INTRODUCTION

The oculomotor nerve bifurcates into the superior and inferior divisions when it reaches the superior orbital fissure. The former supplies the rectus superioris and the levator palpebrae superioris muscles. The latter innervates the medial and inferior rectus muscles, the inferior oblique muscle, the sphincter of the pupil and ciliary body (1). Isolated paralysis of the inferior division of the oculomotor nerve is rarely reported (2-13). Etiologies of this clinical manifestation have been reported to be head trauma (2-4,11), mesencephalic vascular malformation (5), ependymal cyst (6), ischemia following the clipping of a basilar artery aneurysm (7), arteriovenous fistula (9), intraorbital dural arteriovenous malformation or varix (10,12), ophthalmoplegic migraine (11), presumed vasculitis or demyelinating disease (8,11), inflammatory (3,10) and even undetermined disorder (3,11). Now we report a case of diabetic inferior division palsy of the oculomotor nerve, which resolved gradually 2 months after the event.

CASE REPORT

A 26-year-old woman was admitted for an acute onset of the right periocular pain with horizontal binocular diplopia. There was no history of vertigo, nausea, vomiting, facial numbness, tinnitus, dysarthria, dysphagia, choking or hiccups. She had been diagnosed as having type 1 diabetes mellitus (DM) and hypertension at age 24. At the time of admission, she received laser therapy treatment for the PDR of the right eye 6 months later. After the procedure, the visual

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The acuity of the right and left eye was 20/100 and 20/20 with eyeglasses, respectively. Following that, she reported not regularly taking her medications for the control of her diabetes and hypertension during the follow-up period. Then, because of an acute onset of dizziness, right periorcular pain and binocular double vision she visited our emergency room for help. At that point, she reported having had no history of migraine, cranio-facial trauma, systemic vasculopathy or any recent infection.

On admission, her blood pressure was 180/100 mmHg. A check-up of her visual acuity demonstrated a best-corrected acuity of 20/200 OD and 20/25 OS. Ophthalmoscopic examination showed findings compatible with PDR. The pupils were isocoric and reacted properly to both direct and indirect light stimuli. Mild extropia and hypertropia of the right eye were noted in natural position. The right eye movement showed poor adduction with a mild limitation of depression (Figure) while the left eye showed full motility in all directions. There was no blepharoptosis (Figure). Examination of the other cranial nerves was normal. Tendon reflexes of the four limbs showed generalized hyporeflexia. No evidence of abnormal pyramidal, extrapyramidal or cerebellar signs was found. Our clinical impression was isolated inferior division paresis of the right oculomotor nerve.

A complete blood cell count, liver and renal function, electrolytes, uric acid assay and thyroid function were all within normal limits. The erythrocyte sedimentation rate (ESR) was 14 mm/hour. Tests of her fasting blood glucose ranged from 256 to 277 mg/dl and her index of glycosylated hemoglobin was 9.6%. The lipid profile showed dyslipidemia (total cholesterol was 242 mg/dl and triglyceride was 339 mg/dl). Tests for VDRL, anti-acetylcholine receptor antibody and antinuclear...
antibody were all negative. An enhanced computed
tomography (CT) of the head showed no vascular abnor-
mality, mass lesion, or meningeal enhancement. Magnetic
resonance imaging (MRI) with gadolinium diethylenetriamine penta-acetic acid (Gd-DTPA)
enhancement and magnetic resonance angiography
(MRA) did not reveal any lesion in the intrinsic brain-
stem, cavernous sinus, or posterior orbit (findings not
shown). Insulin and anti-hypertensive agents were
administered and some amelioration of both the periocu-
lar pain and the limitation of eye movement were
observed during the admission. In addition, during her
stay we added an antiplatelet medication to her drug reg-
imen as we attributed her medical problems to diabetes-
related vasculopathic cranial mononeuropathy. Two
months after the event, a complete recovery of the right
ocular movement was observed.

DISCUSSION

Our patient’s clinical manifestation was character-
ized by weakness of the right medial rectus, inferior rec-
tus and inferior oblique muscles with normal function of
the superior rectus and levator palpebrae muscles. The
clinical constellation indicated that the palsy was limited
to the inferior division of the oculomotor nerve with
sparing of pupil. Selective involvement of the inferior
division of the oculomotor nerve is rarely reported and
only twenty-two cases have been documented(2-13).

Anatomically, the oculomotor nerve divides into a
superior and inferior division as it reaches the superior
orbital fissure(1). Hence isolated divisional oculomotor
paresis usually indicates a lesion at the anterior cav-
ernous sinus or the posterior orbit. Nevertheless, more
proximal lesions in the subarachnoid space(14) or intrinsic
brainstem(15) have been reported. Based on this, function-
al segregation of the nerve fiber should appear before
division of the oculomotor nerve in the anterior cav-
ernous sinus. On the other hand, topographic distribution
of the fascicles of the oculomotor nerve in the ventral
midbrain tegmentum from lateral to medial has been
proposed as follows: inferior oblique, superior rectus,
levator palpebrae, medial rectus, inferior rectus and
pupillary fibers(13). Selective involvement of the inferior
oblique, medial rectus and inferior rectus muscles in our
patient made intrinsic brainstem lesion unlikely.

With regard to the etiological diagnosis of the oculo-
motor nerve palsy, screening for hyperthyroidism, myas-
thenia, infection and autoimmune disease should be
done. In addition, absence of severe headache and spar-
ing of the pupil with gradual recovery of eye movement
were major against the existence of intracranial aneurysm. Cranial CT scan and MRI revealed no evident
lesion in the brainstem, cavernous sinus, or posterior
orbit. In a patient with similar clinical manifestation,
Ohtsuka and his colleagues, by using a fat suppression
MR technique, revealed a Gd-DTPA enhanced lesion at
the inferior division of the oculomotor nerve. They pro-
posed that the imaging findings might indicate the
inflammatory change of the oculomotor nerve caused by
a preceding viral episode(10). The fact that there was no
enhanced lesion in our study results suggested a vascu-
lopathy in the pathogenesis of the isolated inferior divi-
sional oculomotor paresis in this patient.

Combination of mononeuropathy multiplex and
focal cranial neuropathy in this patient is highly sugges-
tive of a dispersed process such as diabetic vasculitic
disorder. Isolated “nontraumatic”, “noncompressive”
oculomotor nerve palsy has been attributed to in situ
microinfarction of the nerve, a possible complication of
diabetes, hypertension or generalized atherosclerosis(15).
In particular, the pathogenesis of vasculitic oculomotor
nerve palsy has been well studied in the context of dia-
betes. Asbury and his colleagues identified hyalinization
of the arterioles with endothelial proliferative degenera-
tion and resultant stenosis(16). Although the nature of the
lesion has been considered ischemic biologically, direct
evidence of vessel occlusion was rarely found(16).-
Classical oculomotor lesion in patients with diabetes has
reportedly involved the nerve in the subarachnoid(17) or
cavernous portion(18). The lesion is normally located at
the borderzone areas between the blood supplies from the
perforating branches of the posterior cerebral artery, the
posterior communicating and basilar arteries, the tentori-
al and meningeal branches from the meningohypophy-
seal trunk of internal carotid artery, the ramus of the
artery of the inferior cavernous sinus and collateral arteries from the ophthalmic artery\(^{16}\).

A recent study showed that the pupil is affected in only one-third of patients with ischemic injury\(^{18}\). Of patients with relative pupil-involvement, the degree of anisocoria is generally less than 2 mm with prompt reaction to light\(^{18}\). The anatomical basis for the pupil-sparing third nerve palsy is thought to involve vasa vasorum with secondary destruction of central axons and the surrounding myelin sheath\(^{16,17}\). In contrast, the peripherally located pupillary-motor fibers, receiving blood supply from the arachnoid vessels, are spared as the core of the oculomotor nerve is injured by an ischemic condition\(^{16,17}\). In daily clinical practice, a normal pupil implies intrinsic nerve lesion while a dilated pupil suggests extrinsic lesion such as nearby tumor or aneurysm. However, incomplete ophthalmoplegia with pupil sparing has been reported as a sign of aneurysmal compression\(^{19-22}\), none of this class of patients sustained the inferior divisional palsy, as in our patient. To further evaluate a possible extrinsic lesion, MRA was performed and no cerebral aneurysm was ever discovered in our patient. Her ophthalmoparesis remained isolated with some improvement during her hospital stay, and recovered gradually without signs of aberrant regeneration within 2 months. Her outcome is compatible with other reported observations that complete recovery is usually found within 3 months in more than 90% of patients with isolated diabetic oculomotor nerve palsy\(^{26}\).

Clinically, third cranial nerve palsy is one of the most common diabetes related cranial neuropathies\(^{23}\), nevertheless, isolated divisional palsy is still a rarity\(^{24,25}\). Of special note, diabetic superior division palsy has been reported in few patients\(^{24,25}\). Inferior division palsy has been documented in only one multiple sclerosis (MS) patient with long-standing type 1 DM, however the MS demyelinating process was proposed as the pathogenesis in that case\(^{23}\). The presentation of our patient suggests that diabetic ophthalmoplegia may, in an isolated way, affect the superior or the inferior division. In conclusion, although inferior division oculomotor paresis is a rare manifestation, its value in the differential diagnosis of isolated oculomotor palsy should not be overlooked.

**REFERENCES**

20. Kissel JT, Burde RM, Klingele TG, et al. Pupil-sparing oculomotor palsy with internal carotid-posterior commu-