PRACTICE OBSERVED

Practice Research

Hepatitis B in general practice: epidemiology, clinical and serological features, and control

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Abstract

In an outbreak of hepatitis B virus infection in a south Wales village 31 cases were identified over 16 months. Spread of the infection was by sharing needles and by sexual contact. Twenty seven patients were known to have symptoms, of whom nine had an anicteric illness. Generally the illness was mild with no deaths, and there was only one chronic carrier. Two patients were only transiently positive for hepatitis B surface antigen. The outbreak was controlled by vigorous contact tracing and counselling.

Despite an enormous increase in workload for the general practitioner and problems including disposal of contaminated needles, outbreaks of hepatitis B virus infection may adequately be treated in the community.

Introduction

Since the discovery of Australia antigen by Blumberg *et al*¹ much of the epidemiology of hepatitis has been described.² Vertical transmission has been shown from antigenaemic mothers to their offspring.³⁻⁵ Horizontal transmission may occur from close, particularly sexual, contact (either heterosexual^{6,7} or homosexual^{8,9}). Horizontal transmission may also result from inoculation of

infected blood or blood products. This may occur during blood transfusion,¹⁰ as a result of needlestick injury,¹¹ or during intravenous drug abuse when contaminated needles are shared with carriers or acutely infectious people.^{12,13}

In England and Wales laboratory reports of hepatitis B virus infection almost doubled from 1980 to 1984.¹⁴ During this period reported cases from Wales increased from 24 to 54 a year (S Polakoff, personal communication). Much of this increase was due to infections in known intravenous drug abusers. During the summer of 1985 several outbreaks of hepatitis B virus infection occurred in south Wales. One of these was centred on a village in a Welsh mining valley. Most of the patients belonged to one group practice. We describe this outbreak and its transmission within the community and discuss some of the problems in its control.

Patients and methods

Cases were defined as patients who were found to have hepatitis B surface antigenaemia or antihepatitis B antibodies. Fourteen presented to one of us in general practice with clinical disease. Ten other patients with clinical disease presented to other general practitioners. The index case was diagnosed from a blood donation and one further patient diagnosed while receiving inpatient care for another condition. In addition to these cases, more than 150 people presented themselves to, or were discovered by, the primary health care team (general practitioners and health visitors) for screening and counselling. The remaining cases were identified from this group.

A detailed clinical history was taken and a physical examination made of all those presenting to the surgery. In addition to symptoms, patients were questioned about any history of drug abuse, shared needles, and sexual contacts. Blood was taken for liver function tests and testing for serological markers for hepatitis B. Serological testing for hepatitis B markers was by radioimmunoassay.^{15,16} All serum samples were examined for hepatitis B surface antigen (HBsAg) and antihepatitis B core (anti-HBc) IgG antibody. Those samples found to contain HBsAg were examined for hepatitis B e antigen (HBeAg) and anti-HBe. All serum samples from patients with HBs

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antigenaemia were sent to the Virus Reference Laboratory, Central Public Health Laboratory, London, for examination for anti-HBc IgM and delta (δ) antigen and antibody.

Results

OUTBREAK

From May 1985 to September 1986, 26 cases of HBs antigenaemia occurred, 24 of the patients having a related clinical illness. In a further five cases hepatitis B antibodies were detected in the absence of HBsAg. Two of these patients had seroconverted after screening early in the outbreak and three had hepatitis B antibodies when first examined. Of these five patients, three reported a recent illness and one was asymptomatic; no information was available in the remaining case. All 31 cases identified were epidemiologically related. The table gives the dates of onset of illness in the 25 patients with symptoms for whom the date of onset was known.

Month of onset of symptoms in 25 cases of hepatitis B virus infection

	1985								1986							
	May	J	J	A	S	O	N	D	J	F	M	A	M	J	J	A
No of cases	1	1	3	3	7	3	1	1	1	0	2	0	0	0	1	0

Twenty five of the 31 patients, including most of the nucleus group of frequent drug abusers, lived on one council estate. The estate consisted of 490 dwellings built in the early 1960s to accommodate miners transferred from exhausted pits higher up the valley. Unemployment was high owing to closure of the remaining pits.

SPREAD OF INFECTION

All cases were traced to one man (see fig 1). This patient was found to have HBsAg in a blood donation in May 1985; no clinical illness was known. Probably he had contracted the infection from a heroin dealer in the Bristol area, who was a known carrier. This particular dealer had a reputation for "cutting" the heroin and it was usual to sample some, using shared needles, before purchase to test its purity.

From this one case spread occurred to three further patients by shared needle contact. The fourth patient was responsible for most of the remaining cases. All further patients who were intravenous drug abusers shared needles with her. Also four of the 10 patients who were not intravenous drug abusers were sexual contacts of this woman. The remaining six cases were sexual contacts of other patients who were intravenous drug abusers. Only the initial two cases were in people who habitually abused heroin. All the remaining drug abusers used amphetamines. Urinary opiates were detected in one other patient, though this was probably due to cutting of the amphetamine with small amounts of heroin.

The woman in case 4 admitted to sexual contact with nine men during one week in early June. This was immediately after a suction termination of pregnancy and while she was still bleeding. Of these nine contacts, six were screened for serological markers. In five HBsAg was detected, though in one only transiently, giving an attack rate of 83%. Of 15 known sexual contacts of all patients (who were not known intravenous drug abusers), however, 10 (67%) contracted hepatitis B. Of the main group of intravenous drug abusers, who were suspected of sharing needles regularly, 24 out of 35 contracted hepatitis—that is, an attack rate of 68.6%. There were, however, large numbers of other people who were reported as occasional shared needle contacts of this group. None of these developed hepatitis. Figure 1 summarises the spread of the infection through the at risk group.

CLINICAL FEATURES

The incubation period was virtually impossible to determine owing to the frequency of contacts. For sexual contacts of the patient in case 4 this varied between 10 and 14 weeks. In the 30 cases for which any clinical details were available 18 patients (60%) developed acute hepatitis with jaundice, 9 (30%) had an anicteric flu-like illness, and 3 (10%) were apparently asymptomatic.

More detailed clinical information was available on the 14 patients with symptoms who presented to one of us as their general practitioner. Typically

in both anicteric and icteric cases the initial illness was similar to flu. This lasted from 48 hours to a few weeks and was characterised by high temperature, rigors, myalgia, hepatic tenderness, and severe malaise. Seven patients reported polyarthralgia, five a rash, usually morbilliform but including one case of giant urticaria, and 11 symptoms of gastric hyperacidity (especially oesophageal reflux) relieved by antacids. Symptoms became less severe as jaundice developed. Four patients had a relapse of symptoms after their initial clinical recovery. In three cases this coincided with the appearance of anti-HBe. The relapse of symptoms was indistinguishable from the initial illness and was mirrored by an increase in serum transaminase activities.

SEROLOGY

None of the contacts screened early in the outbreak possessed anti-hepatitis B antibodies. Three patients in whom anti-HBc was detected on first testing were all seen at least six months after the start of the outbreak.

FIG 1—Spread of hepatitis B through population at risk. → = Needle contact with or without sexual contact. —→ = Sexual contact only. Figures in symbols refer to first (index case) and fourth patients in series.

Apart from the index case, all those found to be antigenaemic had a related clinical illness. It appeared that the population had not previously experienced hepatitis B infection.

In two cases HBsAg was detected transiently in only one sample. One patient had a clinical illness, the other was asymptomatic. Neither patient developed anti-HBs, anti-HBc, or anti-HBe. The findings were confirmed by the virus reference laboratory. In those cases in which HBsAg was detected before the onset of clinical illness it predated symptoms by two weeks. In no case was δ antigen or antibody detected.

We could follow the antigen state of 19 patients, excluding the two with

transient HBs antigenaemia. Figure 2 shows the persistence of HBsAg and HBeAg after diagnosis. In half the patients HBeAg had cleared from the serum by two weeks. No patient had HBe antigenaemia after 12 weeks. Only half the patients were clear of HBsAg by 10 weeks, one patient remaining positive 18 months after diagnosis. In most cases anti-HBe had developed by the time the first serum sample was negative for HBeAg. One patient, however, had neither antibody nor antigen for over three weeks.

FIG 2—Persistence of hepatitis B antigens in patients' serum after diagnosis.

CONTROL MEASURES

The outbreak was controlled by vigorous tracing of contacts by general practitioners, health visitors, and the community physician. These contacts were then counselled about the mode of transmission of the infection and how it could be prevented. Hepatitis B vaccination was offered only to health care personnel who were likely to come into direct contact with cases. The only exception was the pregnant spouse of a man with the disease and her baby immediately after delivery.

Discussion

The attack rate in this outbreak of hepatitis B virus infection was high compared with previous studies,^{6,7} particularly in the nucleus group of frequent drug abusers. This probably reflects the high frequency of drug and sexual contact within the group and also the total lack of immunity in the population. No cases were identified among people who were not direct contacts of known drug abusers. The lack of spread of the disease away from the main group may reflect the success of control measures.

Generally the illness suffered by patients in this outbreak was clinically mild. Indeed, many of the cases would probably have been missed were it not for the increased awareness of general practitioners. Only one patient became a chronic carrier. There were no deaths or cases of chronic hepatitis. Only one patient was admitted to hospital, and she took her own discharge next day. Only two of the four patients referred for outpatient assessment attended. Probably a proportion of mild cases remained undiagnosed. In adults hepatitis B virus infection may not be as severe an illness as widely believed, certainly in patients not needing admission to hospital. Furthermore, chronic carriage may well be an uncommon complication of acute hepatitis in adults.

Fever, arthralgia, and rash were frequent findings in this study. This combination of clinical features is very similar to glandular fever. Many cases of hepatitis B virus infection may be misdiagnosed as such in general practice. Possibly all cases of Paul-Bunnell negative glandular fever should be screened for HBsAg. Cases of hepatitis B virus infection have been diagnosed in our laboratory in patients with this provisional diagnosis.

Hepatitis B vaccine was not offered routinely to contacts, as a study of sexual contacts vaccinated after exposure had shown no significant reduction in infection rates.¹⁷ It seems that hepatitis B vaccination is not helpful in the control of such outbreaks. Most patients will have been infected before the doctor becomes aware of the problem. By contrast, hepatitis B immunoglobulin has been

shown to be effective.⁷ Supplies are limited, however, and stocks were insufficient to treat such a large group.

As a result of counselling some modification of behaviour was apparent. There was a reduction in drug abuse for a while after the start of the outbreak. Unfortunately, the local and national media spread the erroneous story that the infection was being transmitted by the use of syringes discarded from the local hospital, which minimised the importance of person to person spread. The publicity had one negative effect. Children on the estate who had not started abusing parenteral drugs began stabbing each other with syringe needles to prove that they were not "yellow" about catching hepatitis.

The outbreak had an enormous effect on the workload of the practice (seven doctors, 15 000 patients), particularly in the early weeks. In part this was due to the decision to treat most of the cases in the community and undertake contact tracing and counselling. There were also problems with contaminated needles, special arrangements having to be made with the local general hospital for disposal.

We suggest that, though hepatitis B may be a severe and unpleasant infection, many cases can adequately be treated by the general practitioner in the community. Such outbreaks, especially in smaller communities, may be controlled by contact tracing and counselling. Furthermore, our findings suggest that studies of hepatitis B vaccination based on the assumption that patients will be treated as inpatients^{18,19} may have overestimated the cost effectiveness of vaccination.

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