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# Nursery-associated hepatitis A traced to a male nurse

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SUMMARY

A small epidemic of nursery-associated hepatitis A (HA), in which silently infected children played a cryptic role in transmitting the illness to adult contacts, was investigated. It was found that a male nurse had transmitted HA to four children before he fell ill. He was not heard from for about two months as he was a part-time worker and was admitted to a local hospital in another prefecture. Two of the four silently infected children transmitted HA to their parents who then revealed the presence of nursery-associated HA. The epidemic was terminated by injection of human gamma globulin to nursery children and to antibody-negative parents, nursery staff and other contacts except for two breakthrough cases.

A 34-year-old male nurse (case 1, Fig. 1) of the day-care centre of the Toho University Hospital fell ill with hepatitis A (HA) on 17 February 1983. He was admitted to a local hospital near his home and little was heard from him for two months. A 36-year-old male (case 4) and a 26-year-old female (case 5), both laboratory technicians, fell ill on 13 April, followed by a 27-year-old female office worker (case 6) on 15 April. Both cases 4 and 6 had their children taken care of at the nursery. A nursery-associated epidemic of HA was suspected as three of the four HA patients were closely associated with the nursery. Investigation of case 1 disclosed that he had consumed more than 10 raw oysters 19 days prior to the onset of his illness. Furthermore, hs 33-year-old wife (case 2) and their 1-year-old daughter (case 3) fell ill with HA on 19 March and 5 April, respectively (Fig. 1).

Serological tests for HA virus (HAV) were made employing commercially available kits: HAVAB-EIA (which detects anti-HAV antibody of IgG and IgM classes) and HAVAB-M-EIA (which detects anti-HAV antibody of IgM class) (Abbott Laboratories, Chicago). Forty-eight nursery children were bled on 22 April and were given human gamma globulin, 24 mg/kg of body weight, before the results of serological tests were made available. Eighteen nursery staff members (group 1), 30 mothers and three fathers (group 2) and 34 clinical laboratory staff members and office workers (group 3) were bled on the same day and antibody-negative persons were given 450 mg of human gamma globulin on 26 April.

Four out of ten children aged between 24 and 37 months, who had been taken

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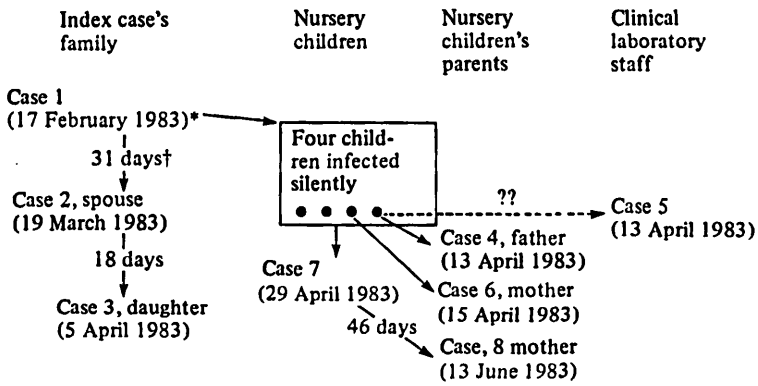


* Figures in parentheses indicate date of onset of illness.
† Figures indicate interval of onset of illness.
Arrows indicate the direction of transmission.

Fig. 1. *Chronological sequence of nursery-associated hepatitis A.*

Table 1. *Prevalence of HAVAB-IgG in different occupational and age groups*

| Occupation | Nursery staff | | Parents of children | | Clinical lab. staff and others | | Overall | |
|---|---|---|---|---|---|---|---|---|
| | Total | Positive | Total | Positive | Total | Positive | Total | Positive |
| Age (yrs) | | | | | | | | |
| 18–29 | 5 | 0 | 6 | 0 | 23 | 3 | 34 | 3 (9) |
| 30–39 | 3 | 1 | 24 | 7 | 9 | 1 | 36 | 9 (25) |
| 40–49 | 4 | 2 | 3 | 2 | 1 | 1 | 8 | 5 (63) |
| 50 and over | 6 | 5† | 0 | 0 | 1 | 1 | 7 | 6 (86) |
| Total | 18 | 8 (44) | 33 | 9 (27) | 34 | 6 (18) | 85 | 23 (27) |

* Figures in parenthesis indicate percentage of total.
† One had non-A, non-B chronic hepatitis.

care of by case 1, were positive for anti-HAV IgM. By contrast, none of 38 children aged between 8 and 23 or 38 and 72 months who had been taken care of by other nursery staff was positive for anti-HAV antibody. The association of antibody-positive children to case 1 was significant ($P = 0{\cdot}0011$, Fisher's direct method). Furthermore, one of the six children negative for anti-HAV antibody who had been taken care of by case 1 became ill with HA seven days after gamma globulin had been given (case 7).

It is significant that none of the four antibody-positive children showed abnormal transaminase tests (SGPT and SGOT) at the time blood was taken for virus antibody studies or had signs and symptoms of HA during the past four months. Two anti-HAV IgM-positive children transmitted HAV to one of their antibody-negative parents (cases 4 and 6). The other two silently infected children did not transmit HAV to their antibody-negative parents or three siblings.

Twenty-three out of 85 people with direct or indirect contact with nursery children were positive for anti-HAV IgG. None was positive for anti-HAV IgM. Group 1 had the highest incidence of 44 per cent, followed by group 2 with 27 per cent and group 3 with 18 per cent (Table 1).

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No significant difference in the incidence was seen between groups 1 *vs* 2 or 3 by chi-square tests. The decreasing incidence of positive antibody was explained by the age of the contacts. Ten out of 18 in group 1 were older than 40 years, 24 of 33 in group 2 were in the age group of 30–39 years, and 23 of 34 in group 3 were in the range of 18–29 years. As a whole, the percentage of antibody-positive persons in each group was lower than or equal to other groups studied in Japan (Suzuki *et al.* 1982; Yano, 1982; Kashiwagi *et al.* 1983). This study indicated that the contacts were not severely exposed to HAV except for a father and a mother who became ill.

The epidemic was terminated by the injection of human gamma globulin except for the child who became ill seven days after the treatment (case 7) and his pregnant mother nurse (case 8) who did not receive human gamma globulin although she had been told of the absence of antibody and the necessity of prophylactic injection. She saw her sick boy in the ward until 12 May, gave birth to a deformed boy five days later and developed HA on 13 June 1983. None of the remaining 43 children and 61 adult contacts who were anti-HAV antibody-negative and received human gamma globulin developed overt hepatitis during the following 5 months of observation, although serological tests for HAV antibodies were not repeated during this period.

It is not easy to pinpoint who brough the silent HAV infection to small children who are usually negative for anti-HAV antibody. However, the fact that four silently infected children and a symptomatic child were all under the care of case 1, and also the fact that he transmitted infection to his own family, strongly indicate that he might have infected four asymptomatic children while they were in his charge.

It is not known how long silently infected children excrete HAV in the stool. The symptomatic cases ususally excrete HAV in the stool 7–10 days prior to the onset of illness and about 1–2 weeks thereafter (Deinhardt & Gust, 1982). The incubation period is usually 3–5 weeks (range 2–6). It is, therefore, possible that the four anti-HAV IgM positive children were infected during 10–16 February and excreted HAV in the stool during the first three weeks of March. Cases 4, 6 and 7 were probably infected during this period and developed HA 3–4 weeks later.

The interval of the onset of illness in the index case's family, 18 and 31 days, and 46 days between cases 7 and 8 can be regarded as reasonable when the duration of excretion of HAV in symptomatic cases is taken into consideration.

It was impossible to determine where case 5 contracted HA. She worked in the same clinical laboratory with case 4, who often brought his silently infected child to the laboratory. But her contact with this child was less than that of the nursery staff who were not infected.

The interval of 19 days between the consumption of raw oysters by case 1 and the development of illness is shorter than the incubation periods of our similar cases (range 22 to 32 days, average 26·9 days) (Ohara *et al.* 1983), but it is not too short to incriminate them as the source of infection. It happened that a companion of case 1 ate the same foods at the same time but remained well as he was positive for anti-HAV IgG. None of the symptomatic cases or silently infected children ate raw oysters during the two months prior to the onset of illness, except for case 1. Any person who is in charge of HAV-susceptible children should refrain from

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consuming foods that might be contaminated with HAV in order to minimize the occurrence of epidemics.

## REFERENCES

Deinhardt, F. & Gust, I. D. (1982). Viral hepatitis. *Bulletin of the World Health Organization* **60**, 661–691.

Kashiwagi, S., Hayashi, J., Ikematsu, H., Kusaba, T., Shingu, T., Hayashida, K. & Kaji, M. (1983). Prevalence of antibody to hepatitis A virus in Okinawa and Kyushu, Japan. *American Journal of Epidemiology* **117**, 55–59.

Ohara, H., Naruto, H., Watanabe, T. & Ebisawa, I (1983). An outbreak of hepatitis A caused by consumption of raw oysters. *Journal of Hygiene* **91**, 163–165.

Suzuki, S., Sato, A., Kojima, H. & Ichida, F. (1982). Epidemiology of hepatitis A. In *Proceedings of the 12th Inuyama Symposium* (*Hepatitis A and Fulminant Hepatitis*), (ed. T. Takahashi *et al.*), pp. 17–21. Tokyo: Chugai Medical Publishing Co. (in Japanese.)

Yano, M. (1982). Epidemiology of hepatitis A. in the same *Proceedings* as above, pp. 29–31.

https://doi.org/10.1017/S0022172400064275 Published online by Cambridge University Press