Magnetic Resonance Imaging in Wallenberg's Lateral Medullary Syndrome

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SUMMARY Four patients with a clinical diagnosis of Wallenberg's lateral medullary syndrome were studied with both Magnetic Resonance Imaging (MRI) and cranial Computed Tomography (CT). Using transverse images and both T1 and T2—weighted sequences, MRI demonstrated a medullary infarction not seen on CT in all four cases. MRI also demonstrated a coexisting cerebellar infarction in three cases which was unsuspected clinically and undetected by CT.

MAGNETIC RESONANCE IMAGING (MRI) is a sensitive diagnostic technique for evaluating the brain and spinal cord.1-10 Indications for neuroimaging with MRI are currently being defined. One clinical setting in which MRI is likely to be useful is in the evaluation of brain stem infarction.11-13 The present study was undertaken to assess the diagnostic efficacy of MRI in patients with Wallenberg's lateral medullary syndrome and to compare MRI with CT.

Subjects and Methods

From September 1984 through February of 1985, four hospitalized patients with Wallenberg's lateral medullary syndrome were evaluated at the University of Iowa Hospitals. These were the only patients with this syndrome seen during that period. Three patients (cases 1, 2, 3) underwent CT and MRI within two weeks of onset of illness. CT scans were performed on a Picker International 600 or 1200 scanner (cases 1, 3, 4) and a Technicare 2060 scanner (case 2). MRI was performed with a .5 Tesla superconducting Picker Vista MR imaging system. Both T1-weighted and T2-weighted multislice images were obtained in the transverse plane in each case. T1-weighted images were performed with either 1) an inversion recovery pulse sequence using an inversion time (TI) of 600 msec and a repetition time (TR) of 2050 msec or 2) a spin-echo pulse sequence using an echo time (TE) of 40 msec and a TR of 2 seconds or greater. The criteria used to diagnose infarction were the presence of a focus of abnormally decreased signal intensity on T1-weighted images and a corresponding area of increased signal intensity on T2-weighted images.

Case Reports

Case 1

A 59-year-old woman developed sudden onset of right-sided ataxia followed by nausea, vomiting, dysphagia and hoarseness. She had a right Horner's syndrome, mild skew deviation with a hypertropic right eye, and torsional nystagmus on primary gaze. Lateral pulsion of the eyes to the right was present. She had a right palatal paralysis and speech was dysarthric. The right corneal response was absent and there was right facial hypalgesia and thermoanesthesia. Left trunk and extremity hypalgesia and thermoanesthesia were present. She had right arm dysmetria and an ataxic gait. CT obtained 5 days after onset was negative. MRI done 48 hours later, demonstrated a right lateral medullary lesion consistent with infarction (fig. 1).

Case 2

A 50-year-old hypertensive man awoke with painful numbness of the right face, nausea, vomiting and dizziness. On arising he had an ataxic gait. He also noted dysphagia and transient diplopia. Examination revealed a right Horner's syndrome and horizontal nystagmus to the right. He had hypometric saccades when looking from left to center and hypermetric saccades with overshoot upon looking from right to center. He had left facial hypalgesia and thermoanesthesia. There was limb ataxia on the left. The gait was ataxic with a tendency to fall to the left. MRI fourteen days after onset, showed lesions in the left medulla and left cerebellum consistent with infarction (fig. 2). Arteriography demonstrated occlusion of the left vertebral artery.

Case 3

A 50-year-old hypertensive man awoke with painful numbness of the right face, nausea, vomiting and dizziness. On arising he had an ataxic gait. He also noted dysphagia and transient diplopia. Examination revealed a right Horner's syndrome and horizontal nystagmus to the right. He had hypometric saccades upon looking from right to center and hypermetric saccades with overshoot upon looking from left to center. There was facial hypalgesia and thermoanesthesia on the right and a decreased right corneal response. He had right vocal cord and palatal paralysis. Sensory examination revealed hypalgesia and thermoanesthesia of the left hemibody up to C2 level. He had hypotonia and ataxia of the right limbs. The gait was ataxic with veering to the right. CT obtained on day four was negative. MRI on day nine, demonstrated infarctions...
FIGURE 1. Transverse MR images through the lower medulla and cerebellum demonstrate abnormal signal intensity in the right lateral medulla compatible with infarction (arrows). A. The lesion is dark with the T1-weighted inversion recovery sequence (TI/TR = 600/2000). B. A corresponding area of increased signal intensity in the medulla is seen at a slightly higher level with the T2-weighted spin echo sequence (TE/TR = 80/2300).

FIGURE 2. Transverse images at two different levels through the lower medulla and cerebellum show cerebellar and medullary infarction. A. Abnormal areas of decreased signal intensity (arrows) are noted in the left medulla and cerebellar hemisphere with the T1-weighted spin-echo sequence (TE/TR = 40/550). B. At a slightly higher level the cerebellar infarction is better demonstrated as a region of increased signal intensity (arrow) posteriorly in the left hemisphere with the T2-weighted sequence. (TE/TR = 80/2000).

in the right dorsolateral medulla and right cerebellar hemisphere (fig. 3).

Case 4

A 64-year-old woman with a history of hypertension, type IV hyperlipidemia and glucose intolerance, had several previous cerebral infarctions (left parietal, left internal capsular and putaminal, right internal capsular) and had recovered with a stable deficit of mild right hemiplegia. Two weeks before admission she developed nausea, vomiting, vertigo, dysarthria, dysphagia and ataxia. Examination revealed mild right hemiparesis, right central VIIth nerve paresis and right-sided hyperreflexia, all consistent with previous infarctions. Her new deficits were a right Horner's syndrome, horizontal nystagmus on right lateral gaze, severe dysarthria, a diminished gag on the right, diminished right corneal response, and ataxia of the right leg and gait. Facial sensation was normal. CT at two and six weeks of onset showed previous cerebral infarctions and no abnormality in the posterior fossa. MRI obtained six days after the second CT demonstrated infarction in the right lateral medulla and right cerebellar hemisphere (fig. 4) as well as the areas of infarction seen on CT.

Discussion

Wallenberg's syndrome results from infarction of the dorsolateral medulla. The clinical features are well described.14-16 Most patients with the lateral medullary syndrome have a good outcome. However, death has been reported secondary to cardiac or respiratory failure.17 Also, those who have a coexistent large cerebellar infarction may die from brain stem compression and acute hydrocephalus.18, 19 Early clinical detection of such patients may not be possible because the lateral medullary syndrome appears much the same whether or not there is coexisting cerebellar infarction.20 MRI
FIGURE 3. Transverse images through the medulla and cerebellum show infarction involving the majority of the right medulla (arrows) and cerebellar hemisphere. There is mild mass effect and midline shift from associated edema. A. The lesion is dark with the T1-weighted inversion recovery sequence (TI/TR = 600/2050). B. The same area appears bright with the T2-weighted spin-echo sequence (TE/TR = 120/2900).

allows early visualization of infarctions7, 13 and permits early intervention, should deterioration occur in those patients with coexisting large cerebellar infarctions.

Our four patients had the clinical findings consistent with Wallenberg's syndrome. In all cases MRI demonstrated a medullary infarction (figs. 1–4). In three cases clinically unsuspected cerebellar infarctions were also demonstrated. In contrast to MRI, CT failed to detect either the brain stem or cerebellar infarction in any of these cases.

MRI depends upon both tissue properties and the order and timing of applied radiofrequency pulses. The sensitivity of MRI in differentiating infarction or other lesions from normal tissue depends primarily on changes in tissue T1 and T2 relaxation times which are related to tissue water content. Infarction without hemorrhage lengthens T1 and T2 beyond normal, approaching the values for fluids. Therefore, regions of infarction typically have a signal intensity more like CSF than surrounding brain regardless of the pulse sequence used. The various pulse sequences can be grouped into those which are T1-weighted (i.e., the appearance depends primarily on the T1 of tissue), T2-weighted and intermediate. T1-weighted images show excellent anatomical detail and contrast between gray and white matter. T1-weighted images show CSF as black, gray matter as gray and white matter as white. Infarctions are darker than surrounding gray or white matter (figs. 1–4A). T2-weighted images are essentially the reverse. Anatomical detail and gray-white contrast are not seen as well but T2-weighted images are extremely sensitive to pathologic processes. On T2-weighted images CSF appears white or light gray and pathologic tissues stand out as bright against the gray of normal brain (figs. 1–4B).

We believe the brain stem is best evaluated with transverse images using both T1 and T2-weighted pulse sequences. With our system (Picker International .5 Tesla magnetic field strength) the best T1-weighted images are obtained with inversion recovery (IR) sequences. T1-weighted images are obtained using a spin-echo sequence with a long repeat time and echo

FIGURE 4. Infarction is demonstrated in a small area of the dorsolateral medulla (arrows) and a larger wedge of the right cerebellar hemisphere. A. The lesion is dark with the T1-weighted inversion recovery sequence (TI/TR = 600/2050). B. The same area appears bright with the T2-weighted spin-echo sequence (TE/TR = 120/2900).
delay. Although our lesions were larger, we believe it will be possible to detect infarctions as small as 2 mm in size in the lateral medulla using standard techniques.

While the lateral medullary syndrome remains a clinical diagnosis based upon a characteristic history and constellation of physical findings, MRI offers improvement in visualization of the medullary infarction. In addition, MRI can demonstrate coexisting cerebellar infarctions which may have previously been clinically unsuspected and undetected by CT. Visualization of such infarctions by MRI can alert the clinician to the potential for serious complications associated with large cerebellar lesions.

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References

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