

An outbreak of infectious hepatitis investigated using radioimmunoassays for hepatitis A virus antibody

By S. J. SKIDMORE

*Regional Virus Laboratory, East Birmingham Hospital, Bordesley Green East,
Birmingham B9 5ST*

S. S. BAKHSI, R. BEEDLE, AND J. KIMBERLEY

Environmental Health Department, 120 Edmund Street, Birmingham B3 2EZ

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SUMMARY

An outbreak of suspected infectious hepatitis in a primary school was investigated using sensitive diagnostic methods for hepatitis A. A total of 116 sera from children were tested for the presence of both IgM and IgG antibodies to hepatitis A (HAV). The results were compared to those obtained for samples from a control school. IgG antibodies were present in 45% and 10% of the children in the outbreak and control schools respectively.

An outbreak of hepatitis was retrospectively recognized in December 1980 when 8 cases of hepatitis A had been reported from the same school in the previous 5 months. It appears to have started in July 1980. The index case was a boy who had recently returned from Pakistan where he had been in contact with several jaundiced people. He was admitted to hospital and the diagnosis of hepatitis A confirmed by the demonstration of anti-HAV specific IgM in his serum. Two siblings who travelled with him were also unwell. A period of three months elapsed before the next clinical case, although it seems likely that other children were affected subclinically during this time. School reopened in September and the first case was reported approximately three weeks later in early October.

In January 1981 it was decided to collect sera from all the children attending the affected school. These were then screened for specific anti-HAV IgM, which is indicative of a recent infection within the previous 3–4 months, and also for the IgG class of antibody, which is evidence of a past infection. One hundred and sixteen children of a total school population of 121 between the ages of 4 and 11 were bled. Control sera were obtained from 108 children in a school where there was no apparent hepatitis A. This school was situated in a similar area to the affected school but was larger, with between 300 and 350 pupils. However, those bled matched the outbreak school sera as closely as possible for place of birth, age, ethnic origin and socioeconomic class.

All sera were tested for the presence of anti-HAV by the HAVAB radio-immunoassay (Abbott Laboratories). The test was carried out according to the manufacturer's instructions. When HAVAB positive, the sera were tested for anti-HAV specific IgM using a solid-phase anti- μ radioimmunoassay test previously described (Busher *et al.* 1981). If the HAVAB test was positive but no specific IgM was detected this was taken to indicate the presence of hepatitis A antibody of the IgG class. Tests for aspartate and alanine transaminases (SGOT and SGPT) were carried out on sera which gave a positive IgM result. (Bilirubin estimations were not performed since none of the children was jaundiced at the time of testing.)

At the time of testing sera from 31 of the children from the affected school gave a positive IgM test. Seven of these had had icteric hepatitis A and the remaining 24 had either anicteric or completely subclinical hepatitis A; three experienced some symptoms, but were not icteric, and four had mildly elevated SGOT and SGPT levels at the time of testing, but were otherwise asymptomatic. Three of the early icteric cases including the index case were already negative for IgM. This was expected since the test for IgM, an efficient marker of recent infection, detects the antibody for only 3–4 months post infection.

A further 17 children were IgG positive, IgM negative, indicating that they had been infected earlier than the preceding 3 months. All children with no immunity to hepatitis A were retested 6 months later in June when only the eleventh clinical case, who became ill in February 1981, showed a seroconversion. In all, 11 children had icteric hepatitis A (Fig. 1). Symptoms were usually malaise with loss of appetite, abdominal pain, vomiting and occasionally fever. Only 2 children were thought sufficiently ill to be hospitalized.

Of the 108 sera from the control school, only 11 (10%) contained IgG and none of these was positive for IgM antibodies to HAV. The 11 immune children comprised three United Kingdom-born Asians and eight caucasians including two pairs of siblings and four others. No history of illness could be found for one pair of siblings, but the other pair had been in close contact with a confirmed case of hepatitis A three years previously.

Compared to the outbreak school, where 45% of the children had some evidence of past infection with hepatitis A, the level of immunity in the control school was low, with 90% of the children susceptible to infection. Therefore it is not surprising that hepatitis A can quickly spread throughout a school, and there have recently been several reports of outbreaks of hepatitis A in primary school children. Outbreaks are more easily recognized in schools, because this is where cases in an area tend to be concentrated. Schools also provide conditions of close contact which facilitate spread of the virus. As the infection is spread by the intestinal/oral route, a high standard of personal hygiene should reduce the spread of infection even in a susceptible population. The toilet facilities in the school investigated are housed in a separate, uncovered block at some distance from the main school buildings. There are no washbasins in the toilet block and the nearest ones in the main building have no hot water or hand-drying facilities. Strict hygiene measures instituted when the outbreak came to notice probably helped in reducing the further spread of infection.

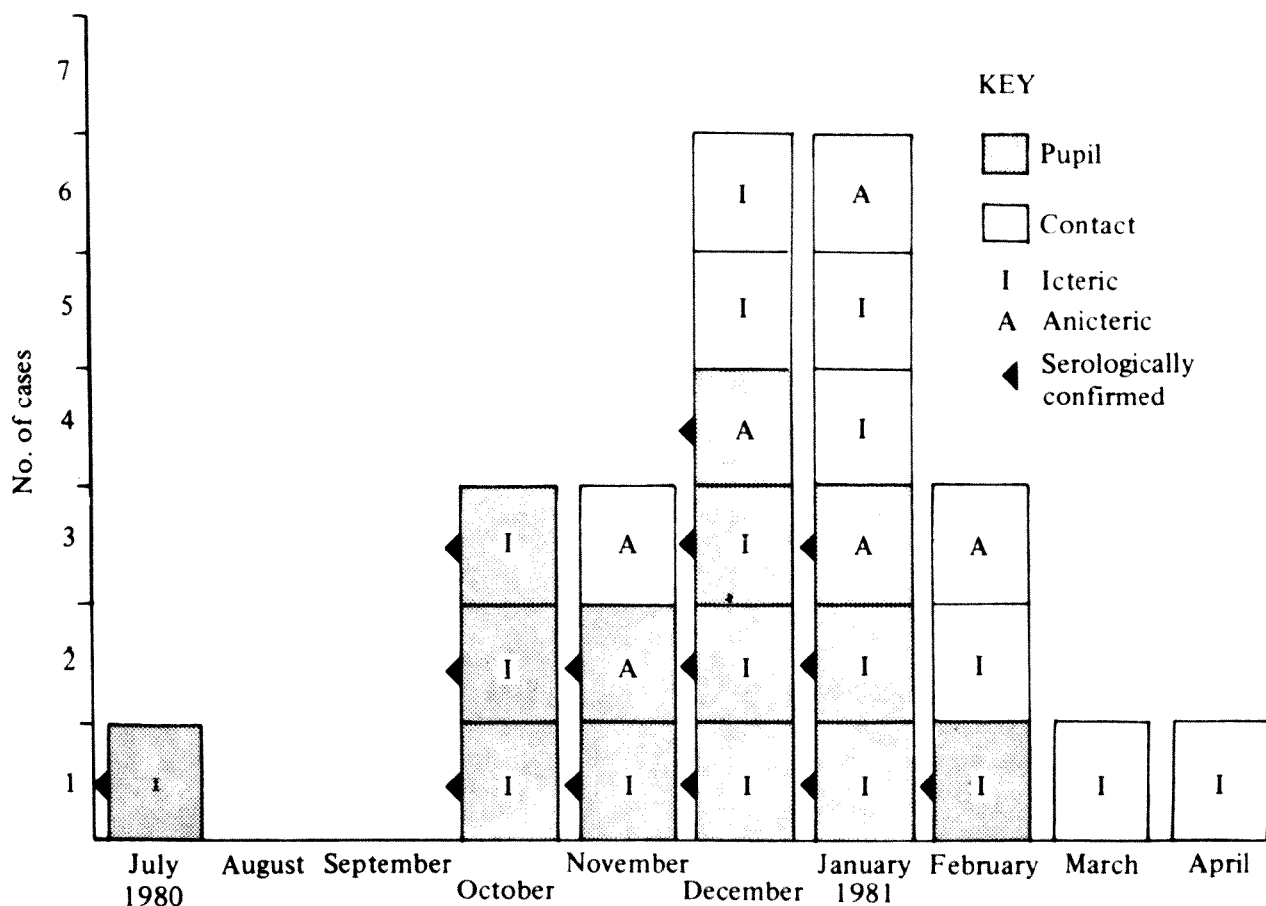


Fig. 1. Development of cases.

The development of cases over a period of 10 months suggests that the virus spread from person to person and was definitely not a common source outbreak. Interestingly, 29 of the 52 who showed immunity had one or more siblings who were also immune. All cases of hepatitis A, whether clinical or not, are infectious, and when further enquiries were made it was found that 10 close contacts of pupils at the school also had a clinical illness compatible with hepatitis A (Fig. 1). Seven of these developed jaundice, but unfortunately none was investigated serologically.

The clinical cases which are fully investigated usually represent only a fraction of those infected, the ratio of subclinical and anicteric clinical cases to icteric cases has been estimated to be approximately 10:1 in children (Zuckerman & Howard, 1979). In the outbreak described here there were 11 icteric cases. In addition there were 24 non-icteric cases in whom recent infection was diagnosed on the basis of HAV specific IgM. This gives an icteric:non-icteric ratio of 1:2. However, of the remaining 81 children from the outbreak school, 17 (21%) were HAV IgG positive, specific IgM negative. The expected number on the basis of the frequency of antibody (10%) in the control school is 8. Thus it is possible that the excess of 9 children in the outbreak school became antibody positive in relation to the outbreak. If these are included as subclinical cases then the icteric:non-icteric ratio is 1:3.

In most cases hepatitis A in children runs a mild, uneventful course as illustrated by this outbreak. It is however, worth bearing in mind that an increasing number

of adults are not immune and that the infection may prove more serious in this population with higher numbers of clinical cases (Noah, 1981, personal communication).

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