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Two Cases of Suspected Arteriosclerotic Optic Chiasmal Syndrome

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Optic chiasmal syndrome due to arteriosclerotic vascular changes is rate. To our knowledge, there has been no report of arteriosclerotic chiasmal syndrome in Korea. In our two cases, other causes of chiasmal syndrome were not detected by MRI, four-vessel cerebral angiography, CSF study, and special laboratory examinations including ANA test, RA factor, and LE cell. With conservative treatment, the visual fields of the two patients are showing progressive improvement. We report here two cases of suspected arteriosclerotic optic chiasmal syndrome.

Key Words: *Chiasmal syndrome, Arteriosclerosis*

INTRODUCTION

Optic chiasmal syndrome represents almost bitemporal field defect due to mass lesions in the optic chiasm or perichiasm, but unusally nontumorous optic chiasmal syndrome has been reported. Recently, we have seen two cases of suspected arteriosclerotic optic chiasmal syndrome.

CASE PRESENTATION

Case I

A 47-year-old male had suffered from visual disturbance for 20 days. There was no history of diabetes mellitus, hypertension, or pulmonary tuberculosis as far as patient claimed. The patient was admitted to the Department of Neurology, Hanyang University Hospital, with a suspected diagnosis of sellar or parasellar tumor and possible aneurysm. On admission, the vital signs were within normal limits.

On neurologic examination, his mentation was alert and his visual acuity was 30/60 in the right eye and 20/50 in the left eye.

His light reflex was intact, but visual field defect with bitemporal hemianopsia was noted in the confrontation test. Fundoscopic examination revealed the disc margins were clear with no abnormal change in the retinal vessels.

In Goldmann's perimetry, typical bitemporal hemianopsia was noticed (Fig. 1-a). In laboratory examination, the CBC, ESR, serum-electrolyte, liver function test, and urine analysis were within normal limits, but fasting blood sugar was 200mg%, HgA_1C, 13.9%.

The RA factor and ANA test were non-reactive and LE cells were not seen. In the lipid study, type IV hyperlipidemia was suggested (total lipid: 645mg/dl, cholesterol: 187mg/dl, triglyceride: 191mg/dl, HDL-chol: 35mg/dl), and the endocrine study provided no evidence of pituitary dysfunction.

A lumbar puncture revealed colorless spinal fluid under normal pressure, unelevated protein, nonexistent pleocytosis, and negative findings on special stain. A plain skull X-ray showed no evidence of calcification in the sellar and parasellar regions. An MRI scan (Hitachi, 0.2T) taken at the time of admission revealed no intrasellar mass or any demyelinating changes (Fig. 2). Four-vessel cerebral angiography was also performed and revealed some tortuous findings of the ophthalmic artery and anterior cerebral artery but no evidence of an aneurysm (Fig. 3).

The patient has been on supportive therapy, including diabetes mellitus and hyperlipidemia control, his visual field has been improving slowly but progressively, and superior bitemporal quadrantanopsia was noted by Goldmann's perimetry two months later. At that time, blood sugar was 100mg%, total lipid, 45mg/dl, cholesterol 170mg%, triglyceride 150mg/dl, HDL-chol 41mg/dl. Five months after onset, he is leading a normal life with an almost normal visual field.

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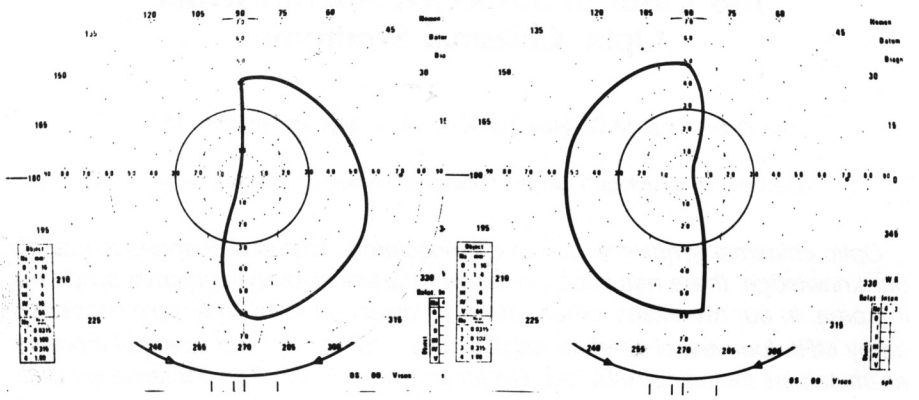

Fig. 1-a) Bitemporal hemianopsia in Goldmann perimetry (Sept. 1989)

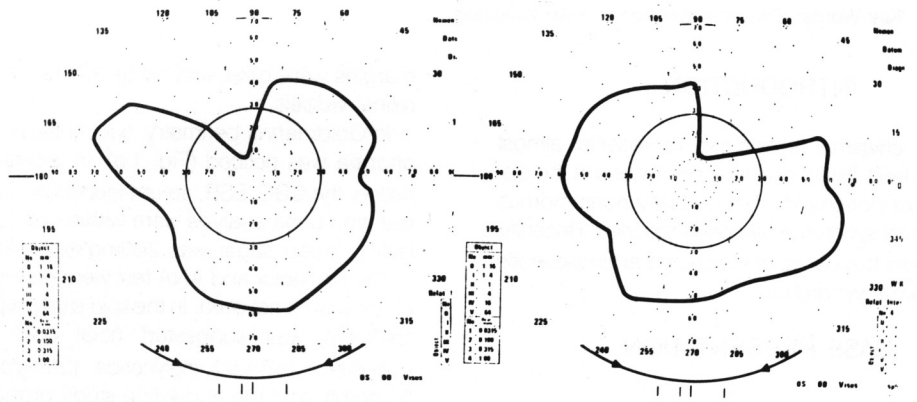

Fig. 1-b) Superior bitemporal quadrantanopsia in Goldmann perimetry (Jan. 1990)

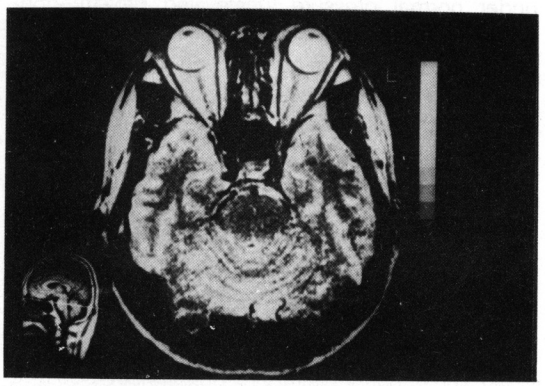

Fig. 2. MRI scan shows no abnormal findings (TR/TE 200/112)

Case II

A 44-year-old male came to the Department of Neurology, Hanyang University Hospital, complaining of narrowing of the visual field for the last 10 days. There was no history of diabetes mellitus, hypertension, or pulmonary tuberculosis on admission as far as the patient claimed. Vital signs were within normal limits. A neurological evaluation revealed bitemporal hemianopsia in confrontation test but was otherwise unremarkable. No color vision abnormalities were noted in either eye. Funduscopic examination was normal. In Goldmann's perimetry, bitemporal hemianopsia was noticed (Fig. 4).

In the laboratory findings, fasting blood sugar was 176mg%, cholesterol 260mg%, HgAIC, 12%, HDL-chol 32mg/dl, and CSF study normal. A hormonal study provided no evidence of pituitary dysfunction. Plain chest and skull radiography were negative.

A computerized tomography scan taken on admission revealed no intrasellar mass. Four-vessel cerebral angiography performed three times on different oc-

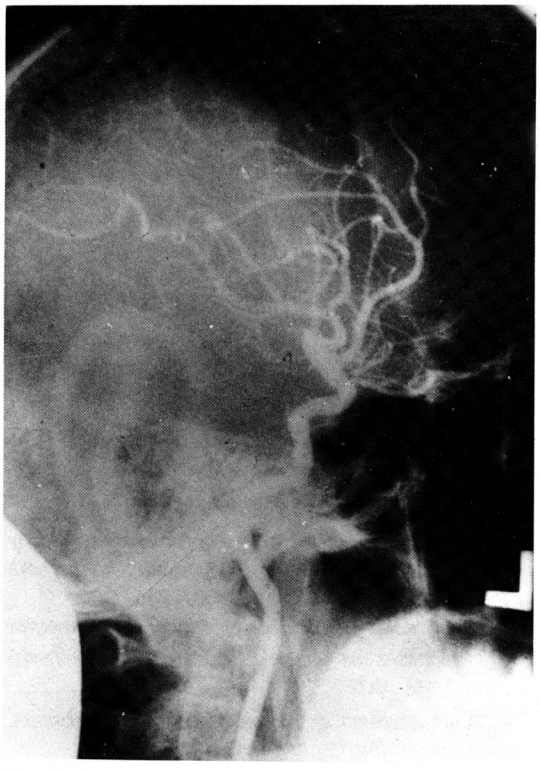

Fig. 3. Four-vessel cerebral angiography shows tortuosities in ophthalmic artery and anterior cerebral artery

casions revealed slight arteriosclerotic changes but no aneurysm was noted. The patient has been on supportive treatment and is improving progressively, enjoying a normal life.

DISCUSSION

The two patients described proved to have no mass lesions around the sellar area including an aneurysm or tumor. Considering their ability to carry on normal activity with improved visual field and their response to supportive treatment, arteriosclerotic chiasmal syndrome is highly suggested.

Since Cushing's chiasmal syndrome, a few reports citing unusual causes of optic chiasmal syndrome have been published, for instance, optic chiasmal arachnoiditis (Bruetch, 1940), multiple sclerosis (Eldermann, 1932), sarcoidosis (Walsh and Smith, 1968), cavernous hemangioma (Manzo Klen, 1979), metastatic carcinoma (Cohen and Lessel, 1979), and intrasellar abscess (Francis, 1971).

In 1930, Cushing described the importance of optic atrophy and bitemporal field defects as indicative of tumors around the sellar. He focused on the mass lesion mainly.

In 1958, Hughes implied that arteriosclerosis in relatively elderly patients can cause a classical chiasmal syndrome.

In 1960, Walsh and Gass called attention to the direct effect of sclerotic anterior cerebral artery on the chiasm, confirmed by a necropsy specimen, in which they found compression and distortion of the chiasm and optic nerve by a sclerotic, elongated, and prolapsed anterior cerebral artery. In 1965, Hilton and Hoyt reported a 68 year old man with slowly progressive chiasmal syndrome caused by arteriosclerotic vascular change and fusiform dilatation of the anterior cerebral artery.

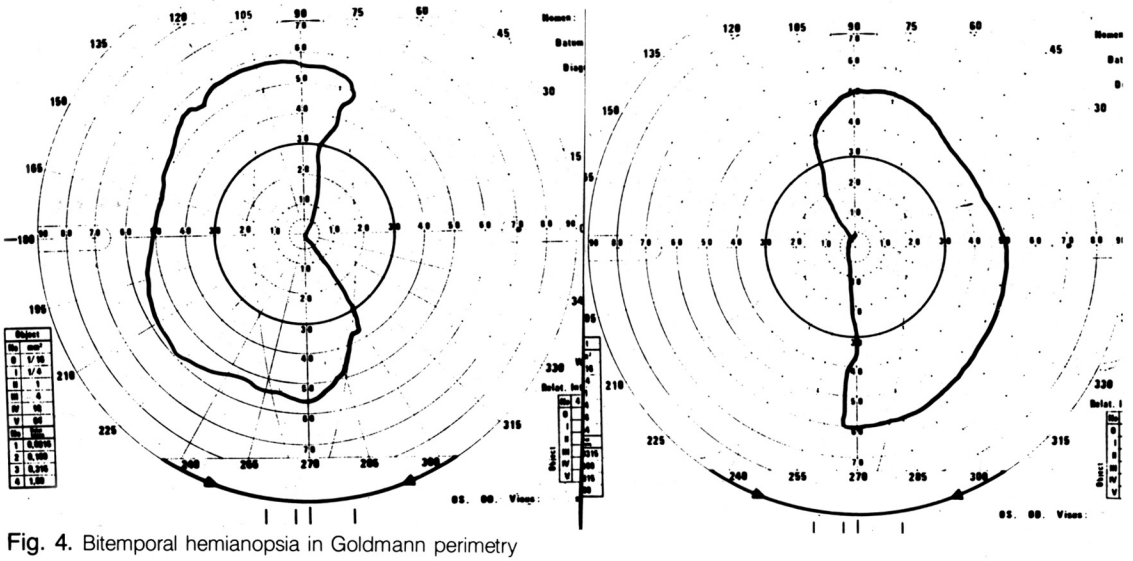

Fig. 4. Bitemporal hemianopsia in Goldmann perimetry

In 1980, Matsuo et al. reported bitemporal hemianopsia associated with sclerosis of the intracranial internal carotid arteries confirmed after intracranial surgical procedure.

The other series of 25 patients with ischemic chiasmal syndrome has been reported (Lee at al., 1975) and divided into five categories:

1) Mechanical compression of the optic chiasmal syndrome by ectatic redundant anterior cerebral or internal carotid arteries
2) Arteriosclerotic optic chiasmal syndrome
3) Optic chiasmal arachnoiditis
4) Various forms of arteritis (especially giant-cell arteritis)
5) Post-partum pituitary necrosis

But they cautioned that a diagnosis of ischemic chiasmal syndrome may be entertained only after a mass lesion is categorically excluded by arteriography and pneumoencephalography.

In our two cases, mass lesion was excluded by MRI and four-vessel cerebral angiography, along with inflammatory chiasmal syndrome by CSF study.

Our first case was similar to optic chiasmal syndrome due to demyelinating disease (especially multiple sclerosis).

A demyelinating cause was difficult to exclude. However, there were no symptoms or signs involving cerebrum, cerebellum, optic nerve, or spinal cord by demyelination. Six cases of optic chiasmal syndrome in multiple sclerosis (Spector et al., 1980) showed a preceding typical fluctuation of clinical symptoms.

In 1969, Bergland described the blood supply of the optic chiasm and divided it into two groups.

First, the superior group was made up of branches from the anterior cerebral artery and, occasionally, the anterior communicating artery.

Second, the inferior group was composed of branches from the internal carotid artery, posterior cerebral artery, posterior communicating artery, and basilar artery. Wollschlaeger et al. (1971) studied the chiasmal blood supply in 630 autopsy specimens, and their findings suggested that there are extensive collaterals supplied to the optic chiasm.

In Case I, the initial field defect represented typical bitemporal hemianopsia. After being treated for diabetes mellitus and hyperlipidemia, the visual field defect improved.

The optic chiasmal syndrome of case I seems to have developed from arteriosclerotic changes of the branches of the inferior group of optic chiasm, considering improvement of the visual field defect (Fig. 1-b) and response to supportive therapy. There have been no reports in Korea to our knowledge of this syndrome, and attention and study to arteriosclerotic optic chiasmal syndrome has not been urged even though there are a few possible cases noted in neurology and the neurosurgical field.

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