MR 'hot nose sign' and 'intravascular enhancement sign' in brain death.

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MR 'Hot Nose Sign' and 'Intravascular Enhancement Sign' in Brain Death

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Summary: Three cases of MR with gadopentetate dimeglumine in patients diagnosed with cerebral death are presented. Observation of an MR "hot nose sign" and an "intravascular enhancement sign" provided additional imaging support in the clinical diagnosis of brain death. The MR findings in brain death include: 1) transtentorial and foramen magnum herniation, 2) absent intracranial vascular flow void, 3) poor gray matter/white matter differentiation, 4) no intracranial contrast enhancement, 5) carotid artery enhancement (intravascular enhancement sign), and 6) prominent nasal and scalp enhancement (MR hot nose sign). Additional modalities for confirming brain death are discussed.

Index terms: Brain, death; Brain, magnetic resonance; Cerebral blood flow

The expansion of organ transplantation programs has led to increased interest in the diagnosis of brain death. However, the diagnosis of brain death continues to be problematic and is often confused by methods that use different criteria (1–3). An analysis of cerebral blood flow is often considered necessary confirmation of the clinical diagnosis of brain death and is frequently performed via nuclear medicine studies (2–6). Internal carotid artery occlusion with increased external carotid blood flow and a finding of the "hot nose sign" on a nuclear medicine study support the diagnosis of brain death (6–13). Magnetic resonance (MR) has been shown to demonstrate herniation and absent cerebral vascular flow void in brain death (14). Enhancement of vessels supplying areas of infarcted brain on MR has been reported and referred to as the "intravascular enhancement sign" (15–17). We present three cases of a hot nose sign and intravascular enhancement sign demonstrated by gadolinium-enhanced MR imaging and discuss the use of MR in relation to other tests to confirm brain death.

Materials and Methods

Three cases of brain death were evaluated by MR. The subjects ranged in age from 21 to 59 years; all were men. Imaging was performed on a 0.064-T permanent magnet (MTP Access, Toshiba America MRI, South San Francisco, Calif). MR studies included T1-weighted spin-echo sequences at 2000/30, 45, 105/2–4 (repetition time/echo time/excitations) and gradient-echo sequences at 68/24/3 with flip angle of 60°. Sagittal and axial imaging planes were completed. Examinations included contrast-enhanced axial T1 (gradient echo) sequence (68/24/3) with 60° flip angle using 0.1 mmol/kg gadopentetate dimeglumine as the final portion of the MR study in each case. Patients were evaluated with full ventilatory support using MR-compatible respirators (AutoVent models 2000 and 3000, Life Support Products, Irvine, Calif). The MR examination was performed before the suspected diagnosis of brain death in one case and confirmed by subsequent radionuclide evaluation suggested on the basis of the MR findings. MR was performed in two subsequent cases after the diagnosis of brain death.

Case Reports

Case 1

A 21-year-old man was admitted after a self-inflicted gunshot wound to the head. He was unresponsive with decerebrate posturing, bradycardia, and a Glasgow coma score of 3. His pupils were fixed and dilated. Computed tomography findings included diffuse cerebral swelling, subarachnoid hemorrhage, and hemorrhage along the path of the bullet from right occiput to left parietal region. MR showed transtentorial and foramen magnum herniation, no intracranial vascular flow void, poor gray matter/white matter differentiation, small subdural hematoma on the left, bullet fragments, and no intracranial enhancement after the administration of gadopentetate dimeglumine. There was prominent enhancement of the nasal region and carotid arteries (Fig 1). A subsequent technetium-99m

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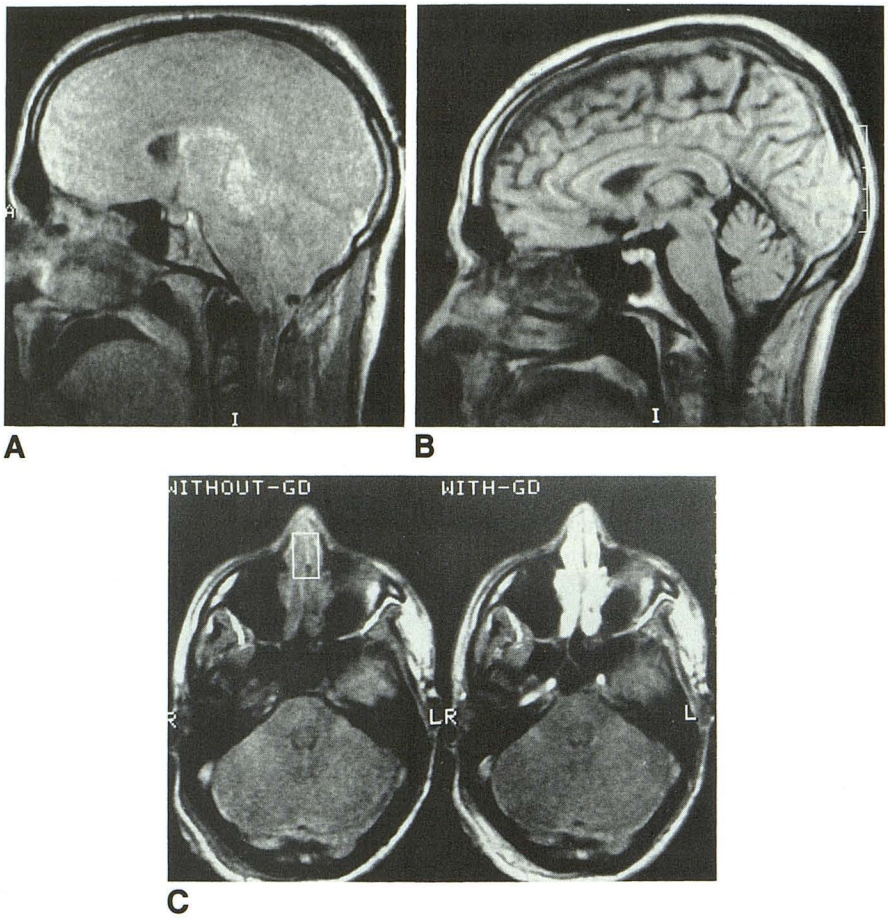

Fig. 1. *A*, T1-weighted sagittal MR image (68/24/60) demonstrating transtentorial and foramen magnum herniation, absent intracranial vascular flow void, and poor gray matter/white matter differentiation in brain death.

B, Healthy age- and sex-matched control subject. Sagittal T1-weighted MR (68/24/60) demonstrating the excellent definition of cortex, white matter structures, such as the corpus callosum and subarachnoid spaces, compared with *A*.

C, T1-weighted axial MR image (68/24/60) before and after gadopentetate dimeglumine demonstrating the MR hot nose sign and associated increased scalp signal intensity in brain death. (Note the markedly increased intensity in the nasal region after gadolinium injection.) There is also an absence of flow void in the carotid arteries with intense enhancement after the injection of gadolinium, indicating an intravascular enhancement sign.

cerebral blood flow study performed on the same day demonstrated findings consistent with the clinical diagnosis of cerebral death, including the hot nose sign (Fig 2).

Case 2

A 28-year-old man was admitted after a self-inflicted gunshot wound to the head. The bullet entered the right parietal area and lodged in the left temporal/parietal area. Computed tomography showed a bullet, bone fragments traversing the bullet pathway, a large right posterior parietal hematoma, intraventricular hemorrhage, generalized edema, fractures of the right and left parietal bones, and minimal shift to the left. After the diagnosis of brain death was made, the MR findings were right parietal intracerebral hemorrhage, intraventricular hemorrhage, intraparenchymal bone fragments, diffuse edema with poor gray matter/white matter differentiation and brain stem herniation, absent cerebral vascular flow void, and no intracerebral enhancement, but prominent enhancement of the nasal region and the carotid arteries after the administration of gadopentetate dimeglumine.

Case 3

A 59-year-old man presented with a history of left cerebral vascular accident 1 year before admission, hypertension, diabetes mellitus, and rheumatoid arthritis. He was admitted unresponsive with an elevated blood pressure. The computed tomography findings included extensive intraparenchymal hemorrhage in the left basal ganglia, intraventricular hemorrhage, subarachnoid hemorrhage, and extensive mass effect. His clinical examination was consistent with brain death.

MR demonstrated hemorrhage in the third, fourth, and lateral ventricles with obstructive hydrocephalus, large intraparenchymal hemorrhage in the left basal ganglia, and centrum semiovale with midline shift, transtentorial and foramen magnum herniation, poor gray matter/white matter differentiation, and absent intracranial vascular flow void. There was no intracranial enhancement, but intense nasal and carotid artery enhancement was seen with gadopentetate dimeglumine.

Discussion

The absence of clinical evidence of cortical and brain stem function when reversible factors such as hypothermia, hypotension, and barbiturates have been eliminated provides the basis for a clinical diagnosis of brain death (2,3,5,14). This diagnosis is further supported by imaging tests, which are usually considered ancillary (1–13).

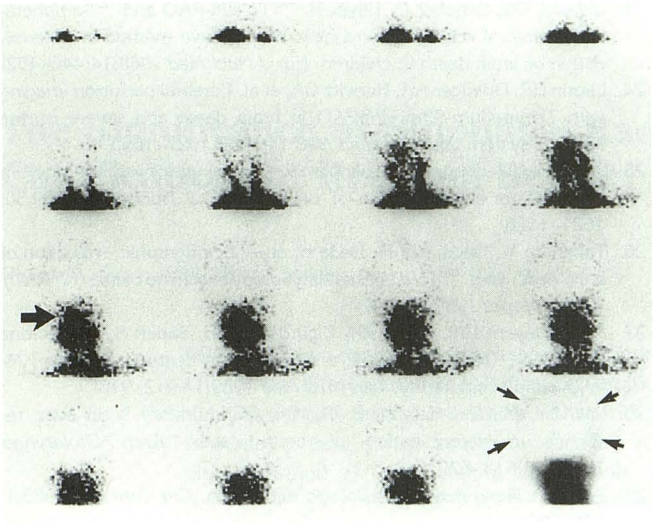

Fig. 2. Anterior projection radionuclide cerebral angiogram (1 second per image for 15 seconds) and immediate static image (last frame), which depicts absent intracranial arterial flow, scalp perfusion (*small arrows*), hot nose sign (*large arrow*), and absent superior sagittal sinus.

Nuclear medicine procedures are commonly used to confirm brain death, and the standard radionuclide cerebral angiogram can be performed with a variety of technetium-99m-based radiopharmaceuticals. The usual positive findings for brain death include no intracranial blood flow in the anterior or middle cerebral circulations, extracranial blood flow over the scalp and face, absent or sometimes weak visualization of the superior sagittal sinus in the venous phase, and a hot spot representing collateral circulation through the center of the face, also referred to as the hot nose sign (6–13).

The value of MR imaging in the evaluation of cerebral ischemia is well established; however, routine MR imaging within the first 24 hours, particularly within the first 8 hours, may have variable reliability (15,18,19). The importance of using a contrast agent such as gadopentetate dimeglumine in the evaluation and detection of early cerebral infarction has been emphasized (15–17). The intravascular enhancement sign has been noted as the earliest finding in cerebral infarction, and a more proximal occlusion may be associated with a lack of intracranial enhancement (15–17). Although the mechanism of arterial enhancement in cerebral ischemia is currently unknown, in part this phenomenon can be attributed to a lack of the normal flow void expected on MR in patent vessels (17,20). The anticipated flow void of the carotid arteries is not present in our cases of brain death, and there is intense enhancement in these vessels, indicating the intravascular enhancement sign.

Perfusion brain agents for single-photon emission computed tomography include I-123 iodoamphetamine and technetium-99m hexamethylpropyleneamine oxime (21–27). The primary disadvantages of these newer agents is their expense (which in our institution results in a study costing slightly more than MR), but they provide a satisfactory evaluation for brain death in the absence of the bolus injection required for radionuclide cerebral angiogram. Diagnostic planar images may be recorded at the bedside with mobile gamma cameras after the injection of one of these agents. Both radionuclide cerebral angiogram and these planar images may be acquired with the same injection.

Other tests that may be of value in the confirmation of brain death include the electroencephalogram, brain stem auditory evoked response, ultrasound, computed tomography, and cerebral angiography (28–41).

A recent case report details noncontrast MR documentation of absent internal carotid flow detected by flow-sensitive gradient-echo sequences and evidence of brain stem herniation in a case of brain death (14). Our report demonstrates that gadolinium-enhanced MR may also reflect decreased cerebral perfusion by marked enhancement of the facial structures, resulting in an MR hot nose sign. Evidence of decreased cerebral perfusion is further demonstrated by the absence of carotid arterial flow void, the intravascular enhancement sign, and a lack of intracerebral enhancement. This precise parallel to the radionuclide cerebral angiogram hot nose in conjunction with evidence of absent intracerebral arterial flow is a useful sign in supporting a diagnosis of brain death. Therefore, the anticipated MR findings in brain death include 1) transtentorial and foramen magnum herniation, 2) absent intracranial vascular flow void, 3) poor gray matter/white matter differentiation, 4) absent intracranial contrast enhancement, 5) carotid artery enhancement (intravascular enhancement sign), and 6) prominent nasal contrast and scalp enhancement (MR hot nose sign).

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