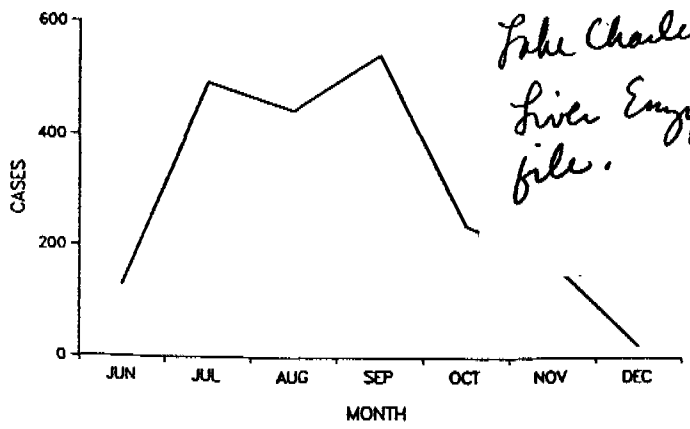
Editorial Note: Non-A, non-B hepatitis, which continues to be a diagnosis of exclusion, is considered to have two distinct forms, which are transmitted by different routes and presumably caused by different viruses. The first, initially recognized as post-transfusion non-A, non-B hepatitis, is seen commonly in North America and Europe, is epidemiologically similar to hepatitis B, and is recognized most commonly after blood transfusions and parenteral drug abuse. The second, enterically transmitted non-A, non-B hepatitis, is transmitted by the fecal-oral route. This disease is known to cause large outbreaks of viral hepatitis and has been reported in the Indian subcontinent (*2-7*), Burma (*8*), and Algeria (*9*). Frequently, large outbreaks have been linked to a fecally contaminated water source or have occurred after heavy rains in areas without systems for adequate sewage disposal. Person-to-person transmission can occur.

Enterically transmitted non-A, non-B hepatitis has several characteristic epidemiologic features. Its incubation period is approximately 40 days (as opposed to 30 days for hepatitis A and 60-180 days for hepatitis B). Clinical disease is common among adults, but infrequent among children. Pregnant women have a dramatically high mortality rate. Large outbreaks of acute viral hepatitis among adults in areas where the population is immune to hepatitis A should alert public health authorities to the presence of enterically transmitted non-A, non-B hepatitis.

FIGURE 2. Reported cases of jaundice among Ethiopian refugees, by month — eastern Sudan, June-December 1985



John Charles
Liver Enzyme
file.

SL 043153

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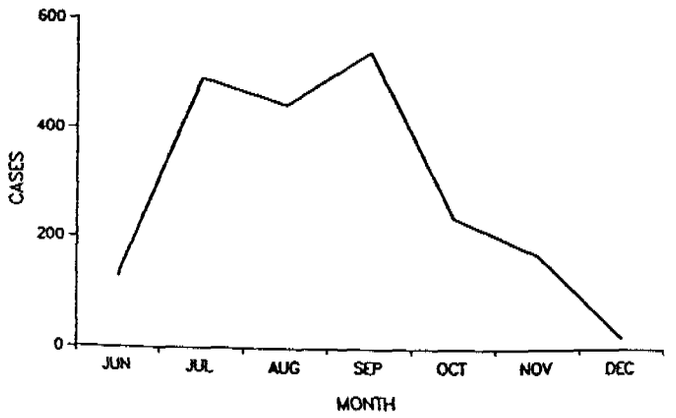

Signs and symptoms of enterically transmitted non-A, non-B hepatitis are similar to those of other forms of viral hepatitis, although generalized pruritus may be more common. The majority of patients who are not pregnant recover completely, and there is no evidence of chronic liver disease as a long-term sequela. Outbreaks of disease may be identified by the suggestive epidemiologic pattern (especially the high mortality rate among pregnant women) and the exclusion, through serologic testing, of other forms of viral hepatitis. Post-transfusion non-A, non-B hepatitis has not been documented in communitywide outbreaks.

Currently, no serologic test is available for diagnosis; however, 27- to 30-nm virus-like particles have been found by IEM in stool samples of patients in the early acute phase of infection (*1,7,10*), and hepatitis can be induced in two different species of primates with this agent. Acute-phase antibody in sera may also be demonstrated by IEM.

In an outbreak situation, emphasis must be placed on preventing transmission. Water sources should be examined for fecal contamination. If the water supply is contaminated, all water should be boiled or chlorinated before consumption. Efforts to reduce person-to-person transmission by improving sanitation should be stressed. Immune globulin (IG) manufactured in the West does not appear to be effective in preventing disease. The efficacy of IG from endemic areas is unknown.

These reports mark the first time that this disease has been described as a problem in refugee camps and the first time that the characteristic virus-like particles have been identified in Africa. Refugee camps represent a fertile setting for the transmission of enterically transmitted non-A, non-B hepatitis. These camps usually have inadequate sanitation and are overcrowded. While contaminated drinking water was not a factor in this outbreak, this problem may exist in other refugee camps. Fecally contaminated, standing rainwater may have facilitated transmission of disease at Tug Wajale B. Finally, refugee camps are sites of contact between susceptible refugees, who may have come from remote areas, and refugees who have come from areas where this virus is endemic. Staff members working in refugee camps are also at risk for acquiring this disease and should be careful to wash their hands after contact with patients and before eating and smoking. Because of poor sanitary conditions in these camps, enterically transmitted non-A, non-B hepatitis, like other enteric diseases, is likely to be difficult to control.

References

1. Decock KM, Bradley DW, Sandford NG, et al. Epidemic non-A, non-B hepatitis in patients from Pakistan. Ann Intern Med 1987;106:227-30.
2. Viswanathan R. Infectious hepatitis in Delhi (1955-56): a critical study—observations in pregnant women. Indian J Med Res 1957(suppl);45:1-76.
3. Sreenivasan MA, Banarjee K, Pandya PG, et al. Epidemiological investigations of an outbreak of infectious hepatitis in Ahmedabad City during 1975-76. Indian J Med Res 1978;67:197-206.
4. Wong DC, Purcell RH, Sreenivasan MA, Prasad SR, Pavri KM. Epidemic and endemic hepatitis in India: evidence for non-A, non-B hepatitis virus etiology. Lancet 1980;2:876-8.
5. Tandon BN, Joshi YK, Jain SK, Gandhi BM, Mathiesen LR, Tandon HD. An epidemic of non-A non-B hepatitis in north India. Indian J Med Res 1982;75:739-44.
6. Khuroo MS. Study of an epidemic of non-A, non-B hepatitis: possibility of another human hepatitis virus distinct from post-transfusion non-A, non-B type. Am J Med 1980;68:818-24.
7. Kane MA, Bradley DW, Shrestha SM, et al. Epidemic non-A, non-B hepatitis in Nepal: recovery of a possible etiologic agent and transmission studies in marmosets. JAMA 1984;252:3140-5.
8. Myint H, Soe MM, Khin T, Myint TM, Tin KM. A clinical and epidemiological study of an epidemic of non-A non-B hepatitis in Rangoon. Am J Trop Med Hyg 1985;34:1183-9.
9. Belabbes H, Benatallah A, Bouguermouh A. Non-A, non-B epidemic viral hepatitis in Algeria: strong evidence for its water spread [Abstract]. In: Vyas GN, Dienstag JL, Hoofnagle JH, eds. Viral hepatitis and liver disease. Orlando Florida: Grune and Stratton, Inc, 1984:637.
10. Balayan MS, Andjaparidze AG, Savinskaya SS, et al. Evidence for a virus in non-A, non-B hepatitis transmitted via the fecal-oral route. Intervirology 1983;20:23-31.