Hepatitis B in a hospital for the mentally subnormal in southern England

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British Medical Journal, 1978, 2, 594-596

Summary and conclusions

The prevalence of hepatitis B viral (HBV) infection was assessed in 340 patients and 268 staff in a hospital for the mentally subnormal in Wessex. Hepatitis B surface antigen (HBsAg), antibody to hepatitis B core antigen, antibody to HBsAg, e antigen (eAg), and antibody to eAg were used as markers of such infection. Forty patients and 10 staff had evidence of recent or current infection, while 149 patients and 50 staff had evidence of past infection. HBV markers were more common in mongols, epileptics, patients with cerebral palsy, and those of lower mental grades and reached a peak after 5-15 years of hospitalisation. eAg was detected in 12 out of 26 patients with HBsAg but in none of the four staff with HBsAg. Abnormal liver function values were found in 24 (60%) of the patients with recent or current HBV infection but in only 30 (19%) of those without HBV markers. Among the staff the prevalence of HBV markers correlated with the duration of employment and degree of contact with patients. Of those with recent or current infection, 4 (40%) had abnormal liver function values compared with 25 (12%) of those without HBV markers. Despite the high prevalence of markers clinically overt hepatitis B was rare.

Introduction

Viral hepatitis is hyperendemic in institutions for the mentally subnormal, and several studies of the prevalence of hepatitis B in mentally retarded patients have been published from the United States. There are no equivalent reports from the United Kingdom, however, and, furthermore, such surveys have not included analysis of the hepatitis B core and e antigen-antibody systems. We report the prevalence of such markers of hepatitis B infection in patients and staff in a Wessex hospital for the mentally subnormal.

Subjects and methods

Patients—Serum and clinical details were obtained from 340 patients resident in the hospital. The patients were housed in 11 villas and one hospital ward. Two of the villas were for children and one was for geriatric patients; the other villas housed patients of varying age but divided according to sex and degree of mental and

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physical disability. The prevalence of mongolism, epilepsy, and severe physical handicap varied from villa to villa, but overall 36 patients were mongols, 116 were epileptic, and 41 had some form of major cerebral palsy. The degree of mental disability varied, but most patients were classed as severely subnormal (287) and the rest as subnormal (53); 243 were male and 97 female, and their ages ranged from 2 to 83 years (mean 38 years).

Staff—Serum was obtained from 268 staff. Of these, 143 were nursing or auxiliary staff in direct physical contact with the patients (nursing staff), and 125 were administrative or ancillary workers with only indirect contact with patients (non-nursing staff). Altogether 105 were male and 163 female, and their ages ranged from 19 to 64 years (mean 40 years). The duration of employment in the hospital and any past history of hepatitis were recorded.

Laboratory investigations—All sera were coded and tested for hepatitis B surface antigen (HBsAg) by passive haemagglutination (Hepatest, Wellcome), antibodies to HBsAg (anti-HBs) by radio-immunoassay (Ausab, Abbott), antibodies to hepatitis B core antigen (anti-HBc) by complement fixation, and e antigen and antibodies (eAg and anti-e) by immunodiffusion. Serum alanine and aspartate aminotransferase and alkaline phosphatase concentrations were measured as tests of liver function. Liver function values (LFVs) were considered to be abnormal if any value exceeded the normal range by 50% or more. Raised alkaline phosphatase concentrations were ignored in those aged below 20 years and those taking anticonvulsant drugs.

The χ^2 test was used to analyse the results.

Results

PATIENTS

Of the 340 patients, 26 (19 male, 7 female) were carriers of HBsAg (table I); 12 of these had eAg but none had anti-e. Thirty-nine had anti-HBc, including all the HBsAg carriers. One hundred and forty-nine had anti-HBs. Overall, 184 (54°_{\circ}) had at least one serum marker of past or present infection with hepatitis B virus (HBV). Anti-HBs

TABLE I—Prevalence of HBV markers in patients (n = 340) and staff (n = 268)

	Anti-HBs		Anti-HBc		HBsAg		eAg		Overall prevalence of HBV markers	
Patients Staff	No 149 50	43·8 18·7	39 10	11·5 3·7	No 26 4	7·6 1·5	No 12*	3.5	No 184 59	54·1 22·0

^{*}All carriers of HBs Ag.

was more commonly found in those with mongolism, epilepsy, and cerebral palsy than in those with no physical abnormalities (P < 0.05), but HBsAg and anti-HBc were more common only in mongols (P < 0.02) (fig 1). All HBV markers occurred more often in those graded severely subnormal than in those graded subnormal (P < 0.05) (table II). The prevalence of HBV markers was relatively constant in patients up to 30 years of age but declined thereafter (table III). The markers were gradually acquired over the first five years in hospital (4.5°_{-0} HBsAg-positive), reached a peak after 10-15 years (16°_{-0} HBsAg-positive), but then became progressively less common (4.4°_{-0} HBsAg-positive after 25 years or more).

Ninety-two of the patients had abnormal LFVs. More of those with HBsAg or anti-HBc had abnormal LFVs than those with anti-HBs or no HBV markers (P < 0.001) (fig 2).

Past medical history and medication did not appear to be relevant. Most of the patients were receiving some form of medication but this did not influence the distribution of HBV markers or abnormal LFVs. Acute hepatitis B was rare and was recorded only three times in the hospital patients between 1970 and 1977. Acute non-B hepatitis was more common: 61 such cases occurred in 1971, and thereafter sporadic cases occurred, averaging one a year. HBV markers were not more prevalent in these patients.

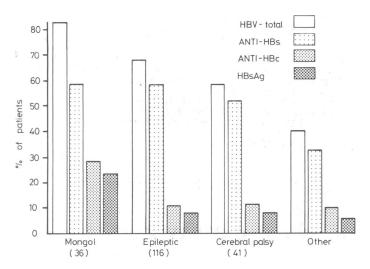


FIG 1—Prevalence of HBV markers in 340 patients subdivided according to physical abnormalities.

TABLE II—Distribution of HBV markers in 340 patients according to mental grade

	Subnorm	al (n = 53)	Severely subnormal (n = 287)				
	No	0.0	No	0			
HBsAg Anti-HBc Anti-HBs	ti-HBc 2		25 37 135	8·7 12·9 47·0			

TABLE III—Distribution of HBV markers in 340 patients with respect to age

Age (years)	No tested	Proportion HBsAg positive		Proportion anti-HBc positive		Proportion anti-HBs positive		
		No	00	No	0,0	No	00	
\$10 11-20 21-30 31-40 41-50 51-60 >60	5 51 77 49 63 54 41	1 7 10 3 3 1	20·0 13·7 13·0 6·1 4·8 1·9 2·4	1 12 17 3 2 2 2	20·0 23·5 22·1 6·1 3·2 3·7 4·9	3 31 42 24 29 13 6	60·0 60·8 54·5 49·0 46·0 24·1 14·6	

STAFF

Four of the 268 staff were carriers of HBsAg; of these, none had eAg and one had anti-e. Ten had anti-HBc and 50 anti-HBs. Overall, 59 (22° $_{0}$) had at least one HBV marker (table I). All HBV markers were more common in nursing than non-nursing staff (P < 0·001) (table IV). Among the nursing staff the prevalence of HBV markers increased with duration of hospital employment. In the first year one-fifth acquired an HBV marker, the prevalence rising to almost half after 10 years or more of hospital service. In the general population of the Wessex region the prevalence of HBsAg is around $0\cdot1^{\circ}_{0}$ and of anti-HBs below 5°_{0} .

Abnormal LFVs were significantly more common in staff with HBsAg (50°_{0}) and anti-HBc (44°_{0}) than in those without HBV markers (12°_{0}) (P < 0.01) (fig 2). Acute hepatitis B was rare, being recorded only twice between 1970 and 1977. Acute non-B hepatitis occurred sporadically but infrequently. No specific precautions were taken to prevent infection other than common-sense hygiene measures.

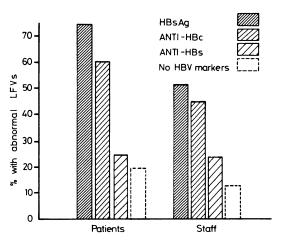


FIG 2—Prevalence of abnormal LFVs in relation to HBV markers in patients and staff.

TABLE IV—Prevalence of HBV markers in staff with (nursing) and without (non-nursing) direct contact with patients

	Anti	Anti-HBs		Anti-HBc		HBsAg		Overall prevalence of HBV markers	
	No	0.0	No	%	No	0:	No	0/	
Nursing (n = 143) Non-nursing (n = 125)	36 14	25·2 11·2	9	6·3 0·8	3	2·1 0·8	44 15	30·8 12·0	

Discussion

Our observations on hepatitis B markers in an institution for the mentally subnormal in southern England are generally in accord with reports from the United States. The greater prevalence in mongols in our series has often been noted before.2 3 The increased prevalence in those of lower mental grade or with physical handicap was suggested by Szmuness et al,4 although their findings were not statistically significant. As in other studies, we noted that HBsAg was most common in those admitted to hospital at an early age and in care for between five and 15 years.4 5 LFVs are much more often abnormal in those with HBsAg than in those without⁵ but are not influenced by anti-HBs.5 We found HBsAg in 22% of the mongols and 6% of patients with other mental disorders, prevalence figures similar to those in other studies.⁵ ⁷⁻⁹ Anti-HBs was found in 58% of the mongols and 40% of patients with other mental disorders; this conflicts with other reports, in which anti-HBs was less common in mongols.3 4

We found that patients and staff with HBV markers rarely had a history of acute hepatitis B and that staff who were in direct contact with patients had a greater prevalence of HBV markers than non-nursing staff, findings that have both been reported. Of particular interest were our observations on anti-HBc, eAg, and anti-e. Anti-HBc is thought to be a marker of HBV replication and may be present in the absence of HBsAg. Anti-HBc is probably a more sensitive marker of viral replication than HBsAg, though this point is still undergoing evaluation. We regarded anti-HBc as indicating current or recent HBV infection and showed that this would be considerably underestimated by looking at HBsAg alone.

Magnius and Espmark¹¹ described a new antigen-antibody system, designated "e," which is found only in the presence of HBsAg. eAg correlates positively with infectivity, severity, and chronicity of liver disease, whereas with anti-e the converse is true.¹²⁻¹⁵ We discovered a high prevalence of eAg and a low prevalence of anti-e in patients with HBsAg. This finding together with the high prevalence of abnormal LFVs suggests

that among patients in this institution the HBsAg carrier state is not benign and is probably associated with a high level of infectivity and chronic liver disease.

Among staff the prevalence of HBsAg was low, although higher than in the general population, eAg was not found, and clinically overt hepatitis B was rare. Nevertheless, half the nursing staff who had served for 10 years or longer had anti-HBs. This suggests that active immunity is acquired with little clinically overt disease. There is therefore no indication for routine passive prophylaxis, but should safe and simple active immunisation become available this should be recommended in such "at-risk" groups. In the event of needle-stick accidents or exposure to similar "extra risks" hepatitis B immune globulin should be given.

We thank G D Searle and Co, High Wycombe, Bucks, for financial support and technical help, and Mr B Cohen, of PHLS, Colindale Avenue, London NW9, for kindly supplying HBcAg for the anti-HBc assay.

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(Accepted 27 June 1978)

Home blood sampling for plasma glucose assay in control of diabetes

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British Medical Journal, 1978, 2, 596-598

Summary and conclusions

Estimation of plasma glucose in home blood samples is needed to improve diabetic control. Sufficiently precise measurements on capillary blood were obtained by (a) storing Reflotest glucose-oxidase strips in a desiccant container before reading and (b) collecting blood samples into a simple vacuum bottle containing potassium fluoride (assay of sodium content indicating volume of plasma collected). The precision of the methods (± 1 SD) was ± 0.35 mmol/l (± 6.3 mg/100 ml). Clinical reliability was assessed by measuring the basal plasma glucose concentration at home on different mornings in patients with maturity-onset diabetes, the day-to-day variation (± 1 SD) being ± 0.73 and ± 0.92 mmol/l (± 13.2 and \pm 16·6 mg/100 ml) respectively.

The mean basal plasma glucose concentration in all 84 patients with maturity-onset diabetes from three general practices was 8 mmol/l (144 mg/100 ml), 44 of the values exceeding 6 mmol/l (108 mg/100 ml). Improving control by monitoring the basal plasma glucose concentration in maturity-onset diabetes might help to prevent diabetic complications.

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Introduction

Current treatment of diabetes does little to prevent complications, possibly because control based on urine glucose estimation is not precise enough. Improved control might be gained by regular measurement of the plasma glucose concentration at home. In maturity-onset diabetes a logical aim is to lower the basal plasma glucose concentration to normal,1 2 while in insulin-dependent diabetes additional plasma glucose estimations after meals are needed to determine the requirement for insulin before meals. Although plasma glucose may be estimated at home by patients with glucose-oxidase strips and a colorimeter,3 4 the need for equipment means that the method is not widely applicable.

In this paper we examine alternative methods for home capillary blood sampling, including a stored glucose-oxidase strip and a new collector bottle. Their clinical use has been validated by fasting plasma glucose measurements in patients with maturity-onset diabetes.

Methods

Reflotest—The colour of Reflotest glucose (Boehringer Mannheim) remains stable when stored under suitable conditions (see Results). Patients prick their finger with an automatic lancet (Autolet; Owen Mumford, Woodstock, Oxon), apply a drop of blood on to a Reflotest strip, wait exactly 60 seconds, wipe off the blood, and send the strip in a desiccator container to hospital or general practice clinic for reading in a Reflomat colorimeter. Each strip is closely inspected and discarded if the colour is uneven, indicating that the single drop of blood has not been correctly applied.

Vacuum collector bottles-Small polyethylene bottles containing approximately 1.2 ml Analar potassium fluoride 100 g/l are sealed after being compressed to produce a partial vacuum. The bottle top, which is covered by a screw cap, contains a shallow depression. After pricking a finger the patient puts a drop of blood into the depression. He then pricks through the drop (with the lancet used to prick his finger) to make a small hole, and when the lancet is withdrawn the