Hepatitis A outbreak associated with kava drinking

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Abstract

Hepatitis A is caused by the hepatitis A virus (HAV), with transmission occurring through the faecal-oral route. In May 2013, a case of hepatitis A infection was reported to a Western Australian regional public health unit, with infection acquired in Fiji. Following this, 2 further cases were linked to the index case by kava drinking and 1 further case was a household contact of a secondary case. This outbreak highlights that the preparation of kava drink and/or the use of a common drinking vessel could be a vehicle for the transmission of HAV. *Commun Dis Intell* 2014;38(1):E26–E28.

Keywords: hepatitis A, outbreak investigation, kava

Introduction

Hepatitis A is an acute infection of the liver caused by hepatitis A virus (HAV). It is predominantly transmitted person-to-person via the faecal-oral route.[1] Sources of infection include contaminated water, infected food handlers and raw or undercooked food sources. A case is considered infectious from a few days before the onset of prodromal symptoms to 1 week after the onset of jaundice or 2 weeks after the onset of the prodromal symptoms, whichever comes first.[2]

In Western Australia, hepatitis A is a notifiable disease. The role of the public health unit (PHU) is to undertake enhanced surveillance of cases to identify the source of infection and to implement measures to reduce further transmission. In the Western Australia Goldfields Region, 36 cases of hepatitis A were reported between 2003 and April 2013, an average of less than 4 cases per year.[3] In May 2013, the PHU was informed of a laboratory confirmed case of hepatitis A in a 40-year-old Fijian male (case 1). The PHU was notified 34 days later of 2 further laboratory confirmed cases of HAV (cases 2 and 3). A 4th case (case 4) was notified in late July.

This report describes the investigation into a cluster of 4 cases of HAV infection.

Methods

Follow-up was conducted for all cases by the PHU staff in accordance with current enhanced surveillance guidelines, using a standardised questionnaire to determine epidemiological links and/or high risk activities. Blood specimens for all cases were sent to the Victorian Infectious Diseases Reference Laboratory (VIDRL), for HAV genotyping to be performed, as is the standard practice for all locally acquired cases of HAV. The investigation was performed as part of routine public health work.

Results

On 26 May 2013, the index case presented with anorexia, nausea, vomiting, lethargy, frontal headache and fever. The index case had travelled to Fiji 4 weeks prior, with symptoms developing 18 days after his return. Clinical examination revealed mild jaundice, noted on the 2nd day of admission. Blood tests showed elevated liver function tests (LFTs) and positive HAV IgM. He was treated symptomatically and his hospital stay was uneventful. He was discharged on day ten.

The PHU interview revealed that during his stay in Fiji, the index case resided in a remote village with poor sanitary conditions including makeshift bathrooms, pit toilets and no running water. He also described consuming street food and untreated drinking water. He had not previously been vaccinated against HAV. The index case did not identify any close contacts who may be at risk of transmission from him, such as immediate family, sexual contacts or persons who consumed food that he had prepared and had not been further cooked. He was provided with verbal and written information regarding HAV, including the infectious period and routes of infection.

On 4 July 2013, 2 further cases of hepatitis A were reported to the PHU by the local hospital medical team. Case 2 presented to the Emergency Department on 1 July 2013 with an 11 day history of fever, myalgia, nausea, vomiting, general fatigue and a 6 day history of jaundice. Investigations showed elevated LFTs and positive HAV IgM and IgG. He was managed symptomatically and was discharged on day four. During interview, case 2 revealed that he had drunk kava with the index case in June, 1 day after the index case was discharged from hospital and 8 days after his onset of jaundice. Case 2 reported that the kava drink was prepared by the index case and they shared a communal drinking vessel.

Case 3 presented to the local hospital the same week as case 2, complaining of a 1 week history of nausea, vomiting and generalised fatigue. He was later noted to be mildly jaundiced with raised LFTs and was positive for HAV IgM and IgG. His recovery was uneventful and he was discharged home on day six. During extensive interviewing with case 3 it was confirmed that he too had shared kava with 2 male friends, later found to be the index case and case 2.

In late July a further case (case 4) was reported to the PHU. Case 4 was the 8-year-old child of case 3. Case 4 presented to the local hospital with a 5 day history of abdominal pain, headache followed by fever, nausea and vomiting. Case 4 had been given HAV vaccination 17 days prior to becoming symptomatic. Hepatitis serology detected hepatitis A IgM and IgG antibodies. Transmission was likely due to normal household activities.

The temporal relationship between all 4 cases is shown in the Figure.

The results of HAV genotyping from blood specimens showed that all 4 cases had the same HAV genotype (IA) and with 100% sequence identity.

Discussion

Kava is an unusual reported vehicle of HAV infection. This outbreak revealed that kava drinking was linked with transmission of 2 cases of HAV infection. Mixing of kava was described by one of the cases as a process by which kava root powder is placed in a muslin cloth and soaked in a vessel filled with cold water. Manual extraction is used, with repeated squeezing of the cloth to extract kava. A second vessel is then used to dip into the mixing vessel and pass around as a communal drinking cup. The source of the HAV was most likely the index case during preparation of kava and/or via the shared drinking vessel.

Multiple interviews assisted in obtaining information to link these cases. At the time of the interview with the index case, a history of kava drinking was not elicited. In contrast, multiple interviews were performed with the subsequent adult cases, which elicited the history of kava sharing. The 3 adult cases were all reluctant to discuss kava use. This reluctance may have been due to cultural differences, a perceived disapproval of the activity or because its importation is restricted. The current standardised questionnaire for hepatitis A is generic and as a result does not identify different cultural practices as a risk factor for the transmission of HAV.

Of concern in this outbreak is the time of apparent infectivity of the index case and that despite provision of written and verbal information to the index case he participated in a high risk activity a day after discharge, facilitating transmission of HAV. The index case participated in kava drinking 8 days after onset of jaundice and 10 days after the onset of his prodromal symptoms. The *Hepatitis A National Guidelines for Public Health Units* outlines the infectious period of cases as being a 'few days before onset of prodromal symptoms to a few days after onset of jaundice and non-infectious 1 week after onset of jaundice or 2 weeks after onset of prodromal symptoms, whichever comes first'.[2] Applying these timeframes to the information provided by the 3 adult cases, case 2 and 3 were exposed outside of the infectious period of case 1. This has implications for the usual advice given to patients. While it is imperative to ensure that an accurate timeline of symptom onset is ascertained, a conservative approach should be used when discussing this timeline with patients. The emphasis should be on preventative measures such as hand hygiene and avoiding high risk activities, rather than the length of time during which they must exercise this caution.

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References

1. Heymann DL ed. *Control of Communicable Diseases Manual*. 19th edn. Washington D.C: American Public Health Association; 2008.
2. Western Australian Department of Health. Hepatitis A: National Guidelines for Public Health Unit, Government of Western Australia, Department of Health OD 0228/09t. 2009. [online]Accessed on 22 October 2013. Available from: http://www.health.wa.gov.au/circularsnew/pdfs/12564.pdf
3. Western Australian Department of Health. Western Australian Notifiable Infectious Diseases Database (WANIDD). Data extracted 1 August 2013.

Figure: Timeline of 4 cases of hepatitis A, Western Australian Goldfields Region, 2013