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Received April 18, 1986.
Received in revised form June 16, 1986.
Accepted June 19, 1986.

CRANIAL MAGNETIC RESONANCE IMAGING IN ECLAMPSIA

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Although the precise neuropathologic basis for eclamptic convulsions remains unclear, intracranial hemorrhage is frequently associated with fatal cases. Magnetic resonance imaging (MRI) is a recently developed neuroimaging technique that appears superior to other processes for defining intracranial anatomy and pathophysiology. This technique has not yet been reported in eclampsia. The authors performed serial MRIs on an eclamptic woman, with findings compatible with previously described neuropathologic changes. ***(Obstet Gynecol 70:474, 1987)***

Although the incidence of eclampsia has decreased progressively over the past few decades, it remains a prominent cause of maternal morbidity and mortality. One of the most frequent mechanisms of maternal death in eclampsia is intracranial hemorrhage. Autopsies in fatal cases may reveal massive hemorrhage, especially in the basal ganglia or pons, although multiple petechial hemorrhages or infarcts in the cortex or subcortical areas are more common. Most clinicians consider cerebral edema a prominent component of eclampsia, but neuropathologic studies have not reported this consistently. Various neuroimaging techniques have been used to evaluate eclamptic patients. Cerebral angiography is capable of demonstrating larger hemorrhages and mass lesions, but is a potentially risky procedure in the severely hypertensive gravida. Computed tomography (CT) can be performed with minimal maternal risk, but still may not demonstrate the multifocal cortical and subcortical hemorrhages described in autopsy series.[1,2]

Magnetic resonance imaging (MRI) has been used extensively in neuroradiologic studies during the past few years, and correlates well with subsequent autopsy findings. The technique provides images in multiple planes, avoids the use of ionizing radiation, and is not associated with any known biohazards.

Although MRI seems to be an appropriate and safe diagnostic procedure in the eclamptic patient, we are unaware of any confirming reports. This study describes a case of eclampsia studied with MRI both in the immediate puerperium and at three months postpartum.

Case Report

A 21-year-old white woman, gravida 1, was transferred to The University of Iowa Hospitals at 31 weeks' gestation because of eclampsia. Thirteen days before referral, her blood pressure was 126/70 mmHg, and she was asymptomatic. In the week before transfer, she had developed a constant headache, which improved somewhat with aspirin. She had experienced several convulsions in the preceding two days (abnormal breathing, frothing at the mouth, stiffening with rhythmic movements of the left arm, and postictal lethargy). She had no history of seizures.

At a local emergency room, her blood pressure measured 140/98 mmHg; there was evidence of proteinuria and fetal cardiac activity. A head CT was described as normal (Figure 1). She had two further seizures at the local hospital, for which she received 5 mg diazepam and 4 g magnesium sulfate intravenously. She was then transferred.

On arrival, she was confused and somnolent, but denied headache, blurred vision, or epigastric pain. Her blood pressure was 160/100 mmHg, and urine output was approxi-

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Supported in part by a grant from the Heed Ophthalmic Foundation.

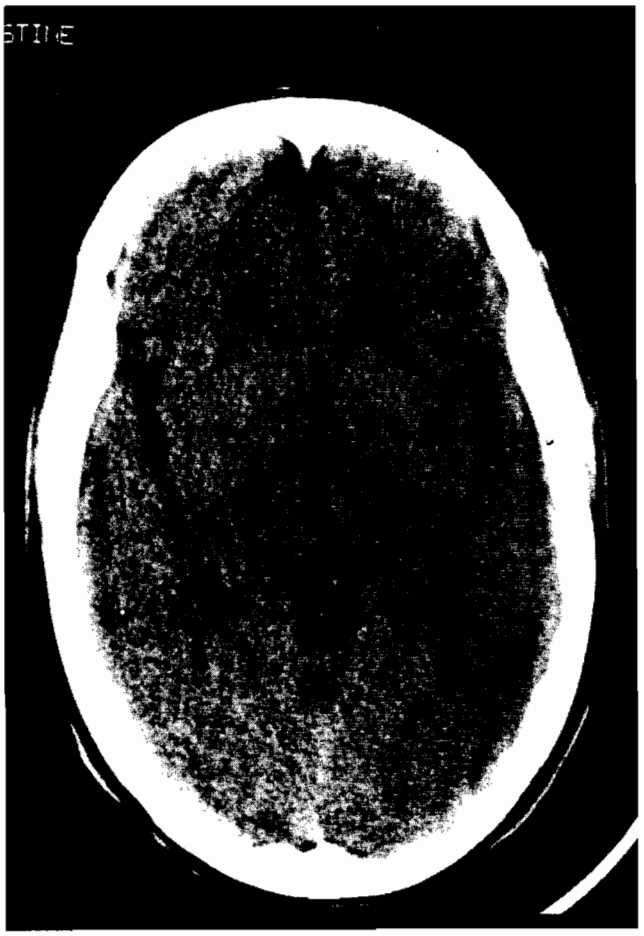

Figure 1. Axial CT sections through the frontal, temporal, and occipital lobes demonstrating no abnormality.

mately 50 mL/hour. She had facial edema with minimal extremity edema. Ophthalmoscopy revealed arteriolar narrowing with a nerve fiber layer hemorrhage in the right eye, although vision was intact. Her lungs were clear to auscultation, and the fundal height was 26 cm. Electronic fetal monitoring revealed a fetal heart rate of 135 beats per minute, without ominous changes. The cervix was 2 cm dilated and 60% effaced, with a vertex at −2 station. Initial laboratory examination revealed 4+ proteinuria, thrombocytopenia (101,000/μL), hyperuricemia (8.4 mg%), and elevated liver function studies (lactate dehydrogenase = 700 IU/mL; aspartase transaminase = 190 IU/mL).

She was continued on intravenous magnesium sulfate at 2 g/hour. Labor was induced effectively by intravenous oxytocin plus amniotomy, and the patient was delivered by forceps, because of persistent fetal bradycardia, of a 1210-g female infant with Apgar scores of 3 and 7 at one and five minutes, respectively. Cord blood gases revealed a venous pH of 7.38 and an arterial pH of 7.24. The infant had an uncomplicated nursery course.

In the first 24 hours postpartum, the patient's vital signs remained stable and she had no further convulsions. At approximately 24 hours postpartum, her urine output increased and there was a slight decrease in blood pressure. Magnesium sulfate was discontinued 36 hours postpartum.

Forty-eight hours postpartum, an MRI of the head was performed using a Picker 0.5 Tesla superconducting unit. Axial and coronal spin echo sequences were obtained with T2 weighting, using a repetition time of 2300 milliseconds and an echo time of 80 milliseconds. Axial inversion recovery images, which are T1-weighted images, used a repetition time of 3050 milliseconds and an inversion time of 500 milliseconds. The study revealed a small area of high-intensity signal on the T2-weighted images within the left temporal region, adjacent to a temporal lobe gyrus (Figures 2 and 3). Multiple other areas of gyral configuration-increased intensity signals on T2-weighted images were seen bilaterally over the occipital lobes and above the convexities of the frontal and parietal lobes (Figure 4). These also displayed a gyriform pattern and were located at the cortical surfaces. These signal abnormalities were slightly more prominent on the right. Corresponding abnormalities were not identified on T1-weighted inversion recovery images. No abnormalities were identified in the subcortical white matter, ventricles,

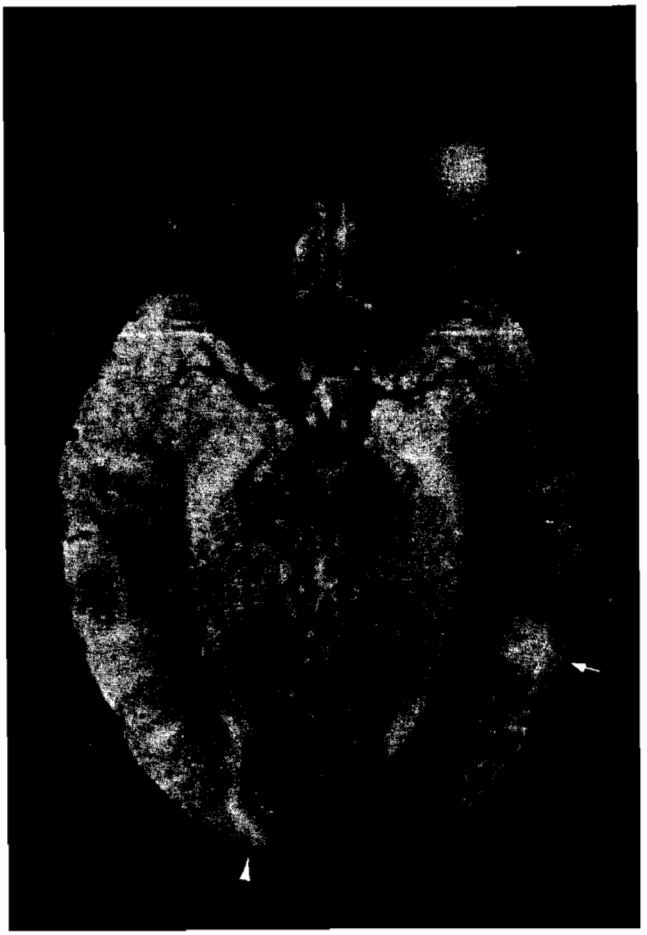

Figure 2. Axial T2-weighted image obtained with 0.5 Telsa MRI unit, on a plane similar to that in Figure 1. A discrete region of abnormally increased signal intensity (prolonged T2) is visible in the left temporal lobe (*arrow*). A second lesion is seen in the right occipital lobe (*arrowhead*) in a gyriform configuration.

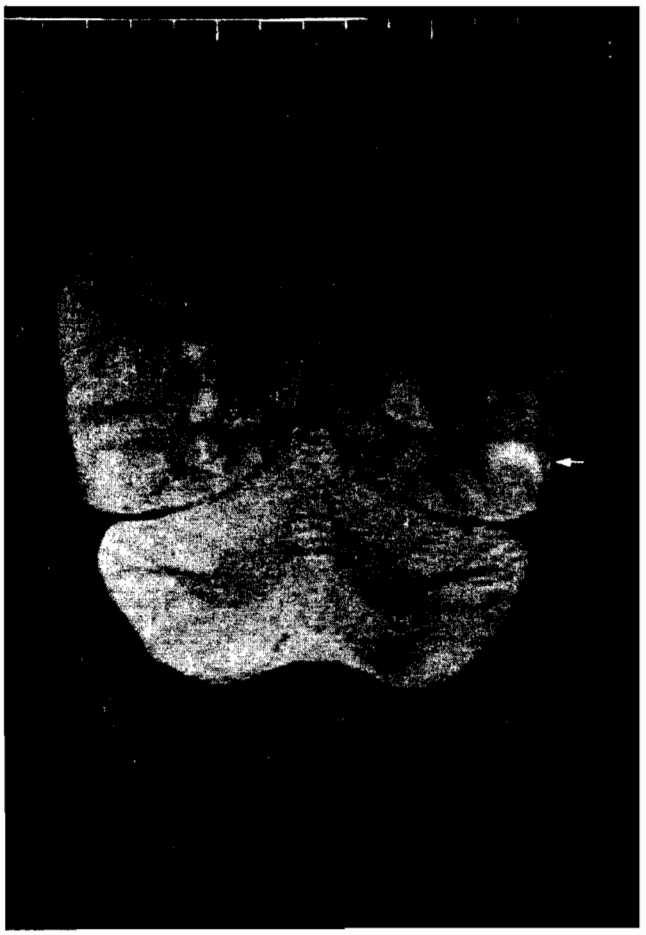

Figure 3. Coronal T2-weighted image. The left temporal lobe lesion has a more gyriform configuration in this plane (*arrow*). There is no associated mass effect.

basal ganglia, or brain stem. An incidental finding was a left maxillary sinus high-intensity signal consistent with sinusitis.

The patient continued to make an uneventful recovery and was discharged on the fifth postpartum day. At that time, her blood pressure was 138/78 mmHg, and serum chemistries were all returning to normal.

At three weeks postpartum, the patient's blood pressure was 118/76 mmHg, and she had returned to work. A complete neurologic and ophthalmologic examination performed at three months postpartum was entirely normal. A follow-up MRI study at this time also revealed no abnormalities.

Discussion

The etiology of eclampsia remains unclear. However, autopsy studies reveal widespread arteriolar vasospasm and thrombosis. Neuropathologic examination of the brain may reveal edema, although the large autopsy series of Sheehan and Lynch[1] did not report this. However, they defined absence of edema as normal brain weight, whereas most neuropathologists establish this diagnosis on histologic grounds rather than on overall weight. In another large autopsy series, Govan[2] described fibrinoid changes as regular findings in the walls of the cerebral vessels. Similar endothelial damage with loss of vascular integrity has been noted in the uteroplacental vasculature[3] and kidney[4] of women with preeclampsia/eclampsia.

Gross and/or microscopic foci of hemorrhage or infarction are also seen in 60–85%[1,2] of eclamptic patients who die within two days of convulsions. However, hemorrhages are seen only in about 20% of women who survive for more than two days.[1] Although massive hemorrhage may occur, especially in the basal ganglia or pons, multiple petechial hemorrhages and/or infarcts in the cortex and subcortical areas are more common.[1,2] In Sheehan and Lynch's large series, the most frequent and characteristic brain lesions were groups of pinpoint cortical petechiae, arranged in streaks running radially in the cortex. They may appear anywhere on the gyral surface and are

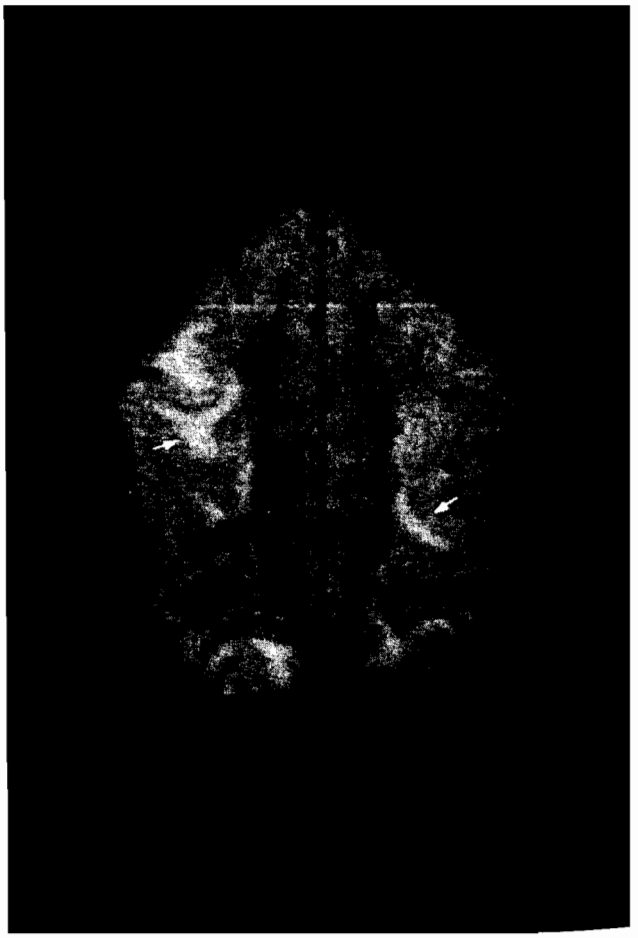

Figure 4. Axial T2-weighted image obtained with 0.5 Telsa MRI unit through the frontal and parietal lobes. Multiple gyriform areas of abnormally increased signals are visible in the gray matter of both hemispheres (arrows identify representative lesions).

usually 2–4 cm in diameter. They are most common in the occipital lobes and least common in the temporal lobes. Many occur in the border zones between major cerebral arterial supplies. There is no symmetry between the hemispheres. Histologically, these lesions are a combination of numerous, very small hemorrhages (0.3–1.0 mm in diameter), capillary stasis and thrombi, and very small infarcts (0.3–1.0 mm in diameter).

The MRI findings on the second postpartum day are consistent in configuration and distribution with Sheehan and Lynch's description of cortical petechiae. However, it is more likely that the visible abnormality on this patient's MRI images represents local edema in response to the local insult. The hyperintense T2 signal abnormality and normal T1 signal pattern seen in the initial scan would also indicate this. Brain edema, as a reflection of increased water content, demonstrates hyperintensity on T2-weighted images and relative hypointensity on T1-weighted images. The failure to observe the T1 signal aberration may be due to the proximity of the lesion to the normally hypointense cerebrospinal fluid-filled gyri. It may also reflect in part the time delay from delivery to initial MRI imaging. Further, hemorrhage would likely appear hyperintense on T1- and T2-weighted images at that time in the patient's clinical course, because of the conversion of deoxyhemoglobin to methemoglobin within the hematoma. The resulting hyperintensity on both T1- and T2-weighted images has been described at low and high field strengths. The complete resolution of signal abnormality on the follow-up scan also suggests resolved edema, because intraparenchymal hemorrhage is known to demonstrate signal abnormalities persisting for at least eighteen months. The size of the individual lesions seen pathologically is below the limits of resolution for the MRI system we used, again arguing that the visible abnormalities are most likely due to edema. Because there is no neuropathologic correlation in this case, it is not possible to say with certainty what these MRI findings represent. All of these considerations emphasize the importance of evaluating more eclamptic women with cranial MRI in the near future.

This case also emphasizes the potential value of MRI in the evaluation of women with preeclampsia/eclampsia. Although the MRI was abnormal in this woman, her CT scan was normal. Such improved sensitivity and possible specificity may be of clinical value in distinguishing between eclampsia and other causes of convulsions in the pregnant woman (seizure disorder, brain tumor, etc). Recent reports[5,6] have described the MRI findings in focal seizures and partial complex seizures. These lesions are commonly located in the temporal lobe, extend beyond the cortex, and are round rather than gyriform, a distinctly different pattern from the one in this case. Likewise, the MRI findings in brain tumors, and the findings from large white-matter hemorrhages sometimes seen in the course of eclampsia, are quite different from those in the current case.[7] Optimum diagnostic capability will ensure optimum treatment for the pregnant woman who develops convulsions. If future reports confirm the increased sensitivity of MRI, it may then be appropriate to recommend it as the technique of choice in evaluating the cerebral pathophysiology of pregnant women with convulsions when the diagnosis of eclampsia is unclear.

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Received June 24, 1986.
Received in revised form October 7, 1986.
Accepted October 20, 1986.