

A review of the 1992-1993 Yellow Fever outbreak in Kenya and future management options

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Abstract

In 1992-1993 Kenya saw its first outbreak of yellow fever in 50 years in the Rift Valley Province. By March 1993, 55 cases of suspected yellow fever were identified, with 34 deaths. This is likely to be an underestimate of infected cases. This has immeasurable impact on the villages of the region, and economic repercussions for the nation. This report examines the management of the outbreak.

Following identification of the causative agent, a mass vaccination campaign and follow-up surveillance was initiated. However, more prompt action could have led to earlier detection of the emerging infection and perhaps limited its extent. There was considerable delay in responding to the initial reports of an unknown haemorrhagic fever. The initial surveillance system omitted a key hospital in the affected region where cases had presented earlier and there was little effort to control the vector mosquito, *Aedes africanus*.

A more efficient reporting and response system may have allowed for earlier recognition of the emerging epidemic. A greater awareness of the clinical syndrome of yellow fever and appropriate early collection of samples for diagnostic confirmation would facilitate the public health response. An education campaign on mosquito avoidance and control could have been directed at the immediate at-risk population. Reliance on an emergency mass vaccination campaign, driven by economic factors in impoverished nations, may allow for temporary control, but sub-optimal immunisation rates allow the opportunity for future outbreaks.

Keywords: Yellow fever, *Aedes africanus*, immunisation, vaccination

Introduction

The yellow fever virus is a flavivirus that may produce symptomatic illness with headache, fever, myalgia, nausea and vomiting. In some 15% of symptomatic infections, jaundice and mucosal bleeding occurs with a mortality of approximately 50% (WHO 2001). Yellow fever has been endemic in tropical West Africa for centuries and causes periodic epidemics. However, yellow fever had not been seen in Kenya since the two reported cases in 1943 (Okello *et al* 1993), until cases began to appear in 1992 and 1993.

In September 1992 cases of haemorrhagic fever appeared in the Kerio Valley region of the Rift Valley Province in Kenya (Sanders *et al* 1998). The cases demonstrated sylvatic transmission, where close contact occurred between humans and epizootic mosquitoes, predominantly *Aedes africanus* and *keniensis*. The cases were predominantly young people from local villages who had exposure to the woodlands area of the valley. Yellow fever virus was suspected to be enzootic in Kenya, as low-level immunity had been found in humans and monkeys (Reiter *et al* 1998). Severe drought in 1991-2 may have brought monkeys into closer proximity to human crops and water supplies within the region, and heavy rains in April 1992 would have increased the number of vector mosquitoes. The villagers in the area were at risk of yellow fever infection as they were non-immunised, despite recommendations by the World Health Organisation in 1988 and 1990 (WHO 1998) to introduce yellow fever vaccination to the Extended Programme of Immunisation for Africa. The potential for a large scale epidemic was realised, as the affected area had a new road constructed through it, and was approximately 250 kilometres from Nairobi, with a non-immune population of 1.5 million.

In January 1993, an epidemiological investigation to identify the cause of the haemorrhagic fever was established. By March 1993, some 55 cases were identified as confirmed or suspected yellow fever, with 34 deaths (Sanders *et al* 1998). In view of the propensity for under-reporting of disease in Africa, it is likely that there were greater numbers of afflicted cases.

The aim of this paper was to critically examine the management of the yellow fever outbreak in Kenya in 1992-1993. It explores the factors that lead to the outbreak and proposes a management plan to prevent future recurrences.

Social impact of the outbreak

The impact of this disease outbreak in the community is multi-faceted. The medical cost of hospitalisation can be difficult to quantify – 49% of the cases were diagnosed retrospectively (Sanders

et al 1998). Large outbreaks have the potential to overwhelm local health services, although this did not appear to be the case in Kenya, where most cases were identified late in the course of the outbreak and presented to a number of health facilities. The financial burden of hospitalisation, diagnostic testing and supportive treatment must be borne by the government or the patient's family. The loss of a productive member of the village (84% of cases were less than 40 years of age (Sanders *et al* 1998)) may have economic consequences for the village – loss of income, labour, and parents.

The outbreak of a potentially fatal disease has an impact on the psyche of the nation. Fear of nationwide spread of the disease can result in misinformation and bias. This outbreak in 1992-1993 was initially attributed to highland malaria (Kimani 2002, Okello *et al* 1993) - a more reassuring explanation for Kenya's unvaccinated population. The nation also suffered from the stigmata of a yellow fever outbreak. Tourism is a leading contributor to Kenya's income. Traditionally, tourist numbers fall once a disease outbreak reaches international recognition. This was seen following the 1992 death of a Briton from malaria in Kenya. Poor publicity resulted in 101,000 less British tourists to Kenya in 1992-1994, and an estimated loss of US \$104 million (Marine Medical Systems 1997). Kenya has now been declared a high-priority yellow fever region and the International Certificate of Vaccination against Yellow Fever is required by visitors entering and leaving Kenya. Unfortunately, forged certificates are being produced (Siringi 2002).

Management of the outbreak

When cases of haemorrhagic fever began to appear in the Kerio Valley region, the health authorities responded by establishing an investigative team to determine the cause of the outbreak. However, there was considerable delay between the reporting of cases and confirmation of yellow fever as the causative agent (Reiter *et al* 1998). The first cases were notified to the Kenyan Ministry of Health in September 1992. It was not until late January 1993 that serological testing at the Kenya Medical Research Institute (KEMRI) identified the cause as yellow fever, and samples were sent to the Centers for Disease Control and Prevention (CDC) in the USA for confirmation. Then a combined task force was established (Kenya Ministry of Health, KEMRI, WHO, CDC) to investigate the outbreak. Sentinel surveillance centres were established in five hospitals near the region. Hospital staff were advised to report suspect cases, utilizing a standard case-definition. They were also asked to report any cases of haemorrhagic fever dating back to September 1992. Surviving suspect cases were interviewed by the investigation team, and past medical records were examined. Blood samples were obtained where possible and sent to KEMRI and CDC for serology and virus isolation. However, only 47% of identified haemorrhagic fever cases had samples available for serological confirmation of yellow fever infection.

A mass vaccination campaign for the immediate at-risk population was implemented. This approach is similar to that taken by other African countries, such as Liberia, Nigeria, and Cote d'Ivoire, which have experienced yellow fever epidemics in recent years. It can be very effective, as seen in Sierra Leone in 1995 when one case was reported near the border with Liberia, which had experienced an epidemic several weeks before. Following rapid response and mass vaccination, no further cases were reported in Sierra Leone that year (WHO 1996).

Deficiencies are evident in the management of this outbreak. There was considerable delay in recognition of cases of yellow fever. This may have been due to inexperience in the primary health workers of the region and their failure to recognize symptoms. Clinical diagnosis of yellow fever is difficult in Africa, where many diseases have similar early presentations eg, malaria, hepatitis, typhoid, rickettsial disease. Furthermore, a generation of health workers had never seen a case of yellow fever in Kenya. These health workers were educated in recognizing haemorrhagic fever when the investigation team arrived. This could have been done sooner, with the provision of screening forms and questionnaires while the response team was being organized. The cases occurred in a rural area, where delayed presentation often occurs from reluctance to travel long distances to medical facilities, and cultural differences, where villagers may opt to see a local traditional healer before seeking western style medical centres. An educational campaign targeting the villagers and encouraging early presentation to hospital could have been implemented early in the response effort.

When cases were suspected to be yellow fever, there were delays in confirming the virus as the causative agent. Specimens were not collected early in the outbreak, and although cases of haemorrhagic fever were being reported from September 1992, there was no concerted effort to establish a diagnosis. Local laboratory facilities were not capable of performing the necessary diagnostic tests, and so specimens were sent to Nairobi and USA for further analysis. To add to the

confusion, some samples were found to be positive for Ebola Zaire antibody, but it was not determined whether this was past or recent infection (Okello *et al* 1993).

There was a marked delay in the government response to the disease outbreak – a period of almost five months. Possible reasons have already been alluded to. It is known that the political landscape had been unstable at the time. Kenya's first multi-party elections were held in December 1992 (Mulli 2005). There had been ethnic fighting in the Rift Valley Province before the election and a parliamentary committee report was being formulated.

The sentinel surveillance system initially established excluded a mission hospital in the affected region. It was later revealed that six fatal cases of haemorrhagic fever had presented to this hospital dating back to August 1992, but were not included in the investigation (Sanders *et al* 1998). Exclusion of this hospital introduces the possibility of subsequent cases of yellow fever remaining undiagnosed. The surveillance system was expanded to 13 sites by mid-1993 and up to 18 sites by 1994 (Sanders *et al* 1996).

Proposed Management Plan

Arboviral disease outbreak involves the interaction of hosts, vectors, pathogens and the environment. In responding to an outbreak, each of these factors needs to be considered in determining methods of breaking the transmission cycle.

The vector, *Aedes africanus*, was a known zoophilic mosquito. Although mosquito control methods had been used in Africa to limit the spread of malaria (Killeen *et al* 2004), DDT was officially banned in Kenya in 1985, and dieldrin was banned in 1992, due to concerns over accidental poisoning and environmental damage, despite still being widely available (Wandiga 2001). However, the use of organophosphates, such as malathion, in the situation of a potentially devastating epidemic would be reasonable. Mosquito control methods may help delay the spread of disease whilst immunisation programmes are being implemented, and have been used extensively in urban dengue epidemics (WHO 2000). They could have been implemented early during the Kenyan yellow fever outbreak to control adult mosquitoes and prevent the development of an intermediate transmission cycle in the villages. Simple, well-tried methods include fogging of vegetative areas (used for plantation workers in South America), and this could have been done in the areas surrounding the villages in Rift Valley. Spraying of the village houses may reduce the possibility of an urban cycle becoming established by anthropophilic vectors feeding on a viraemic patient.

It is conceivably difficult to destroy the mosquito breeding sites, as *Aedes africanus* is known to be tree-dwelling and was captured at heights of six metres in the woodlands canopy (Reiter *et al* 1998). Widespread destruction of the environment to diminish the habit of the vector mosquito or monkey host is not acceptable. However, much could be done to limit man's interaction with the mosquito habitat. A basic option would be to change the behaviour of the villagers in the affected area. If food was supplied to the village during the outbreak, then less people would need to venture into the woodlands for hunting and gathering. Alternatively, hunting responsibilities could be delegated to those villagers known to be immunised against yellow fever. Once the vector was known, villagers could have been instructed to avoid the woodlands during peak mosquito biting times. The use of permethrin-impregnated mosquito nets (D'Alessandro *et al* 1995), long-sleeved clothing and DEET based repellent are proven anti-mosquito measures, and could have been promoted. Supplies of such items may have been obtained from the local government or military. The aim of these interventions would be to minimize the risk of an urban cycle.

One of the notable features of this outbreak was the delay in presentation and diagnosis of cases. The villagers could be better educated through a media campaign to be alert to the signs and symptoms of yellow fever, and encouraged to attend medical facilities promptly should illness occur. The role of education and information during disease outbreak cannot be underestimated. Villagers could be provided with pamphlets and brochures in a local dialect. Public noticeboards could carry warnings and instructions on mosquito avoidance and what to do with suspected yellow fever cases. In communities with poor literacy, group teaching sessions could be arranged through trained volunteers or non-government organisations. In areas like Kerio Valley with its new roads (and therefore portal for viraemic humans), warning signs could be erected to warn travellers and visitors of the yellow fever risk. Such signs are used throughout the world, including remote areas in Australia, to warn of potential arbovirus transmission.

Mass emergency vaccination campaigns are an effective way of controlling sylvatic epidemics, as seen in Liberia in 2004. A stockpile of yellow fever vaccine could be utilized in such outbreaks, and WHO

recommendations were for one million doses to be stored (and rotated) for such events in a region (WHO 1998). This number was found to be grossly insufficient, and the recommendation has been changed to store six million vaccines. (12) Integration of yellow fever vaccination into Kenya's EPI is still recommended, and this did occur after 2000, although coverage is only partial (WHO 2003). Integration into a pre-existing programme could cost as little as US 17 cents per vaccine (Henderson 1998). However, many African nations, even in 2005, fail to achieve adequate immunization cover for the EPI diseases, let alone additional vaccine-preventable diseases. Unfortunately, frequent yellow fever outbreaks can result in diversion of stockpiles intended for the EPI, as occurred in 2001 (Africa News Service 2001).

Most notable in the YF outbreak in Kenya, was the delayed diagnosis and realization that an outbreak was occurring. The public health administration was found to be deficient and unprepared for a yellow fever outbreak. No suitable surveillance system existed for disease outbreak, and an incomplete one was hastily established. Direct communication between the Ministry of Health and all health facilities, and simultaneous training sessions of all health care workers within the affected region, would have expedited the identification of suspect cases. The establishment of direct contact public health telephone numbers or an electronic medium for disease notification may have made it more obvious to the ministry that an outbreak was emerging. Education of health workers was required, and did occur, although it was six months later before training workshops had been completed for the expanded surveillance programme (Sanders *et al* 1996). These one-day workshops would be better implemented at all health facilities within a region at the outset of the epidemic.

Further delays occurred when specimens were not available or readily processed for disease confirmation. Specimens needed to be forwarded to other centres for analysis. The availability of rapid tests, such as ELISA for IgM, at a local level would allow more prompt response whilst awaiting confirmation. The cost of upgrading small laboratories may be too burdensome, but regional centres like KEMRI should have the ability to provide confirmation testing. In 1994, an epidemiological support unit at KEMRI was established, and laboratory personnel undertook further training at CDC in Colorado (Sanders *et al* 1996).

In the long-term management of vector-borne disease, a sustainable plan to minimize human infection is required. Appropriate urban planning and reducing encroachment upon native habitats could be addressed in the long-term scheme of reducing interaction between man and vectors.

The implementation of a disease outbreak response in an impoverished country is difficult where resources and infrastructure are poor, mistrust and misinformation abounds, and where political upheaval or conflicting health priorities are confounding issues. Money and infrastructure are of paramount importance. Over the past few decades, many NGO, international agencies, philanthropists like Bill Gates (Dawes 2000) and corporate organisations have contributed to financial and medical aid to Africa. Continued lobbying and motivation to see programmes implemented and maintained is crucial to the survival of any health disaster plan in Africa. The international community plays a large role; British Airways has a partnership with UNICEF to collect donations (British Airways 2005). Tourists to the region could be charged a levy specifically to combat vaccine-preventable diseases. Certainly, considerable financial assistance is required to repair the 'holes' in the system that could see Kenya experience another yellow fever outbreak.

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