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Hepatitis E Outbreak, Dadaab Refugee Camp, Kenya, 2012

To the Editor: Hepatitis E virus (HEV) is transmitted through the fecal-oral route and is a common cause of viral hepatitis in developing countries. HEV outbreaks have been documented among forcibly displaced persons living in camps in East Africa, but for >10 years, no cases were documented among Somali refugees (*1*,*2*). On August 15, 2012, the US Centers for Disease Control and Prevention (CDC) in Nairobi, Kenya, was notified of a cluster of acute jaundice syndrome (AJS) cases in refugee camps in Dadaab, Kenya. On September 5, a CDC epidemiologist assisted the United Nations High Commissioner for Refugees (UNHCR) and its partners in assessing AJS case-patients in the camp, enhancing surveillance, and improving medical management of case-patients. We present the epidemiologic and laboratory findings for the AJS cases (defined as acute onset of scleral icterus not due to another underlying condition) identified during this outbreak.

Dadaab refugee camp is located in eastern Kenya near the border with Somalia. It has existed since 1991 and is the largest refugee camp in the world. Dadaab is composed of 5 smaller camps: Dagahaley, Hagadera, Ifo, Ifo II, and Kambioos. As of December 2012, a total of 460,000 refugees, mainly Somalians, were living in the camps; >25% were recent arrivals displaced by the mid-2011 famine in the Horn of Africa (*3*). Overcrowding and poor sanitation have led to outbreaks of enteric diseases, including cholera and shigellosis (*4*); in September 2012, an outbreak of cholera occurred simultaneously with the AJS outbreak.

During July 2–November 30, 2012, a total of 339 AJS cases were reported from the camps and 2 nearby villages: 232 (68.4%) from Ifo II, 57 (16.8%) from Kambioos, 26 (7.7%) from Ifo, 12 (3.5%) from Dagahaley, 10 (3.0%) from Hagadera, and 1 each (0.6%) from the nearby Kenyan villages of Biyamadow and Darkanley. The epidemic curve of the outbreak is shown in the Figure.

Of the 339 AJS case-patients, 184 (54.3%) were female. The overall median age was 23.5 years (range 1 month–91 years). The median age among female and male residents was 24 years and 20 years, respectively. Among the 134 women of reproductive age (15–49 years), 72 (53.7%) reported being pregnant; the median gestational age was 17.4 weeks (range 8.7-35.3 weeks). Death was reported for 10 of the 339 case-patients (case-fatality ratio 2.9%), 9 of whom were postpartum mothers (case-fatality ratio 12.5%) and 1 a 2-year-old child.

Serum samples were obtained from 170 (50.1%) AJS case-patients for testing at the Kenya Medical Research Institute/CDC laboratories in Nairobi, Kenya. Of the 170 samples, 148 were tested for hepatitis E virus (HEV) IgM by using an ELISA

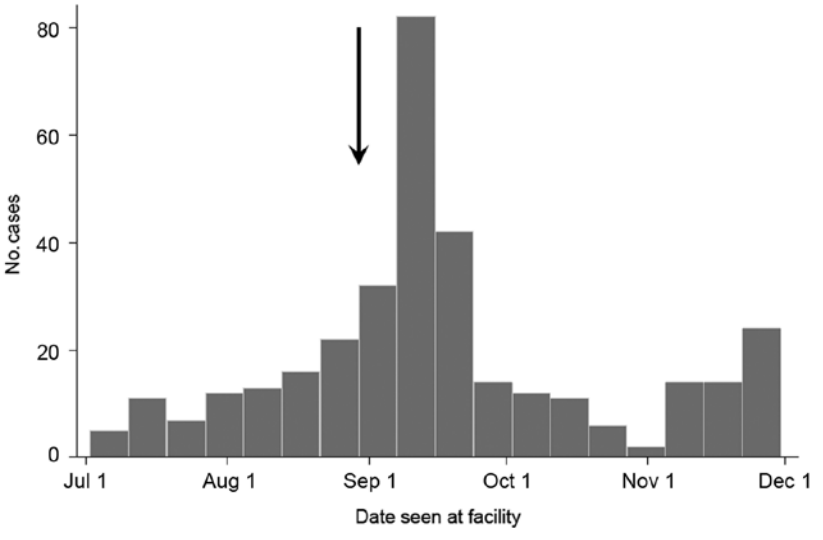

Figure. Cases of acute jaundice syndrome, Dadaab, Kenya, July–November 2012. The arrow indicates the point at which outbreak control measures (e.g., construction of new latrines and hygiene messaging) were initiated by health authorities.

(Diagnostic Systems, Saronno, Italy), and 93 were tested for HEV RNA by using the GeneAmp Gold RNA PCR Reagent Kit (Applied Biosystems, Foster City, CA, USA). Of the 170 samples tested, 131 (77.1%) were positive for HEV IgM, HEV RNA, or both: 120 (81.1%) of 148 tested for HEV IgM and 48 (51.6%) of 93 tested for HEV RNA were positive. In response to the outbreak, UNHCR and partners initiated control measures, including training of health care workers, increasing community awareness, improving hygiene promotion activities, and hastening latrine construction.

The outbreak also affected refugee resettlement to the United States and other countries. At the onset of the outbreak, ≈100 Dadaab refugees per month were scheduled for US resettlement. The incubation period for HEV is 15–60 days (*5*); thus, there was concern that refugees could become ill in transit or within weeks of US resettlement. Acute HEV infection, including progression to fulminant hepatitis, had been reported among travelers returning from regions where the disease is endemic (*6*). As a precaution, the International Organization for Migration and CDC conducted heightened AJS surveillance during pre-departure and arrival health screenings. As of February 2013, no cases of AJS were reported among refugees from Dadaab who resettled in the United States.

Dadaab has faced grave insecurity: aid workers were abducted from the camp in late 2011, and Dadaab has experienced numerous blasts from explosive devices (*7*). Thus, UNHCR and CDC have been limited in their capacity to collect data and conduct a thorough outbreak investigation to identify risk factors. An earlier study in the Shebelle region of Somalia suggested an increased incidence of HEV during the rainy season and elevated risk for infection in villages dependent on river water (*8*). Further evaluation is needed to identify the risk factors for HEV transmission and HEV-associated deaths in this region, including the role of person-to-person transmission. UNHCR and CDC investigations of HEV outbreaks in refugee camps in southern Sudan may provide data to answer these questions.

HEV is believed to have infected humans for centuries (*9*); however, the reemergence of the disease in refugee camps is a major concern because of the difficulty in implementing effective preventive measures under camp conditions. Point-of-care tests will be useful for rapidly detecting outbreaks and could potentially save lives. The progress made in developing effective vaccines is encouraging (*10*). Once available, HEV vaccination should be prioritized in this population, especially for pregnant women.

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Wild Poliovirus Importation, Central African Republic[1]

To the Editor: Since the Global Polio Eradication Initiative was launched in 1988, indigenous transmission of wild poliovirus (WPV) has been interrupted in all countries except Afghanistan, Pakistan, and Nigeria (*1*). However, during 2003–2011, outbreaks resulting from importation of WPV occurred in 29 previously polio-free countries in Africa, including Central African Republic (CAR) (*1–3*). In 2011, 350 WPV cases were reported from 12 countries in Africa, a 47% decrease from the 657 cases reported by 12 countries in Africa in 2010 (*1*).

In CAR, the last case of poliomyelitis caused by indigenous transmission of wild poliovirus was reported in 2000, but importation of WPV type 1 has been reported (*4*). We describe the importation of WPV1 and WPV3 into CAR during successive events in 2008, 2009, and 2011.

To investigate importation of WPV into CAR, we conducted a study using fecal samples collected from patients in CAR who had acute flaccid paralysis (AFP) during 2008–2011. The samples were analyzed for virus isolation, typing, and intratypic differentiation at the Regional Reference Laboratory for Polio, Institut Pasteur de Bangui, using World Health Organization (WHO) standard procedures (*5*). Isolated WPV strains were sent to the Centers for Disease Control and Prevention (Atlanta, Georgia, USA) or the National Institute for Communicable Diseases (Johannesburg, South Africa) for sequencing according to WHO guidelines (*6–8*). Cases were classified as laboratory confirmed or polio-compatible according to WHO recommendations; a polio-compatible case was defined as AFP for which stool samples were not adequate or a situation in which the patient was lost to follow up or had residual paralysis 60 days after testing.

Of 141 AFP cases from 2008, three, from Bangui, Ouham, and Ouaka districts, were laboratory confirmed as WPV1; this cluster was designated B2D1B (Figure). Sequencing results showed that the virus in this cluster belonged to the South Asia A (Indian) genotype, which was circulating in Angola and Democratic Republic of Congo at that time (Figure).

Of 163 AFP cases from 2009, 14 in Ouham-Pende district were laboratory confirmed as WPV3; this cluster was designated D2B2B1. Sequencing results showed that the virus in this cluster belonged to West Africa B genotype, which was circulating in Nigeria and southern Chad at that time (Figure).

Of 142 AFP cases from 2011, four in Ouham district were laboratory confirmed as WPV1; this cluster was designated I6C2B4C1A2. Sequencing results showed that the virus in this cluster belonged to West Africa B genotype, which was circulating in south Chad and Nigeria at the time (Figure).

The importation of wild poliovirus strains into CAR appeared to follow 3 different routes. In 2008, WPV1 originated from Democratic Republic of Congo and was first detected in the capital, Bangui, which is located in

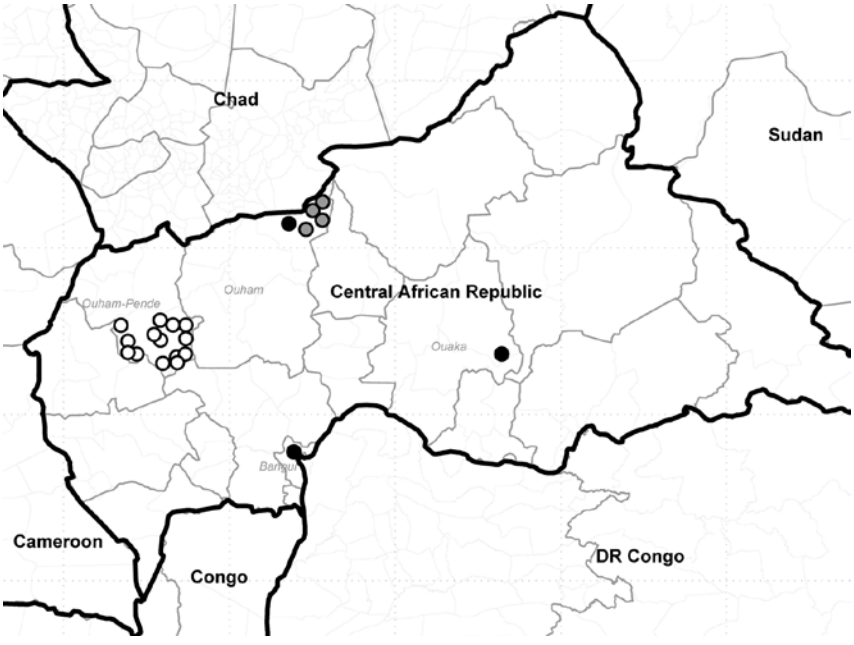

Figure. Clusters of polio cases caused by wild poliovirus importations, Central African Republic, 2008–2011. Each circle represents 1 case of acute flaccid paralysis confirmed as polio. Black circles, cluster B2D1B, 2008 poliovirus (PV) type 1 SOAS importation from Democratic Republic of Congo (DR Congo); white circles, cluster D2B2B1, 2009 PV3 WEAF-B importation from Nigeria and southern Chad; gray circles, cluster I6C2B4C1A2, 2011 PV1 WEAF-B importation from southern Chad.

[1]Data from this report were presented to the Global Polio Laboratory Network, Geneva, Switzerland, and at the First International Conference of the African Society of Laboratory Medicine, 2012 Dec 1–7, Cape Town, South Africa.