Local transmission of hepatitis E virus in Australia: implications for clinicians and public health

Outbreak indicates that HEV should be considered in cases of acute hepatitis. irrespective of the patient's travel history

n this issue of the MJA, Yapa and her colleagues report an outbreak of locally acquired hepatitis E virus (HEV) in Australia.1 The authors describe 24 cases of serologically confirmed HEV infection in people who had not travelled overseas during the HEV incubation period. Of these, 17 individuals could be epidemiologically linked to a single restaurant; HEV sequencing studies were consistent with a single source outbreak. Seven other locally acquired infections were also identified that were not linked to this restaurant; in one case, HEV RNA was detected in a pork liver sausage locally produced in Australia. HEV has previously been detected in Australian pigs.2



HEV causes an acute, self-limiting hepatitis, similar to hepatitis A virus (HAV) infection, and can be difficult to distinguish clinically from other causes of acute liver injury.3 Infection during pregnancy is associated with a particularly high mortality rate. As for HAV, resolution of HEV infection generally confers protective immunity. HEV is endemic in many low and middle income countries in Asia and Africa, with seroprevalence rates of up to 20-45% reported in adults. 4-8 Outbreaks are typically cyclic, and often associated with seasonal heavy rainfall, because of the disruption of clean water supplies. HEV genotypes 1 and 2, which infect only human beings, are responsible for most outbreaks. Travel to endemic countries remains a cardinal clue in the differential diagnosis.7

Locally acquired HEV infection has been thought to be very rare in Australia. However, there have been a number of sporadic cases reported in Western countries, not associated with a history of travel to diseaseendemic areas.8-10 An unexpectedly high prevalence of HEV-specific antibodies among blood donors has been observed in Europe and the United States. Undercooked pork and wild boar products have been implicated as sources of zoonotic transmission, typically with genotype 3 HEV, as described by Yapa and colleagues.1 Genotype 3 HEV has also been associated with chronic hepatitis in immunosuppressed individuals, including organ transplant recipients,7,11,12 in whom chronic

hepatitis E may cause progressive liver disease.3 Reducing immunosuppression or treatment with ribavirin or pegylated interferon-α can achieve viral clearance; very recently, sofosbuvir was shown to have antiviral activity against HEV.13

The report by Yapa et al¹ indicates the need to consider locally acquired HEV as a cause of acute hepatitis in Australian patients. We suggest HEV serological testing should be requested in cases of acute hepatitis where the initial diagnostic panel is negative, including for people who have not travelled abroad. Reference laboratories can perform HEV nucleic acid testing, but HEV RNA is generally only detectable in blood during the first week of infection, and in stool for 2 weeks after symptom onset; faecal shedding generally corresponds with infectiousness. All suspected and confirmed cases require notification to public health agencies. The reported case series also reinforces the need to adhere to best practice food preparation standards.

The report of locally acquired HEV infection in this edition of the Journal requires a considered Australian response, to educate clinicians, to provide laboratories with the necessary resources, and to promote public health measures that limit food-borne transmission of the virus. This outbreak lasted 9 months and shows that HEV transmission should be considered an ongoing risk in Australia.

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