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Abbreviated Reports

MR of Acute Subarachnoid Hemorrhage

It is reported that MR is poor for visualizing acute (<90 hr) subarachnoid hemorrhage [1, 2]. We report a case of positive MR findings in acute subarachnoid hemorrhage and describe possible mechanisms.

Case Report

Three days before presentation, a 57-year-old woman noted a sudden onset of a mild headache that resolved in the evening. The next day, a sudden, severe suboccipital headache without accompanying neurologic deficits developed; this headache did not resolve. She was admitted to another hospital the next day and a CT scan showed blood in the prepontine, interpeduncular, and suprasellar cisterns (Fig. 1A). No intraparenchymal hemorrhage was noted. The patient was transferred to our institution the next day for further evaluation and management.

At the time of admission, the patient complained of a continuing severe headache but was alert, conversant, and without focal neurologic findings. A four-vessel cerebral angiogram showed no significant arterial spasm, aneurysms, or vascular malformations. MR performed on a 0.3-T imager 50 hr postictus showed increased signal in the perimesencephalic and interpeduncular cisterns that was isointense to brain parenchyma on a T1-weighted image (Fig. 1B). A repeat MR 72 hr postictus showed no further changes. A third MR study performed 10 days postictus showed hyperintensity of the

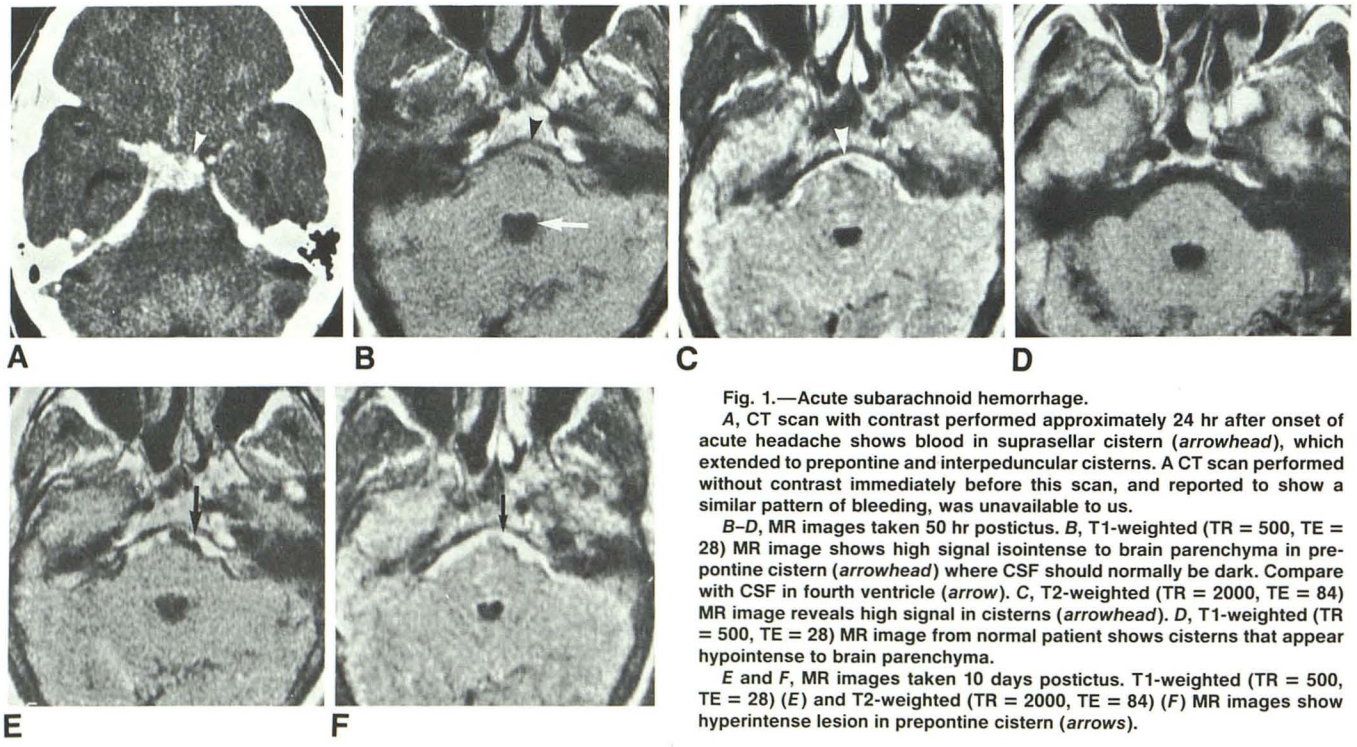

Fig. 1.—Acute subarachnoid hemorrhage.

***A*, CT scan with contrast performed approximately 24 hr after onset of acute headache shows blood in suprasellar cistern (*arrowhead*), which extended to prepontine and interpeduncular cisterns. A CT scan performed without contrast immediately before this scan, and reported to show a similar pattern of bleeding, was unavailable to us.**

***B–D*, MR images taken 50 hr postictus. *B*, T1-weighted (TR = 500, TE = 28) MR image shows high signal isointense to brain parenchyma in prepontine cistern (*arrowhead*) where CSF should normally be dark. Compare with CSF in fourth ventricle (*arrow*). *C*, T2-weighted (TR = 2000, TE = 84) MR image reveals high signal in cisterns (*arrowhead*). *D*, T1-weighted (TR = 500, TE = 28) MR image from normal patient shows cisterns that appear hypointense to brain parenchyma.**

***E* and *F*, MR images taken 10 days postictus. T1-weighted (TR = 500, TE = 28) (*E*) and T2-weighted (TR = 2000, TE = 84) (*F*) MR images show hyperintense lesion in prepontine cistern (*arrows*).**

cisterns in both the T1- and T2-weighted images (Figs. 1E and 1F). The patient improved symptomatically, with progressive lessening of her headache, and was discharged on the 13th day postictus.

Discussion

Although MR is often superior to CT in the evaluation of subacute and chronic subarachnoid hemorrhage, it is thought to be poor for assessment of acute subarachnoid hemorrhage [1–4]. In our patient, we found that a region of the subarachnoid space anterior to the midbrain, which should have been dark on the T1-weighted image, was isointense to the brain parenchyma. A CT scan of the same region performed before MR confirmed that this area represented acute hemorrhage.

It has been shown that the presence of deoxygenated blood in CSF does not appreciably decrease the T1 relaxation time [1]. Despite the paramagnetic nature of the ferrous ion in deoxyhemoglobin, CSF mixed with hypoxic red blood cells does not show a significant shortening of T1 relaxation time. The shortening in T1 relaxation time associated with the formation of methemoglobin does not become maximal until 90 hr after the hemorrhage [1, 5].

It is possible that in our patient adequate formation of methemoglobin within 48 hr of the hemorrhage caused the resultant hematoma to become isointense to brain parenchyma. Two lines of evidence, however, suggest that the observed shortening of T1 relaxation time in acute subarachnoid hemorrhage is not governed solely by the oxidation state of heme iron and the gradual accumulation of methemoglobin.

In acute intraparenchymal hemorrhage, changes in the oxidation state of iron from the desaturation of oxyhemoglobin to paramagnetic deoxyhemoglobin result in preferential T2 shortening [5]. Unlike the T1 shortening characteristic of methemoglobin, the decrease in T2 relaxation time associated with formation of deoxyhemoglobin lowers signal intensity on both T1- and T2-weighted images. This phenomenon is potentially limited in subarachnoid hemorrhage by the significant dilution effects of CSF; and the low-signal appearance of CSF in T1-weighted images further masks any preferential T2 relaxation effects. We would not expect the increased signal seen in our T1- and T2-weighted images (Figs. 1B and 1C).

Second, Cohen et al. [6] and Rapoport et al. [7] have shown that the formation of thrombi can cause a significant decrease in T1 relaxation during the acute hemorrhagic episode. The mechanism of T1 shortening during thrombus formation is unclear, and, to our knowledge, the effect of protein clotting factors on T1 relaxation has not been studied.

At present, we can only hypothesize that T1 shortening within the area of subarachnoid hemorrhage may represent a combination of paramagnetic effects from methemoglobin augmented by protein deposition within the thrombus, which alters the proton-electron dipole-dipole interactions of water molecules. MR performed 10 days postictus showed hyperintense lesions in the same locus on both the T1- and T2-weighted images consistent with the T1 shortening resulting from formation of methemoglobin during the subacute stage (approximately 1 week postictus) of a hematoma.

MR imaging has already been shown to be superior to CT for the evaluation of subacute and chronic intracranial hematomas. As we gain further experience with the MR appearance of acute subarachnoid hemorrhage, MR may play a greater role in the evaluation of acute subarachnoid hemorrhage.

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