readily available in most hospitals. It provides a high concentration of oxygen with either spontaneous or controlled ventilation and allows the operator to adopt a reasonably comfortable posture, enabling him to hold the mask and giving freedom of vision.

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(Accepted 27 March 1986)

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Papillitis and hepatitis B

We report a case of acute hepatitis B virus infection that presented as acute bilateral optic disc papillitis.

Case report

A 22 year old homosexual man presented with a three week history of deteriorating vision in his right eye, painful eye movements, swollen neck glands, and lassitude. Over the past four years he had had rubella, infectious mononucleosis, genital herpes simplex, non-specific urethritis, gonorrhoea, and

Results of liver function tests

	25 Jan	28 Jan	22 Feb	20 Apr	26 Apr	16 May	15 June	4 Aug
Anti-HBc	—		+					—
HBsAg	+	+	+	+	+	+	+	—
HBeAg	+	+	+	+	—	—	—	+
Anti-HBe					—	—	+	—
Anti-HBs								
Bilirubin (μmol/l) (normal <19)	7			10	9		6	
Alkaline phosphatase KAUI (normal=20-110)	100			100	90		100	
Alanine transferase (IU/l) (normal <20)	15			95	27		13	

Conversion: SI to traditional units—Bilirubin: 1 μmol/l ≈ 0.06 mg/100 ml.

tonsillitis. He had never had jaundice or hepatitis, but three months earlier his regular partner had acute hepatitis B virus infection.

On examination there was generalised lymphadenopathy with firm, non-tender nodes 1-2 cm in diameter. Ocular examination showed visual acuities corrected with glasses of 6/18 (for distance) N5 (for near vision) on the right and 6/5 N5 on the left, his colour vision, pupillary reactions, and extraocular movements being normal. There was a central scotoma in the right field but the left was full, and there were cells in the anterior vitreous bilaterally. Both fundi showed noticeable disc swelling, dilated capillaries, arterial sheathing, haemorrhage in the nerve fibre layer, retinal pigment epithelial change, and macular star formation.

Investigations showed haemoglobin concentration 1.42 g/l, white cell count $6.6 \times 10^9/l$ with 50% lymphocytes with atypical lymphocytosis, negative results from Paul Bunnell test, negative syphilis serology, the presence of hepatitis B surface antigen and hepatitis B e antigen (table), and the following viral titres: Epstein Barr virus IgG 1/20, IgM 1/8; cytomegalovirus IgG 1/32, IgM negative; and toxoplasmosis 1/16. These titres remained unchanged at three months. The table gives the results of liver function tests. A computed tomogram of the brain was normal and a cervical node biopsy showed reactive hyperplasia.

The patient remained well without jaundice, and his lassitude, lymphadenopathy, visual function, and serological results improved steadily (table). When examined six months after presentation he had minimal lymphadenopathy and ocular examination was normal.

Stored serum samples were subsequently examined for antibodies to the human T lymphotropic virus type III/lymphadenopathy associated virus (HTLV-III/LAV). A serum sample taken in January 1983 was negative, but a sample taken in December 1983 was positive. He admitted to having had several new sexual partners between August and December 1983.

Comment

Viral disease may be associated with papillitis with or without evidence of intraocular inflammation.¹ Papillitis has been reported in association with rubella,² herpes zoster,³ chicken pox, mumps, influenza, poliomyelitis, coxsackie virus,¹ and infectious mononucleosis.⁴ One patient reported by Walsh and Hoyt exhibited bilateral optic neuritis after hepatitis B virus infection but suffered persistent disturbance of his colour vision, and the role of hepatitis in the development of his optic neuritis was uncertain.⁵

Our patient had unequivocal clinical and serological evidence of acute hepatitis B infection with an identical time course to the development of papillitis. there were no clinical features of demyelination or raised intracranial pressure.

Lymphadenopathy is common in the early stages of acute hepatitis B infection. As our patient was homosexual it was thought, initially, that his lymphadenopathy might have been unrelated to hepatitis B infection and represented a concurrent persistent lymphadenopathy syndrome secondary to HTLV-III/LAV infection. The subsequent finding of antibodies to HTLV-III/LAV could mean that the patient was immunocompromised at the time of his acute hepatitis B infection and perhaps staged an exaggerated lymph node response because of this. He was, however, negative for HTLV-III/LAV antibodies at presentation, and the lymphadenopathy, ocular features, and immunological abnormalities all cleared after resolution of his acute hepatitis B infection. Although he may have had seronegative HTLV-III/LAV infection, it seems more likely that he acquired HTLV-III/LAV infection when he became sexually active again.

This patient had acute papillitis and lymphadenopathy coincident with serological evidence of acute hepatitis B virus infection. This suggests that hepatitis B virus may be associated with the development of papillitis.

We thank Professor Dame Sheila Sherlock, Dr D Geraint James, and Dr E Graham for permission to report this patient under their care and for their invaluable help in the preparation of this paper.

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(Accepted 1 April 1986)

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