
Brain Death: MR and MR Angiography

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Summary: Four patients in whom brain death was identified on the basis of neurologic and electroencephalographic findings were examined with MR imaging and MR angiography. MR images showed diffuse swelling of the cerebral gyri and cerebellar cortex, with prolongation of both the T1 and T2 signal (representing hypoxic ischemic brain injury), downward displacement of the diencephalon and the brain stem (central and tonsillar herniation), and loss of flow void in the intracranial portions of both internal carotid arteries. MR angiograms did not show the intracranial vessels above the level of the supraclinoid portion of the internal carotid arteries. MR angiography and MR imaging are noninvasive and reliable methods for use in determining brain death.

Index terms: Brain, death; Brain, magnetic resonance; Magnetic resonance angiography; Forensic medicine

Brain death is the state of irreversible cessation of all cerebral and brain stem functions resulting from edema and massive destruction of brain tissue despite continued cardiopulmonary activity maintained by advanced life-support systems and mechanical ventilation (1, 2). The increasing refinement and implementation of organ transplantation procedures requires an unequivocal diagnosis of brain death for legal and moral reasons (3–17). The method of diagnosing brain death should be free of error, easily and quickly performed, and verifiable. We reviewed the magnetic resonance (MR) imaging and MR angiographic findings in four patients in whom brain death had been diagnosed and evaluated the diagnostic role of these imaging techniques.

Materials and Methods

Among the four patients whose findings we studied, the cause of brain death was head injury in two, cerebral hypoxia resulting from asthma in one, and subarachnoid hemorrhage in one. The diagnosis of brain death was made on the basis of neurologic and electroencephalographic (EEG) findings.

The brains of all patients had been examined with single-photon emission computed tomography (SPECT) using 1.11 GBq of ^{99m}Tc hexamethyl-propyleneamine oxime (HMPAO) immediately before or after MR imaging. MR imaging and MR angiography were performed with a 1.0-T MR system (Magnetom Impact; Siemens, Erlangen, Germany) while the patients were ventilated by means of a Monahan 225/SIMV volume ventilator combined with the MR unit (Monahan). Electrocardiographic (ECG) monitoring was also used. Axial T1-weighted and T2-weighted and coronal or sagittal T1-weighted or T2-weighted images were obtained. For T1-weighted images, a spin-echo technique was used with parameters of 500/15/1 (repetition time/echo time/excitations). For T2-weighted images, a fast spin-echo technique was used with parameters of 3700–4000/85–90/1, a 5-mm section thickness, a 2.5-mm intersection gap, a 21- to 23-cm field of view, and a 256 × 256 matrix.

MR angiograms were obtained with three dimensional time-of-flight (TOF) fast imaging with steady-state precession sequences (45–55/10/1), a 20° to 25° flip angle, a 14 × 14-cm field of view, and a 64-mm section thickness. Magnetization transfer saturation was used to suppress the signal from the brain parenchyma and a tilted optimized nonsaturating excitation was used to enhance the in-flow effect of the arteries.

Results

The clinical data and the MR imaging and MR angiographic findings of the four patients are given in the Table. On neurologic examination, no patient had a cephalic or brain stem response and none had evidence of spontaneous respiration. All patients had flat EEGs and died within 4 days after the MR examination. Brain SPECT showed no uptake of radioactivity in the cranial cavity (the so-called hollow skull) in any of the four patients, which suggested brain death (Figs 1 and 2) (8). Common MR findings included diffuse brain swelling, especially gyral swelling, associated with diffuse prolongation of T1 and T2 signal in the cerebral gyri and cere-

Received June 7, 1995; accepted after revision September 8.

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AJNR 17:731–735, Apr 1996 0195-6108/96/1704–0731 © American Society of Neuroradiology

MR, MR angiographic, and SPECT findings in four patients in whom brain death was diagnosed

| Case | Age, y/Sex | Clinical Diagnosis | MR Findings | MR Angiography | SPECT |
|------|------------|--------------------------------|---|----------------|-------|
| 1 | 30/F | Cerebral hypoxia due to asthma | Diffuse gyral swelling, central and tonsillar herniation, loss of ICA flow void | NV | NIR |
| 2 | 45/M | Head injury | Diffuse gyral swelling, central and tonsillar herniation, contusion of bilateral frontal lobes, loss of ICA flow void | NV | NIR |
| 3 | 23/F | Head injury | Diffuse gyral swelling, central and tonsillar herniation, multiple cerebral white matter injury, loss of flow void in the mid ICA | NV | NIR |
| 4 | 50/M | Subarachnoid hemorrhage | Diffuse gyral swelling, intraventricular hematoma, central and tonsillar herniation, loss of ICA flow void | NV | NIR |

Note.—SPECT indicates single-photon emission computed tomography; ICA, internal carotid artery; NV, no visibility of the ICA and its branches above the level of the supraclinoid portion; and NIR, no intracranial radioactivity.

bellar cortex, representing hypoxic/ischemic brain damage; downward displacement of the diencephalon and brain stem (central and tonsillar herniation); and loss of flow void in the intracranial portions of both internal carotid arteries (ICAs) (Figs 1 and 2). In all four cases, MR angiograms showed no intracranial vessels above the level of the supraclinoid portion of the ICA (Figs 1 and 2). However, the cervical and petrous portions of the ICA were seen in three cases and the cervical portions of the vertebral arteries were depicted in one case.

Discussion

The diagnosis of brain death can usually be made by physical examination to detect coma, the absence of cephalic reflexes, and the absence of spontaneous respiration, and by a flat EEG (1, 2). Established clinical criteria usually suffice, but even objective tests such as the EEG may be inaccurate in comatose patients owing to drug-induced hypothermia, intoxication, and shock. Conventionally, serial EEGs and radionuclide perfusion studies, either planar imaging or SPECT, have been used to confirm the diagnosis of brain death in an appropriate clinical setting (4–13). The absence of cerebral blood flow is generally accepted as a definite sign of brain death (3–17). The reason for the cessation of cerebral blood flow after brain death is uncertain. Although an intracranial pressure higher than the mean arterial pressure is not the sole explanation for the absence

of cerebral perfusion, such an increase of intracranial pressure resulting from diffuse brain edema is thought to be the primary cause of the cessation of blood flow (5).

Cerebral vascular studies, such as contrast angiography, radionuclide cerebral angiography, and SPECT, have been used to confirm and document the clinical diagnosis of brain death (3–13). Recently, brain SPECT with ^{99m}Tc HMPAO has commonly been used for this purpose (4–13). Several authors have documented the MR findings in patients with brain death (15–18), including Orrison et al (17), who identified six signs: (a) transtentorial and foramen magnum herniation, (b) absence of the intracranial vascular flow void, (c) poor gray matter/white matter differentiation, (d) no intracranial enhancement, (e) carotid artery enhancement (the intravascular enhancement sign), and (f) prominent nasal and scalp enhancement (the “hot nose” sign). Although we did not perform contrast-enhanced MR imaging, we also noted the first three signs in our patients. However, these three findings alone should not be considered indicative of brain death, since transtentorial and foramen magnum herniation as well as poor gray matter/white matter differentiation may also be observed in patients with severe hypoxic brain damage in the absence of brain death.

The MR angiographic features of brain death have been documented in a report by Aichner et al (16). TOF MR angiography relies on the flow of fully magnetized spins into the imaging section or volume to differentiate vascular struc-

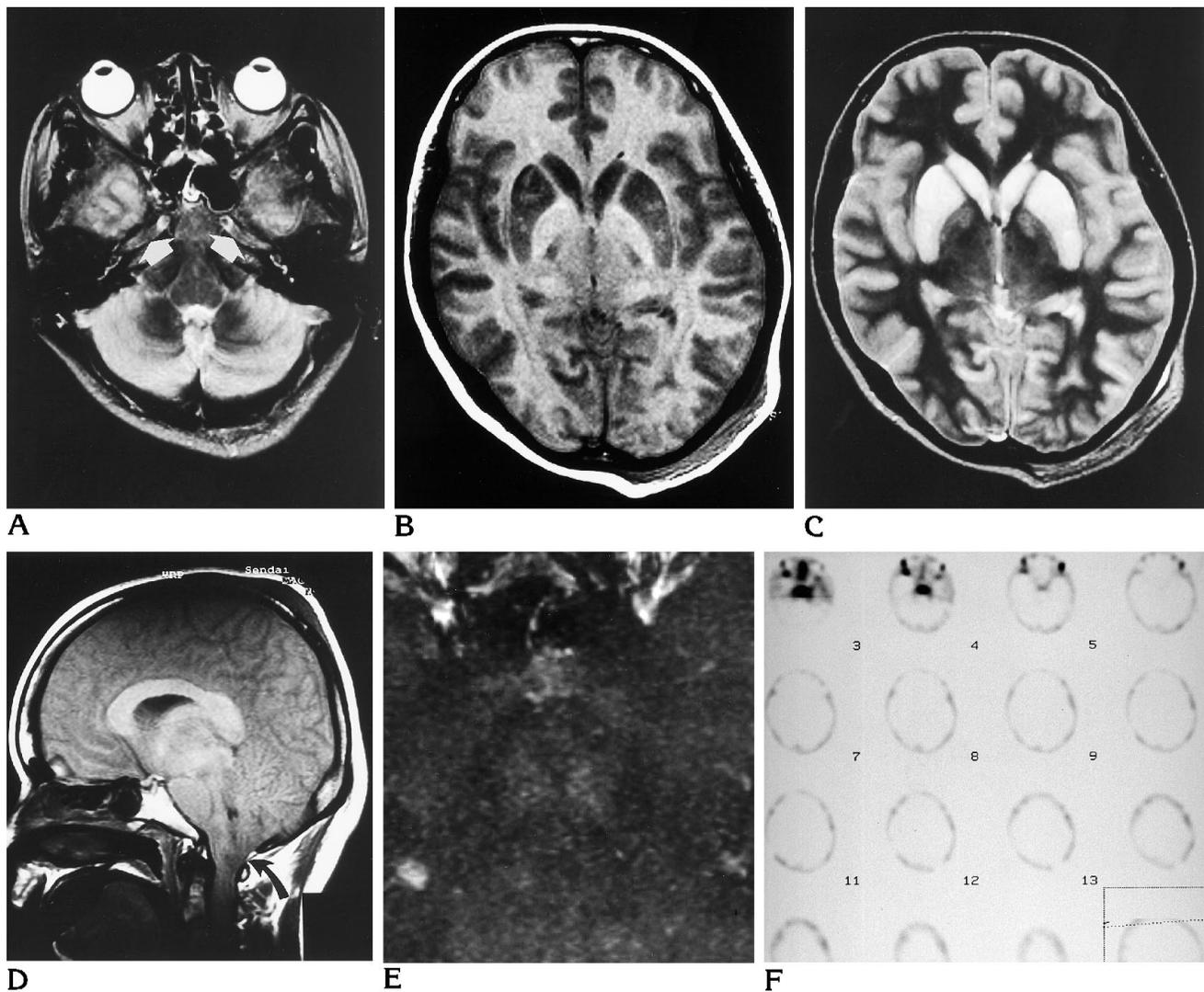


Fig 1. Case 1.

A, Axial T2-weighted MR image (3700/90) shows lack of flow void in the petrous portion of both internal carotid arteries (*arrows*). Both cerebellar hemispheres and the vermis appear hyperintense.

B, Axial T1-weighted MR image (500/15) shows hypointensity of the putamina and cerebral gyri along with effacement of the cerebral sulci, representing hypoxic brain damage.

C, Axial T2-weighted MR image (3700/90) at the same level as in B shows hyperintensity of the putamina and cerebral gyri.

D, Midsagittal T1-weighted MR image (500/15) shows downward displacement of the medulla oblongata and cerebellar tonsils through the foramen magnum (tonsillar herniation, *arrow*) as well as effacement of the cerebral sulci and cerebellar fissures.

E, MR angiogram (basal view) shows no intracranial arteries.

F, Brain SPECT with ^{99m}Tc HMPAO shows no intracranial radioactivity ("hollow skull"), a finding consistent with brain death.

tures from stationary tissue. The advantages of 3-D TOF MR angiography include high spatial resolution, relatively short imaging time, short echo time, and high signal-to-noise ratio and contrast-to-noise ratio (Turski P, "Magnetic Resonance Angiography: Central Nervous System Applications," In: *Syllabus: Special Course in Neuroradiology*, Oak Brook, Ill: RSNA 1994; 31-46). Our results suggest that absence of the

ICA above the supraclinoid portions of these vessels and absence of their intracranial branches on MR angiograms is specific to brain death, since this absence was noted exclusively in patients with brain death. Although 3-D TOF MR angiography has a reduced sensitivity to slow blood flow, this technique still seems to provide reliable information for the diagnosis of brain death.

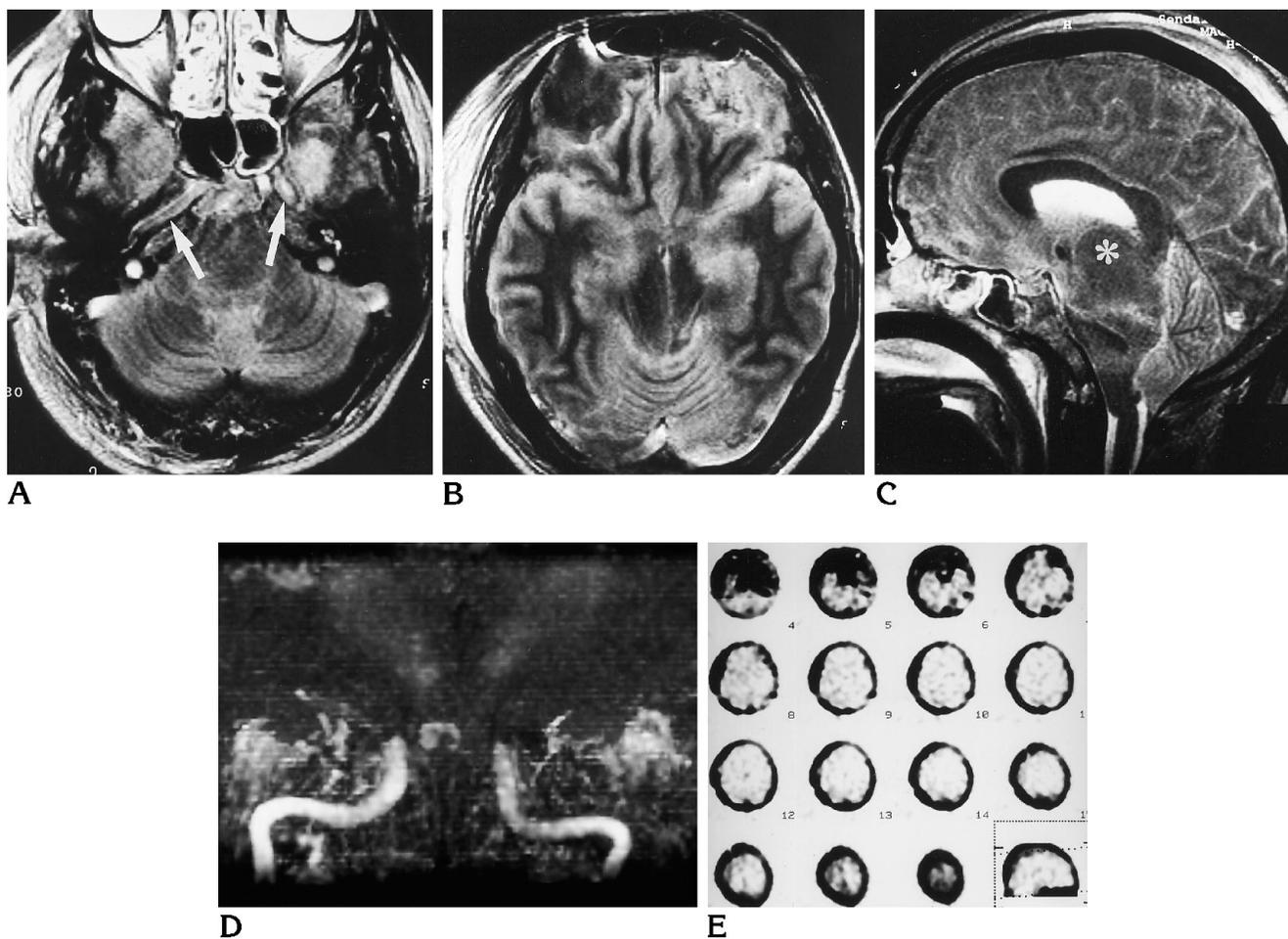


Fig 2. Case 2.

A, Axial T2-weighted MR image (3700/90) through the posterior fossa shows lack of flow void in both internal carotid arteries (arrows), hyperintensity of the cerebellum, a tight posterior fossa, and contusion of the left temporal lobe.

B, Axial T2-weighted MR image (3700/90) through the midbrain shows diffuse hyperintensity and swelling of the cerebral gyri along with marked compression and distortion of the midbrain, damage to the parahippocampal gyri, and contusion of the left frontal lobe.

C, Midsagittal T2-weighted MR image (3700/90) reveals downward displacement of the diencephalon (asterisk), brain stem, and cerebellum (central and tonsillar herniation). Contusion of the base of the frontal lobe can also be seen.

D, MR angiogram (frontal view) shows no cerebral vessels above the level of the supraclinoid portion of the internal carotid arteries.

E, Brain SPECT with ^{99m}Tc HMPAO shows a "hollow skull."

In conclusion, loss of flow void in the intracranial ICA, central and tonsillar herniation, and diffuse brain swelling on MR images along with absence of any cerebral vessels above the level of the supraclinoid portions of the ICA on MR angiograms are findings suggestive of brain death. In particular, MR angiography provides a noninvasive and reliable method for the diagnosis of brain death.

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