CASE REPORTS

Bilateral Fascicular Third Nerve Palsy in Posterior Circulation Stroke

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ABSTRACT

Third nerve palsy is bilateral in only about 10% of cases, of which one in five cases is due to brainstem stroke. Bilateral oculomotor nerve palsy as an isolated clinical finding after brainstem stroke is extremely rare. We present a case of severe bilateral fascicular oculomotor nerve palsy due to distal basilar occlusion and subsequent midbrain infarction of cardioembolic origin. The patient required mechanical aids and subsequent ptosis surgery to relieve complete ptosis at least unilaterally.

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Introduction

Mesencephalic infarction is rare, encompassing about 1% of cases in major stroke registries, of which only 10% are bilateral.¹ One in three mesencephalic strokes affect third nerve function.^{2,3}

Third nerve palsy in general is bilateral in about 10% of cases, of which one in five cases is due to brainstem stroke.⁴ On the other hand, bilateral oculomotor palsy as an isolated clinical finding after brainstem stroke is extremely rare. A review in 2002 on isolated cranial nerve palsies in cerebrovascular brainstem disease reported no bilateral cases.⁵ In most cases, other symptoms due to more widespread brainstem or thalamic lesions accompany deficits in ocular motility and tend to dominate the clinical picture.^{6,7}

Here, we present a rare case of bilateral fascicular oculomotor nerve palsy after midbrain stroke with only minor clinical deficits beyond ocular motility following the acute stage. The patient had to undergo a prolonged rehabilitation programme including botulinum toxin treatment of ocular muscles and unilateral ptosis surgery.

Case report

The 54-year-old patient acutely developed double vision, dysarthria, vertigo, left-sided motor weakness, and severe disturbance of consciousness. Blood

pressure was almost normal (143/83 mmHg) and no vascular risk factors had been known before. In an outside hospital, distal basilar occlusion without obvious infarction was demonstrated by diagnostic computed tomography imaging. The right vertebral artery appeared hypoplastic and supplied the territory of the posterior inferior cerebellar artery only.

Systemic thrombolysis with 70 mg rtPA was started and the patient was then transferred to a second hospital in order to perform endovascular thrombectomy. On arrival there, symptomatic improvement was noted (no disturbance of consciousness anymore, NIH stroke scale score only 3), and diagnostic cerebral angiography demonstrated that the thrombus at the basilar head had already dissolved following systemic thrombolysis. Magnetic resonance imaging (MRI) of the brain later demonstrated multiple ischemic lesions in the paramedian mesencephalon bilaterally, in rostral cerebellum bilaterally, in the paramedian thalamus on the right, and a spot-like infarction in the left occipital lobe (Figure 1). Probably, both the regions of the oculomotor nucleus (Figure 1d and e) and the oculomotor nerve fascicles exiting the mesencephalon (Figure 1b and c) were affected bilaterally.

There were imaging findings suggesting previous cerebral infarctions. According to the patient, 3 months earlier, he had transiently experienced a left-sided haemianopia, dysarthria,

Figure 1. Magnetic resonance imaging of acute right thalamic and bilateral brainstem stroke. (a) Coronal FLAIR imaging demonstrates cytotoxic oedema in right paramedian thalamus and bilateral paramedian mesencephalon. (b,c) Diffusion weighted imaging (b) and corresponding T2 imaging (c) of bilateral anterior paramedian mesencephalic and minor left cerebellar infarction. (d,e) Diffusion weighted imaging (d) and corresponding T2 imaging (e) of bilateral paramedian mesencephalic infarction.

and left-sided weakness. An MRI scan had been reported normal and no further diagnostic steps had been taken.

No angiographic or neurosonological pathology affecting the left vertebral artery was shown, in particular no signs of vertebral artery dissection. A diagnostic work-up to identify the source of embolic basilar artery occlusion made a cardioembolic origin probable. Transcranial ultrasound studies after agitated saline had been injected intravenously revealed a single embolic signal at rest and about 20 embolic signals following Valsalva manoeuvre. Right-to-left shunt following Valsalva manoeuvre due to patient

foramen ovale was confirmed by transoesophageal echocardiography. A hypermobile septal aneurysm was present, but there was no intracardiac thrombus or aortic atheroma. Left ventricular function and flow velocity in the left atrial appendage were normal. Importantly, no crural venous thrombosis was detected by duplex ultrasonography. Apart from a single supraventricular run over only seven beats no relevant cardiac arrhythmia was detected in several 24-h electrocardiographies. Blood lipids, coagulation screening, and other blood tests were unremarkable, except for slight hyperuricaemia. Therapy with acetylsalicylic acid and a statin was initiated.

Dysarthria subsided after a few days. After discharge from the hospital and during the rehabilitation phase that followed, the sequelae of bilateral third cranial nerve palsy prevailed. The most disturbing feature was complete ptosis that required mechanical aids, e.g. adhesive strips, to keep either eye open. Only the right eye could be moved almost until midline while the left eye remained in abduction. Both pupils were mydriatic, on the right more than on the left side, and no reaction to light was present on the right side. There was exotropia of the right eye and vertical eye divergence. Convergence paresis and complete vertical gaze palsy were also present. In later examinations, an incomplete ocular tilt reaction was suggested by a slight skew deviation of the left eye over the right one and a slight ocular torsion to the right, without head tilt. Due to oculomotor disturbance, there was dizziness and unsteadiness of gait. Monopodal stance on the left side was unsteady, but balance and postural control improved after dedicated physical therapy. Slightly reduced fine motor skills and disturbed diadochokinesis of the left hand were treated by ergotherapeutic intervention.

There were minor neuropsychological deficits of tonic alertness, divided attention, and working memory that were irrelevant for everyday function and quality of life. Post-stroke depression responded to psychotherapy. Dizziness improved and a slight improvement of the adduction of right eye could be observed during rehabilitation. One year after the acute stroke, botulinum toxin injection to the lateral rectal muscle of the right eye led to an improvement of the adduction of the right eye and some saccadic improvement, but still no stereoscopic vision was achieved when both eyes were open. Prismatic glasses provided some benefit. A support bracket attached to the glasses helped to keep one eye open. Later, ptosis reduction was accomplished surgically by frontal muscle suspension on the right side.

Ten months later, the patient suffered an infarction in the right frontal lobe which showed minor haemorrhagic transformation after systemic thrombolysis. No other embolic source and no signs of paraneoplastic disease were detected during repeated follow-up examinations. The patient recovered from a left-sided hemiparesis and neglected very well, and underwent endovascular occlusion of the patient

foramen ovale without complications. During a further follow-up of 5 months, no new cerebral ischemic events have been observed.

Discussion

Bilateral oculomotor palsy as the predominant clinical finding after brainstem stroke is rare, as other symptoms due to widespread brainstem, thalamic, or posterior stroke dominate over deficits in ocular motility.^{6,7}

The oculomotor nerve nucleus is a paired nucleus in the mesencephalic tegmentum containing motor neurons for four individual ocular muscles, including the contralateral superior rectus muscle.⁸ Eyelid elevation is governed by the unpaired central caudal nucleus situated medially and dorsally of the main nucleus. The pupillary sphincter is innervated by the Edinger-Westphal nucleus which is situated posterior to the oculomotor nucleus, or by more dorsally located neurons.⁸ Thus, unilateral third nerve palsy at the nuclear level may give rise to bilateral ptosis and bilateral superior rectus superior weakness in 50% of cases, while innervation of the other contralateral ocular muscles is spared in unilateral lesions.⁸ Fibres of the third nerve traverse the ipsilateral red nucleus and leave the midbrain ventrally near the midline.

The paramedian area of the rostral midbrain is supplied by superior medial mesencephalic branches of the posterior cerebral artery.⁹ The superficial layers of the ventral paramedian tegmentum may also be supplied by direct branches from the basilar artery.

Paramedian ischemic lesions in the dorsal midbrain tegmentum may affect the oculomotor nucleus bilaterally.¹⁰ A similar clinical picture may arise from paramedian midbrain haematoma¹¹ or possibly from compression of the bilateral nerve exit by a dolichoectatic basilar artery.¹²

Midbrain infarction might also cause bilateral vertical gaze palsy, as seen in our case,¹³ or bilateral internuclear ophthalmoplegia in more paramedian lesions.¹⁴ In the case presented here, MRI (Figure 1) suggested bilateral fascicular lesions which affected both the third nerve nuclei and their emergent fibres. Fascicular nerve lesions due to more lateral infarcts are characterized by

weakness of adduction and vertical restriction of the ipsilateral eye, ptosis, and a mydriatic pupil.¹

Recently, a more widespread palsy of ocular motility has been described as a reverse locked-in syndrome,¹⁵ which we consider a rather misleading term. Nevertheless, the sequelae of bilateral oculomotor nerve palsy can be persistently disabling and deserve intensive neurorehabilitatory efforts including prisms, occlusion, scanning exercises, refraction, and in some cases surgical management,¹⁶ in order to provide symptom relief and to regain some level of functional independence during everyday activities.

Patient consent

The patient described in this case report consented in written form to publication of all data and imaging files related to the case.

Declaration of interest

The authors declare that there are no conflicts of interest. The authors alone are responsible for the writing and content of the article.

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