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Radiologic-Clinical Correlation
Millard-Gubler Syndrome

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Millard-Gubler syndrome, first described in 1858, is caused by a lesion at the ventral part of the pons and manifests itself as a unilateral facial and/or abducens palsy with contralateral weakness of the arm and leg (1, 2). These classical descriptions were based on postmortem examinations and stated that hemorrhage or tumor was the common cause (1, 3). We describe a patient with Millard-Gubler syndrome caused by an ischemic stroke in the right pons. Precise anatomic confirmation was obtained by computed tomography (CT) and magnetic resonance (MR). Our report compares the neuroradiologic findings with the classical neuroanatomic correlation (4).

Case Report

A 76-year-old man presented with sudden weakness of the left arm and leg and slurred speech. He had a history of essential hypertension, ischemic heart disease, and insulin-treated type II diabetes mellitus. On examination he was oriented with slight dysarthria. Ocular movements were normal. There was a peripheral right facial palsy and flaccid left hemiparesis. Deep tendon reflexes were brisk on the left side, and plantar responses were extensor on the left. The remainder of the physical and neurologic examination was normal.

Postcontrast brain CT scan performed 3 days after admission demonstrated a low-density lesion in the right anteromedial pons (Fig 1A). MR of the brain showed a well-defined area in the right anteromedial pons, which was homogeneously hypointense on short-repetition-time/short-echo-time images (Fig 1B) and hyperintense on long-repetition-time/long-echo-time images (Fig 1C).

The patient's condition improved slowly. On examination 2 months later cranial nerves were normal, and there was a spastic hemiparesis involving essentially the left arm.

Discussion

Millard-Gubler syndrome, one of the classical pontine-crossed syndromes, was initially described in autopsy specimens by the French school of neurology in the last century (5, 6). Originally the cause of the neurologic deficit was believed to be tumoral (1). Our report emphasizes the possible vascular cause of the

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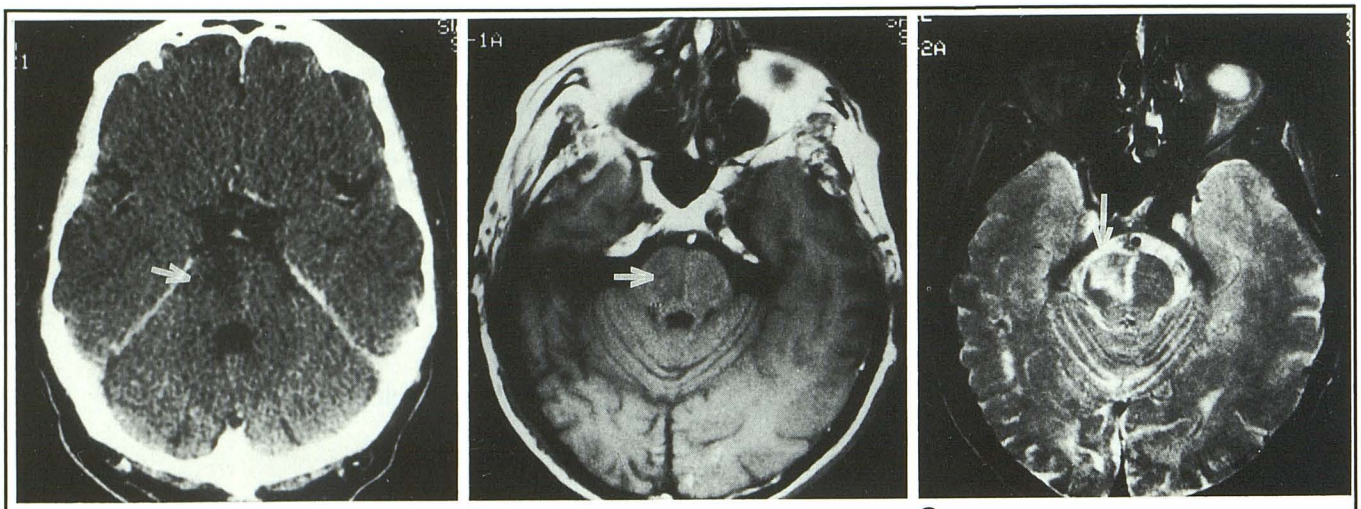

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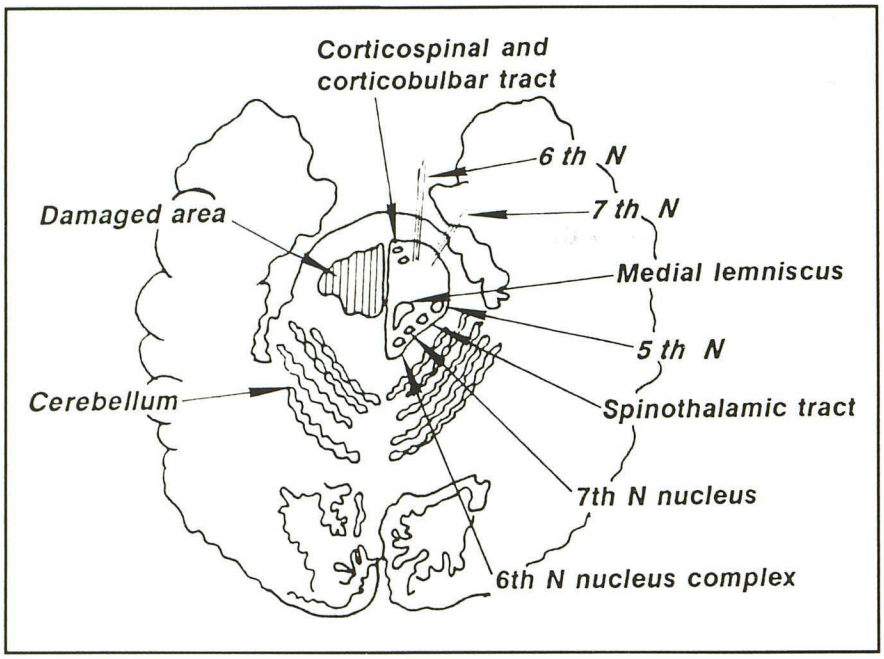

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Fig. 1. The lesion located at the ventromedial part of the right lower pons appears as a hypodense lesion on axial unenhanced CT (*A*); low signal intensity on short-repetition-time/short-echo-time axial MR (400/10/2 [repetition time/echo time/excitations]) (*B*); and high signal intensity on long-repetition-time/long-echo-time axial MR (2000/80/1) (*C*). Diagram of the lesion on the left side, normal anatomy of the region, and relevant structures on the right side (*D*).

syndrome (2, 7, 8). In our case the precise anatomic location was identified with imaging.

The neuroanatomic pathways present at the inferior pontine level are represented in Fig 1D, in the central region of the ventral pons course the corticospinal and corticobulbar tracts. The medial lemniscus lies posteriorly on each side of the median raphe. Nuclei of the VI and VII nerves are found in the dorsal portion of the pons: their fibers pass through the pontine tegmentum and emerge anteriorly at the cerebellopontine angle. The spinothalamic tract occupies a position in the anterolateral tegmentum, medial to the descending tract and nucleus of the V nerve (9).

The lesion causing Millard-Gubler syndrome and demonstrated in Fig 1 involves the ventromedial part of the pons, which contains the corticospinal tract and the fascicular intrapontine portion of the VII nerve, causing ipsilateral paralysis of the facial nerve and contralateral hemiplegia. Fibers of the VI nerve, sometimes involved in the classical descriptions (1), were spared in our case, as in Gubler's first case, because of the slightly anterior course of the VI nerve within the substance of the pons (4). The medial lemniscus and the spinothalamic tract are spared in this syndrome, as it is shown in Fig 1D, thus explaining the absence of sensory impairment.

TABLE 1: Pontine-crossed syndromes, anatomic and neurologic correlation

Syndromes		Millard-Gubler	Foville	Raymond
Site in pons	Ventral	+		+
	Tegmentum		+	
Structures involved	Corticospinal tract	+	+	+
	VI			
	nerve		+	
	roots	±		
	fibers			+
	VII			
	nerve		+	
	fibers	+		
	Gaze center		+	
Signs	Gaze palsy		+ to side of lesion	
	Abducens palsy	± ips	+ ips	+ ips
	Facialis	+ ips	+ ips	
	Hemiplegia	+ con	+ con	+ con

Note.—± indicates sometimes; ips, ipsilateral; and con, contralateral.

Other pontine-crossed syndromes have been described and remain part of the classical neurologic heritage. They are briefly summarized, according to their initial descriptions, in Table 1 (4). Later reports describe more extensive lesions (1) or combine these pontine syndromes (2).

These syndromes are unique because the disease involves the brain stem above the decussation of the pyramidal and spinothalamic tracts; *the cranial nerve signs are therefore ipsilateral to the lesion, and the long tract signs contralateral, resulting in a crossed syndrome.*

Foville syndrome manifests usually as gaze palsy to the side of the lesion, ipsilateral abducens, and facial palsy and contralateral hemiplegia (2, 4, 5). Crossed hemihypesthesia and ipsilateral cerebellar signs may be present in the more complete forms (1).

In the Raymond syndrome abducens paralysis is associated with contralateral hemiplegia (4, 10). Sensory impairment (deep and superficial) and cerebellar signs with rubral, athetotic, or ballistic movements of the contralateral limbs are included in this syndrome in later reports (1).

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