicion of the cause of this patient's illness and he alerted the team in the high-security laboratory and warned them of the impending arrival of specimens for urgent attention. Immediately on their receipt in the laboratory, suspensions were prepared from the blood and urine of the patient and inoculated into tissue cultures and into litters of baby mice, which thereafter were examined daily. Specimens of blood and urine were also prepared for electron microscopic examination, but this failed to show identifiable virus particles.

One week later the baby mice began to sicken. Some were sacrificed and the films made from the brains of the mice gave a positive result on immunofluorescent tests for Congo virus, but negative results for Marburg, Lassa and Rift Valley fever viruses, thus confirming the provisional diagnosis of Congo-Crimean haemorrhagic fever. During this week several thousand ticks were collected in the nature reserve by Dr P. Jupp for identification and further investigation.

**Congo-Crimean haemorrhagic fever**

Congo-Crimean haemorrhagic fever was first observed in the Crimea by Russian scientists in 1944 and 1945. At that time it was established by studies in human volunteers that the aetiological agent was filtrable and that the disease in man was associated with the bite of the tick *Hyalomma marginatum*. The agent was detected in the larvae and in adult ticks, as well as in the blood of patients during the fever. This agent, presumably a virus, was not maintained in the laboratory and was lost.

Congo virus was first isolated in Africa from the blood of a febrile patient in Zaire in 1956. In 1967 Simpson et al. described 12 cases of a feverish illness of which 5 were laboratory infections; the virus was isolated by the inoculation of blood into newborn mice. Simpson showed that these viruses were similar to the one isolated in 1956. Casals then showed that the viruses isolated in cases of Crimean haemorrhagic fever and the Congo virus were serologically indistinguishable and demonstrated that other virus strains from Central Asia, the USSR and Bulgaria were similar. The virus has been classified as a Nairovirus in the genus *Bunyavirus* in the family Bunyaviridae. It contains RNA and is inactivated by lipid solvents and detergents.

Laboratory studies have shown that Congo virus is related to Hazara virus isolated from ticks in Pakistan, and to Nairobi sheep disease virus; together they form the Nairobi virus group.

In Africa the virus has been isolated from a variety of animals, including cattle, sheep, goats, hares and hedgehogs, and from a number of ticks which parasitize them, including *Hyalomma* sp., *Amblyomma variegatum*, *Boophilus decoloratus* and *Rhipicephalus* sp.

The most important transmitters of the infection to man are species of the genus *Hyalomma*, the life history of which is shown in Fig. 1. The larval and nymphal stages of some species parasitize birds, including migratory birds, some of which fly from south-eastern Europe to South Africa and thus may carry the infection over long distances. To verify whether this actually happens will require further study of the ticks and their hosts in South Africa and on their way from Europe.

Recently outbreaks of Crimean-Congo haemorrhagic fever in Pakistan's, Iraq and Dubai involved members of hospital staff. In an outbreak in Rawalpindi, Pakistan, in January 1976, 5 members of the hospital staff who had close contact with the fatal index case contracted the infection and 2 died. There were 7 tertiary cases, 5 of which were among members of the hospital staff and 2 were family contacts; all of them recovered. In Baghdad, Iraq, in 1979 a medical officer and a nurse were infected in hospital by spread from the index case; all 3 patients died. In Dubai in November 1979 the index patient died in the casualty ward of the Rashid Hospital soon after admission. There were 5 secondary cases with 2 deaths.

**Clinical picture**

The infection is usually transmitted to man by the bite of a tick, as in the case of the patient reported here, but an increasing number of cases have occurred among the medical and nursing staff caring for patients in hospital and in laboratory personnel carrying out investigations of these patients. In these cases the infection has apparently been acquired by contagion, particularly by contact with the patient's blood or blood-contaminated specimens. Exposure to the blood of infected animals, especially cattle and sheep, has led to severe and often fatal infections.
The incubation period is 2 - 7 days. The onset of the illness is sudden, with fever, chills, severe muscular pains, headache, vomiting and pain in the epigastric and lumbar regions. A haemorrhagic state develops from the 3rd to the 5th day and manifests as petechial haemorrhages or purpura in the skin, and bleeding from the mucous membranes manifests as epistaxis, haemoptysis, haematemesis, melaena and haematuria. At this stage the conjunctivae are injected, the face is flushed and the tongue is dry, often coated with dry blood. The pulse is slow in the beginning, but with continuing loss of blood becomes fast and feeble; the blood pressure drops and the heart sounds become weak — clear signs of impending shock and vascular collapse. The liver is enlarged and tender and there is tenderness over the epigastrium and splenic region. In patients who recover, the temperature falls between the 10th and the 20th day and bleeding stops, but convalescence is prolonged up to 4 weeks or longer. In fatal cases, death from massive haemorrhage and cardiac arrest occurs, usually 7 - 9 days after the onset of the illness. Massive haemorrhage into the gastro-intestinal tract, with scattered haemorrhages into the viscera, is found at autopsy.

The diagnosis is suggested on clinical grounds when the patient has a history of a tick bite or of exposure to ticks in the environment, and after an incubation period of 2 - 7 days develops an illness of sudden onset of muscle pains, headache, fever and a rapidly evolving severe illness with the development of a haemorrhagic state with bleeding from the mucous membranes and petechiae in the skin, associated with thrombocytopenia and leucopenia.

The diagnosis may be confirmed in the laboratory by intracerebral inoculation of baby mice with blood of a patient; the mice sicken about 1 week after inoculation. The virus is identified by using known specific Congo virus antisera in an immunofluorescent test. The development of antibodies in patients' serum as the illness progresses may be demonstrated in immunofluorescent tests using chamber slides with tissue culture cells infected with Congo virus.

The authors are grateful to Professor O. W. Prozesky, Director of the National Institute for Virology, and to Dr R. Swanepoel, Dr K. Struthers, Mrs E. Rossouw and Miss G. McGillivray, staff members of the high-security laboratory, and to Dr P. Jupp of the Arbovirus Unit of the National Institute for Virology for undertaking, as a matter of urgency, the investigations which led to the incrimination of the Congo virus as the cause of this patient's fatal illness. The authors are grateful to Dr E. R. Podlashuk, who first called attention to this patient, Mrs Podlashuk, who identified the tick as a species of *Hyalomma*, and to Mrs M. Anderson, who prepared the chart of its life cycle.

REFERENCES