SHORT REPORTS

Hepatitis B in a West Indian population in the United Kingdom

The prevalence of markers of hepatitis B virus infection shows considerable geographic variation, often being high in tropical areas where the standard of living is low. While the high prevalence in the far east is maintained by vertical transmission from mother to baby in the perinatal period,[1] in Africa infection is usually transmitted horizontally during infancy.[2]

In the West Indies the prevalence of carriage of hepatitis B surface antigen (HBsAg) is 0-4·1%,[3,4] varying between islands and according to the sensitivity of the test used, but we are unaware of any report on prevalence among West Indians in the United Kingdom. To assess the need for screening for markers of hepatitis B virus in this group we determined the prevalence among West Indians and their descendants attending a department of genitourinary medicine in the district of Lambeth.

Methods and results

Serum samples were collected from 454 heterosexual West Indian negroes (195 born in the West Indies and 259 first generation, born in Britain of West Indian descent) and 180 white heterosexuals attending the department of genitourinary medicine at this hospital during February and March 1985. Each was tested for HBsAg by radioimmunoassay (Ausria II, Abbott, North Chicago, Illinois). Positive results were confirmed using 0·1% modified reverse passive haemagglutination, and these serum samples were then tested for hepatitis B e antigen (HBeAg) and antibody (HBeAb) by radioimmunoassay. All samples negative for HBsAg were tested for antibody to surface antigen (HBsAb). A sample with a result higher than a 50 IU control was considered to be positive for HBsAb, as was one below the 50 IU control but with a result above the cut off for the kit if it was positive for antibody (IgG) to core antigen (HBcAb). Random samples negative for other markers of hepatitis B virus were also tested for HBcAb by enzyme immunoassay (Corzyme, Abbott). Statistical analysis was by χ^2 with Yates's correction.

The table compares the prevalence of markers of hepatitis B virus infection in native West Indians with that in first generation patients of West Indian descent born in Britain and white patients.

All markers were more common in native West Indians; 18·5% of this group were positive for surface markers compared with 2·7% of first generation West Indians ($p<0·001$). This difference persisted when men and women were analysed separately. Of the eight native West Indians (4·1%) with HBsAg, two were HBeAg positive and five HBeAb positive, and one had no HBe markers. Only one first generation West Indian (0·4%) was positive for HBsAg ($p<0·025$), and he was also HBeAg positive; he denied exposure to other risk factors for infection with hepatitis B virus. Although HBsAg was not detected in any white patients, five (two men and three women) had HBsAb. One of these subsequently gave a history of vaccination against hepatitis B virus, one was a soldier, and two had a history of jaundice.

Hepatitis B virus (HBV) markers in native West Indians, first generation West Indians born in Britain, and white patients

Ethnic group	Country of birth	Mean (SD) age (years)	No (%) positive for: HBsAg	HBsAb	HBcAb	(%) Positive for ≥1 HBV marker
Negro (West Indian)	West Indies	33·0 (8·9)	8/195 (4·1)	28/195 (14·4)	2/43 (4·7)	23
Negro (West Indian)	United Kingdom	22·4 (5·1)	1/259 (0·4)	6/259 (2·3)	0/44	2·7
White	United Kingdom	27·8 (8·0)	0/180	5/180 (2·8)		2·8

Comment

These results confirm a relatively high prevalence of hepatitis B infection in native West Indians but not in those of West Indian descent born in the United Kingdom. The 0·4% prevalence of carriage of HBsAg in the first generation West Indians approaches the 0·2% found in the indigenous British population,[5] indicating that this West Indian population adopted the British pattern of infection with hepatitis B virus over only one generation.

The results suggest that screening for HBsAg should be undertaken among native West Indians but not their descendants born in Britain. Such a policy would result in considerable financial saving in areas like Lambeth, which has a large West Indian population.

The disparity in the prevalence of hepatitis B virus markers between the native West Indian population and those born in Britain suggests that perinatal transmission from mother to baby is uncommon among West Indians and that environmental factors in the tropics may be more important.

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Gall bladder contraction induced by cholecystokinin: bolus injection or infusion?

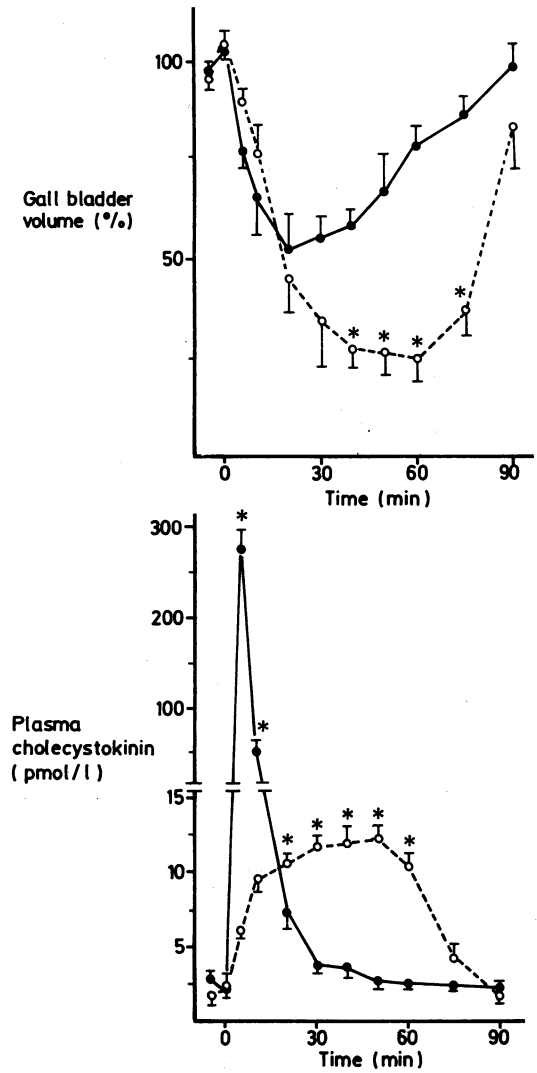
Inducing contraction of the gall bladder has been reported to be of value in diagnosing biliary tract disorders.[1] This procedure may facilitate the diagnosis of adenomyomatosis of the gall bladder, small gall stones, and biliary tract obstruction. Furthermore, measurement of gall bladder emptying in response to cholecystokinin may detect impaired emptying, as found in acalculous gall bladder disease.[2] Both intravenous bolus injection and infusion of cholecystokinin have been used to induce gall bladder emptying.[1,2] Since a rapid and strong contraction is needed we have compared the time course and degree of gall bladder contraction induced by bolus injection and infusion of similar amounts of cholecystokinin. We have also measured the plasma concentrations of cholecystokinin to obtain more insight into the mechanism of gall bladder contraction in response to this hormone.

Subjects, methods, and results

On two separate mornings six fasting healthy volunteers (four men) aged 20-27 years were studied in random order. On one morning cholecystokinin (Kabi Diagnostica, Studsvik, Sweden), 1 Ivy dog unit/kg, was administered by intravenous bolus injection over one minute, while on the other morning the same dose of cholecystokinin was administered as a continuous infusion over 60 minutes. Images of the gall bladder were obtained in duplicate by real time ultrasonography at −5, 0, 5, 10, 20, 30, 40, 50, 60, 75, and 90 minutes.[3] Gall bladder volumes were measured as described using a computer.[3] Each time the gall bladder was imaged blood samples were taken for measurement of cholecystokinin. Plasma cholecystokinin concentrations were measured by a sensitive and specific radioimmunoassay employing antibody T204, which binds to all biologically active forms of the hormone.[4] Results were expressed as the mean and one standard error of the mean. Statistical analysis was by Student's *t* test for paired samples. Informed consent was obtained from all the subjects.

Both bolus injection and infusion of cholecystokinin induced significant decreases in gall bladder volume. Infusion of cholecystokinin induced a strong contraction reaching a plateau for at least 30 minutes, whereas after bolus injection maximum gall bladder contraction was followed rapidly by relaxation

(figure). Maximum individual gall bladder contraction during infusion (79 (SEM 5)%) was significantly greater ($p<0.05$) than that after bolus injection (58 (5)%). Furthermore, when the variability of gall bladder volumes at the time of mean maximum contraction (that is, 20 minutes after bolus injection and at 60 minutes during infusion) was analysed gall bladder contraction after bolus injection ranged from 5% to 71% compared with 57% to 89% during infusion. Peak increments in plasma cholecystokinin during infusion (10·7 (SEM 0·7) pmol/l; 42 (2·7) ng/l), however, were significantly smaller ($p<0.0001$) than those after bolus injection (275 (25) pmol/l; 1·1 (0·1) μg/l). Plasma cholecystokinin concentrations during infusion were significantly raised above basal value in all samples obtained during the 60 minute infusion period ($0.0001<p<0.05$), whereas after bolus injection plasma cholecystokinin concentrations were significantly raised for only 25 minutes ($0.0001<p<0.05$) (figure). After bolus injection of cholecystokinin all subjects experienced transient abdominal discomfort, cramps, nausea, and a feeling of facial flushing, whereas no side effects were reported during infusion of the hormone.



Effects of one minute bolus injection (●) and 60 minute infusion of cholecystokinin (○) on gall bladder volumes (top) and plasma cholecystokinin concentrations (bottom).

*Significant difference between bolus injection and infusion of cholecystokinin (see text).

Conversion: SI to traditional units—Cholecystokinin: 1 pmol/l≈3·9 ng/l.

Comment

This study shows that a 60 minute infusion of cholecystokinin is a more efficient stimulus of gall bladder contraction than a bolus injection of the same dose. Gall bladder contraction during infusion of cholecystokinin was not only stronger but also more consistent and longer lasting than that after bolus injection. Hence this procedure may facilitate the visualisation of biliary tract abnormalities and is an important technique for the diagnosis of impaired gall bladder contraction. Furthermore, that maximum gall bladder contraction during cholecystokinin infusion is maintained for at least 30 minutes makes the exact timing of gall bladder imaging less critical.

Our finding that a 60 minute infusion of cholecystokinin is a more efficient stimulus than a bolus injection of the same dose is the more interesting because peak plasma concentrations of cholecystokinin were substantially lower during infusion than after bolus injection of the hormone. Significant increases in plasma cholecystokinin, however, were observed for longer during infusion than after bolus injection. We therefore conclude that both the degree and the duration of the rise in plasma cholecystokinin are important for inducing gall bladder contraction. The relative ineffectiveness of the large increase in plasma cholecystokinin after bolus injection in inducing a more efficient gall bladder contraction may be related to various factors, such as sluggishness of the gall bladder to respond to stimuli and spasm of the cystic duct induced by supraphysiological plasma cholecystokinin concentrations.[5] Interestingly, plasma cholecystokinin concentrations found during infusion of the hormone were comparable to those observed after intraduodenal instillation of fat and were accompanied by similar degrees of gall bladder contraction.

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Lack of correlation between fulminant form of viral hepatitis and retrovirus infection associated with the acquired immune deficiency syndrome (AIDS) in drug addicts

The observation in the past two years of a spread of infection of the acquired immune deficiency syndrome (AIDS) in our area[1] and a remarkably increased use of intravenous drugs in cases of viral fulminant hepatitis (not entirely explained by the wide circulation of hepatitis viruses in drug addicts, as the prevalence rises from 20% in uncomplicated cases to about 70% in fulminant ones)[2] has led us to investigate a possible role of AIDS associated retroviruses in determining this condition.

Patients, methods, and results

We investigated antibodies to AIDS associated retroviruses in 18 consecutive patients referred to our department over the past 24 months (May 1983-May 1985) for viral fulminant hepatitis: 13 had hepatitis B virus, six of whom had hepatitis delta coinfection and three hepatitis delta superinfection, and five had non-A, non-B virus (15 men aged 17-24); 13 of whom had a declared history of prolonged use of intravenous drugs. We also examined 156 cases of acute viral hepatitis as control patients (90 had hepatitis B virus, of whom 28 had associated hepatitis delta, and 66 had non-A, non-B virus), made up of 78 consecutive drug addicts and 78 patients matched by age, sex, and geographical provenance, admitted to our department during the same period.

Diagnoses were made according to international criteria and confirmed by necropsy in the 15 patients who died (83%). Antibodies to AIDS associated retroviruses were determined, as previously described,[3] by indirect immunofluorescence on a human T cell line infected with AIDS associated retroviruses (E11/HUT-78), controlling specificity of the test on non-infected HUT-78 cells.

Among our 18 patients with fulminant hepatitis, one of the 13 who were drug addicts and none of the five who were not drug addicts had antibodies against AIDS associated retroviruses. Among the 156 patients with uncomplicated acute hepatitis, six of the 78 who were drug addicts had antibodies but none of the 78 who were not drug addicts had them. Antibodies against AIDS associated retroviruses were thus significantly more common among drug addicts than