

Pontine Cavernoma Hemorrhage Leading to Millard-Gubler Syndrome

Serdar Kesikburun, MD, İsmail Safaz, MD,
and Ridvan Alaca, MD

From the Department of Physical Medicine and Rehabilitation, Gülhane Military Medical Academy, Turkish Armed Forces Rehabilitation Center, Ankara, Turkey.

Financial disclosure statements were filed and no conflict of interest has been reported by the authors or any individuals that control the content of this article.

0894-9115/11/9003-0263/0

American Journal of Physical Medicine & Rehabilitation

Copyright © 2011 by Lippincott Williams & Wilkins

DOI: 10.1097/PHM.0b013e3181e29e8e

A 27-yr-old woman with a right hemiplegia accompanying a left peripheral facial paralysis and diplopia was admitted to our rehabilitation center with a diagnosis of Millard-Gubler Syndrome (MGS). Her initial complaint had been one of the rapid onset of double vision 1 mo earlier. Four days after this, contralateral limb weakness and facial palsy developed. After ophthalmologic and neurologic examinations, a hemorrhage in the brain stem caused by a cavernous hemangioma that extended over a large area of the left side of the pons was detected with magnetic resonance imaging (Fig. 1). After neurosurgery to excise the cavernoma, the patient was referred to our department for rehabilitation. In her neurologic examination at presentation she could not look outward with her left eye because of paralysis of the abducens, and her left facial muscles were completely paralyzed because of facial nerve paralysis. There was loss of right upper and lower limb strength, and she was unable to walk independently. All muscle stretch reflexes were hyperactive on her right side. She did not have sensorial impairment, and her cognitive abilities were normal. After 1 mo of rehabilitation, including strength training, balance, and gait exercises, she was capable of walking independently with an ankle-foot orthosis and a tripod cane.

MGS is a crossed brain stem syndrome that affects the face on the side of the lesion and the limbs on the opposite side. The components of MGS are ipsilateral facial and abducens nerve paralysis as a lower motor lesion at the cranial nerve nucleus level and hemiplegia of the contralateral limbs caused by the involvement of the corticospinal tract before its crossing over.¹ It is usually caused by

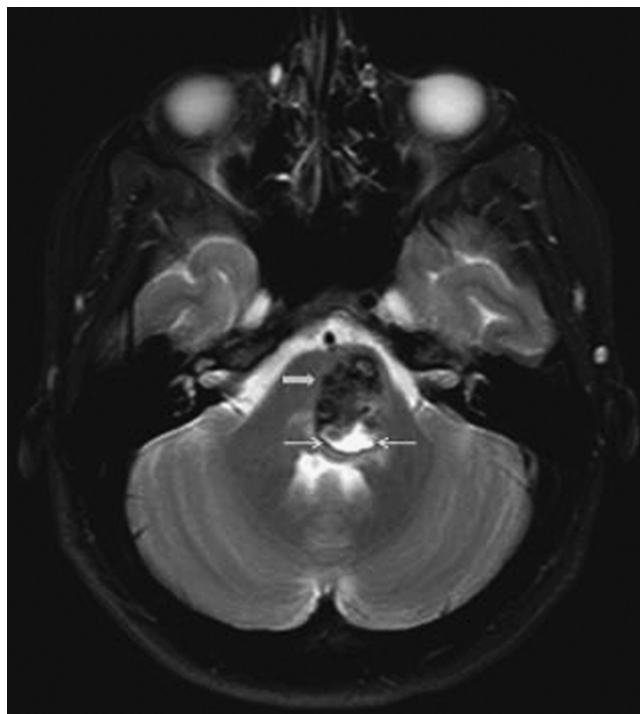


FIGURE 1 Magnetic resonance imaging scan of the brain stem shows the hemorrhage (thin arrows) that resulted from a cavernous hemangioma (thick arrow) that extended over a large area of the left side of the pons.

lesions of the basal portion of the caudal part of the pons, which are extensive enough to involve the corticospinal tract and include the fibers of the abducens and facial nerves. Structural injury of the pons in MGS is caused by tumors, bleeding, and, rarely, by infarction secondary to occlusion of the basilar artery.² Patients with MGS may also have other neurological abnormalities associated with involvement of structures next to the facial nerve nucleus, such as contralateral hemiparesis and contralateral cerebellar ataxia.^{2,3}

REFERENCES

1. Silverman IE, Liu GT, Volpe NJ, et al: The crossed paralyses. The original brain-stem syndromes of Millard-Gubler, Foville, Weber, and Raymond-Cestan. *Arch Neurol* 1995;52:635-8
2. Onbas O, Kantarci M, Alper F, et al: Millard-Gubler syndrome: MR findings. *Neuroradiology* 2005;47:35-7
3. Gandhavadi B: Millard-Gubler syndrome: Electrophysiologic findings. *Arch Phys Med Rehabil* 1988;69:980-2

All correspondence and requests for reprints should be addressed to: Serdar Kesikburun, MD, TSK Rehabilitasyon Merkezi 06530 Bilkent, Ankara, Turkey.