

Omer Onbas
Mecit Kantarci
Fatih Alper
Leyla Karaca
Adnan Okur

Millard–Gubler syndrome: MR findings

Received: 15 April 2004
Accepted: 24 September 2004
Published online: 13 January 2005
© Springer-Verlag 2005

O. Onbas (✉) · M. Kantarci · F. Alper
L. Karaca · A. Okur
Department of Radiology,
School of Medicine, Atatürk University,
Terminal mah. Somunoglu cad, Esatpasa
Apt B Blok No: 3/7, Erzurum, Turkey
E-mail: oonbas@hotmail.com
Tel.: +90-442-2361212
Fax: +90-442-2361301

Abstract We present magnetic resonance imaging findings in Millard–Gubler syndrome. In our case, Millard–Gubler syndrome was described first by magnetic resonance angiography findings. MR imaging revealed an infarct in the left side of the ventral pons. Clinical examination and MR imaging revealed Millard–Gubler syndrome.

Keywords Infarction ·
Millard–Gubler syndrome ·
MRA

Introduction

Millard–Gubler syndrome (MGS) is caused by infarction of the pons involving the 7th cranial nerve and fibers of the corticospinal tract. It is usually seen in brainstem tumor or bleeding, but rarely as infarction. The characteristic of this syndrome is crossed paralysis affecting the limbs on one side of the body and the face on the opposite side.

To our knowledge, although there have been cases of MGS in the literature [1–5], no such case had been reported previously with 3D time-of-flight MR angiography (3D TOF MRA) findings.

Case report

A 56-year-old male was admitted to our department because of complete left facial weakness and contralateral right side numbness for 6 months. The symptoms were limited to the lower portion of the face, where the muscles were paralyzed. There was no problem with his intelligence and consciousness. On neurological

examination, he showed left facial paralysis and hemiparesis on the right side. Ocular movements were normal. The deep tendon reflexes were exaggerated on the right side. Blood cholesterol was 260 mg/dl. The remaining laboratory results were normal.

Without contrast axial T1-weighted MR images revealed a hypointense area in the left ventral part of the pons (Fig. 1). The axial T2-weighted constructive interference of steady state (CISS) sequence showed a hyperintense infarct located ventromedial on the left part of pons without peripheral edema and stenosis of basilar artery (Fig. 2). 3D TOF MRA showed stenosis of basilar artery (Fig. 3). Diffusion MRI revealed an old ischemic lesion in the ventral pons (Fig. 4). He was, clinically and radiologically, diagnosed as having MGS resulting from an infarction of the ventral pons.

Discussion

Millard–Gubler syndrome is characterized by ipsilateral facial palsy, probably involving the root fibers and with contralateral hemiplegia or hemiparesis resulting from

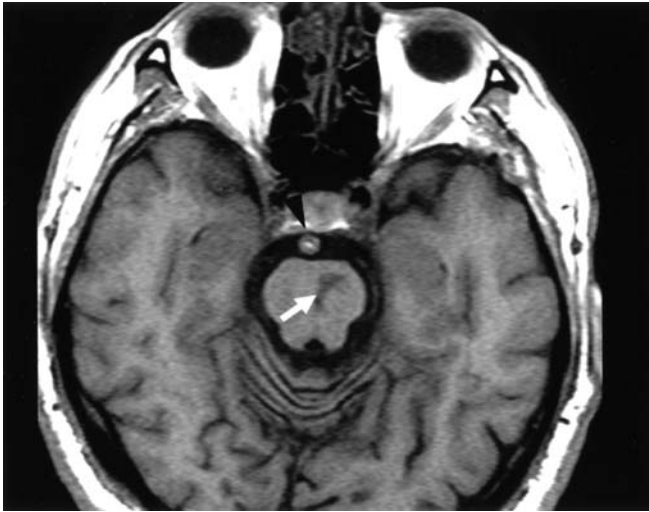


Fig. 1 Axial T1-weighted MR images show hypointense area in the left ventral part of the pons (*white arrow*) and stenosis of basilar artery (*black arrow head*)

involvement of the corticospinal tract. The disturbance was described by Millard in 1855 and by Gubler in 1856. The characteristics of this syndrome are crossed paralysis affecting the limbs on one side of the body and the face on the opposite side [3, 5, 6].

Augustine Millard (1830–1915) first proposed that direct facial paralysis accompanied by contralateral hemiplegia was a sign of pontine hemorrhage [6]. MGS results from a lesion of the medioinferior pons. The facial weakness is due to a lower motor neuron lesion at the nucleus level. The abducens nerve passes through to the medial pons to the ventral surface of the pons.



Fig. 2 Axial T2-weighted constructive interference of steady state sequence shows hyperintense infarct located ventromedial on the left part of pons without peripheral edema (*white arrow*) and stenosis of basilar artery (*black arrow head*)

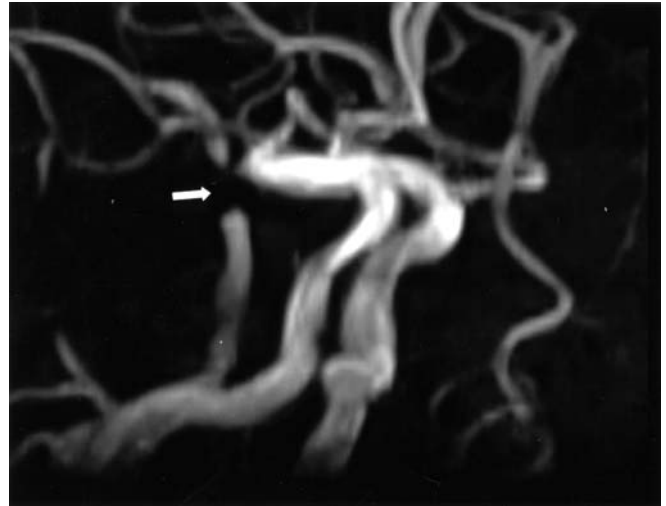


Fig. 3 3D TOF MRA shows stenosis of basilar artery (*white arrow*)

Therefore, MGS is often associated with abducens nerve palsy, contralateral cerebellar ataxia, one-and-a half syndrome and similar syndromes [4, 5]. Most patients with MGS show other associated neurological abnormalities, since many nuclei or fibers exist next to the root fibers of the facial nerve. It is generally seen in brainstem bleeding and tumors, but rarely in infarction. This infarct results from occlusion of the penetrating branches of the basilar artery into the pons [1, 6]. The cause of the event was a rare pons infarct. Our case had isolated facial paralysis and right hemiparesis, but the eye movements were normal and the eye muscles were not affected due to restriction of the infarct to the ventral

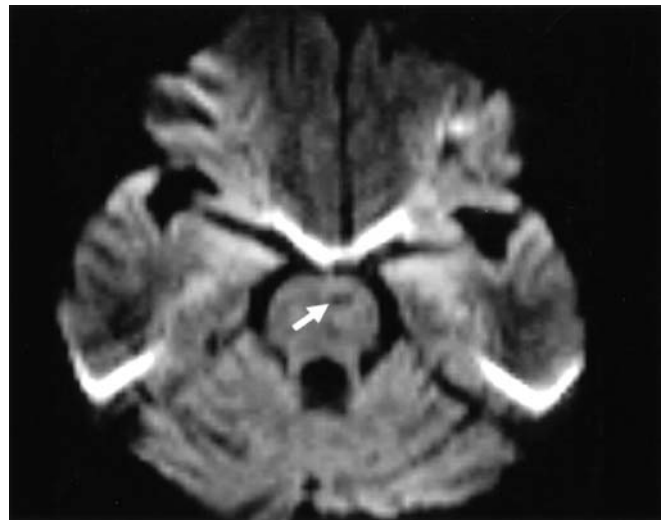


Fig. 4 True diffusion ($b = 1000$) image shows an ischemic lesion in the ventral pons (*white arrow*)

pons and absence of dorsal spread. This lesion probably affected the corticospinal tract and facial nerve fascicle.

Clinico-anatomical correlations have usually been reported in neuropathological cases. In some cases reported previously, computerised tomography (CT) and classical MR imaging were used in the diagnosis of MGS [1–5]. 3D TOF MRA is routinely used in stroke work up to detect arterial occlusion. It is a non-invasive technique that allows dynamic assessment of the cranial circulation [7, 8]. Diffusion MRI and routine MR images gave us information about diagnosis with infarct images, but they did not give information about the etiology of MGS.

As demonstrated with classical T1 and T2 weighted imaging, we confirmed the infarct with diffusion. Also, we have highlighted with 3D TOF MRA that the cause

of infarct was an occlusion in the basilar artery. We presented this first case showing isolated MGS diffusion and 3D TOF MRA. Our aim was to show MRI and 3D TOF MRA correlations in patients with the MGS with a ventral pontine infarct.

In conclusion, in clinically suspected MGS cases, the cause should be taken as pons infarct and in order to determine this, after the routine MR sequence, diffusion MRI and MRA should be added. 3D TOF MRA, which is a non-invasive technique, provides prognostic information by showing occluded arteries and their levels for MGS patients. This information may help a neurologist to develop strategies for patient treatment. In addition, it may provide valuable information about the degree and location of affected parts of pons.

References

1. de Seze J, Lucas C, Leclerc X et al. (1999) One-and-a-half syndrome in pontine infarcts: MRI correlates. *Neuroradiology* 41:666–669
2. Hashizume Y, Kishimoto H, Iijima S (1984) Occlusion of the basilar artery. A clinical and pathological study of thirteen autopsied cases. *Acta Pathol Jpn* 34:29–40
3. Matlis A, Kleinman Y, Korn-Lubetzki (1994) Millard–Gubler syndrome. *Am J Neuroradiol* 15:179–181
4. Gandhavadi B (1988) Millard–Gubler syndrome: electrophysiologic findings. *Arch Phys Med Rehabil* 69:980–982
5. Yasuda Y, Matsuda I, Sakagami T et al. (1993) Pontine infarction with pure Millard–Gubler syndrome: precise localization with magnetic resonance imaging. *Eur Neurol* 33:331–334
6. Silverman IE, Liu GT, Volpe NJ et al. (1995) The crossed paralyses the original brain-stem syndromes of Millard–Gubler, Foville, Weber, and Raymond–Cestan. *Arch Neurol* 52:635–638
7. Burdette JH, Ricci PE, Petitti N et al. (1998) Cerebral infarction: time course of signal intensity changes on diffusion-weighted MR images. *Am J Radiol* 171:791–795
8. Yang JJ, Hill MD, Morrish WF et al. (2002) Comparison of pre- and post-contrast 3D time-of-flight MR angiography for the evaluation of distal intracranial branch occlusions in acute ischemic stroke. *Am J Neuroradiol* 123:557–567