Bilateral optic neuritis in ankylosing spondylitis

Sir,

Ocular involvement in ankylosing spondylitis is common, particularly in uveitis. It occurs in 25-30% of patients at some point during the course of their disease (1). However, involvement of the retina or optic nerve is very rare. We describe here a patient with ankylosing spondylitis and bilateral optic neuritis.

In April 1992, a 42-year-old woman was seen at the Rheumatology Clinic of Seoul National University Hospital because of low back pain. In 1981, she developed pain in the lower back and buttocks, which improved with the intermittent administration of over-the-counter medications. In early 1992, she visited a hospital due to posterior neck pain which was diagnosed as ankylosing spondylitis. In April 1992 she was referred to our hospital with pain and stiffness in the neck, dle and lower back, and right hip. A complete blood cell count showed white blood cells 10,900/mm\(^3\) (segmented neutrophils 71%, lymphocytes 24%, monocytes 2%, eosinophils 2%, and basophils 1%), hemoglobin 11.9 g/dl, and platelets 230,000/mm\(^3\). The Westergren erythrocyte sedimentation rate (ESR) was 65 mm/hr. Rheumatoid factor was negative. Antinuclear antibody was weakly positive at 1:40 dilution. Radiographs of the pelvis and lumbar spine showed bilateral sacroiliitis (Fig. 1) and syndesmophytes. An HLA-B27 test was positive. Fenoprofen 600 mg tid and sulfasalazine 0.5 g bid were prescribed, which led to gradual improvement of the arthralgia. In March 1997, her neck pain worsened with a Westergen ESR of 83 mm/hr. Fenoprofen was replaced by indomethacin. She remained relatively well until July 1997 when she developed a visual disturbance in the left eye. She visited a private ophthalmology clinic, where a visual field examination revealed a defect in inferior altitudinal visual field. This visual deficit improved slowly and spontaneously, but two weeks later she suddenly developed blindness accompanied by severe pain in the right eye, together with frontal headache.

Ophthalmologic examination at our hospital revealed that the visual acuity was finger count in the right eye and 0.8 in the left. There was an afferent pupillary defect in the right eye. The intraocular pressures and slit-lamp examination results were unremarkable in both eyes. The disc was not edematous on ophthalmoscopic examination. The patient’s ESR was 52 mm/hr and her C-reactive protein was 0.6 mg/dl (normal < 0.5). Fluorescein angiography showed a faint filling defect around the disc margin in both eyes. A visual evoked potential (VEP) study showed prolonged latency of deflection. Computed tomography and magnetic resonance imaging of the brain were normal. The patient was admitted to hospital with a diagnosis of optic neuritis and received i.v. methylprednisolone pulse therapy (1 g/day).

Fig. 1. AP view of the pelvis shows bilateral sacroiliitis.

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for 3 days, followed by oral prednisolone 60 mg daily. Prednisolone was tapered over 2 months, and then discontinued. Visual acuity slowly improved and the eye pain disappeared. Seven months after the onset of symptoms, ophthalmologic examination revealed a visual acuity of 0.7 in the right eye and 1.0 in the left. The visual field defect returned to normal, although mild residual visual blurring was still present in the right eye.

Optic neuritis is often idiopathic, but may occur in association with demyelinating disease such as multiple sclerosis or bacterial, viral, mycotic, and parasitic infections. It has been on rare occasions associated with systemic inflammatory conditions such as systemic lupus erythematosus (2), Behçet’s disease (3) and relapsing polychondritis (4-7). Optic neuritis is clinically characterized by acute or subacute visual loss, often associated with retrobulbar pain or pain with eye movement. It usually affects patients at a relatively young age (15 - 45 years). Loss of visual acuity, decreased color vision, and a central scotoma are generally present in the affected eye. An afferent pupillary defect almost invariably occurs in the acute phase, and the VEP usually shows a prolonged latency (3, 8). The clinical course is characterized by a rapid deterioration of vision followed by steady recovery, with most patients gradually recovering their vision over the next several weeks.

In our ankylosing spondylitis patient, ischemic optic neuropathy and optic neuritis were the primary possibilities to be considered in the differential diagnosis. Ischemic optic neuropathy is described as an infarction of the optic nerve head. It usually affects patients 50 to 70 years of age, showing a sudden and painless onset. Visual acuity varies from 1.0 to no light perception, and altitudinal or arcuate visual field defects are typical. Ophthalmologic examination shows a swollen optic disc, often with hemorrhages. There is usually little or no recovery of lost visual function. The patient’s age and the pattern of visual field defect, as well as the lack of recovery, are helpful to distinguish this condition from optic neuritis (3, 8).

Our patient was 47 years old and presented with pain in the right eye and frontal head. She experienced a sudden onset of visual loss and showed a nearly complete recovery over time, both typical characteristics of optic neuritis. Fundus examination revealed no swelling in the disc. Although a faint filling defect at the disc margin was detected on fluorescent angiography, it did not correspond with the visual field defect. Therefore, the visual field defect was not considered to be ascribable to vascular insufficiency. These findings favor the diagnosis of optic neuritis rather than ischemic optic neuropathy. Optic neuritis and multiple sclerosis (MS) are known to be associated (9), and a relationship between ankylosing spondylitis and MS has been reported (10). Therefore, the possibility of MS had to be considered in our patient. Although she did not show any neurological signs indicative of MS until August 1998, the optic neuritis in our patient could have represented the initial presentation of MS. The absence of oral ulcer, genital ulcer, or skin symptoms pertinent to Behçet’s disease, and the limitation of joint symptoms to the axial joints, exclude the possibility of this disease in our patient, however.

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