

Binocular vertical double vision in a diabetic patient

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CASE REPORT

A 73-year-old woman was admitted to our neurology department complaining of acute onset of binocular vertical double vision. She reported having diplopia for one week. She was not complaining of headache, nausea, or vomiting. She had no ocular history of trauma or ocular misalignment. The patient noticed a sudden onset of vertical diplopia, especially when she gazed in the lower-left direction. She did not have any other symptoms, and a neurological examination revealed no other findings. On examination, her visual acuity was 20/20 bilaterally. Pupil sizes and light reflexes were normal; there was no relative afferent

pupillary defect. Ptosis was absent in both eyes. Slit-lamp biomicroscopy, intraocular pressure measurements, and ophthalmoscopy were normal. There were no optic atrophy and optic disc oedema. Results of automated (Humphrey) visual field tests were normal. Extraocular motility examination was performed (*figure 1*).

WHAT IS YOUR DIAGNOSIS?

See page 310 for the answer to this photo quiz.

Figure 1. Extraocular movements in nine cardinal positions. Left downward eye movement is limited, left eye shows hypertropia and infraduction deficit



ANSWER TO PHOTO QUIZ (ON PAGE 309)

BINOCULAR VERTICAL DOUBLE VISION IN A DIABETIC PATIENT

DIAGNOSIS

The patient had a history of type 2 diabetes mellitus (DM), hypertension and hyperlipidaemia for five years. She had been on treatment with antihypertensive, antilipidaemic and oral hypoglycaemic agents and had poor glycaemic control. Fasting blood glucose was 16.7 mmol/l. Results of intravenous infusion of edrophonium (10 mg) and forced duction tests were negative. Computed tomography, magnetic resonance imaging (MRI) with and without contrast agents and diffusion MRI scans did not show any abnormalities, including the midbrain and the orbit. Also, MRI angiography did not reveal an aneurysm or arteriovenous malformation.

She was discharged after normalisation of blood glucose concentrations. Over the next one month, the patient's diplopia resolved completely and there was no residual ophthalmoplegia (*figure 2*).

In the light of these data, the diagnosis was considered to be isolated inferior rectus muscle (IRM) palsy. The differential diagnosis of an isolated IRM palsy includes orbital lesion (orbital pseudotumour, tumour, traumatic or postsurgical restrictive disease, thyroid ophthalmopathy), neuromuscular junction lesion (myasthenia gravis), multiple sclerosis, congenital, partial third nerve palsy and idiopathic causes.^{1,2} The cause of most IRM palsies is believed to be microvascular ischaemia, frequently associated with DM or systemic hypertension. Microvascular third nerve palsies

are frequently quite painful but usually resolve after two to four months.^{3,4}

Other reasons for the IRM palsy were excluded. In the differential diagnosis, we suspected that vascular ischaemic lesion related to DM was causing this clinic manifestation. Improvement in the patient's clinical condition after the regulation of blood glucose supported our diagnosis.

CONCLUSION

The diagnosis is isolated IRM palsy from microvascular ischaemia involving the oculomotor nucleus caused by type 2 DM.

REFERENCES

1. Lee AG, Tang RA, Wong GG, et al. Isolated inferior rectus muscle palsy resulting from a nuclear third nerve lesion as the initial manifestation of multiple sclerosis. *J Neuroophthalmol* 2000;20:246-7.
2. Takano M, Aoki K. Midbrain infarction presenting isolated inferior rectus nuclear palsy. *Rinsho Shinkeigaku* 2000;40:832-5.
3. Bortolami R, D'Alessandro R, Mani E. The origin of pain in 'ischemic-diabetic' third-nerve palsy. *Arch Neurol* 1993;50:795.
4. Lee DK, Kim JS. Isolated inferior rectus palsy due to midbrain infarction detected by diffusion-weighted MRI. *Neurology* 2006;66:1956-7.

Figure 2. Extraocular movements in nine cardinal positions



Improvement of patient's clinical condition after the regulation of blood glucose. There is no limitation in any gaze direction.