In July 1982 he developed fever, cough productive of blood stained mucopurulent sputum, malaise, and weight loss. A chest x ray film showed an abscess in the lingula and associated pulmonary infiltrates. The results of microbiological examinations of the sputum were persistently negative. No improvement was seen despite repeated courses of antibiotics. Bronchoscopy showed no obstruction but friable submucosal plaques. Pneumocytosis was diagnosed histologically and the lung lesion resolved with high dose cotrimoxazole.

In January 1983 he was admitted; he was febrile with encephalopathy and had a generalised rash three weeks after an episode of shingles in the thoracic 5-6 dermatomes. Cerebrospinal fluid protein was 2.29 g/l and the fluid contained 294 white cells ($0.294 \times 10^9/l$), of which 90% were lymphocytes. No bacteria or viruses were isolated. This disseminated zoster infection responded to parenteral acyclovir, but recovery was complicated by a recurrence of oesophageal candidiasis. In October 1983 he developed two painful indurated lumps on the chest wall. A biopsy specimen showed a squamous carcinoma, from which he died in November 1983. The primary site was not identified.

A persistent increase in suppressor and decrease in helper peripheral blood T cell numbers was found with the commercially available monoclonal antibodies OK T 3, OK T 4, and OK T 8 (Ortho Diagnostics, Raritan, New Jersey). The total lymphocyte count was $1.72 \times 10^9/l$ (control $1.05-3.05 \times 10^9/l$), of which $1.12 \times 10^9/l$ (control $0.8-1.63 \times 10^9/l$) were OK T 3 positive (T cells), $1.07 \times 10^9/l$ (control $0.26-0.82 \times 10^9/l$) OK T 8 positive (suppressor), and $0.23 \times 10^9/l$ (control $0.54-1.11 \times 10^9/l$) OK T 4 positive (helper). The helper suppressor ratio was 0.2 (normal 1.0-3.0). Total serum IgG, IgA, and IgM concentrations were normal. The patient had negative specific tests for syphilis, a negative time test and was HbsAg negative. Screening for autoantibodies including rheumatoid factor, antinuclear factors, and DNA antibodies gave negative results.

Comment

Our patient suffered from a series of unusual opportunistic infections and malignancy accompanied by the pattern of immune paresis described in the acquired immune deficiency syndrome (AIDS).^{1,2} He did not fall into one of the recognised risk groups and he had no known contact with anyone from such a group. He acquired the immune paresis three years before the first description of AIDS in the USA. Possibly he represents one of the presumably rare and as yet unrecognised individuals from whom the current epidemic in at risk populations arose. Alternatively, his acquired immune deficiency may have resulted from Crohn's disease. His first infection predated clinical evidence of Crohn's by more than a year. Alterations in T lymphocyte populations in the highly specialised gut mucosal immune system occur in Crohn's disease³ and anergy to skin test antigen has been shown. Studies of circulating T lymphocyte populations have yielded conflicting results but a recent study using monoclonal antibody techniques found no change in helper or suppressor T cell populations, regardless of disease activity, site, extent, or mode of treatment.⁴ Finally, transfusion of blood and its products is a possible mode of transmission of acquired immune deficiency syndrome.⁵ He received multiple transfusions only after Crohn's disease presented one year after the first infective episode. We believe that acquired immune deficiency syndrome was the primary event in our patient.

We thank Dr S A Cairns for his advice and encouragement during the preparation of this paper and Dr N Williamson and Dr P Johns and their staff at the subregional immunology laboratories at Preston Infirmary for their expert technical help.

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(Accepted 28 February 1984)

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Do chillies influence healing of duodenal ulcer?

Chillies are generally believed to be harmful to patients with duodenal ulcer. Consequently, these patients are advised to take a relatively bland diet containing little or no spices and chillies. This view is perhaps based on experimental findings that red chilli powder damages the gastric mucosa. Intragastric instillation of red chilli powder in human volunteers caused a considerable increase in the deoxyribonucleic acid content of gastric aspirate, suggesting exfoliation of the surface epithelial cells.¹ This effect has been reproduced with capsaicin, the active principle of chillies.² The results concerning acid production are conflicting: one study showed no effect³ while another found considerably increased acid concentrations in both controls and patients with duodenal ulcer after intragastric infusion of red chilli powder.⁴ The effect of chillies on the healing rate of duodenal ulcer has not been determined, however, and to examine this we performed a controlled trial on patients in hospital.

Patients, methods, and results

Fifty consecutive patients with endoscopically proved duodenal ulcer were studied. None of the patients had been on recent specific antiulcer treatment and none had ulcer related complications such as haemorrhage or perforation. None had had gastric surgery or suffered from any systemic illness. Patients were admitted to the hospital and were randomly allocated to one of the two groups. Group 1 consumed a normal hospital diet (without any chillies) and took liquid antacid (Aludrox MH) containing aluminium hydroxide 61 g/l and magnesium hydroxide 20 g/l (neutralising capacity 2.3 mol/l) in a dose of 15 ml one hour and three hours after each meal, six times a day. Group 2 in addition to the above treatment received 3 g red chilli powder (1 g with each meal) a day. Treatment was continued for four weeks. The patients were assessed clinically each week, and symptoms were recorded on a form. Patients underwent endoscopy after four weeks and the findings were categorised as: (a) healing of duodenal ulcer with or without residual duodenitis and (b) non-healing of ulcer (irrespective of its size). The examiner doing the endoscopy was unaware of the treatment received by the patient.

The results are shown in the table. Weekly comparison of symptomatic relief showed no real difference between the two groups throughout the study period. After four weeks of treatment ulcer healing was identical (80%) in both groups. Patients receiving chillies showed no gastric mucosal abnormalities such as hyperaemia or erosions at endoscopy. Two out of 25 patients in group 2 had mild intolerance to chillies and two from group 1 and four from group 2 developed mild constipation, but none had to stop treatment.

Clinical details and endoscopic healing rate in the two groups of patients with duodenal ulcer

	Group 1	Group 2
No of patients	25	25
Age (yr)		
Mean (SD)	36.8 (11.1)	32.6 (12.8)
Median	35	28
Men: women	24:1	24:1
Duration of symptoms (yr)		
Mean (SD)	4.92 (4.58)	5.28 (4.9)
Median	3	4
Range	1-15	1-15
No of smokers	19	17
No of patients with symptomatic relief in:		
Week 1	15	13
Week 2	18	20
Week 3	20	21
Week 4	23	22
No with endoscopic healing	20	20

Comment

In the present study 3 g of red chilli powder was given daily, which is the usual amount consumed in India.³ Antacid was administered in a dose of 15 ml six times a day since we find this to be the optimal quantity required for healing of duodenal ulcer.⁵ In previous studies red chillies have damaged the gastric mucosa.^{1,2} In those experiments, however, the chilli powder or capsaicin (its active principle) was instilled directly into a fasting stomach by means of a tube.^{1,2} Moreover, the evidence of damage was transitory and was not observed once the infusion was stopped.² In our study red chillies were found not to influence the healing of duodenal ulcer. Moreover, we did not observe any gastric mucosal damage in the form of hyperaemia or erosions. In view of these findings we recommend that patients with duodenal ulcer may consume a normal diet and that bland food is unlikely to serve any useful purpose.

The financial and material help of John Wyeth and Brothers Ltd is gratefully acknowledged.

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(Accepted 29 March 1984)

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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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(Accepted 11 April 1984)

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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.