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Dysphagia

Magnetic Resonance Imaging for Evaluating Neurogenic Dysphagia

Won S. Kim, M.D.,[1] David Buchholz, M.D.,[2] Ashok J. Kumar, M.D.,[1] Martin W. Donner, M.D.,[1] and Arthur E. Rosenbaum, M.D.[1]

Departments of [1] Radiology and Radiological Sciences and [2] Neurology, The Johns Hopkins Medical Institutions, Baltimore, Maryland, USA

Abstract. Dysphagia due to CNS pathology usually stems from one of two patterns of disease: (1) bilateral corticobulbar tract dysfunction ("pseudobulbar palsy") or (2) pontomedullary dysfunction ("bulbar palsy"). Computed tomography (CT) has proved to be useful for evaluating the brainstem in patients with neurogenic dysphagia. Nonetheless, artifacts are common in CT imaging of the posterior fossa. Also, direct sagittal imaging is not usually obtainable by CT in adult patients.

Magnetic resonance imaging (MRI), in contrast to CT, simultaneously gathers sequential images in the same plane and can obtain direct reconstructions in any plane of interest. MRI has proven to be more sensitive than CT in demonstrating lesions of the brain, such as demyelinating (e.g., multiple sclerosis) and ischemic diseases, (Brant-Zawadzki et al. 1984, Bradley et al. 1984, Bydder et al. 1982, Sheldon et al. 1985) as well as neoplastic masses that may produce neurogenic dysphagia (Lee et al. 1985, Zimmerman et al. 1986).

Five patients with dysphagia are reported for whom MRI was valuable in detecting and characterizing their lesions of the brainstem and the cerebral hemispheres.

Key words: Neurogenic dysphagia – Magnetic resonance imaging – Computed tomography

Case Reports

Case 1

A 12-year-old girl in previously excellent health acutely developed symptoms of brainstem dysfunction shortly after recovery from a febrile, viral-type illness. The symptoms included dizziness, dysphagia, dysarthria, ataxia, diplopia, and right hemiparesis. Her dysphagia was characterized by coughing and choking episodes produced by both solid and liquid foods. Aspiration pneumonia developed before cessation of oral intake, and nasogastric tube feeding was instituted.

On neurologic examination, conjugate left lateral gaze was impaired, and there were rotatory nystagmus, right palatopharyngeal weakness, left tongue weakness, dysarthria, dysphagia, and mild right body weakness and incoordination. Blood tests, including a "vasculitis screen," were normal, except for an elevated erythrocyte sedimentation rate of 55 mm/h and a positive ANA at an initial titer of 1:320 and subsequent titer of 1:40. PPD was negative. Serial cerebrospinal fluid studies, including oligoclonal bands, cytopathology, and VDRL, were also normal, except for a slightly elevated total protein level. Visual and auditory evoked responses were also normal.

A CT scan suggested left sphenoid sinusitis, but sinus exploration demonstrated only polypoid changes. The brain appeared normal (Fig. 1a). Cinepharyngoesophagography revealed severe diffuse pharyngeal paresis with consequent laryngeal penetration and aspiration of the contrast material retained in the pharyngeal recesses.

MRI showed a hyperintense signal in the medulla and sphenoid sinus on the T2 weighted images (T2WI) (Figs. 1b, 1c); the T1 weighted images (T1WI) were unremarkable (Fig. 1d).

The patient was treated with dexamethasone for 3 weeks during which she improved. Follow-up cine pharyngoesophagography showed complete resolution of the pharyngeal paresis. Oral feeding was resumed without complication. MRI was repeated 10 weeks after the initial scan and showed a slight decrease in the size of the brainstem lesion (Fig. 1e). The final diagnosis was postinfectious brainstem encephalitis. The patient has been well for 4 months of follow-up.

Case 2

A 50-year-old woman was admitted for the evaluation of dysphagia after uncovering a hypodense "cystic mass" of the lower brainstem on CT. Ten years ago, the patient developed acute dysphagia that lasted several weeks and then resolved spontaneously. No cause was found then for her swallowing difficulty. Following this recrudescence of her dysphagia, dysarthria developed the day after. Neurologic examination at that time revealed bilateral 12th nerve palsies. MRI elegantly demonstrated an olive-shaped mass with signal characteristics of a fluid dissimilar in signal to cerebrospinal fluid. The mass com-

Address reprint requests to: Martin W. Donner, M.D., Department of Radiology, Johns Hopkins Hospital, 600 N. Wolfe Street, Baltimore, MD 21205, USA

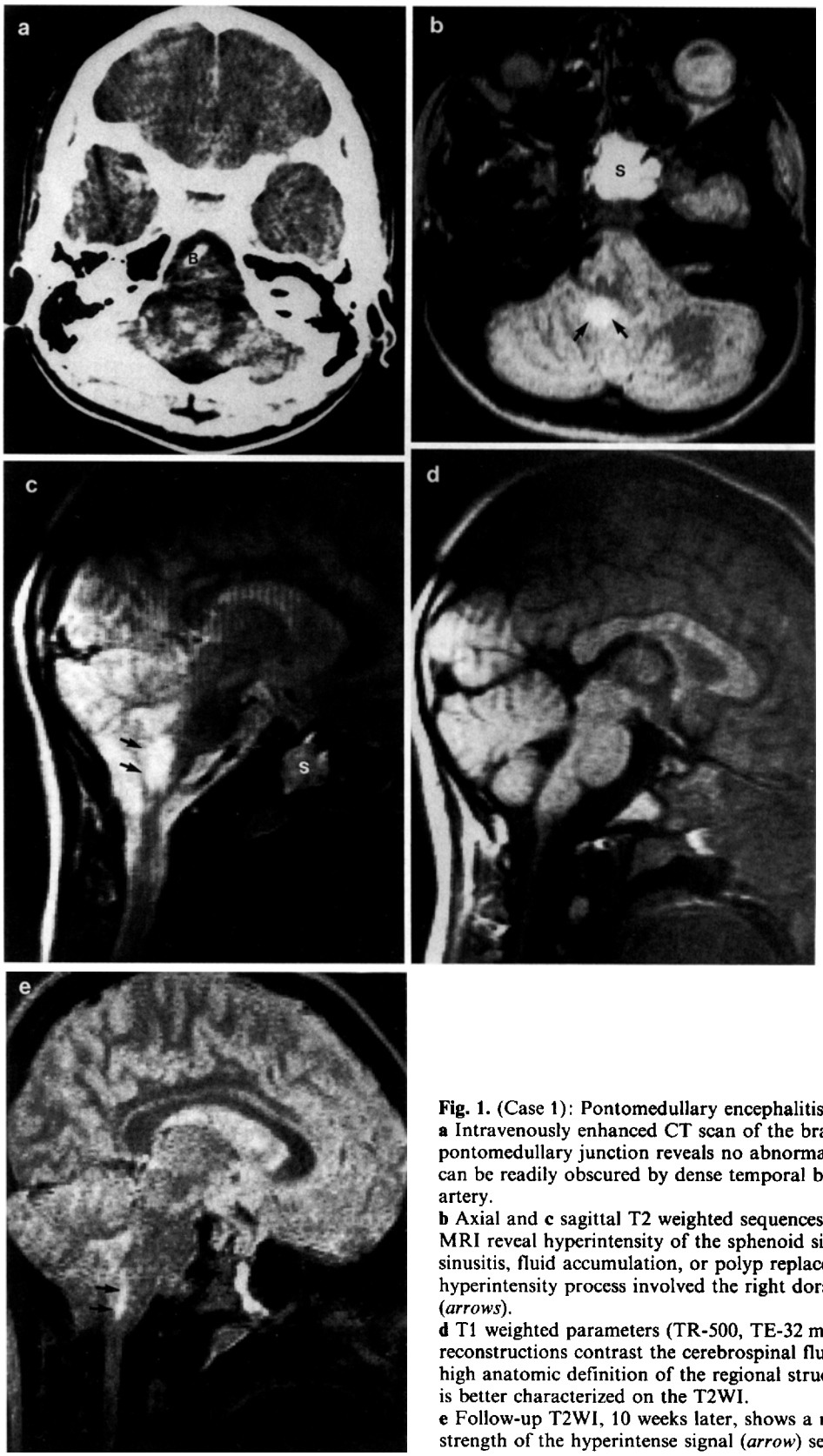

Fig. 1. (Case 1): Pontomedullary encephalitis:
a Intravenously enhanced CT scan of the brain at the level of the pontomedullary junction reveals no abnormality. However, subtle lesions can be readily obscured by dense temporal bone artifacts; *B* = basilar artery.
b Axial and **c** sagittal T2 weighted sequences (TR-2500, TE-64 msec) on MRI reveal hyperintensity of the sphenoid sinus (*S*) consistent with sinusitis, fluid accumulation, or polyp replacement. In the medulla, a hyperintensity process involved the right dorsal aspect of the medulla (*arrows*).
d T1 weighted parameters (TR-500, TE-32 msec) using sagittal reconstructions contrast the cerebrospinal fluid better, thereby providing high anatomic definition of the regional structures, but the lesion per se is better characterized on the T2WI.
e Follow-up T2WI, 10 weeks later, shows a reduction in the size and strength of the hyperintense signal (*arrow*) seen in **c**.

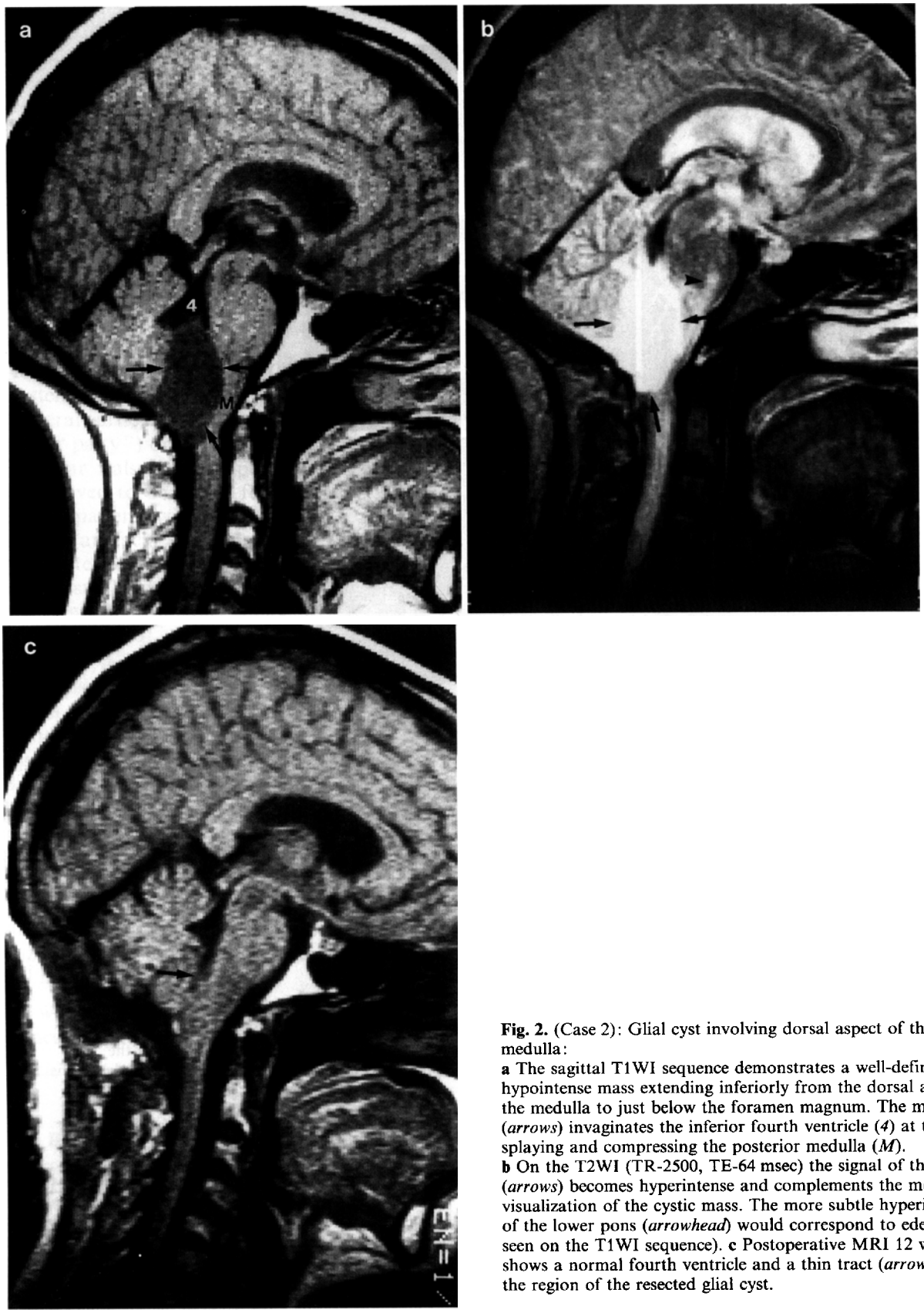

Fig. 2. (Case 2): Glial cyst involving dorsal aspect of the medulla:
a The sagittal T1WI sequence demonstrates a well-defined hypointense mass extending inferiorly from the dorsal aspect of the medulla to just below the foramen magnum. The mass (*arrows*) invaginates the inferior fourth ventricle (*4*) at the obex splaying and compressing the posterior medulla (*M*).
b On the T2WI (TR-2500, TE-64 msec) the signal of the mass (*arrows*) becomes hyperintense and complements the morphologic visualization of the cystic mass. The more subtle hyperintensity of the lower pons (*arrowhead*) would correspond to edema (not seen on the T1WI sequence). **c** Postoperative MRI 12 weeks later shows a normal fourth ventricle and a thin tract (*arrow*) defining the region of the resected glial cyst.

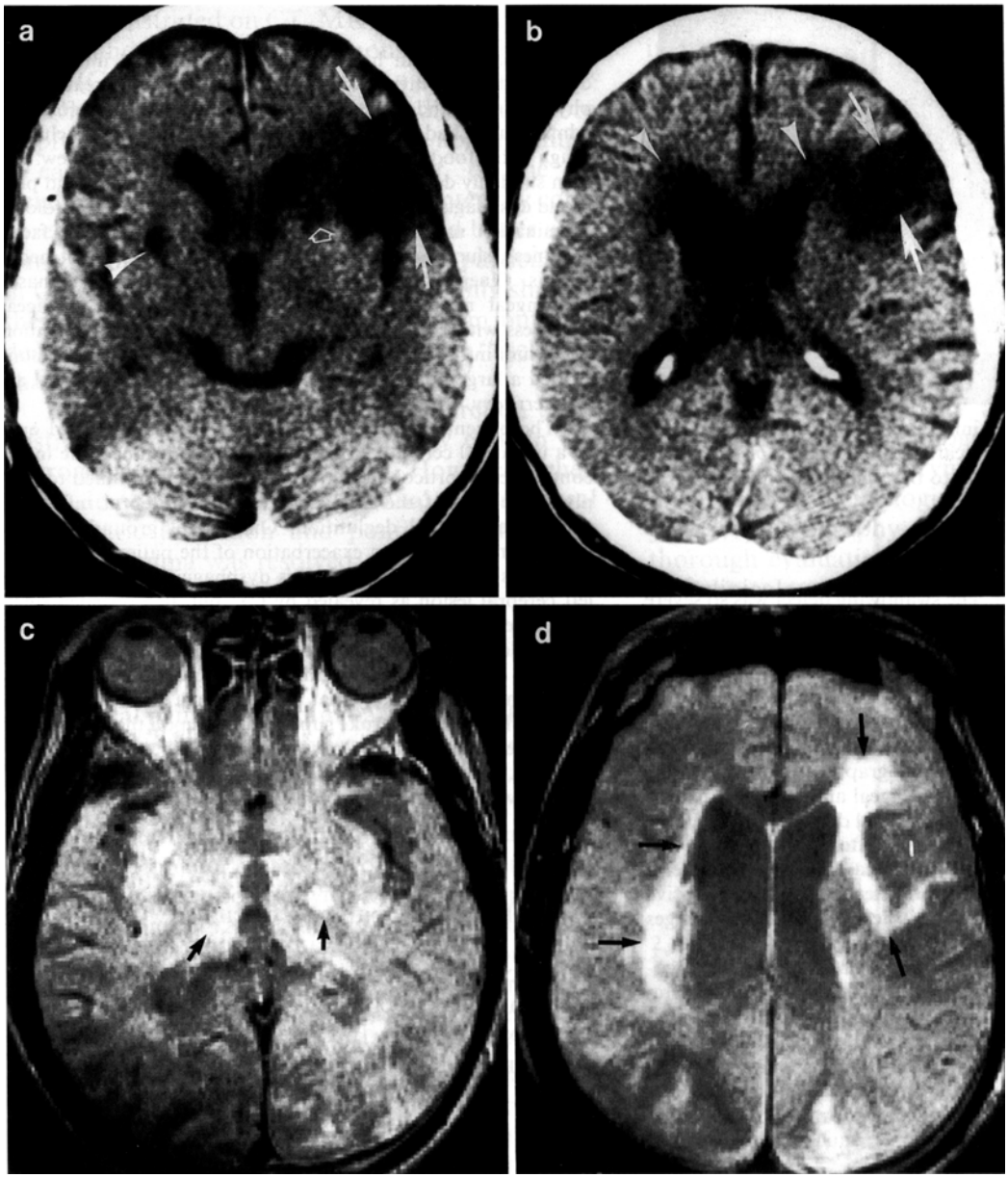

Fig. 3. (Case 3): Basal ganglia and hemispheric infarctions:
a Section at the level of the third ventricle and inferior frontal horns shows basal ganglia infarcts involving the left caudate nucleus (*open arrow*), right putamen (*arrowhead*), and the left frontoparietal lobes (*solid arrows*).
b Section at high lateral ventricular level shows that bilateral paraventricular white matter hypodensities (*arrowheads*) surround the frontal horns consistent with infarction, edema, or demyelination. Arrows point to large frontoparietal lobe infarct.
c The T2WI axial MR slice that still includes the third ventricle (different scanning angle) shows discrete thalamic lesions (*arrows*) better than CT.
d This MR slice corresponding to the CT (Fig. **b**) shows bilateral peri- and paraventricular high signal intensity (*arrowheads*) consistent with infarction. MRI shows broader zone of white matter involvement than CT. A portion of the peripheral zone of hypodense frontoparietal lobe infarct on CT (Fig. **b**) becomes isointense (*i*) on MRI with the adjacent brain due to a different nature of the infarct of this region (liquifactive necrosis).

pressed the medulla and lay immediately inferior to the fourth ventricle that it abutted (Figs. 2a, 2b). At surgery, a yellowish cystic mass was resected. Histopathologic examination demonstrated that glial cells lined the cyst, and there was no evidence of neoplasm.

The patient's symptoms of dysphagia and dysarthria resolved in the immediate postoperative period. An MRI performed 12 weeks after operation showed only a residual thin space to indicate where the cyst had been (Fig. 2c).

Case 3

A 75-year-old man with hypertension and two prior episodes of transient left hemiparesis reported the acute onset of dyspha-

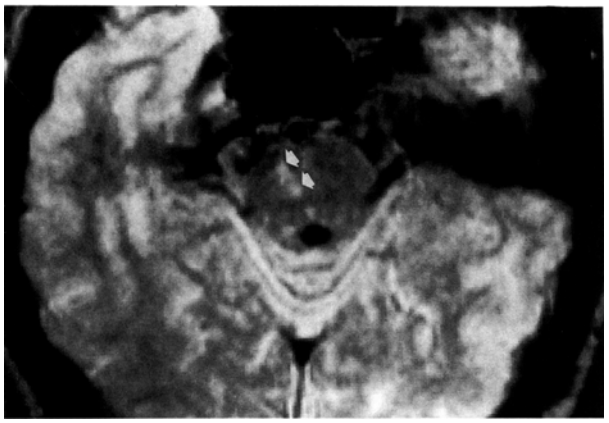

Fig. 4. (Case 4): Brainstem infarction: Pontine lesions, infarcts on MRI, are represented by scattered high signal (*bright*) intensities (*arrows*) centered largely to the right of the midline.

gia, dysarthria, and ataxia. The swallowing defect was so severe that a small sip of water caused prolonged laryngospasm. The neurologic examination showed mild facial weakness, moderate dysarthria, right palatopharyngeal weakness, left hyperreflexia with a left Babinski sign, and moderate gait ataxia. The noncontrast head CT scan revealed bilateral basal ganglia and left frontotemporoparietal hypodensities consistent with infarction (Figs. 3a, 3b). Cinepharyngoesphagography demonstrated diffuse involvement, with severe pharyngeal and laryngeal paresis resulting in reflux of the contrast material during every swallow. Thus, nasogastric tube feeding was initiated.

MRI scanning confirmed the CT scan findings of bilateral hemispheral infarctions, but more clearly showed extensive periventricular abnormalities on the T2 weighted images (Figs. 3c, 3d). No brainstem infarction was seen.

The patient's dysphagia is attributable to bilateral corticobulbar tract disruption ("pseudobulbar palsy") from infarction, rather than to a brainstem origin, as initially surmised clinically. The patient underwent swallowing rehabilitation guided by videofluoroscopy. Within 6 weeks he was able to feed himself safely by mouth.

Case 4

A 77-year-old hypertensive woman presented with the sudden onset of dysphagia and dysarthria. She had previous transient unilateral neurologic deficits that involved both sides of the body, but at different times. Her neurologic examination on admission showed only mild facial paresis, hyperactive gag reflex, and "wet", mildly slurred speech. Intravenously enhanced CT scanning revealed no definite abnormality. Cinepharyngoesophagography demonstrated difficulty in initiating swallowing caused by moderate diffuse pharyngeal and laryngeal weakness; laryngeal penetration of the contrast material followed. Nasogastric tube feeding was then initiated.

MRI scanning showed bilateral pontine (Fig. 4) and subcortical infarctions. These lesions were thought to account for the patient's dysphagia; however, the relative contributions from bilateral subcortical (corticobulbar tract) and brainstem dysfunction could not be precisely differentiated. The patient is presently undergoing videofluoroscopy-guided swallowing rehabilitation.

Case 5

An 81-year-old man had previously experienced intermittent dysphagia due to myasthenia gravis that was being treated with physostigmine and prednisone. For several months before this admission, he had been able to tolerate a normal diet, including tough solid foods that he would carefully cut and chew. He then suddenly developed severe right hemiparesis and had profound dysphagia requiring nasogastric tube feeding. Neurologic examination revealed a right visual field defect, right facial weakness, slurred and hoarse speech, and a dense right hemiparesis. Cinepharyngoesophagography showed marked nasopharyngeal regurgitation, diffuse pharyngeal and laryngeal weakness with consistent reflux into the larynx, and retention of barium in the pharyngeal recesses. A head CT scan demonstrated a large infarction involving the left middle cerebral artery territory.

The patient's severe dysphagia could not be ascribed only to a unilateral cerebral stroke. In that circumstance, the intact contralateral corticobulbar tract would have continued to supply both sides of the brainstem with the cerebral influences needed for normal deglutition. On clinical grounds, it also seemed unlikely that an exacerbation of the patient's myasthenia gravis was responsible for his dysphagia. MRI showed the left cerebral lesion as revealed by CT, but also revealed small right cerebral and pontine infarcts. Although pontine dysfunction could have caused dysphagia in this case, the concomitant onset of dysphagia, right hemianopsia, and right hemiparesis made it much more likely that the dysphagia had resulted from an acute left cerebral stroke superimposed upon the pre-existing right cerebral infarction. Thus, bilateral corticobulbar tract impairment was induced. The patient was referred for rehabilitation of swallowing and for right-sided motor dysfunction.

Results

Two of the five patients discussed, Cases 1 and 4, had normal CT examinations and abnormal MRI scans (Table 1). Case 1, a patient with brainstem encephalitis clinically, had a grossly abnormal MRI examination. In Case 4, the numerous brainstem and subcortical infarcts were detected only on MRI. Of the three cases in which abnormalities

Table 1. Pertinent findings of the brain

	Computed tomography	Magnetic resonance imaging
Case 1	Normal	Lesion in pons and medulla
Case 2	Cystic mass adjacent to fourth ventricle	Sagittal views show cystic mass elevating and compressing fourth ventricle
Case 3	Bilateral lacunar and periventricular infarctions; normal brainstem	Bilateral infarctions, greater degree of involvement than seen on CT; normal brainstem
Case 4	Normal	Bilateral subcortical infarction and pontine infarction
Case 5	Left cerebral infarction	Left and right cerebral infarction; pontine infarction

were demonstrated on CT, MR detected additional lesions and more clearly demonstrated the mass lesion (Case 2) and the surrounding structures.

The T2 weighted images were distinctly more helpful in the MRI detection of the abnormalities in each of the five cases. However, on the T1 weighted images, cerebrospinal fluid intensity compared to that of the brainstem afforded better anatomic morphologic characterization, particularly in Cases 1 and 2. In Case 3, MRI not only showed the normal appearance of the brainstem but also demonstrated additional periventricular "ischemic changes" not shown by CT.

In Case 5 the single unilateral infarction demonstrated by CT could not clinically explain the neurogenic basis for dysphagia. However, when MRI demonstrated the additional contralateral hemispheric infarction and pontine infarcts, the clinical dilemma was resolved.

Discussion

The clinical neurologic examination competes only with the clinical cardiac examination for accuracy and reliability in the practice of medicine. Nonetheless, concerning neurologic accuracy in the era of angiography, the famed Boston neurosurgeon, Dr. James Poppen, stated on rounds that "the best neurologist is the person with the angiographic syringe (the angiographer)." CT revolutionized the neuroradiologic armamentarium when it provided the first in vivo noninvasive visualization of the brain. However, posterior fossa artifacts generated from the dense temporal bones preclude excellent visualization of the brainstem even on the most advanced CT scanners. CT cisternography (the intrathecal injection of a nonionic contrast medium via lumbar or cervical puncture, with gravity delivery of the contrast medium to the intracranial subarachnoid space followed by CT) can define the contours of the brainstem with high fidelity. However, the intrinsic structure of the brainstem cannot be resolved as it can with medium- and especially high-field strength (1.5 Tesla) MRI. MRI provides detailed visualization of the larger tracts and nuclei of the brain stem (Flannigan et al. 1985). Such detailed information becomes important in explaining the causes for disease of the brainstem, including dysphagia.

The capacity to obtain any projection (reconstruction) of the brain by MRI has proved to be an important asset of the technique. The option of obtaining direct sagittal views of the posterior fossa by MRI cannot be duplicated by CT. Case 2 (Fig. 2) illustrates the relationships in the sagittal plane of the posterior fossa glial cyst to the fourth ventricle. The surgical planning and operation are aided by such additional perspectives that are not available from CT.

When a patient presents with dysphagia, the causes for consideration are often numerous. Usual causes are physiologic and/or anatomic abnormalities of the pharynx and esophagus. A detailed history, neurologic examination, and pharyngoesophagography are usually required in the thorough evaluation of dysphagia. When there is the clinical suspicion of neurologic dysfunction, CT or MRI is also indicated. Because MRI is superior to CT in sensitivity and contrast discrimination, it is recommended that MRI be selected as the imaging procedure of choice for neurogenic dysphagia.

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