Magnetic Resonance Imaging Characteristics of Hyperacute Intracerebral Hemorrhage

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Abstract

Background—Magnetic resonance imaging (MRI) of the brain is highly sensitive for detecting intracerebral hemorrhage (ICH). Understanding of the imaging characteristics of hyperacute ICH is limited secondary to availability and increased time to scan relative to other imaging modalities.

Objective—In this report, we present the MRI characteristics of two patients who developed ICH while on the MRI table.

Methods—A 44-year-old women presented with week-long headache associated with blurry vision and photophobia. A second patient, a 38-year-old woman, presented with headache. Both patients developed ICH while on the MRI table. We studied the imaging characteristics of hyperacute ICH in these two patients. Postcontrast T1 sequence obtained showed contrast extravasation from two spots in the right caudate nucleus with ventricular extension.

Results—Hyperacute intracerebral hemorrhage has a characteristic appearance on MRI. The morphology of the hyperacute ICH that we presented confirms an asymmetric growth pattern. It also showed that the area of hemorrhage appeared to consist of multiple distinct compartments. These features suggested that expansion of ICH may be compounded by mechanical disruption of secondary blood vessels as the hematoma expanded. We can also confirm that hyperacute intraventricular hemorrhage can be revealed by gadolinium-enhanced MRI.

Conclusion—MRI is able to detect hyperacute ICH and our findings demonstrate an asymmetric pattern of hematoma evolution. MRI with contrast is sensitive to detect hyperacute intraventricular hemorrhage.

Keywords

Intracerebral hemorrhage; magnetic resonance imaging; fluid attenuated inversion recovery; hematoma

INTRODUCTION

Magnetic resonance imaging (MRI) of the brain is highly sensitive for detecting intracerebral hemorrhage (ICH) and compared with computed tomography (CT) scan, it is better suited to detect structural abnormalities associated with ICH [1,2]. MRI characteristics of ICH from the acute to chronic type are well described and enable neuroradiologists to reliably estimate the age of hemorrhagic lesions in the brain; however, understanding MRI characteristics of hyperacute ICH is limited secondary to lower availability, increased time to scan and complexity of MRI relative to other imaging modalities.

In this report, we present the MRI characteristics of hyperacute ICH in two patients who developed ICH while on the MRI table.
CASE REPORTS

Patient 1

A 44-year-old woman with history of hypertension presented with week-long headache associated with blurry vision and photophobia. Initial examination revealed no neurologic deficits, although the patient was found to have elevated blood pressure (BP) of 207/110. While en route to the CT scanner, the patient experienced a generalized tonic-clonic (GTC) seizure, which was treated with lorazepam and a loading dose of intravenous levetiracetam. CT scan of the head was negative for acute abnormality, including infarction or ICH. Lumber puncture was performed and negative for subarachnoid hemorrhage or intracranial infection. MRI of the brain was planned for further evaluation. The patient remained neurologically stable but hypertensive with a BP of 170/95 prior to MRI. After the MRI was partially completed, the patient experienced a second brief GTC seizure while on the MRI table. The seizure was controlled and the MRI resumed with a delay less than 30 min.

A coronal T2 fluid-attenuated inversion recovery (FLAIR) sequence obtained before the seizure revealed bilateral parieto-occipital hyperintense lesions, which were consistent with posterior reversible encephalopathy syndrome (PRES). There was no evidence of hemorrhage on MRI sequences obtained pre-seizure. However, the MRI sequences obtained post-seizure revealed an expanding right basal ganglia hemorrhage with local mass effect. Figure 1 shows before and after images of the seizure event. After identification of ICH, an emergent decompressive hemicraniectomy was performed. The patient’s modified ranking scale (mRS) score at the time of discharge was 4, and her 6-month follow-up mRS score improved to 2.

Patient 2

A 38-year-old woman, 4 weeks postpartum presented with headache for one day. Initial CT scan of the head was negative for acute abnormality, including infarction or ICH. A contrast-enhanced MRI of the brain was planned for further evaluation. Part of the images obtained prior to contrast injection—including diffusion weighted imaging (DWI), T2, T2 FLAIR and T2*-weighted gradient recall echo (GRE) sequences—were unremarkable. T1 sequence obtained after GRE, within 3 minutes, showed small subtle hypointense areas in right caudate nucleus. Succeeding postcontrast T1 sequence obtained showed contrast extravasation from two spots in the right caudate nucleus. Succeeding postcontrast T1 sequence obtained showed contrast extravasation from two spots in the right caudate nucleus with ventricular extension. The location of hemorrhage on postcontrast T1 sequence was consistent with the subtle hypointense area noted on T1 sequence (Figure 2). Emergent EVD was placed. Although the patient had prolonged hospital stay, she made a remarkable neurological recovery.
DISCUSSION

Our findings are consistent with previous reports that the appearance of hyper-acute ICH on MRI is composed of a central core, a periphery, and a surrounding rim. The core was isointense to hyperintense on DWI and T2*-weighted GRE, respectively. The periphery of this lesion was hypointense on DWI and T2* GRE images and represented deoxygenated hemoglobin at the periphery of the hematoma, which was detected secondary to its paramagnetic properties [3]. We were able to confirm that this rapidly de-oxygenated hemoglobin was present as early as 30 minutes after the onset of ICH. The surrounding rim of hyperintensity on T1- and T2-weighted images represented vasogenic edema, which was secondary to the degradation of hemoglobin with the release of toxic materials that caused an increase in blood brain barrier permeability [4].

The morphology of the hyperacute ICH that we presented confirms an asymmetric growth pattern. The cause of this asymmetric expansion could be multifactorial, and theories include local differences in tissue resistance to ICH growth, and trajectory of blood flow from primary as well as secondary ruptured vessels [5]. The significance of the dark spots visible on periphery of hematoma in T2* GRE images (Figure 1c, arrows) was unclear and may represent secondary active rupture of blood vessels surrounding the hematoma, or simply a susceptibility artifact. To our knowledge, this finding on MR imaging has not been previously reported. Further examination of T2*-weighted GRE in the first case and post contrast T1 sequence in the second case revealed that the area of hemorrhage appeared to consist of multiple distinct compartments. These features suggested that expansion of ICH may be compounded by mechanical disruption of secondary blood vessels as the hematoma expanded and distorted peripheral parenchyma, as opposed to an isolated primary vessel rupture. This confirms the previous observation by Qureshi and colleagues.[6]

The reported incidence of ICH in PRES is 15.2–17% [7]. The high incidence of ICH in PRES is likely secondary to hemodynamic disequilibrium associated with elevated blood pressure causing disruption of blood brain barrier [8]. Given this theory, it is interesting that the ICH in our case appears to arise in a vascular territory separate from the PRES features. Our second case also illustrates MRI characteristics in hyperacute ICH with intraventricular extension where contrast enhancement in parenchyma and ventricles represent hemorrhage. It confirms that hyperacute intraventricular hemorrhage can be revealed by gadolinium-enhanced MRI.

CONCLUSION

In conclusion, MRI is able to detect hyper-acute ICH and our imaging demonstrates an asymmetric pattern of hematoma evolution, which may be caused by the rupture of secondary blood vessels in the periphery of an expanding hematoma. In addition, postcontrast T1 sequence can reveal hyperacute intraventricular hemorrhage.
DISCLOSURES
The authors received no financial or material sources of support for this report. Dr. Iftikhar, Dr. Rossi, Dr. Goyal, Dr. Khan, and Dr. Zand have nothing to disclose. Dr. Arthur has served as a consultant for Codman, Medtronic, Microvention, Sequent, Siemens, Stryker; and has received research support from Siemens and Sequent.

ACKNOWLEDGMENTS
The authors wish to thank Andrew J. Gienapp, BA (Department of Neurosurgery, University of Tennessee Health Science Center, Memphis, TN; Medical Education, Methodist University Hospital, Memphis, TN) for technical and copy editing, preparation of the manuscript and figures for publishing, and publication assistance with this manuscript.

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