


Hyperacute intracranial hemorrhage with extensive contrast extravasation and rapid hematoma expansion imaged at onset with magnetic resonance imaging

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Abstract

Evaluation for intracranial hemorrhage is a common indication when performing imaging of the head in the emergency setting. We present a rare case of active, spontaneous extravasation of blood into the brain parenchyma, which evolved during a magnetic resonance imaging examination. A 70-year-old woman who had no previous history of hypertension or trauma underwent magnetic resonance imaging of the brain for confusion and dysarthria. Initial imaging sequences did not demonstrate an acute cerebral hemorrhage; however, subsequent fluid-attenuated inversion recovery, T2- and T1-weighted sequences demonstrated a progressively enlarging signal abnormality. Extravasation of contrast was noted after the administration of gadolinium-based contrast, indicating active intraparenchymal hemorrhage. A computed tomography scan was performed after magnetic resonance imaging to confirm the findings.

Keywords

Intracranial hemorrhage, spontaneous, extravasation, hematoma expansion, magnetic resonance

Introduction

Spontaneous intracranial hemorrhage (SICH) presents as severe headache and altered mental status with no antecedent head trauma. Dependent on the underlying etiology, SICH can affect any age group and is associated with high morbidity and mortality.¹ Therefore physicians, including radiologists, should be familiar with the imaging appearance of this critical diagnosis on any imaging modality to provide urgent treatment.

Generally, a noncontrast computed tomography (CT) of the head is the initial study of choice and sufficient to diagnose/rule out acute intracranial hemorrhage (ICH). If hemorrhage is detected, CT angiography (CTA) and contrast-enhanced magnetic resonance imaging (MRI) are generally performed to evaluate for vascular or other underlying pathology, respectively.¹ Interestingly, prior studies have shown that MRI is as accurate as CT for the detection of acute hemorrhage.² In this report, we present a case of an extensive spontaneous lobar hemorrhage that was not present on initial MRI sequences and demonstrated rapid hematoma expansion during the course of the examination. Although very few cases of SICH imaged with MRI at onset have been described,^{3–5} this case shows hematoma expansion to an extent not previously reported.

Case report

A 70-year-old female was brought to the hospital by her family for acute confusion without previous history of hypertension or trauma. The patient had a remote history of a left temporoparietal ICH managed with decompressive craniectomy, clot evacuation, and subsequent cranioplasty. Before her acute mental status change, the patient was started on aspirin for a prior transient ischemic attack and antiepileptic medications after a first time seizure a few months ago. The patient had otherwise been doing well and lived completely independently.

The initial workup was completed at an outside hospital. It comprised a noncontrast head CT that did not show acute hemorrhage, and a metabolic/infectious workup which was also negative. Additionally, an electroencephalogram (EEG) was performed that revealed high electrical activity but no definite seizure. Three days after her initial presentation, the patient was then transferred to our hospital at her family's request.

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A repeat noncontrast head CT after her arrival in the emergency department failed to demonstrate any acute abnormalities, and the patient was admitted to our institution. During her second day of hospital admission she developed aphasia, and EEG monitoring demonstrated focal left-hemispheric status epilepticus. Subsequently, on day four of admission and after 2 days of a seizure-free period, an MRI brain scan was obtained to investigate for any new structural brain injury. Before the patient went to the MRI scanner, she was responsive and reportedly in good condition. Following the MRI, the patient was more lethargic and nauseous with a new headache.

Review of the contrast-enhanced MRI at first demonstrated no territorial infarct on diffusion-weighted imaging, and b0 images did not show any signal abnormality in the right temporooccipital region (Figure 1(a), 00:14). Next, sagittal pre-contrast T1-weighted sequence showed no mass effect or signs of acute hemorrhage (Figure 1(b), 00:15). Subsequently, on fluid-attenuated inversion recovery (Figure 1(c), 00:17) and T2-weighted sequences (Figure 1(d), 00:20), a 2.7 cm hyperintensity was seen in the medial right temporooccipital region, which further increased in size on gradient recalled echo (GRE) imaging (Figure 1(e), 00:23) and pre-contrast T1-weighted imaging (Figure 1(f), 00:26). After intravenous gadolinium administration (Figure 1(g), 00:31; (h), 00:35), there was corresponding immediate and avid enhancement. The signal intensity of the enhancement was similar to the adjacent dural venous sinus with internal

regions of hypointense signal changes, likely indicating active bleeding. Also noted were numerous, scattered old lobar microhemorrhages indicating cerebral amyloid angiopathy (CAA). A follow-up noncontrast head CT (Figure 2, 03:07) showed further increase in this ICH measuring 7.4 cm in the largest extent, with intraventricular extension.

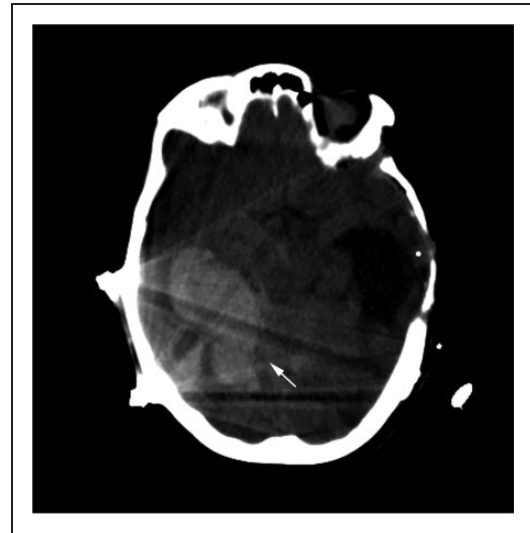


Figure 2. Noncontrast head computed tomography in subdural window obtained 2.5 h after the magnetic resonance imaging (MRI) demonstrates a large intraparenchymal hemorrhage (arrow) centered in the right temporal lobe causing mass effect and measuring up to 7.4 cm. Note multiple electroencephalogram leads causing extensive streak artifacts.

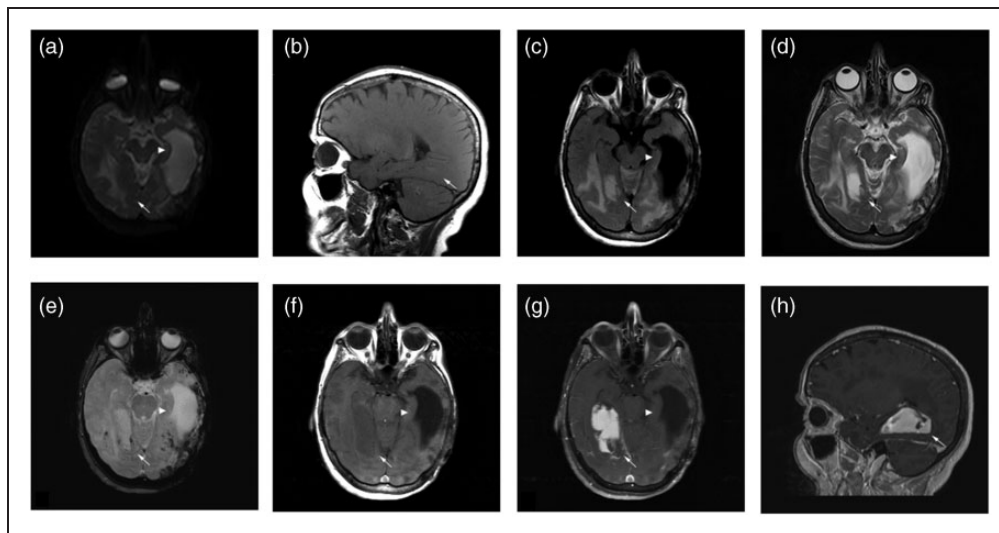


Figure 1. Evolving spontaneous intracranial hemorrhage (arrows) imaged at onset on a 1.5T magnetic resonance imaging (MRI) unit. Note left fronto-parieto-temporal cranioplasty changes with marked subjacent encephalomalacia (arrowheads) related to prior left temporoparietal intracranial hemorrhage and clot evacuation. (a) b0 Image (00:14) shows no signal abnormality in the right temporooccipital region. (b) Sagittal T1-weighted image (00:15) shows no mass effect or signs of acute hemorrhage. (c) Axial fluid-attenuated inversion recovery (00:17), (d) T2-weighted (00:20), and (e) gradient echo (00:23) images show a new, enlarging hyperintensity in the right temporooccipital region. This abnormality is further increasing in size and shows isointense signal intensity compared to grey matter on (f) pre-contrast T1-weighted sequence (00:26), and marked enhancement consistent with extravasation ((g) and (h)) after contrast administration (00:31 and 00:35).

Because of these acute findings the patient was transferred to the intensive care unit. Upon discussion with the patient's family about the unfavorable prognosis, comfort care was provided.

Discussion

SICH accounts for approximately 15% of all strokes and has a mortality rate ranging from 35–52% within the first month. Moreover, approximately half of this mortality occurs within the first 24 h, underlining the importance of accurate diagnosis and management in the emergency department.^{1,6}

Our case demonstrated multiple foci of susceptibility in a lobar distribution, suggesting CAA as the underlying etiology of the bleeding. Lobar hemorrhage is frequently associated with normal blood pressure; likely, the most common etiologies include arteriovenous malformation in young patients and CAA in elderly patients.⁷ In contrast, spontaneous hypertensive hemorrhage usually occurs in deep brain regions, namely in the putamen, thalamus, cerebellum, and pons.^{1,7}

The imaging appearance of hyperacute blood on spin echo T1- and T2-weighted MRI sequences (i.e. isointense on T1-weighted sequences and hyperintense on T2-weighted sequences) is well demonstrated in our case. T2*-weighted GRE sequences and, in recent years, susceptibility-weighted imaging (SWI) have been shown to increase sensitivity for identifying ICH. SWI, a type of GRE sequence, uses phase differences in addition to T2*-weighted contrast and is more sensitive than conventional T2*-weighted GRE sequences for detecting ICH.^{1,7} Our findings on the GRE sequence are similar to prior reports, with the center of the hematoma demonstrating hyperintense signal, comparable to proteinaceous fluid. Simultaneously there is signal loss in the hematoma periphery due to a higher concentration of deoxyhemoglobin and associated susceptibility, which is starting at the periphery and progressing towards the hematoma center.^{5,8} In our case, this process can be noticed as early as 6 min after hemorrhage onset.

After contrast administration, there was extensive active contrast extravasation due to breakdown of the blood-brain barrier,³ suggesting ongoing bleeding.⁹ Active bleeding, which may occur for hours after symptom onset, has been associated with the high level of early neurological decline after ICH.¹ Since hyperacute hemorrhage demonstrates isointense signal on pre-contrast T1-weighted sequences, a potential pitfall is to confuse active extravasation with an enhancing brain tumor.¹⁰ If available, a comparison with prior studies is recommended to evaluate for any prior existing lesion. Additionally, the absence of perihematomal edema with precise hematoma margins suggests that the findings are acute and not secondary to an underlying neoplasm.⁷ A follow-up CT can be

obtained to confirm the findings and assess for hematoma expansion.

Hematoma size is one of the most significant predictors of outcome, and therefore avoidance of hematoma expansion is one of the main goals of ICH management.¹¹ Brain imaging scans for cerebral hemorrhage enable assessment of its growth and detection of underlying pathology which, if present, may need further surgical treatment. In this context, the spot sign has been described on CTA as the presence of tiny enhancing foci within an acute hemorrhage. The spot sign is an indicator of active bleeding and is linked with hematoma enlargement and poor outcome.^{1,7,9} While best known on CTA, Schindlbeck et al. demonstrated the spot sign in acute ICH could also be diagnosed with contrast-enhanced dynamic T1-weighted MRI at 3T.⁹

In summary, we present an extremely uncommon and unique case of SICH presenting with active contrast extravasation and rapid hematoma expansion, which was not evident on initial MRI sequences. Due to further advances in technology, MRI will likely become more available in the emergency setting and play an even more important role. Hence, radiologists will likely be confronted with hyperacute hemorrhage on MRI more often and should be familiar with its appearance.

Conflict of interest

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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