Epidemiological characteristics of a prolonged hepatitis E outbreak in three refugee camps in South Sudan

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Background

MSF suspected a hepatitis E outbreak in Jamam camp, Maban County, South Sudan, in early July 2012 after reports from routine household surveillance that two pregnant women and one child had died from “yellow eyes”. In subsequent weeks, cases were detected in the newly-settled neighbouring Batil and Gendrassa camps. Laboratory confirmation of hepatitis E virus genotype 1 came in August 2012. Here we describe MSF’s response, the epidemiological characteristics of the outbreak and the impact of water, sanitation and hygiene (WASH) interventions in controlling the epidemic. This study met the criteria approved by the MSF Ethics Review Board for analysis of routinely collected programme data.

Project

The case definition was scleral icterus (jaundice) and all suspected hepatitis E cases were referred to MSF facilities. MSF’s response was mainly clinical case management (supportive care - there is no treatment for hepatitis E), active case-finding using outreach teams and targeted screening of pregnant women. Partner agencies were responsible for WASH activities to control faecal-oral transmission. MSF gap-filled these activities with additional water purification, latrine construction, hand-washing equipment, soap distribution and health education.

Epidemiological characteristics and outcomes

The first peaks were observed in early September 2012 but larger second peaks emerged in January 2013. By the end of January 2013, 5370 cases, including 272 (5.1%) pregnant/post-partum women, had been recorded. Of these, 610 (11.4%) were admitted to hospital due to severe complications such as hepatic failure and coma and 115 died (2.1%) – i.e. a clinical attack rate of 7.7% and a case fatality rate of 2.1%. The case fatality rate among pregnant/post-partum women was more than 5x higher at 11.4%. Females predominated slightly (52.5%) and most symptomatic cases were young adults (15-49 years). 65% of all cases were recorded in the newer, larger Batil camp and hepatitis E appears to have spread to all villages within the camps. Potential reasons for the second epidemic peaks have been explored. There was a different spatial distribution between the first and second peaks but this could be explained by population movement. A variable WASH response and significant person-to-person transmission may have played a role in the second peak whereas a non-hepatitis E infectious agent and an influx of new susceptible refugees probably did not.
Conclusions

Prospective community surveillance was critical for the early detection of this outbreak and for continued active case-finding and health education. Community engagement was key for prevention activities and acceptance of medical interventions. However, the failure to control this outbreak was likely due to a late and inadequate WASH response, as in previous hepatitis E outbreaks. If timely and effective WASH responses are unfeasible then new hepatitis E vaccines should be explored for refugee settings.