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Hepatitis B virus infection in thalassaemia major treated in London and Athens

Chronic liver disease is a common complication of β thalassaemia major.¹ This complication might result from iron overload or blood-borne viral infection, or both. We investigated the incidence of hepatitis B virus infection in two groups of patients with β thalassaemia major on identical iron chelation treatment—one in an area endemic for hepatitis and one in a non-endemic area.

Patients, methods, and results

Twenty nine thalassaemic patients of Greek, Turkish, or Indian origin living in London and 29 Greek patients living in Athens were studied. Twenty three of the patients living in London had been born in the UK and had received all their transfusions there. All patients had been transfused since infancy at intervals of four to six weeks. Subcutaneous desferrioxamine had been started five years before this study and at that time ferritin concentrations and serum aspartate transaminase (AST) activities had been recorded. Parents and siblings of 17 of the London patients were also studied. Hepatitis B surface antigen (HBsAg), anti-HBs, antibodies to hepatitis B core antigen (anti-HBc), and IgG antibodies to hepatitis A virus were tested by radioimmunoassay (Abbott). Serum ferritin concentrations were measured by radioimmunoassay and AST activities by SMAC-Technicon. Statistical analysis was performed by measuring the difference of sample means of unequal sizes.

One London patient was HBsAg positive; 12 had anti-HBs, anti-HBc, or both. No Athenian patient had HBsAg, but 28 had anti-HBs, anti-HBc, or both. No London patient but 12 Athenian patients had antibodies to hepatitis A virus (table). AST activity was raised in 11 London patients and in 22 Athenian patients, with higher mean activities in Athens than in London ($p < 0.02$). Athenian patients also had higher AST activity at the beginning of chelation treatment ($p < 0.005$). There was no difference in AST activities before and after chelation treatment within either group. In London four of the 16 patients with no markers of hepatitis B virus and seven of the 13 with markers had raised AST activities. The AST values in the London patients with hepatitis B markers were similar to those in the Athenians with hepatitis B markers, although the latter group had higher AST values than London patients with no hepatitis B markers ($p < 0.005$). The initial ferritin concentrations had been higher in the Athenian patients,

but at the time of the study they were similarly raised in both groups ($p < 0.02$). In both groups ferritin concentrations fell significantly after chelation treatment, particularly in the Athenian patients ($p < 0.001$). All family members of two of the London patients with hepatitis B virus had no hepatitis B markers.

Comment

A high percentage of thalassaemic patients both in Athens and London have antibodies to hepatitis B virus antigens. Passive transfer from transfusion is unlikely since anti-hepatitis B virus antibodies predominate over anti-A virus antibodies in thalassaemic patients, whereas the reverse occurs in blood donors.^{2,3} The high incidence of markers of hepatitis B virus in London was unexpected since the prevalence of HBsAg positivity in the normal population is very low.³ The family study in London patients showed that the possibility that infection had been acquired from relatives was unlikely. The most likely source was transfused blood, although donors are currently screened for HBsAg by radioimmunoassay both in Greece and in the UK. Our patients could have acquired hepatitis B infection either when less sensitive screening was used or from donors positive for anti-HBc, possibly associated with infectiousness,⁴ which is not routinely assessed in blood donors.

Hepatocellular injury could result from iron overload since, in spite of chelation treatment, only three patients reached normal ferritin concentrations. Our results show, however, that abnormal AST activities are associated with evidence of hepatitis B infection rather than with evidence of iron overload as reflected by raised ferritin concentrations. Thus hepatitis B virus probably contributes to chronic liver damage in thalassaemic patients. This should be confirmed by studying prospectively markers of hepatitis B virus and biochemical and histological variables. Furthermore, other bloodborne viral infections (cytomegalovirus, Epstein-Barr, and non-A non-B virus) should be investigated. The high prevalence of hepatitis B infection and its possible association with chronic liver disease, however, might be an argument for active immunisation of thalassaemic patients at diagnosis.

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Viral markers, aspartate transaminase, and ferritin blood values in thalassaemic patients treated in London and Athens

	No of patients	Sex men:women	HBsAg	Anti-HBs	Anti-HBc	Anti-HBs, anti-HBc	Anti-HAV	1978-9		1982-3	
								AST* range (mean)	Ferritin† range (mean)	AST* range (mean)	Ferritin† range (mean)
London	29	18:11	1	2	3	7	0	17-120 (42.1)	1000-17 700 (7015)	17-144 (45.8)	400-9000 (2736)
Athens	29	18:11	0	3	2	23	12	17-207 (74.4)	2500-20 000 (10 079)	21-157 (68.2)	75-5000 (2176)

*Normal range: 7-40 IU/l. †Normal range: women 14-178 μ g/l, men 39-340 μ g/l.