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Sir,

Giant cell arteritis—part of a spectrum of autoimmune disease?

Primary biliary cirrhosis and hypothyroidism are autoimmune diseases with a female preponderance. Giant cell arteritis (GCA) is a vasculitis, possibly of autoimmune aetiology. It is a rare cause of third nerve palsy. We report a case of biopsy-proven GCA causing painful third nerve palsy in a patient with biopsy-proven primary biliary cirrhosis and hypothyroidism. We discuss GCA as part of a spectrum of autoimmune disease.

Case report

A 68-year-old lady, with hypothyroidism and primary biliary cirrhosis (PBC) (Figure 2a), presented to eye emergency clinic with painful third nerve palsy (Figure 1) without pupillary involvement. The ESR was elevated at 68 mm. The diagnosis was presumed to be due to temporal arteritis, and high-dose oral steroid was started. A temporal artery biopsy (Figure 2b) performed next day was positive for GCA. Headaches improved steadily on treatment. ESR (Erythrocyte sedimentation rate) dropped significantly to normal levels 1 week after starting high-dose steroid. Third nerve palsy fully recovered by the fifth month.

Comment

GCA is the most common form of systemic vasculitis in adults,[1] affecting medium and large-sized arteries.

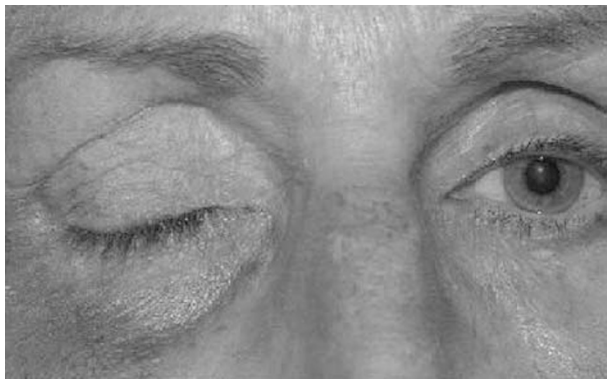

Figure 1 Total ptosis of right eye due to third nerve palsy.

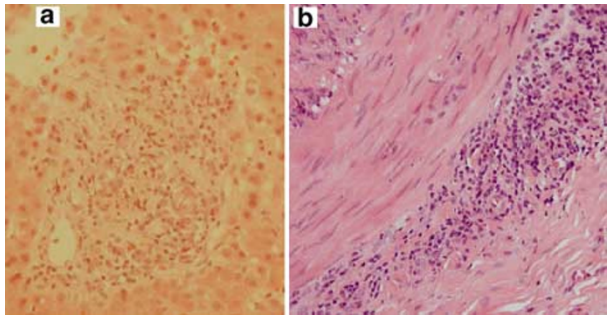

Figure 2 (a) High-power photomicrograph of a granuloma within a portal tract. (b) Medium-power photomicrograph of temporal arterial wall showing intimal proliferation, inflammation, and giant cells.

Immunological processes have been implicated in the development of GCA. Deposits of immune complexes and complement have been found in some temporal artery biopsies.[2] Anti-IgG activity has also been identified in artery biopsy specimens.[3] Recently, a model for the pathogenesis of GCA proposed by Weymann and Goronzy[4] suggests a cell-mediated aetiology. Approximately 30% of patients with GCA have neurologic manifestations.[5]

PBC is an autoimmune disease, leading to progressive destruction of small intrahepatic bile ducts. A survey among a cohort of patients with PBC showed that it is associated with an increased risk of other autoimmune disorders.[6] A survey of thyroid function in patients with PBC revealed the presence of thyroid antibodies in 26% of patients.[7] A nationwide survey in Japan found autoimmune thyroiditis to be associated with primary biliary cirrhosis in 5.8% of cases.[8] Gordon and Isenberg suggest that there is an overlap between polmyalgia rheumatica (PMR) and GCA with autoimmune thyroid dysfunction.[9] Dent and Edwards,[10] in their series of 250 patients with autoimmune thyroiditis, noted PMR or GCA in 2.8% of patients. Gagnerie *et al*[11] report PBC, GCA, and PMR in a single patient. The common

histopathological feature of PBC and GCA is granulomatous inflammation. Thus, even though occurrence of GCA along with PBC and hypothyroidism is rare, awareness of this association may aid the management of this potentially blinding condition in elderly patients.

Competing interests: None

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Sir,

Didanosine-induced retinopathy in adults can be reversible

Didanosine (DDI), a purine analogue used in the treatment of human immunodeficiency virus (HIV) disease, has been associated with pancreatitis, peripheral neuropathy, and retinopathy,[1,2] the latter being attributed to irreversible retinal pigment epithelium (RPE) loss accompanied by partial loss of the choriocapillaris and neurosensory retina in the mid-periphery.[3]

We present a case of a HIV patient with a clinical and electrophysiological diagnosis of DDI retinopathy (DDIR), whose electrophysiological abnormalities improved following DDI withdrawal.

Case report

A 53-year-old HIV positive Caucasian male presented with difficulty with his peripheral vision under scotopic conditions. His medical history included HIV disease, shingles, oral hairy leukoplakia, seborrheic dermatitis, and non-insulin-dependent diabetes with peripheral neuropathy but no diabetic retinopathy. At presentation, he had been HIV positive for 12 years with a $CD4^+$ T-cell count of 224 cells/μl and an undetectable viral load. He was on highly active antiretroviral treatment (HAART) with Didanosine, Tenofovir, Ritonavir, and Saquinavir, the Didanosine (400 mg od) for 5 years. Other medication included Gabapentin, Domperidone, Aciclovir, and Co-trimoxazole.

On examination visual acuity was 6/6 in each eye, with normal pupillary reactions. Anterior segment examination was unremarkable with normal intraocular pressures.

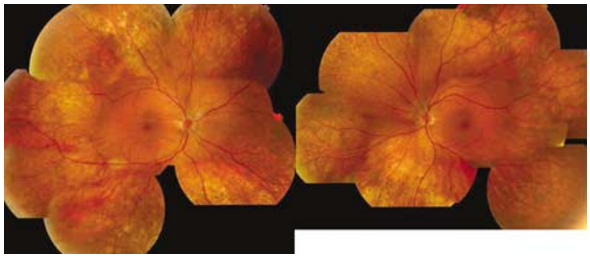

Figure 1 Fundus photographs of the right and left eye show extensive midperipheral atrophy at the level of the RPE in keeping with DDI retinopathy.