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The epidemiology of acute hepatitis A in North Queensland, 1996-1997

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Abstract

Details on all cases of hepatitis A notified in North Queensland in 1996 and 1997 were prospectively collected. There were two substantial outbreaks and a total of 225 cases during this period. The total incidence rate (per 100,000) was 11.0 in 1996 and 27.0 in 1997. Aborigines and Torres Strait Islanders constituted 29% of cases and had incidence rates of 75.2 and 62.7 per 100,000 for 1996 and 1997 respectively. Thirty-nine cases (17.3%) were admitted to hospital for a total of 202 bed-days and a 4 year old died with fulminating hepatitis. A probable source of infection was identified for 69% of cases. The common risk categories for infection were: living in or visiting a rural Aboriginal or Torres Strait Islander community, injecting drug use, contact with a known case of hepatitis A, and travel to countries with endemic hepatitis A. *Commun Dis Intell* 1999;23:120-124.

Introduction

Infection with the hepatitis A virus (HAV) causes considerable morbidity in North Queensland. (Figure 1 illustrates the geograpical extent of Far North Queensland and the North Queensland Public Health Zone.) For example, Far North Queensland was subjected to a prolonged community-wide epidemic from 1992 to 1994 (Figure 2). During this epidemic numerous episodes of transmission in child day-care centres were documented and many occupational exposures were identified. [1,2,3]

An inactivated hepatitis A vaccine was first licensed in Australia in 1993 and recommendations for its use were subsequently published by the National Health and Medical Research Council (NHMRC).[4] The Tropical Public Health Unit (TPHU) promoted vaccination of at-risk groups, including staff at child day-care centres and some health care providers, in response to the Far North Queensland epidemic.

This prospective study was undertaken to describe the current epidemiology of hepatitis A in North Queensland, and to reassess the risk factors for what is now a vaccine preventable disease.

Methods

The TPHU collected details on all notified cases of hepatitis A in the North Queensland Public Health Zone for 1996 and 1997 (Figure 1). The Zone has a population of 592,000, 8.1% of whom are Aborigines or Torres Strait

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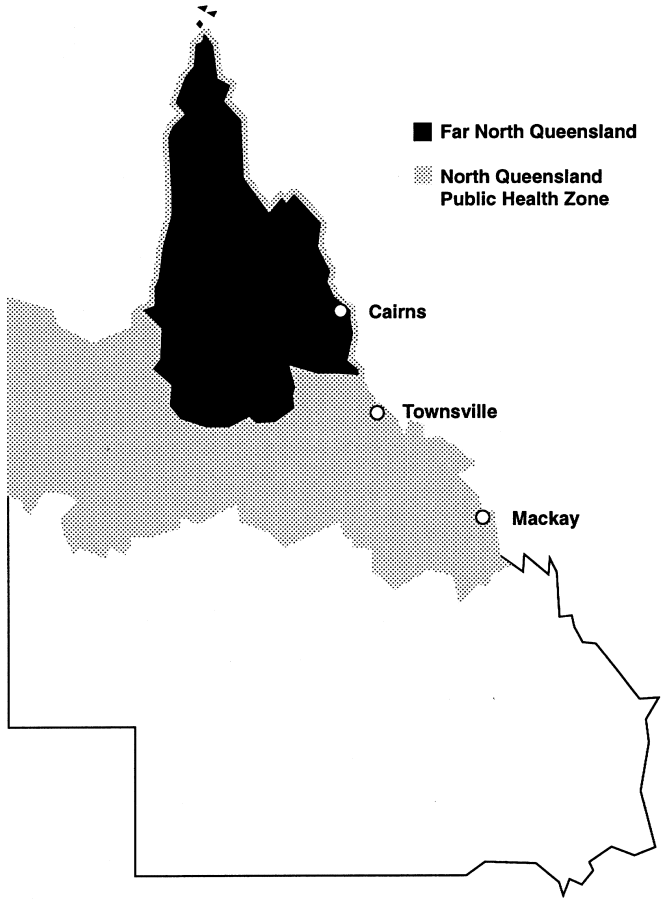
Figure 1. Map of North Queensland



Islanders. [5] Acute hepatitis A is a notifiable disease in Queensland and all public and private laboratories in North Queensland are therefore required to notify the TPHU when IgM antibodies to hepatitis A (anti-HAV IgM) are detected in serum. In addition, clinicians are requested to report any clinical case of acute hepatitis, regardless of whether confirmatory serology is available. All cases in this report were thus confirmed by the presence of anti-HAV IgM in the absence of recent vaccination, or had an illness consistent with hepatitis and an epidemiological link to a serologically confirmed case.[6]

The staff at TPHU contacted the treating doctor of each notified case and, when possible, interviewed the patient directly. The following details were sought: onset date of illness, age, sex, race, address, occupation, potential risk categories for infection,[7] management, immediate health outcome and contacts eligible for hepatitis A prophylaxis. Normal human immunoglobulin (NHIG) was provided to contacts in conjunction with the treating doctor and further public health measures were instituted as required.

Incidence rates were calculated for the total and the indigenous population in North Queensland using

Figure 2. Notifications of hepatitis A in Far North Queensland, by quarter of year

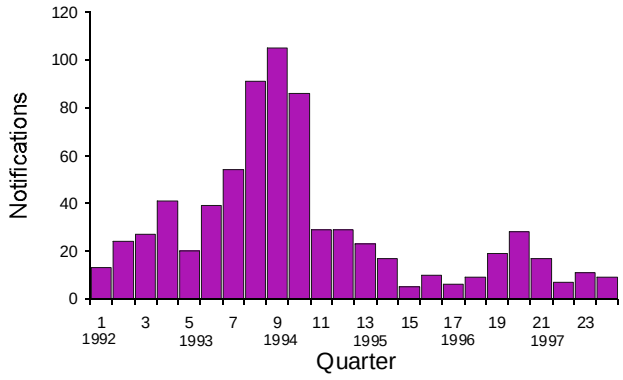


Figure 3. Notifications of hepatitis A, North Queensland, 1996-97

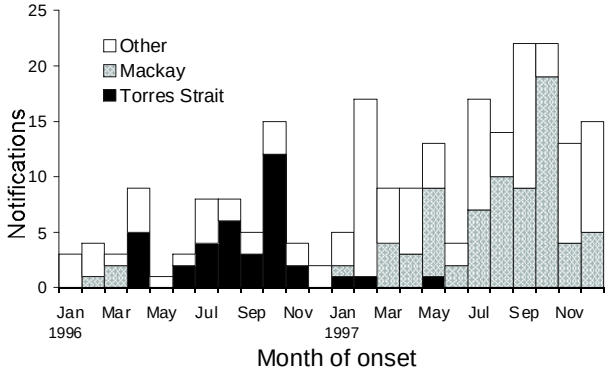
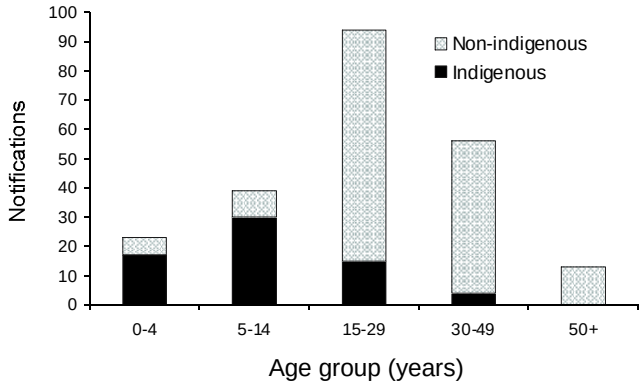


Figure 4. Notifications of hepatitis A, North Queensland, 1996-97, by age and indigenous status



denominator data from the 1996 Australian Bureau of Statistics Census.[5]

Results

A total of 225 cases were notified; 65 in 1996 and 160 in 1997. Total incidence rates were thus 11.0 and 27.0 per 100,000 persons for 1996 and 1997 respectively. Only one case was not serologically confirmed. In addition to sporadic cases, there were two substantial outbreaks; one in the Torres Strait during 1996 (34 cases) and a second in Mackay during 1997 (72 cases) (Figure 3). There were also discrete clusters in two rural Aboriginal communities during 1997, both resulting in 9 notifications. The majority of cases were Caucasian (151), but Aboriginal and Torres Strait Islanders were clearly over-represented as they accounted for 29% (66) of all cases. Incidence rates for indigenous persons were 75.2 and 62.7 per 100,000 persons for 1996 and 1997 respectively.

Hepatitis A was more common in children and young adults, and the mean age of Aboriginal and Torres Strait Islander cases (12.6 years) was significantly lower than that of non-indigenous cases (30.0 years, $p<0.001$) (Figure 4). There were more male than female cases (140 and 85 respectively).

Thirty-nine cases (17.3%) were admitted to hospital, for a total of 202 bed days (median length of hospital stay 4 days, range 1-18 days). Four people required transfer from a peripheral hospital to a regional base hospital and a 45 year old Australian resident was evacuated by air from Papua New Guinea (PNG) to Cairns acutely ill with hepatitis A.

There was one death during 1997. A 4 year old Aboriginal boy developed severe hepatic encephalopathy due to fulminating hepatitis A and died in the Royal Childrens Hospital in Brisbane before liver transplantation was possible.

Risk categories indicating a probable source of infection were identified for 69% of cases, a possible source for a further 2%, and for 29% the source was unknown. Some cases had more than one risk factor identified (Table 1). For example, among those who had contact with a known case, 17 were also identified as injecting drug users, 7 were in the rural indigenous group, 2 had travelled overseas to HAV endemic areas and 1 had child day-care contact. Only a few cases in other groups had multiple risk factors. People were allocated to risk categories on the basis of available information and misclassification may have occurred for some cases. For example, it is likely that some cases had unrecognised contact with an HAV-infected person and that not all injecting drug users (IDUs) were identified.

The most common risk category indicating a probable source of infection was either living in or visiting a rural Aboriginal or Torres Strait Islander community. The majority of people infected in these communities were

Table 1. Risk category for source of hepatitis A infection, North Queensland, 1996-97

Probable source of infection	Number	Percent
Rural indigenous community resident or visitor	59	26
Contact with a known case	43	19
Injecting Drug Use	41	18
Overseas travel to an endemic country	17	8
Urban indigenous community resident	10	4
Child day-care contact (children, staff and parents)	10	4
Homosexual male	4	2
Oysters (Wallis Lake outbreak)	4	2
Sewage exposure	1	0
Total cases with one or more probable risk categories	155	69
Possible source of infection		
Cleaner	5	2
Unknown source	65	29
Total notified cases	225	100

local indigenous residents, but two non-indigenous residents (a doctor and a hospital clerk) and three visitors also acquired HAV infection. Other common risk categories were injecting drug use (IDU), contact with a known case of hepatitis A, and travel to overseas countries with endemic hepatitis A.

A total of 41 cases were identified as IDUs, 34 of whom were involved in the Mackay outbreak. A variety of different drugs, including amphetamines and heroin, were injected by the affected IDUs and no common drug source was identified. Very few cases reported sharing needles, but many described sharing other objects such as cigarettes and bongs. Many lived in houses with other IDUs. During 1997 several other outbreaks of HAV infection in IDUs were identified in southern Queensland but no epidemiological links with the Mackay outbreak were established.

Seventeen cases probably acquired their infection overseas. None had received HAV vaccination or NHIG prior to travel. The most common regions implicated were Papua New Guinea (9 cases) and South East Asia (3 cases).

Other risk categories accounting for small numbers of cases included urban indigenous residents, association with child day-care centres, homosexual males, eating oysters implicated in an outbreak [8] and exposure to sewage. Cleaners could feasibly be exposed to HAV in their workplace and were over-represented as an occupational group among cases; they were included as a possible risk category.

Discussion

Hepatitis A results in considerable morbidity and expense for the North Queensland community. The death of a 4 year old child during this period also indicates how serious this infection can be. While the reported case fatality rate for HAV infection is low (<1/1000), higher rates have been reported in children under the age of 5 years (1.5/1000) and adults over the age of 50 years (27/1000).[9] As a safe and effective vaccine is available, there is a need to consider whether more can be done to prevent HAV infection in north Queensland.

This report identifies a number of key population groups that continue to be at increased risk of contracting HAV infection. These include rural and urban indigenous community residents and visitors, IDUs and people travelling to endemic regions overseas.

Many rural Aboriginal and Torres Strait Islander communities have endemic HAV infection. Nearly all residents (98.5%) from a sample of remote Aboriginal communities in the Northern Territory (NT) were immune to HAV by the age of 10 years when assessed in 1994.[10] In such endemic circumstances HAV infection is usually acquired in early childhood and confers life-long immunity. Acute infection at this early age is often mild and anicteric, thus the disease may be largely invisible despite very high real incidence rates. Clinical notifications will considerably under-estimate infection in such communities.

Paradoxically, hepatitis A initially becomes a more obvious problem as communities experience lower incidence rates in response to changes such as improved sewage disposal, housing and water supply. Fewer early childhood infections result in a susceptible pool of older children, adolescents and adults, and infection in this older group is more likely to be clinically apparent. The outbreak that occurred in the Torres Strait during 1996 and the clusters of cases seen in several rural communities during 1997 probably reflect this transitional process. Further outbreaks and clusters can be expected as this transition continues.

The hepatitis A vaccine has been used to interrupt transmission in communities with high incidence rates but its use needs to be tailored to specific situations. Following the NT seroprevalence study noted above, it was concluded that vaccination was not indicated in those communities as only 1.5% of people over the age of 10 years of age were susceptible to HAV.[10]

There is thus a need for appropriate seroprevalence studies before considering an immunisation program in indigenous communities in North Queensland. Other issues such as the expense of the vaccine and the logistics involved in maintaining such a program would also need to be considered. Community-wide HAV vaccination programs often target young children (aged 3 to 5 years) on the basis that they are a vulnerable group, and because they are recognised as key transmitters of disease within a community.[11]

Non-indigenous staff employed in indigenous communities are also at risk of HAV infection. The NHMRC currently recommends HAV vaccination for teachers and health staff in remote indigenous communities,[12] and this should be arranged prior to their arrival in the community.

Injecting drug use is well recognised as a risk category for HAV infection [7, 13] but had not previously been associated with an outbreak in North Queensland. The exact mechanism by which infection is acquired is unclear, but is likely to involve the associated lifestyle and possibly the use of shared equipment such as bongs. Although the NHMRC recommends that IDUs be considered for HAV vaccination,[12] they are likely to be a difficult group to access.

The number of cases in travellers to endemic areas is of concern. Hepatitis A is the most frequent infection in travellers that can be prevented by immunisation,[14] and it should be possible to more readily identify and target this group for vaccination prior to travel.

Ten cases were associated with child day-care centres; 4 were children in care, 1 was a day-care staff member and 5 were parents of children in care. All were isolated cases and all but four had another risk factor. There were no outbreaks in child day-care centres, which is in marked contrast to the 1992-94 epidemic in Far North Queensland.[1,2]

There were 4 cases attributed to oysters from the Wallis Lakes in New South Wales, but no other food-borne cases were identified.

In conclusion, we have identified a number of important risk factors for HAV infection in North Queensland. There is a need for greater use of the HAV vaccine to protect at risk groups identified in the current NHMRC guidelines including IDUs and travellers to endemic areas. Vaccination also needs to be further considered for some indigenous groups.

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Report of the Australian National Polio Reference Laboratory
1 January to 31 December 1998

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Abstract

The Australian National Polio Reference Laboratory was established at the Victorian Infectious Diseases Reference Laboratory (VIDRL) in late 1994 to carry out virological confirmation of the eradication of poliomyelitis in Australia. The laboratory is responsible for transporting samples from all Australian patients with acute flaccid paralysis (AFP) to VIDRL for poliovirus culture, identification and intratypic differentiation. The laboratory also performs polio serology on selected serum samples from AFP patients when faecal samples are not available. In 1998, faecal specimens were received from 11 patients with AFP. Adenovirus type 2 was isolated from 1 patient and an untypable non-polio enterovirus from another. No viruses were isolated from the other 9 patients. Since 1995, over 820 isolates have been transported to VIDRL from laboratories in five Australian states for testing. Three hundred and seventy three (45%) were confirmed as Sabin vaccine-like polioviruses, 416 (51%) were non-polio enteroviruses and 24 (3%) yielded no virus or viruses other than enteroviruses. Eight polioviruses are still uncharacterised. *Commun Dis Intell* 1999;23:124-128.

Introduction

In 1988, the World Health Assembly passed a resolution which committed the World Health Organization (WHO) to the global eradication of poliomyelitis by the year 2000. The eradication strategy is three-fold: routine and supplementary immunisation, surveillance and, where required, outbreak response.[1,2]

Almost all countries of the six WHO regions are committed to polio eradication. Each country is required to verify the absence of wild poliovirus circulation in the presence of high quality surveillance. Acute flaccid paralysis (AFP) has been proven to be a sensitive indicator for detecting wild poliovirus and was used successfully in the Americas prior to certification of that region as being free of wild poliovirus.[3] In a country where polio is not endemic, there is normally a background incidence of at least one AFP case for every 100,000 children under 15 years of age per year. Surveillance of cases of AFP by most recently endemic and some non-endemic countries of the Western Pacific has resulted in the documentation of AFP rates of at least one per 100,000.[4] Based on Australia's population