



Figure 2 Cervical computed tomography angiography: 3D reconstruction showing intimate contact between the styloid process and the left internal carotid artery.

In cases where there is concomitant neurological focus, it is advisable to perform a Doppler or dynamic angiography, which shows the carotid deterioration induced by head movements. In our case, the ultrasound study made an early diagnosis of carotid dissection possible.

An elongated process can be treated by surgical excision, preferably through an extraoral approach¹⁰. As in spontaneous or non-traumatic carotid dissections, treatment should be anticoagulation unless it is contraindicated.

Our patient is an example that ES may occur as a complication of acute carotid dissection. When faced with cervicofacial pain related to a neurological focus, it is important to consider carotid deterioration due to an elongated styloid process.

Presentations

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L.M. Cano*, P. Cardona and F. Rubio

Servicio de Neurología, Hospital de Bellvitge, L'Hospitalet de Llobregat, Barcelona, Spain

*Corresponding author

E-mail: lcano@bellvitgehospital.cat (L.M. Cano).

Bilateral optic neuropathy in an HIV patient

Neuropatía óptica bilateral en un paciente con infección por el VIH

Dear Editor:

The differential diagnosis of vision loss in a patient infected with human immunodeficiency virus (HIV) is extensive; it covers a variety of processes affecting from the cornea to the visual cortex. Both antiretroviral medication and that used to treat opportunistic infections have increased survival of patients with HIV, but they are significantly toxic¹. Most cases of optic neuritis in patients with HIV

infection are usually caused by opportunistic infections, such as cryptococcosis, histoplasmosis, herpes zoster, cytomegalovirus and syphilis². Some cases of primary central nervous system lymphoma have also been described³.

Case report: 34-year-old male diagnosed 5 years earlier with HIV infection, currently with an acceptable immune status (369 CD4+ and HIV RNA viral load of 690 copies/ml). In treatment with antiretroviral therapy for 4 years; in the last 18 months, the procedure followed was zidovudine 300 mg/12 h, lamivudine 150 mg/12 h, lopinavir 400 mg/12 h and ritonavir 100 mg/12 h. He had presented pneumonia from *Pneumocystis carinii* at the time of diagnosis, but since then had not suffered any other opportunistic infection and did not follow preventive treatment. He had used multiple toxic substances: cocaine, heroin, cannabis, tobacco and benzodiazepines, which he had stopped 3 years

earlier. He had no other personal or family history of vision loss.

In the course of 2 weeks, the patient presented a painless, progressive bilateral vision loss that led to a significant decrease in visual acuity. Ophthalmologic examination showed very poor visual acuity, since the right eye saw only movement of the fingers and the left eye counted fingers at 50 cm. He presented an afferent pupillary defect in the right eye. Intrinsic and extrinsic eye movements were normal. He presented a normal intraocular pressure and the fundus was normal, with no evidence of retinal disease, although the evolution revealed bilateral optic atrophy. Apart from the visual deficit, the neurological examination found no data on focal neurological signs. The temporal arteries were normal.

Analyses: systemic blood analysis, coagulation study, sedimentation rate, normal vitamin B12 and folic acid. Biochemistry: glucose, kidney function, ions, hepatic enzymes, thyroid hormones, proteinogram, antinuclear antibodies, angiotensin converting enzyme, tumour markers, vitamin B1 and B6 without significant alterations. Hypercoagulability study with homocysteine fell within normal limits.

Lumbar puncture: opening pressure, 22 cmH₂O; glycorrachia, 57 mg/dl (blood glucose, 79); proteinorrachia, 23 mg/dl, 1 leukocyte/μl. Absence of malignant cells; aerobic, anaerobic and Lowenstein cultures were negative. Serology for *Brucella*, syphilis, Lyme disease and cryptococcal antigen were negative. PCR for herpes virus was negative.

Genetic study of Leber hereditary optic neuropathy mutations was negative. Brain and optic nerve MRI were normal. Visual evoked potentials: reduction in the amplitude and lengthening of the latencies in both eyes consistent with severe bilateral optic neuropathy. Perimetry: absolute scotoma in the central 30° of both eyes.

The patient was treated with prednisone at doses of 1 mg/kg/day in decreasing doses for 3 weeks and acetazolamide at doses of 250 mg/day for 12 months; there was no improvement of the visual deficit, which remained stable over the 2 years of follow-up.

Leber hereditary optic neuropathy is a maternally inherited mitochondrial disorder characterised by painless, consecutive vision loss in both eyes. Antiretroviral therapy (mainly with zidovudine) may occasionally cause mitochondrial toxicity and trigger the clinical manifestations⁴. It has been suggested that the effect of zidovudine on mitochondrial function would depend more on treatment time than on the dose used⁵. However, in our patient the genetic study of this neuropathy was negative.

It has been suggested that HIV infection itself may produce damage to the optic nerve in these patients. There are cases described in which the beginning of antiretroviral therapy was associated with the recovery of optic neuritis from the HIV⁶. There have been reports of optic neuritis with a relapsing-remitting course similar to that shown in multiple sclerosis in which treatment with corticosteroids appears to be effective⁷. In cases where neither treatment produces an improvement in vision, as in the case of our patient, the clinical case and the developmental pattern are consistent with a mechanism of microvascular ischemia on the optic nerve head⁸.

Opportunistic infections were reasonably excluded in our patient by cerebrospinal fluid analysis. Toxic substance abuse may have played an important role in the pathophysiology of optic nerve damage, as others authors have suggested⁹. Mild intracranial hypertension in the absence of orthostatic headache and the evolution of the condition do not indicate that this cause has an essential contribution to the visual impairment.

The cause of optic neuropathy in our patient seems to be the HIV infection itself. However, both antiretroviral treatment and the history of toxic substance abuse, and mild intracranial hypertension to a much lesser extent, might have contributed in some way. In the differential diagnosis of optic neuropathy, we must not forget HIV infection¹⁰, both as a form of presentation and in cases already diagnosed.

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P.E. Jiménez Caballero^{a,*} and M. Serviá Candela^b

^a *Servicio de Neurología, Hospital Virgen de la Salud, Toledo, Spain*

^b *Análisis Clínicos, Hospital Virgen de la Salud, Toledo, Spain*

*Corresponding author.

E-mail: pjimenez1010j@yahoo.es (P.E. Jiménez Caballero).