The investigation of a 'cluster' of hepatitis B in teenagers from an Indigenous community in North Queensland

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Abstract

Background: In early 1999, five teenagers from the same Indigenous community were notified as having hepatitis B. Hepatitis B vaccine should have been offered to this cohort of teenagers in a 'catch-up' program during the late 1980s when they were of pre-school age.

Objectives: To determine the vaccination status of residents of the community born between 1981 and 1985 (inclusive) and to ascertain the prevalence of markers of hepatitis B infection and carriage in the incompletely vaccinated teenagers in this cohort.

Methods: Community health records were examined to identify all residents in the study cohort. Immunisation records were obtained from local hospital records and from a statewide computerised vaccination database. Serological tests for markers of hepatitis B infection and carriage were performed on blood samples from the incompletely vaccinated teenagers.

Results: Only 44% of 235 teenagers who had their vaccination status assessed were fully vaccinated. One hundred and eleven (47%) of the cohort had not received any hepatitis B vaccine. Over 90% of the incompletely vaccinated had been infected with the hepatitis B virus and 26% of these were hepatitis B carriers.

Conclusions: Despite the availability of an effective hepatitis B vaccine and the recommendation for a catch-up program, the pre-school aged cohort of children at the community were not effectively targeted for vaccination. Hepatitis B remains a consequential infection in Indigenous communities in North Queensland.

Implications: Initiatives to control hepatitis B need to be enhanced within existing maternal and child health, sexual health, alcohol and drug and chronic disease management programs.

Chronic infection with the hepatitis B virus (HBV) can cause liver disease including cirrhosis and primary hepatocellular carcinoma. The risk of chronic HBV infection – the so-called 'carrier' state – is determined by the age of infection, being highest (up to 90%) following infection in early infancy but much lower (5-10%) following infection acquired in adulthood.[1]

Prior to the introduction of hepatitis B vaccination, Indigenous Australians were recognised as being at high risk for HBV infection and the hepatitis B carrier state.[2] In order to prevent HBV infection, and therefore to minimise their risk of developing the carrier state, vaccination of newborn Indigenous infants was initiated in Queensland in late 1985.[3] This neonatal programme was supplemented in 1987 with a catch-up programme for Indigenous children up to 10 years of age.[3]

The detection of HBV surface antigen (HBsAg) in a patient's serum is a notifiable diagnosis in Queensland.[4] Chronic HBV infection is defined as the persistence of HBsAg in a patient's serum for at least 6 months.[1] However, for practical reasons the surveillance of chronic HBV infection is often based upon any detection of HBsAg provided that there is no evidence, either clinical or serological, of acute infection.[4]

Between February and March 1999, the Tropical Public Health Unit (TPHU) was notified of five HBsAg positive teenagers, all from the same Indigenous community in North Queensland. There was insufficient serum from three of the teenagers for further studies, but the remaining two had evidence of acute HBV infection. The latter two cases bought this apparent 'cluster' of hepatitis B notifications to the attention of TPHU and prompted further enquiries.

Although the catch-up hepatitis B vaccination program, undertaken in the late 1980s, was implemented on-site at the primary school in the community, local health staff could not recall whether it specifically targeted the pre-school aged children. Because of this uncertainty and because some of these children, now teenagers, were becoming infected with HBV, an investigation was undertaken. The objectives of this investigation were (i) to determine the hepatitis B vaccination status of the then pre-school aged cohort of children at the community, and (ii) to determine the prevalence of HBV infection and carriage in the incompletely vaccinated individuals in this cohort.

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Methods

With the approval of the Community Council, all residents of the community born between 1981 and 1985 (inclusive) were identified from the community health centre records. The vaccination status of each person in this cohort was then ascertained from a computerised database maintained by Queensland Health and from records held at the local hospital.

To be 'fully' vaccinated an individual was required to have received three doses of hepatitis B vaccine. No consideration was given to the intervals between the doses of vaccine, or the ages at which they were given, in the assessment of the vaccination status.

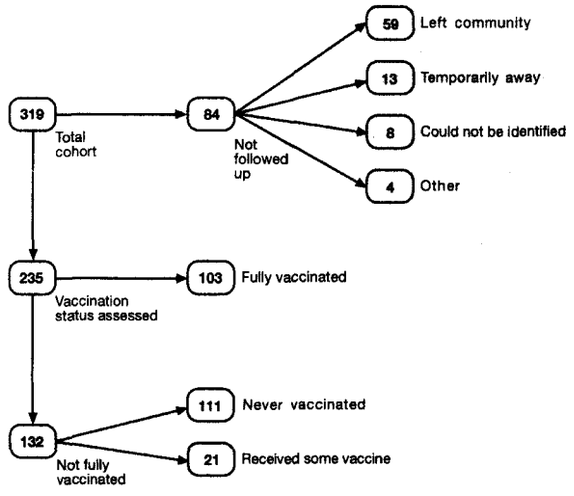
Then with the consent of either the individual or his/her guardian a venous blood sample was taken from the 'incompletely' vaccinated individuals (ie. those who had not received three doses of vaccine) for testing for markers of infection (ie. antibody to the HBV core antigen [anti-HBc]) and carriage (ie. HBsAg). In those individuals who were HBsAg positive, the HBV e antigen (HBeAg) status was also determined (HBeAg is a serological marker of high-level infectivity[1]).

Commercially available enzyme immunoassay kits were used for all the HBV tests (AxSYM®, Abbott Laboratories, Abbott Park, Illinois).

Results

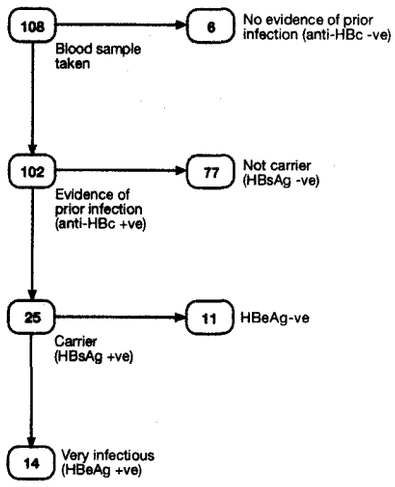
A total of 319 teenagers who had been born between 1981 and 1985 (inclusive) were identified. Of these, 235 (74%) had their vaccination status assessed. Of the 84 whose vaccination status was not assessed, 70% had left the community (Figure 1). One hundred and three (44%) of those assessed had been fully vaccinated and 132 were incompletely vaccinated. One hundred and eleven (84%) of the latter had never received any hepatitis B vaccine (Figure 1).

Figure 1: The vaccination status of the cohort of teenagers born 1981–1985 (inclusive) resident at the community.



Blood was collected for HBV serological studies from 108 incompletely vaccinated teenagers. One hundred and two (94%) had evidence of prior HBV infection and 25 (26%) of these were HBsAg positive. Because 24 of the HBsAg positive teenagers had no symptoms of acute hepatitis it is assumed that they were all chronic carriers of HBV. Fourteen (56%) of these carriers were highly-infectious (ie. HBeAg positive) (Figure 2).

Figure 2: The markers of prior hepatitis B virus infection and carriage among incompletely vaccinated teenagers at the community.



For practical reasons some of the teenagers had a blood sample collected before all the vaccination records had been accessed. As a consequence, 14 fully vaccinated teenagers had HBV serological studies undertaken. Of these, 10 (71%) had evidence of prior infection, and 4 were HBsAg positive. All of the latter were HBeAg positive. The percentage of these fully vaccinated teenagers with a marker of prior infection (71%) was lower than that demonstrated (94%) in the incompletely vaccinated teenagers (Fishers exact test, $p<0.05$).

Discussion

Many Indigenous peoples,[5,6] including Indigenous Australians,[7] were (prior to the introduction of hepatitis B vaccine) at considerable risk of developing HBV-associated hepatocellular carcinoma. Fortunately, hepatitis B vaccines have been shown to be effective in preventing HBV carriage for at least 10 years after vaccination, even in previously hyperendemic regions.[8,9] Evidence is beginning to accumulate that these vaccines can prevent hepatocellular carcinoma.[10]

The available evidence suggests that although some Indigenous Australian infants became infected perinatally, from their carrier mothers, most HBV infections in Indigenous children occurred in later years from horizontal child-to-child transmission, particularly in the pre-school aged years.[11] It is plausible that overcrowding and suboptimal personal hygiene leading to a high prevalence of skin infections contributed to this horizontal transmission;[12] HBsAg and HBeAg have been isolated from exudates from skin ulcers and impetiginous lesions in other Indigenous peoples.[13,14] This horizontal child-to-child transmission provided a rationale for the recommended catch-up hepatitis B vaccination programme.

Hepatitis B vaccines are very effective in preventing HBV infection, even in high risk populations, when administered from birth.[1,15] Although a catch-up vaccination programme would have obviously been too late for those infants infected either perinatally or in the early months of life, it should have protected a considerable portion of the pre-school aged children. Indeed, although the sample size was small, fewer of the fully vaccinated teenagers (who were inadvertently bled) had evidence of previous HBV infection than that seen in the incompletely vaccinated teenagers.

This investigation has revealed that the catch-up vaccination programme was not effectively delivered to the pre-school aged children at the community. Less than half of the then pre-school aged children had received three doses of hepatitis B vaccine. Indeed the investigation suggests that the catch-up programme for pre-school aged children did not occur at all. The median age of the first dose of vaccine in the children who received some doses was 6.1 (4.2 – 9.25) years which indicates that the vaccinations that did occur took place once the children reached primary school.

Intense transmission of HBV to these unvaccinated children occurred, resulting in extremely high prevalences of markers of prior infection and carriage. Indeed, these prevalences are among the highest ever reported among Indigenous Australians.[2,3]

Each carrier teenager was counselled about the infection, about its mode of transmission, how to care for him/herself and how to minimise the risk of transmission to household, sexual and community contacts. Each carrier was advised to have annual hepatitis B serological and liver function tests. The latter can be useful for triggering 'brief interventions' should they suggest alcohol abuse.[16]

The investigation provides two salutary reminders. Firstly, regardless of the efficacy and availability of any intervention, it remains essentially useless unless there are services that are committed to deliver it effectively to the target population. Fragmented and poorly coordinated services continue to blight Indigenous health in some parts of Australia.[17]

Secondly, hepatitis B remains a consequential infection in Indigenous communities in north Queensland and probably elsewhere. Initiatives to control hepatitis B need to be enhanced within existing maternal and child health, sexual health, alcohol and drug and chronic disease management programmes. There is a need to ensure that all Indigenous infants are fully vaccinated against hepatitis B and the other vaccine preventable diseases.[18]

Addendum

Seventeen (68%) of the 25 HBsAg positive teenagers have been retested >6 months after the original blood test. All 17 remain HBsAg positive indicating that they are indeed 'true' carriers. Of the 12 HBeAg positive teenagers who have been retested, all remain HBeAg positive.

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