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Original Articles

Clinical Observation on Oculomotor Nerve Palsy Treated by Moxibustion

Hyun-Su Woo, Dong-Min Seo, Jong-Deog Kim, Sang-Min Park, Hyun-Dong Kim[1], Im-Jeung Byun[2], Chang-Hwan Kim

Department of Acupuncture & Moxibustion, College of Oriental Medicine, Kyung-Hee University,
Department of Classics & Medical History, College of Oriental Medicine, Sang-Ji University[1],
Department of Acupuncture & Moxibustion, College of Oriental Medicine, Kyung-Won University[2]

Objective : Moxibustion is one of the primary remedies in traditional oriental medicine. We successfully treated a one patient who complained of oculomotor nerve palsy related to diabetes mellitus with moxibustion.

Methods : We performed moxibustion treatment on the protuberances surrounding the Mok-chang acupoint and adjacent frontal scalp of the patient' s head at the same time every day and observed the recovery of eye movement and changes of ptosis.

Results : After about 1 week of moxibustion treatment, there were some changes of ptosis and eyeball movement. The patient felt better opening his eyes than before and seemed to reduce his paralytic condition. After 14 days, ptosis was remarkably improved, although slight diplopia remained, and eye movement had recovered to almost normal. Furthermore, his blood glucose was on the decrease and revealed an average 120 ㎎/*dl*.

Conclusions : Moxibustion treatment performed on the Mok-chang acupoint remarkably improved ptosis and limited eye movement arising from oculomotor nerve palsy related to diabetes mellitus. We hope moxibustion is used for treating nerve palsies and similar diseases in the future. *(Korean J of Oriental Med 2003;24(4):149-153)*

Key Words: oculomotor nerve palsy, moxibustion, Mok-chang

Introduction

Diabetes mellitus is the most common endocrine disease in the world today; millions of people the world over suffer from this disease. It is characterized by a disruption in glucose metabolism, resulting in short and long term complications that affect multiple organ systems, particularly the eyes, kidneys, nerves, and blood vessels.

Diabetic neuropathy is one of the most common complications of diabetes mellitus and can affect any part of the nervous system. Patients with diabetic neuropathy may experience numbness, paresthesias, severe hyperesthesias, or pain. Diabetic neuropathy classically presents as a sudden wrist or foot drop or ophthalmoplegia involving cranial nerves Ⅲ, Ⅳ, and Ⅵ[1].

Partial ophthalmoplegia due to third nerve palsy with an intact pupil is a frequent cause of diplopia observed in diabetic patients. Pupillary muscle involvement, such as anisocoria and loss of light reflex, is usually

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Correspondence to:Woo Hyun-su, Kyung-Hee Oriental Medical Center 1st Hoe-gi-dong, Dongdaemun-Gu, Seoul, Korea; Tel: 82-2-958-9198, Fax: 82-2-958-9210, E-mail: mari10@hanmail.net

uncommon in this diabetic cranial mononeuropathy[2].

We present here the case of a man with diabetes mellitus who suddenly developed diplopia, anisocoria, ptosis of eyelid, and defective light reflex. The patient was successfully treated with moxibustion and herbal medication, as reported in this paper.

Report of the case

A 56-year-old man visited our hospital complaining of complete ptosis, diplopia, and eye movement limited in all directions. He also complained of headache and dizziness. He had a history of hypertension, diabetes mellitus and hyperlipidemia. Since 1999, he had suffered from myocardial infarction and had undergone percutaneous transluminal coronary angioplasty (PTCA) in 2001. He did not smoke but sometimes drank, about once or twice a week.

On 30th March 2003, feeling dizziness, headache and general weakness, he was admitted to Daejen University Oriental Medical Hospital and received a diagnosis of cerebral infarction at both periventricular white matter (PVWM) locations. According to his wife, at that time there was no any sign of ptosis, diplopia, or limited eye movement, but, 2 days later, those symptoms suddenly occurred.

During his stay at Daejen University Oriental Medical Hospital, there was no improvement of symptoms. On 16th April, he transferred to our hospital.

Laboratory studies at admission showed elevated blood glucose of 165 ㎎/㎗, elevated glycosylated hemoglobin (HbA1C) of 8.1% and an abnormal lipid profile with a total cholesterol of 261 ㎎/㎗, and triglycerides of 247 ㎎/㎗. All other laboratory values were within normal limits.

Vital signs were pulse rate of 80 beats per minutes, blood pressure of average 130/75 ㎜Hg, body temperature of 36.7 ℃ and a respiratory rate of 20. There were no carotid disorders. Neurologic assessment revealed both eye ptosis with the inability to gaze upward or downward or for medial, lateral movement. Pupil reflex was intact. The fundi were normal with no signs of diabetic retinopathy. Extremity motor assessment revealed normal tone and power. There was no evidence of any gait abnormalities.

Radiological examination included computed tomography (CT) and magnetic resonance imaging (MRI). CT scan of the head showed generalized atrophy and a focal region of low density in the PVWM. To get more precise imaging, MRI of the head was performed. A sagittal short repetition time (TR), short echo time (TE), long TR with short and long TE axial images, and coronal T1-weighed and, axial views on T1- and T2-weighed and FLAIR (fluid attenuated inversion recovery) MR images of the head were obtained. MR images showed mild cerebral atrophy and a focal region of signal abnormality in the left fronto-temporal lobe and quite focal low attenuation in the central pons and PVWM. The left fronto-temporal lobe attenuation likely represented a previous infarct. The central pons lesion low density was suspicious for an age-indeterminate infarction. There was no evidence of a recent brain stem infarction.

The patient was admitted for further evaluation of symptoms and other treatments. We decided to apply moxibustion to his condition. When we deliberated over selection of the useful acupoint, we found a protuberance surrounding the Mok-chang acupoint and adjacent frontal scalp. We knew Mok-chang had been used for ophthalmological disease and orbicularis oculi muscle disease, so we decided to choose this point for treatment.

After about 1 week of moxibustion treatment, there were some changes of ptosis and eyeball movement. The patient felt better at opening his eyes, and his mild headache and dizziness disappeared. After 14 days,

ptosis was remarkably improved, diplopia slightly remained and eye movement recovered to almost normal. Much better, his blood glucose was on the decrease and revealed average 120 mg/dl.

Discussion

The oculomotor nerve innervates all of the muscles controlling movements of the eye except for the lateral rectus (cranial nerve Ⅵ) and the superior oblique (Ⅳ).

Thus, it is responsible for all movements of the eye apart from abducting - lateral rectus - and looking down whilst adducted - superior oblique.

The parasympathetic supply of the eye is also from the oculomotor nerve. This maintains elevation of the eyelid, constricts the pupil and allows broadening of the lens via levator palpebrae superioris, sphincter pupillae and the ciliary muscle, respectively.

Full assessment of oculomotor nerve function involves testing of movement, reaction to light, and accommodation. If all of these are normal, "PERLA" may be written in the notes - pupils equal, reactive to light and normal accommodation.

Oculomotor nerve palsy generally causes mydriasis, adduction impairment and upward/downward gaze palsy in the affected eye. Paralysis of the third cranial nerve affects the medial, superior, and inferior recti, and inferior oblique muscles. Dysfunction of the levator palpebrae and superior rectus function, innervated by the ipsilateral and contralateral subnucleus, is easy to treat. However, the medial rectus, inferior rectus, and inferior oblique function and pupillary components are innervated by ipsilateral subnucleus[3]. The eye is incapable of movement upwards, downwards or inwards, and at rest the eye looks laterally and downwards owing to the overriding influence of the lateral rectus and superior oblique muscles respectively. The reduced response of levator palpebrae superioris results in ptosis, a drooping of the upper eyelid.

The third nerve palsy with pupillary sparing is often termed the medical third palsy and often has an ischemic or diabetic aetiology. This third nerve palsy secondary to ischemic changes from diabetes would not show up as a "stroke" on imaging studies.

The pathology is due to small vessel infarctions on the inner portion of the oculomotor nerve (cranial nerve Ⅲ). The symptoms begin acutely with weakness of part or all (partial vs. complete) of the eye muscles supplied by the third nerve. There may be pain associated with eye movement depending on where the infarct occurred. A classic DM lesion spares the pupil, that is, it will constrict with light. One study in the literature reports that of 61 patients studied with isolated CN Ⅲ palsy, 23 (38%) had symptoms related to ischemic nerve palsy. All of those patients recovered in 3 months. One additional study reported an association with headaches and CN Ⅲ palsy[4].

The differential diagnosis for sudden-onset impairment with oculomotor nerve neuropathy includes diabetic neuropathy, vasculopathy secondary to hypertension, polyarteritis nodosa, posterior communicating artery central aneurysm with cranial nerve Ⅲ compression, systemic lupus erythematosus and neuropathy of unknown etiology (1). In this case, there were symptoms rarely observed in a diabetic patient with preexisting peripheral neuropathy and hypertension in the context of uncontrolled diabetes as evidenced by his elevated admission blood glucose and HbA1C.

Radiologically, no evidence of brain stem infarction was detected on MRI or CT.

Significant case reports of diabetic neuropathies involving various cranial nerves have been observed[5,6]. Simultaneous diabetic polyneuropathy is a much rarer entity, with only 15 patients observed in 10 case reports in the literature[7]. Only 4 of these 15 patients showed

simultaneous oculomotor and trigeminal nerve neuropathies, and in 14 of these cases, the neuropathies occurred bilaterally[8].

Diabetic neuropathy is a common complication of diabetes mellitus. It may be classified as a symmetric distal polyneuropathy, autonomic neuropathy, diabetic amyotrophy or asymmetric neuropathy[9]. In our case, simultaneous bilateral cranial neuropathies are consistent with a diabetic symmetric distal polyneuropathy. The sudden-onset, progressive ptosis, elevated HbA1C, diplopia, inability to gaze upward or downward, and limited eye movement indicates spontaneous bilateral multiple cranial neuropathies involving the oculomotor nerve. Diabetic mononeuropathies typically involve cranial nerve Ⅲ, Ⅳ, Ⅵ, resulting in extraocular muscle paralysis (partial or complete) and diplopia[9]. Diabetic cranial nerve Ⅲ, Ⅳ, Ⅵ, Ⅶ palsies have been shown to have an incidence of 0.97% in the diabetic population over a 25-year period.

The most common syndrome is cranial nerve Ⅲ palsy with pupillary reflex sparing, a feature distinguishing this condition from a compressive neuropathy. This syndrome can involve more than 1 cranial nerve, and spontaneous recovery generally occurs in 3 to 12 months.

Some studies have reported, of the patients with diabetic or idiopathic palsies, regardless of pupillary involvement, about 68% had improvement of the oculomotor paresis within 4 weeks, 96% within 8 weeks, and 100% within 12 weeks of the onset of symptoms. They showed patients with pupil-sparing oculomotor nerve palsies should be considered for extensive neuroradiologic evaluation only if there is deterioration or failure to improve within 4 to 8 weeks[10].

Moxibustion is one of the therapeutic methods of traditional oriental medicine.

Moxibustion stimulation was performed by burning a moxa cone of about 4 mg weight placed on the shaved skin[11], and heat generated by burning herbal preparations containing Artemisia vulgaris (mugwort) to stimulate acupuncture points. This method is safe, cheap, and almost free of side effects.

Mok-chang is an acupuncture point of the gall bladder meridian. Mok-chang is known in treatment of ophthalmological disease and orbicularis oculi muscle disease.

When we considered selection of a useful acupoint, we found a protuberance surrounding the Mok-chang acupoint and adjacent frontal scalp of the patient, so we selected this point and got a remarkable result.

Of course, this is only one case of using moxibustion to treat nerve palsy, but there seems to a significant relationship between moxibustion and nerve palsy syndromes. That is why we report this case. To define this point, more further studies and case reports are needed.

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