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Case Report

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Summary: We describe a case of hyperacute subarachnoid and intraventricular hemorrhage from a ruptured aneurysm, which occurred while the patient was undergoing MR imaging. Compared with CSF, the blood in the subarachnoid space had slightly lower signal intensity on T2*-weighted gradient-echo images and increased signal intensity on T2-weighted spin-echo images. This finding differs from the generally accepted MR appearance of intracranial hemorrhage and should be recognized to ensure proper patient care.

Hyperacute intracranial hemorrhage is an infrequent finding on MR imaging studies, and only a few cases of active bleeding occurring with a patient inside an MR unit have been reported. Freshly extravasated blood is generally considered to be isointense relative to CSF on T2-weighted images; therefore, subarachnoid hemorrhage (SAH) cannot be identified with this sequence. With fluid-attenuated inversion recovery (FLAIR) MR images, SAH becomes hyperintense and conspicuous by virtue of nulling the signal intensity from the CSF adjacent to the bleed. Studies have demonstrated that the FLAIR sequence is very sensitive to SAH and probably more accurate than CT (except in the posterior fossa). A high accuracy with gradient-echo (GRE) T2*-weighted images in the detection of acute SAH was recently reported.

We present a patient who bled acutely while in the imaging machine and discuss the MR imaging appearance of SAH on images obtained by using different pulse sequences. This appearance should be kept in mind; although SAH that occurs while patients are undergoing MR studies is rare, it needs to be recognized to ensure proper patient care.

Case Report

A 47-year-old woman had an episode of seizures. Physical examination findings were negative except for paresis of the right abducens nerve. A brain CT study was obtained, and the findings were normal. Results of routine laboratory studies were unremarkable. Because of cranial nerve palsy, MR imaging study of the brain was performed 2 weeks later. Before the study, she was fully alert but complained of a moderate headache. When the gadolinium-based contrast agent was given, the patient's mental status rapidly changed, and she was not completely responsive. She then became restless during the first contrast-enhanced sequence.

Nonenhanced T1-weighted images showed that the basilar cisterns and the fourth ventricle were completely filled with a material that was hypointense relative to the brain stem, and hyperintense relative to the CSF, with a dependent level in the third ventricle (Fig 1A). On spin-echo (SE), proton density–weighted, T2-weighted, and FLAIR images, the cisterns were of higher signal intensity compared with the CSF (Fig 1B, D, and 1F). On T2*-weighted GRE images, the cisterns had slightly lower signal intensity compared with that of the CSF (Fig 1E). Contrast-enhanced T1-weighted images demonstrated pooling of the gadolinium-based contrast material in the region of the anterior communicating artery (ACoA) (Fig 1G).

Hyperacute SAH, presumably from a ruptured ACoA aneurysm, was diagnosed, and the patient was immediately taken to the angiography suite. A lobulated wide-neck ACoA aneurysm was found (not shown), and surgical clipping immediately followed. Postoperatively, she developed right-sided hemiparesis, and an acute infarct in the left anterior cerebral artery territory was revealed on CT scans. She was discharged home with residual paresis, and her neurologic status remained stable for the following 18 months.

Discussion

The diagnosis of SAH has historically depended on the results of CT or lumbar puncture, as MR imaging was considered to be insensitive to blood in the subarachnoid space. Currently, CT is still widely and most commonly used to exclude intracranial hemorrhage. If clinically suspected, SAH could be reliably diagnosed by means of MR imaging and its source detected on an MR angiogram. MR may obviate diagnostic intraarterial angiography, at least in some cases.

A number of studies in both clinical and animal models have recently demonstrated that FLAIR is an accurate MR imaging technique in the diagnosis of SAH and that it is more sensitive than nonenhanced CT (1–4). GRE T2*-weighted images can depict intracranial hemorrhage in animal models within minutes of its onset (2, 5). T2*-weighted sequences are also sensitive for acute and hyperacute intracerebral hemorrhage in patients, within less than 30 minutes from the onset (6, 7). A recent study demonstrated the high accuracy of GRE T2*-weighted imaging in the detection of acute (within 4 days) and subacute SAH; the accuracy actually surpassed that of FLAIR imaging (8). Traditionally, many have thought that hyperacute SAH may not be depicted on either SE or

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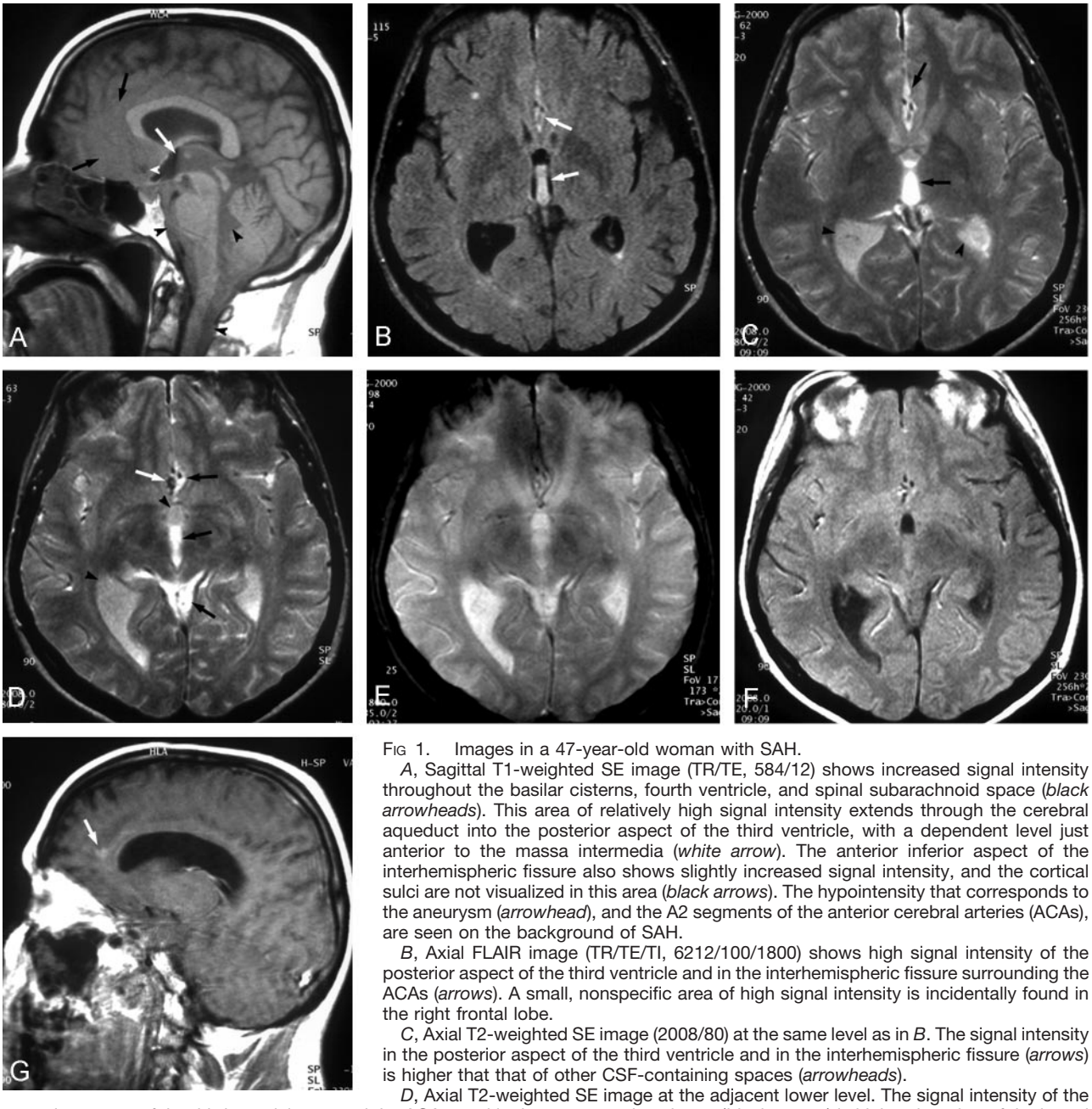

FIG 1. Images in a 47-year-old woman with SAH.

A, Sagittal T1-weighted SE image (TR/TE, 584/12) shows increased signal intensity throughout the basilar cisterns, fourth ventricle, and spinal subarachnoid space (*black arrowheads*). This area of relatively high signal intensity extends through the cerebral aqueduct into the posterior aspect of the third ventricle, with a dependent level just anterior to the massa intermedia (*white arrow*). The anterior inferior aspect of the interhemispheric fissure also shows slightly increased signal intensity, and the cortical sulci are not visualized in this area (*black arrows*). The hypointensity that corresponds to the aneurysm (*arrowhead*), and the A2 segments of the anterior cerebral arteries (ACAs), are seen on the background of SAH.

B, Axial FLAIR image (TR/TE/TI, 6212/100/1800) shows high signal intensity of the posterior aspect of the third ventricle and in the interhemispheric fissure surrounding the ACAs (*arrows*). A small, nonspecific area of high signal intensity is incidentally found in the right frontal lobe.

C, Axial T2-weighted SE image (2008/80) at the same level as in *B*. The signal intensity in the posterior aspect of the third ventricle and in the interhemispheric fissure (*arrows*) is higher that that of other CSF-containing spaces (*arrowheads*).

D, Axial T2-weighted SE image at the adjacent lower level. The signal intensity of the posterior aspect of the third ventricle, around the ACAs, and in the supravermian cistern (*black arrows*) is higher than that of the lateral ventricles and the anterior portion of the third ventricle (*arrowheads*). The larger flow-void area posterior to the ACAs (*white arrow*) was proved to correspond to the ACoA aneurysm on a subsequent intraarterial angiogram (not shown).

E, Axial T2*-weighted GRE image (800/35; flip angle, 25°) at approximately the same level as in *D*. The signal intensities within the third ventricle are reversed compared with those in *D*. The posterior aspect of the third ventricle is of lower signal intensity. This hypointensity is mild, presumably due to small amount of deoxyhemoglobin, which is partly counterbalanced by extremely high T2 signal intensity.

F, Axial proton density–weighted SE image (2008/20) at the same level as in *D*. The signal intensity within the posterior portion of the third ventricle, in the interhemispheric fissure, and in the supravermian cistern, is approximately the same as that in the gray matter.

G, Sagittal contrast-enhanced T1-weighted SE image (584/12) shows pooling of gadolinium-based contrast agent just anterior to corpus callosum (*arrow*). Homogeneously low signal intensity of the CSF is present in the lateral ventricle. The image quality is compromised by motion.

fast SE (FSE) T2-weighted images because it remains isointense relative to CSF (1–4).

The findings in our patient are in accordance with those in the current literature, except that we were better able to visualize the hemorrhage on T2-weighted images. As expected, FLAIR images revealed the hyperintense blood on the background of the dark CSF. The decreased signal intensity of hyperacute SAH was seen on T2* images as it has been described in experimental animal studies.

The generally accepted model for the appearance of intracerebral hemorrhage on MR images attributes

the changes in the signal intensity pattern in evolving hemorrhage to the products of iron metabolism and to the integrity of the red blood cell (6, 7). As oxyhemoglobin passes from arterial blood with high oxygen tension to tissue with low oxygen concentration, the molecule becomes deoxygenated. Because of its unpaired electrons, deoxyhemoglobin is a paramagnetic substance. Susceptibility differences between diamagnetic tissue and paramagnetic deoxyhemoglobin, confined within erythrocytes, result in rapid spin dephasing that leads to signal intensity loss with T2-weighted sequences; this finding is more obvious on T2*-weighted images than on others (6, 7).

Some have proposed that the inability to visualize SAH on SE images is related to the relatively high oxygen tension in the CSF, which prevents the formation of deoxyhemoglobin (9). However, studies in animal models (2, 5) and the findings in our patient indicate that the signal intensity loss on T2*-weighted images may be observed immediately after the onset of SAH. We think that, in our patient, the high volume of extravasated blood may have been responsible for this finding by allowing portions of the clot to become relatively isolated from the oxygen-rich CSF. The hypointensity on T2*-weighted images was mild in our case (Fig, E), and the probable reasons are a small amount of deoxyhemoglobin and an extremely strong T2 prolongation, as shown on SE images (Fig 1C and D). The signal intensity loss that deoxyhemoglobin created was therefore counterbalanced by a kind of a T2 shine-through effect.

Fresh blood has the signal-intensity characteristics of a proteinaceous solution, which lead to high signal intensity on T2-weighted images. However, this appearance is typically masked by CSF, which is of even higher signal intensity. In our patient, the extravasated blood was surprisingly hyperintense relative to CSF. In an article by Perl et al (3) we noticed that hyperacute SAH and parenchymal hemorrhage appear to have signal intensity higher than that of the CSF on T2-weighted SE images in an animal model. To our knowledge, this observation has not been previously described, and we are not aware of an explanation for this phenomenon. The hemoglobin concentration and the hematocrit level in our patient's peripheral blood were within normal limits; therefore, those factors cannot account for the findings. Since the degree of interaction of water with macromolecular structures governs the efficiency of water proton relaxation, it may be possible that the ideal match for the applied sequence is incidentally created in these cases, giving rise to an extremely high signal intensity on T2-weighted images. These findings could also indicate that the generally accepted model of intracranial hemorrhage might need some adjustments.

To our knowledge, one case of a hyperacute SAH during clinical MR imaging study has been reported (10). In this case, the blood was of high signal intensity on FLAIR images, and presumed contrast-enhanced blood extravasation was also seen. In this report by Küker et al, the hemorrhage could not be detected on FSE T2-weighted images. The volume of hemorrhage was much smaller than that of our patient, and it was considered to be a nonaneurysmal perimesencephalic SAH. Also, from their report, the exact time of the MR imaging study was not clear, and protracted bleeding may have started many hours before the study; this may have led to substantial dilution of the extravasated blood. In our case, the different appearances of hyperacute SAH on T2-weighted images may be explained by three possible factors: different field strengths (1T as opposed to 1.5 T); different MR imaging sequences (SE instead of FSE); and perhaps most importantly, relatively rapid massive hemorrhage. Also, if gadolinium-based contrast material is administered before acquisition of T2-weighted images in the case of an ongoing SAH, its high concentration may lead to signal intensity loss, as seen in the urine in the bladder or as seen on perfusion-weighted images. We think that, in our case, the aneurysm ruptured just minutes to 1 hour before the patient was placed in the imaging machine. FSE and SE sequences differ in a number of ways because of the multiple refocusing pulses of FSE sequences, with decreased susceptibility being the most prominent difference. Increased field strength results in increased signal intensity and increased susceptibility; both of these effects may also substantially affect the signal-intensity pattern on the MR images.

Spilling of contrast-enhanced blood into CSF spaces during brain MR imaging has been reported two more times, and it was intraventricular in both cases (11, 12). No signal-intensity abnormalities were noted on nonenhanced images, the hemorrhage appears to have occurred after the injection of gadolinium-based contrast agent in both of these reported cases.

Conclusion

MR imaging in our patient demonstrated that hyperacute SAH was of high signal intensity on FLAIR images, low signal intensity on T2*-weighted images, and increased signal intensity relative to the CSF on T2-weighted SE images. Awareness of these findings led to prompt treatment of this patient.

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