restriction of adduction remained but was stable. There was no proptosis with the exophthalmometer and sensation over the right ophthalmic division of the fifth cranial nerve had returned to normal, although a minor reduction in sensation of the maxillary division persisted. Fields and optic discs remained normal.

Comment

The association of intracranial cartilaginous tumour and Ollier's disease is rare and there are only a handful of similar cases published. Chondromas and chondrosarcomas usually arise from the skull base and are located in the middle cranial fossa. Sarwar[2] reported a patient with Ollier's disease presenting with diplopia and impaired vision due to chondroma arising from the sphenoid. Horizontal diplopia was also the presenting symptom in the cases reported by Traflet *et al*[3] and Reuter and Weber.[4] The patient in the latter report also suffered severe ipsilateral headache associated with nausea and vomiting and paraesthesias in the distribution of the trigeminal nerve. These features are obviously comparable to the case we report. Pospiech and coauthors[5] reported a sellar chondroma associated with Ollier's disease presenting as a chiasmatic syndrome. This patient had head pain, vomiting, and visual failure.

The main differential diagnosis in the reported case was meningioma although the relatively avascular tumour appearance on carotid angiography was against this. When the tumour contains calcification craniopharyngioma must be considered and when positioned more posteriorly (clivus/cerebellopontine angle) radiological differentiation from chordoma may be difficult.[2]

Our patient was treated by complete surgical tumour removal with no radiological evidence of recurrence to date. Surgery is the mainstay of treatment in these tumours which are not considered chemosensitive or radiosensitive.[6]

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1 Ollier L. De la dyschondroplasie. *Bulletin Societe de Chirurgiens de Lyon* 1900; **93**: 23–4.
2 Sarwar M, Swischuk LE, Schechter MM. Intracranial chondromas. *Am J Roentgenol* 1976; **127**: 973–7.
3 Traflet RF, Babaria AR, Barolat G, Doan HT, Gonzalez G, Mishkin MM. Intracranial chondroma in a patient with Ollier's disease. *J Neurosurg* 1989; **70**: 274–6.
4 Reuter K, Weber AL. Parasellar chondrosarcoma in a patient with Ollier's disease. *Neuroradiology* 1981; **22**: 151–4.
5 Pospiech J, Mehdorn HM, Reinhardt V, Grote W. Sellar chondroma in a case of Ollier's disease. *Neurochirurgica* 1989; **32**: 30–5.
6 Souhami R, Tobias J. *Cancer and its management*. Oxford: Blackwell, 1986: 395–6.

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A rare cause of visual loss in AIDS patients: central retinal vein occlusion

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Loss of vision in AIDS patients is most commonly associated with cytomegalovirus (CMV) retinitis.[1] Central retinal vein occlusion (CRVO) is an uncommon condition which has been reported once previously to cause visual loss in a patient with AIDS.[2] In this report we describe an additional case of CRVO in an AIDS patient who presented with acute loss of vision temporally related to treatment of anaemia with recombinant human erythropoietin (r-HuEPO).

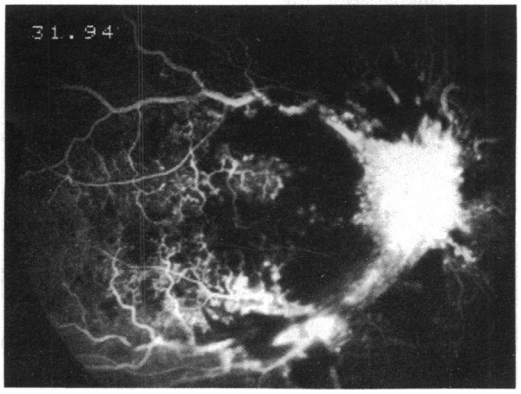

Figure 1 Fluorescein angiography of the right eye showing extensive retinal haemorrhages, blurred disc margins, and dilated veins.

Case report

A 31-year-old woman first tested positive for HIV infection in 1986. She acquired the infection during a 9 year period of intravenous drug use. Her illness had been complicated by multiple hospital admissions for *Pneumocystis carinii* pneumonia, *Staphylococcus aureus* pneumonia, and septicaemia. At the time of presentation her medications included zidovudine, fluconazole, acyclovir, dapsone, imipramine, clonazepam, and r-HuEPO. r-HuEPO had been started 2 weeks before CRVO for anaemia associated with zidovudine treatment.

At presentation she complained of sudden, painless loss of vision in the right eye. The visual examination showed significant diminution of

vision in the right eye to only hand movement at 3 feet, but the corrected vision in the left eye was 20/30. There was no anterior segment neovascularisation and the anterior chamber angle was grade IV open. The intraocular pressures were normal in both eyes. Funduscopic examination revealed typical finding of ischaemic CRVO, which was supported by fluorescein angiography as shown in Figure 1. Laboratory investigations at the time of the diagnosis showed haemoglobin of 7·9 g/l, haematocrit of 26·4%, WBC of 800×10^6/l, and platelets of 30×10^9/l. Extensive coagulation investigation including antithrombin III, heparin-cofactor II and protein S, protein C, antiphospholipid antibodies, and circulating immune complexes (CIC), did not show any abnormalities.

The patient was followed without treatment for 1 year, her vision remained unchanged in the left eye, but showed some improvement in the right eye; she was able to count fingers at 1 foot.

Comment

This is the second case in the literature of CRVO causing visual loss in a patient with AIDS. CMV retinitis remains the most common cause of loss of vision in AIDS patients.[1] The prognosis and the treatment of CRVO and CMV retinitis are quite different.[1 3] The diagnosis in our patient was suspected on funduscopic examination and supported by fluorescein angiography (Fig 1). CRVO occurred in this patient in the absence of underlying cardiovascular disease or any significant coagulation defects that can lead to thrombosis.[2 4] As in the previously reported case, our patient developed CRVO in conjunction with the treatment of the anaemia. In the present case, the anaemia was treated with r-HuEPO. Thrombosis of arteriovenous fistulas and native veins has been reported in patients in chronic haemodialysis during the course of treatment with r-HuEPO.[5] The haematocrit did not rise significantly in this case (24% to 26·4%), however, other rheological effect of r-HuEPO such as increased platelet aggregation could still contribute to CRVO in AIDS patients who are likely to have an abnormal retinal microcirculation.[1 3 5]

Finally, CVRO in this case, as well as in the case presented by Tiech *et al*,[2] occurred in patients known to have AIDS. It remains unclear as to whether or not the CRVO may precede significant AIDS related illness.

We thank Jan Isaacs for her secretarial skills.

1 Palestine AG, Polis MA, Daomet MD, Baird BF, Falloon J, Kovacs JA, *et al.* A randomized, controlled trial of foscarnet in the treatment of cytomegalovirus retinitis in patients with AIDS. *Ann Intern Med* 1991; **115**: 665–73.
2 Tiech SA, Sonnabend J. Central retinal vein occlusion in a patient with AIDS. *Arch Ophthalmol* 1988; **106**: 1508–9.
3 Kohner EM, Laatikaineu L, Oughton J. The management of central retinal vein occlusion (CRVO). *Ophthalmology* 1983; **90**: 484–7.
4 Cansos RT, Zon LI, Groopman JE. Anticardiolipin antibodies associated with HTLV-III infection. *Br J Haematol* 1987; **65**: 495–8.
5 Castasi S, Passerini P, Campise MR, Graziani G, Cesana B, Perisic M, *et al.* Benefits and risks of protracted treatment with human recombinant erythropoietin in patients having hemodialysis. *BMJ* 1987; **295**: 1017–20.

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Persistent visual changes following hashish consumption

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Lasting changes in vision following drug misuse were recently described by Levi and Miller.[1] In one of the reported patients, visual alterations occurred as a result of marijuana misuse only. We observed a subject who presented visual changes which started the day following discontinuation of a 5 year long period of continuous hashish consumption, and which had lasted for months. Visual symptoms were strikingly increased on physical effort or when reading from a brightly lit paper.

Our observation corroborates the recent suggestion by Levi and Miller,[1] that lasting, harmful effects of cannabinoid consumption on visual function may have been previously underestimated.

Case report

A 23-year-old man complained of visual changes lasting for 8 months. Although mentally rather well balanced, when aged 18 he started smoking hashish for social reasons, and went on smoking several times a day, uninterrupted for 5 years. He denied having used any other drug. On the day following discontinuation of drug misuse, he noted the occurrence of changes in visual function, consisting mainly in perception of small black and white spots flickering randomly at high frequency. The phenomenon was described as similar to interference on a television screen. These visual phenomena were markedly increased by either starting physical effort or reading from a brightly lit white paper, making everyday life difficult. The patient also described a reduction in capacity for mental concentration, and in depth perception, and the occurrence of visual perseveration after looking at bright objects. In addition, he occasionally noted a feeling of disconnection between himself and