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Myasthenia gravis: an unusual cause of diplopia in polymyalgia rheumatica

Sirs,

Polymyalgia rheumatica (PMR) is a clinical syndrome in elderly patients characterised by aching in the proximal portions of the extremities and torso, morning stiffness and evidence of systemic inflammation, such as an elevated erythrocyte sedimentation rate (ESR) or C-reactive protein. PMR typically responds rapidly to low-dose prednisone. Giant cell arteritis (GCA) complicates the course of PMR in a varying proportion of patients (6% to 30%) (1). Ocular lesions in GCA include ischemic optic neuropathy (the most common ocular manifestation), retinal ischemic lesions and ophthalmoplegia, with or without diplopia which has been reported in approximately 12% of cases (2). We report here a case of a woman with PMR who developed diplopia as a consequence of myasthenia gravis (MG).

A 69-year-old woman previously diagnosed with knee osteoarthritis and monostotic Paget's disease of bone in the left tibia which started in June 2000 with aching and pain in the muscles of the neck, shoulders, lower back and hips, anorexia, malaise, a raised ESR (89 mm at the first hour), and C-reactive protein of 3.06 mg/dl, without headache, jaw claudication, or visual manifestations. Prednisone 15 mg daily was started with a favourable response and a progressive tapering of the dose was initiated.

She remained asymptomatic until February 2003, when she complained of diplopia. The patient was referred by her general practitioner to an ophthalmologist. After a complete evaluation that disclosed a normal fundus without signs of isquemic optic neuropathy, a cranial and orbital computer tomography scan and a neurophysiologic study were ordered. CTwas normal but neurophysiologic study revealed an increased fatigability of the extraocular muscles with evidence of disease activity on routine muscle sampling (final mean of consecutive differences 56.1 ms). Antibodies against acetylcholine receptors were positive at a concentration of 7.40 nmol/L (normal range < 0.40 nmol/L). A biopsy of temporal artery was discarded when MG was diagnosed based on the neurophysiologic study and the presence of antibody anti-acetylcholine receptor. The patient was initially treated with oral prednisone 60 mg daily, with significant symptomatic improvement. After the diagnosis of MG, piridostigmine was started and the dose of glucocorticosteroid rapidly tapered. The patient remains asymptomatic with piridostigmine bromide 60 mg tid and oral prednisone at a dose of 7.5 mg qd.

A close relationship exits between GCAand PMR. Both conditions affect the same population of patients and frequently occur in the same individual. Due to the predominant involvement of branches of the carotid artery, visual symptoms and signs are common in GCA. Although the most common ocular symptom in GCA is visual loss, diplopia may be the first manifestation of GCA complicating PMR (3-6). In GCA, diplopia usually results from an ocular motor nerve paresis.

MG is a relevant cause of recent-onset diplopia in the general population (7). It is a condition caused by autoantibodies against the human nicotinic acetylcholine receptor, which results in impaired neuromuscular transmission and muscle weakness, either developing or becoming more evident with exertion. Although in cases of inappropriate control of the symptoms, immunosuppressive treatment (high-dose prednisolone) is indicated, the first-line treatment of MG consist of oral anticholinesterase agents (8, 9). Therefore differentiating the two conditions GCA and MG is critical to avoid unnecessary therapy with glucocorticosteroids. The present case emphasises that, although the first possibility in a patient with PMR who develops diplopia is GCA, other causes of ocular symptoms must be reasonably ruled out (Table I).

Table I. Causes of diplopia.

- Monocular diplopia
  - Optical problem
    - Refractive error
    - Keratoconus
    - Cataract
  - Psychogenic or functional
- Binocular diplopia
  - Myoneural junction
    - Myasthenia gravis
  - Restriction of extraocular muscles
    - Inflammation (orbital miositis)
    - Infiltration (thyroid ophthalmopathy or metastatic disease)
    - Entrapment (blow-out fracture of the orbital floor)
  - Disorders of the ocular motor nerves
    - The oculomotor nuclear complex (third nerve)
      - Aneurysms
      - Vascular disease (hypertension, atherosclerosis, diabetes mellitus, temporal arteritis)
      - Trauma and tumor (pituitary tumor, cavernous sinus meningioma, metastasis)
      - Parasellar mass lesions
      - Aberrant regeneration
    - Trochlear nerve (fourth cranial nerve)
      - Trauma
      - Vascular diseases (hypertension, atherosclerosis, diabetes mellitus).
      - Aneurysms (cavernous internal carotid artery or basilar artery).
      - Hydrocephalus
      - Sequelae to intracranial surgery
    - Abducens nerve (sixth nerve)
      - Infarction
      - Aneurysms
      - Tumor
      - Trauma
      - Leptomeningitis
      - Multiple sclerosis
    - Multiple ocular motor nerve palsies
      - Nasopharyngeal carcinoma
      - Granulomatosous inflammatory processes ("Tolosa Hunt" syndrome)
      - Lymphoma
      - Pituitary tumors
      - Meningiomas
      - Chordomas
    - Internuclear ophthalmoplegia
      - Brainstem glioma (in children)
      - Multiple sclerosis (in adults)

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