A community-wide hepatitis A outbreak in the Shoalhaven region, New South Wales

Timothy Heath[1,2]*, Desolie Lovegrove*[3]*, Victoria Westley-Wise*[3] *and Christine Roberts*[2]

Abstract

We investigated a community-wide outbreak of hepatitis A virus infection in the Shoalhaven region of the New South Wales south coast. Epidemiological features of the outbreak suggested that transmission was predominantly person-to-person. These included: the prolonged course of the epidemic, the bimodal age-specific attack rate, the lower socioeconomic risk groups affected, and the large proportion of cases who reported prior contact with another case. Although widespread use of post-exposure immunoprophylaxis appeared to be effective in preventing symptomatic infection in individuals, it did not rapidly halt the outbreak. We review methods of mass intervention for community-wide outbreaks of hepatitis A virus infection, and define priorities for investigation of future outbreaks. *Comm Dis Intell 1997;21:1-4.*

Introduction

The Shoalhaven is a coastal region in south-eastern New South Wales, and is one of four local government areas in the Illawarra Area Health Service (the Illawarra). The townships of Nowra and Bomaderry are located just north and south of the Shoalhaven River (combined population of 22,000). In late February 1996, the Illawarra Public Health Unit received the first of a series of notifications of hepatitis A virus (HAV) infection. Following 13 further HAV notifications over the following month, active surveillance was commenced in the Shoalhaven and surrounding districts, and an investigation was undertaken to examine whether the outbreak was caused by a common source, and whether any populations could be targeted for mass vaccination.

Methods

In late March, all general practitioners, hospital chief executive officers, emergency and infection control departments, and pathology laboratories in the Shoalhaven region were notified of the outbreak and asked to report new or suspected HAV infections. Cases were defined as persons who had either lived in or visited the Shoalhaven any time after 6 January 1996 (seven weeks prior to notification of the first case), with:

- Demonstration of HAV specific IgM in any single serum sample, or a four-fold rise in HAV IgG

1. Western Sector Public Health Unit, 13 New Street, North Parramatta New South Wales 2151
2. National Centre for Epidemiology and Population Health, Australian National University, Canberra
3. Illawarra Public Health Unit, Wollongong

Contents

A community-wide hepatitis A outbreak in the Shoalhaven region, New South Wales 1
Timothy Heath, Desolie Lovegrove, Victoria Westley-Wise and Christine Roberts

Surveillance data in *CDI* 5
Margaret Curran and Ana Herceg

CDI Instructions to authors 9
Communicable Diseases Surveillance 10
Overseas Briefs 16

titres in sequential sera (a definite case); or

- Household contact of definite case and new onset of jaundice or abnormal liver function (elevated transaminases), or abdominal pain and fever (a probable case).

Adult cases were defined as those aged 18 years and over. A contact was defined as any person who stayed overnight in the same household, shared meals with a case in the same household, or was the sexual partner of a case during their infectious period. The infectious period of a case was defined as two weeks before until one week after onset of symptoms. All staff and children attending child-care centres were considered contacts if they were in proximity to a case during the case's infectious period.

Age, sex, Aboriginality, residential address, food handling, contact with child-care, hospital admission, mode and date of notification and diagnosis were recorded from interviews undertaken with all cases. A more detailed structured questionnaire was completed for 35 cases, which recorded case activities during the two to seven weeks prior to onset of symptoms, including known or suspected risk factors for HAV. We obtained HAV notification data for the years 1991 to 1995 from the New South Wales Health Infectious Diseases Surveillance System database.

Age-specific rates were calculated using the Australian Bureau of Statistics (ABS) estimated mid-year populations for 1994. ABS 1991 census data were used to calculate rates for the Aboriginal population, and to compare case demographics with the entire Shoalhaven population. Analyses were performed using Epi Info version 6.03.

Interventions

Regular updates of the outbreak's progress were sent to local general practitioners, hospitals and laboratories. Information sheets, which outlined HAV's mode of transmission and promoted hand washing, were distributed to cases, contacts, schools and workplaces. Information was also provided to the Aboriginal Land Council, Aboriginal health workers, and Nowra Community Health. All child-care centres in the area were reminded that HAV vaccination is recommended routinely for child-care workers, and efforts were made to make this available. In accordance with New South Wales Health guidelines, intramuscular normal human immunoglobulin (NHIG) was recommended for household, sexual, and child-care contacts of cases. In one child-care centre, where three definite cases of HAV occurred, NHIG was recommended for all 80 children and for all staff. The local council and Illawarra Public Health Unit issued press releases in early May, which explained the mode of transmission of HAV and promoted hand washing.

Results

To 20 May 1996, fifty-eight HAV cases were identified in the Shoalhaven, a crude rate of 76 per 100,000 population. This compared with 12 cases notified in the rest of the Illawarra Area Health Service during the same period, a rate of 3.3 per 100,000 population. During the preceding years 1991 to 1995, there had been five HAV notifications in the Shoalhaven region.

There were 55 definite and three probable cases. The progress of the outbreak up to 20 May is illustrated in Figure 1. Laboratories and doctors notified 52% and 40% of cases respectively; the remaining cases were notified by hospital infection control staff or were detected during case investigations. Most cases (64%) were residents of Bomaderry or Nowra, but smaller clusters were encountered in a number of neighbouring coastal townships. The sex distribution of cases was approximately equal (47% female). The median age was 19 years (range 2-55). The majority of cases were adults (52%), but notification rates were highest for children aged 5-14 years (Figure 2). Twenty-four were school children, and three were in child-care. Eighteen (31%) cases were Aboriginal, a rate of 1,200 per 100,000 Aboriginal population. No cases were commercial food handlers. Twelve (22%) were admitted to hospital.

The more detailed questionnaire found that respondents were more likely to be Aboriginal, living with children or with six or more people, or unemployed than the entire Shoalhaven population (Table). During the period that infection probably occurred - two to seven weeks prior to onset of symptoms - 18 (51%) cases had contact with another definite case. Although five cases had eaten raw oysters, these cases did not appear related to each other (the oysters were harvested from rocks at different sites along the Shoalhaven coastline). Recreational swimming was not a frequent

Figure 1. Hepatitis A cases in the Shoalhaven region, by week of notification, to 20 May 1996

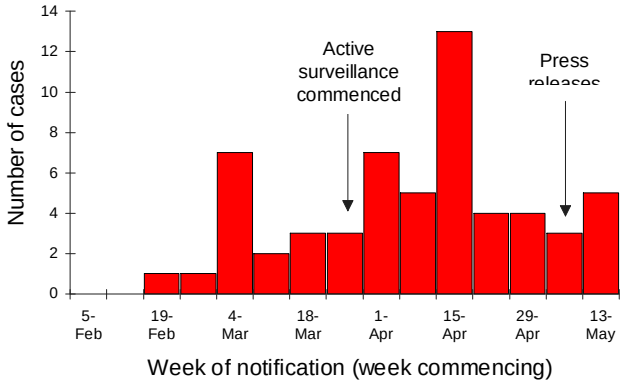
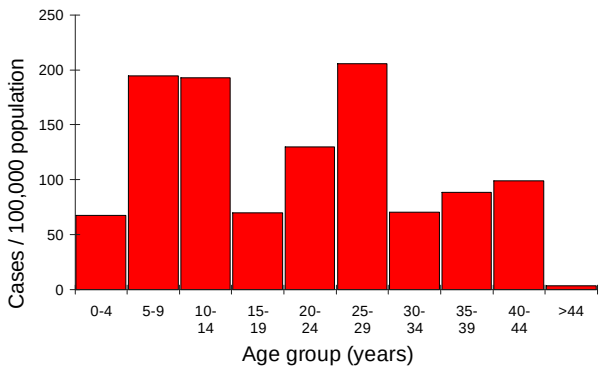


Figure 2. Hepatitis A notification rates by age group, Shoalhaven region, 1 January to 20 May 1996



exposure, and no two cases swam in the same pool, river, or at the same beach. Ten cases ate at restaurants during this period, but the maximum number eating in the same restaurant was four. Only one case had drunk unchlorinated ground water from a stream. No cases had received prophylactic NHIG in the three months preceding their illness, and none had travelled to hepatitis A endemic areas. All cases were Australian born.

Sixteen of the 35 cases were adults. Of these adults, six (37%) were also known to be hepatitis C virus (HCV) seropositive by enzyme immunoassay, and three (19%) gave a history of recent injecting drug use. Cases were not tested routinely for HCV seropositivity. Three of the six known HCV seropositive adults were admitted to hospital, but hospital admission was not significantly more frequent for HCV positive cases than for other adults (Fishers Exact Test; p=0.39). No cases reported being homosexual. Twelve adults (75%) were unemployed.

Table. Socio-demographic characteristics of hepatitis A outbreak survey respondents, compared to Shoalhaven population

Socio-demographic characteristic	Outbreak survey (n=35)	(%)	Shoalhaven[1] population %
Male	15	43	50
Aboriginal	8	23	2.2
School student	16	46	-
Living in a household with children:			
aged < 5 years	20	57	8.3[3]
aged < 2 years	12	34	-
who attend a child care centre	6	17	-
Live in house with six or more people	11	31	3.2[3]
Unemployed (adults[2] n=16)	12	75	16.5[4]

1. Australian Bureau of Statistics 1991 census.
2. Aged 18 years or over.
3. Per cent households.
4. Per cent labour force.

Discussion

Mode of transmission

Community-wide outbreaks of HAV infection are often prolonged and difficult to control. Usually they persist for six to 18 months, until the pool of susceptible persons is exhausted[1,2,3,4].We believe that person-to-person transmission was responsible for this community-wide outbreak of HAV infection in the Shoalhaven. Several epidemiologic features support this view. First, the rise and fall of the epidemic curve was characteristically slow. Second, the bimodal age-specific attack rate we observed, affecting children aged 5-14 years and young adults aged 25-29 years is typical of person-to-person transmission[1,5,6]. The dominant source of infection in community wide outbreaks is thought to be asymptomatic children under five years of age, especially those attending child care, who spread infection to their older siblings and parents. Third, as we observed in this instance, lower socioeconomic groups such as the unemployed, those living in large crowded households, and families with a large proportion of young children are often over represented in person-to-person transmitted outbreaks of HAV infection. Religious and ethnic minorities, Aboriginal populations, and injecting drug users (IDUs) are also at higher risk[1,2,5,6,7]. Fourth, the proportion of cases (51%) reporting prior contact with a definite case in this outbreak was typical of person to person transmission.[1,3,6,7]. We did not undertake extensive questionnaires looking for possible food sources, because the epidemiology of cases did not suggest a food-borne source. Food-borne outbreaks generally have a more abrupt onset, a more prominent peak, tend to affect the adult restaurant-going population, and are usually of shorter duration than outbreaks resulting from person-to-person transmission[8,9,10].

Six of the cases in this outbreak were known to be HCV seropositive, including three who reported recent injecting drug use (IDU). Other investigations have found that IDUs are at increased risk, although it is not clear why[7,11]. It may relate to increased faecal-oral transmission due to poor hygiene, or feasibly parenteral transmission of HAV may occur via unsterile injecting techniques. We noted that several cases in this outbreak reported the communal use of 'bongs' (marihuana smoking devices), and this could facilitate faecal-oral spread. We were unable to formally evaluate these hypotheses because of the small numbers of cases involved. Half of the HCV seropositive cases in this outbreak were hospitalised for severe hepatitic symptoms. Although there were few HCV cases in our study, it has been previously noted that IDUs with chronic hepatitis are more susceptible to severe hepatitis A[12].

Methods of hepatitis A outbreak control

Numerous studies have shown that post-exposure immunoprophylaxis using NHIG reduces the incidence and severity of HAV infection in contacts[1,13]. Symptomatic secondary infection is prevented in 90% of contacts who receive NHIG within ten days following exposure. Some have expressed concern that the efficacy of immunoprophylaxis may be reduced in developed countries, because the concentration of HAV specific IgG in NHIG is decreasing, although so far this has not been accompanied by reports of reduced efficacy[13,14]. During the Shoalhaven outbreak we did not observe any symptomatic infection amongst contacts who received standard dose NHIG prophylaxis within ten days of exposure.

Post-exposure NHIG does not appear to control established community-wide outbreaks of HAV infection. The Shoalhaven outbreak continued despite its widespread use. There has been one report of successful outbreak control using an 'expanded and targeted' program of post-exposure prophylaxis, where contacts of both suspected and confirmed cases were given NHIG[2]. However, our experience supports the more widely held view that while passive immunisation of contacts protects individuals, it does not halt an established community-wide outbreak[1,3,7]. This is probably

because a large proportion of infection occurs asymptomatically.

Because post exposure NHIG has failed to halt community-wide HAV outbreaks, mass administration of NHIG has been trialed. However, mass immunoprophylaxis has met with mixed enthusiasm, and its usefulness remains controversial. It does appear to be successful when there is a clearly defined population at risk, such as an individual school, child-care centre or an isolated community[4,15,16] . However, there are concerns that deferring HAV infection without providing lasting immunity may allow more severe HAV infection to occur later in adult life, and that the community remains susceptible to future outbreaks.

These concerns have prompted recent attempts to control HAV outbreaks by vaccinating entire communities using inactivated hepatitis A vaccines. Several uncontrolled interventional studies have reported promising results, suggesting that mass vaccination can prematurely halt outbreaks, and that a single dose of vaccine can achieve this[17,18,19]. One large scale study in Alaska showed that it was possible to halt outbreaks in communities where high immunisation coverage was achieved[18]. Another large campaign in the United States of America, which only targeted school children for vaccination, has also claimed success[17]. However, while theoretically attractive, it remains uncertain what effect single dose mass HAV immunisation will have upon long-term population immunity and HAV epidemiology.

Given the absence of well defined, accessible risk groups, should all school children in the Shoalhaven region have been vaccinated? There are approximately 12,000 children aged 5-14 years in the region, so even vaccinating this group would entail great cost and major logistic difficulties. The vaccine alone (Havrix, SmithKline Beecham) costs about $35 per dose - that is $420,000 for these children. Even so, it may be that mass vaccination is a worthwhile intervention for community-wide HAV outbreaks. Prospective estimation of the costs incurred by future community-wide HAV outbreaks would help in deciding whether mass vaccination is justifiable. In the absence of such data we decided against mass vaccination, and without a definitive intervention the outbreak continued for ten months. By October 1996, although the number of new cases appeared to be subsiding, the case count was 98, there were two instances of secondary spread from this outbreak to areas outside the Illawarra to Sydney and the Australian Capital Territory.

In the United States of America, universal childhood hepatitis A vaccination has been proposed, because it is thought that vaccinating adult risk groups will not reduce the majority of cases - asymptomatic children - and because it is believed to be the only strategy capable of eliminating HAV infection[20]. The advent of combination vaccines which include both HAV and hepatitis B antigens, if effective in infants, will make this approach more attractive.

Acknowledgements

The authors thank the following for their assistance with this investigation: the Illawarra Public Health Unit staff, Shoalhaven District Memorial Hospital Infection Control, Nowra Community Health staff and Aboriginal health workers, council officers, school and child-care directors, Dr Anthony Capon (Western Sector Public Health Unit), Dr Peter Newton (Illawarra Path Centre), Dr Jeff Hanna (Tropical Public Health Unit, Cairns), and notifying laboratories and general practitioners.Tim Heath's Master of Applied Epidemiology Program position is jointly funded by Western Sector Public Health Unit and the Commonwealth Department of Health and Family Services.

References

1 Shaw FE, Sudman JH, Smith SM, *et al*. A community-wide epidemic of hepatitis A in Ohio. *Am J Epidemiol* 1986;123:1057-1065.

2 Gildon B, Makintubee S, Istre GR. Community-wide outbreak of hepatitis A among an Indian population in Oklahoma. *South Med J* 1992;85:9-13.

3 Majeed FA, Stuart JM, Cartwright KA, *et al*. An outbreak of hepatitis A in Gloucester, UK. *Epidemiol Infect* 1992;109:167-173.

4 Pavia AT, Nielsen L, Armington L, *et al*. A community-wide outbreak of hepatitis A in a religious community: impact of mass administration of immune globulin. *Am J Epidemiol* 1990;131:1085-1093.

5 Crusberg TC, Burke WM, Reynolds JT, *et al*. The reappearance of a classical epidemic of infectious hepatitis in Worcester, Massachusetts. *Am J Epidemiol* 1978;107:545-551.

6 Mosley WH, Speers JF, Chin DY. Epidemiologic studies of a large urban outbreak of infectious hepatitis. *Am J Public Health* 1963;53:1603-1617.

7 Stone S, Erikson B, Alexander M, *et al*. Characteristics of epidemic hepatitis A in Baltimore City: implications for control measures. *Md Med J* 1993;42:995-1000.

8 Lowry PW, Levine R, Stroup DF, *et al*. Hepatitis A outbreak on a floating restaurant in Florida, 1986. *Am J Epidemiol* 1989;129:155-164.

9 Rosenblum LS, Mirkin IR, Allen DT, *et al*. A multifocal outbreak of hepatitis A traced to commercially distributed lettuce. *Am J Public Health* 1990;80:1075-1079.

10 Warburton AR, Wreghitt TG, Rampling A, *et al*. Hepatitis A outbreak involving bread. *Epidemiol Infect* 1991;106:199-202.

11 Jin A, Bardsley J. Intravenous drug use and hepatitis A: an investigation of an outbreak. *Can J Public Health* 1990;81:79-81.

12 Akrividias EA, Redeker AG. Fulminant hepatitis A in intravenous drug users with chronic liver disease. *Ann Intern Med* 1989;110:838-839.

13 Stapleton JT. Passive immunisation against hepatitis A. *Vaccine* 1992;10:S45-S47.

14 Gay NJ, Morgan-Capner P, Wright J, *et al*. Age-specific antibody prevalence to hepatitis A in England: implications for disease control. *Epidemiol Infect* 1994;113:113-120.

15 Bull AR, Kimmance KJ, Parry JV, Perry KR. Investigation of an outbreak of hepatitis A simplified by salivary antibody testing. *Epidemiol Infect* 1989;103:371-376.

16 Hanna J. Hepatitis A in a child day-care centre. *Comm Dis Intell* 1993;17:73-75.

17 Averhoff F, Shapiro C, Hyams I, *et al*. Effectiveness of inactivated hepatitis A vaccine (VAQTA) to interupt a community wide hepatitis A outbreak. In *Procedings of IX Triennial International Symposium on Viral Hepatitis and Liver Disease*, Rome 1996.

18 McMahon BJ, Beller M, Williams J, *et al*. A program to control an outbreak of hepatitis A in Alaska using an inactivated hepatitis A vaccine. In *Procedings of 34th Interscience Conference of Antimicrobial Agents and Chemotherapy*, Florida 1994.

19 Prikazsky V, Olear V, Cernoch A, *et al*. Interruption of an outbreak of hepatitis A in two villages by vaccination. *J Med Virol* 1994;44:457-459.

20 Shapiro CN, Coleman PJ, McQuillan GM, *et al*. Epidemiology of hepatitis A: seroepidemiology and risk groups in the USA. *Vaccine* 1992;10:S59-62.