

Case Report

Diffusion-weighted Imaging Shows Cytotoxic and Vasogenic Edema in Eclampsia

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Summary: In eclampsia, MR imaging shows reversible T2 hyperintensities in a parietal and occipital distribution. Findings on diffusion-weighted images suggest that these abnormalities are areas of vasogenic edema. We describe the presence of both cytotoxic and vasogenic edema, as detected by diffusion-weighted imaging, in a woman with eclampsia. Follow-up MR imaging showed that the regions of cytotoxic edema progressed to cerebral infarction. This case suggests that diffusion-weighted imaging allows the early detection of ischemic infarcts in patients with eclampsia.

The neuroradiologic hallmarks of eclampsia are reversible abnormalities that appear hypodense on CT studies and hyperintense on T2-weighted MR images in a subcortical, predominantly parietal and occipital distribution (1, 2). In previously reported cases of eclampsia, diffusion-weighted MR images have shown these areas to have a high apparent diffusion coefficient (ADC) value, suggestive of vasogenic edema (3–5). We describe the presence of both cytotoxic and vasogenic edema, as detected on diffusion-weighted MR images, in a woman with eclampsia. This finding has, to the best of our knowledge, not been previously described in patients with eclampsia.

Case Report

A 24-year-old woman with a history of schizophrenia presented when 38 weeks' pregnant with proteinuria, pedal edema, and a blood pressure of 160/110 mm Hg. Labor was successfully induced 24 hours after admission. However, the next day, she experienced a generalized tonic-clonic seizure, severe hypertension (highest blood pressure, 220/120), and the eclampsia syndrome of hemolysis, elevated liver enzymes, and low platelets. The patient was disoriented and diffusely hyperreflexic, but had no localizing neurologic findings other than a right Babinski sign on plantar stimulation.

A brain MR study revealed extensive bilateral hyperintense signals on T2-weighted images in the parietal and occipital

lobes (Fig 1A). These areas were predominantly hypointense on diffusion-weighted images (Fig 1B), and an ADC map showed free motion of water (Fig 1C), consistent with vasogenic edema. However, within these abnormalities, two smaller areas of diffusion-weighted hyperintensities were apparent (Fig 1B). The ADC map showed these areas to have restricted motion of water and to be consistent with cytotoxic edema (Fig 1C). Trace ADC values in the normal-appearing white matter and gray matter were $0.707 \pm 0.08 \times 10^{-3}$ mm²/s and $0.859 \pm 0.06 \times 10^{-3}$ mm²/s, respectively. The vasogenic edema had an ADC value of $1.955 \pm 0.12 \times 10^{-3}$ mm²/s. The lesion in the left parietal gray matter had an ADC value of $0.98 \pm 0.02 \times 10^{-3}$ mm²/s. An MR venogram showed no evidence of venous sinus thrombosis.

She was treated with sodium nitroprusside, magnesium sulfate, and mannitol. The next day, a mild right hemiparesis became apparent, which gradually resolved. Over the following days, she remained poorly interactive but less agitated and showed continued improvement. Three months after her admission, only a mild facial asymmetry was apparent, suggestive of a possible right lower facial paresis. A follow-up brain MR study at that time showed only two small areas of encephalomalacia on T1-weighted and fluid-attenuated inversion recovery (FLAIR) images in the left posterior parietal cortex and resolution of all other previously noted abnormalities (Fig 1D).

Discussion

The trace ADC value of $1.955 \pm 0.12 \times 10^{-3}$ mm²/s of the vasogenic edema represented an expected increase in the diffusion of water as compared with normal white matter. The region of restricted diffusion, as suggested by the diffusion-weighted image and ADC map, was not supported by the quantitative value of $0.98 \pm 0.02 \times 10^{-3}$ mm²/s as compared with gray matter. Because we know the region progressed to infarction, there is a possible explanation for this apparent dichotomy. The patient was imaged 48 hours after the onset of symptoms and therefore the region of infarction may have been in the subacute phase with regard to changes in the diffusion properties. Some studies suggest that within 48 hours the ADC values of an infarct may increase as cells lyse and cystic encephalomalacia develops (6, 7). Another explanation for the mildly elevated ADC value could be partial volume effects, since this infarct was anatomically small and averaging with adjacent vasogenic edema could have occurred.

The pathophysiology of eclampsia has remained incompletely understood (3, 5). A prevailing theory proposes that eclampsia is a variant of hypertensive encephalopathy (1). It is thought that the develop-

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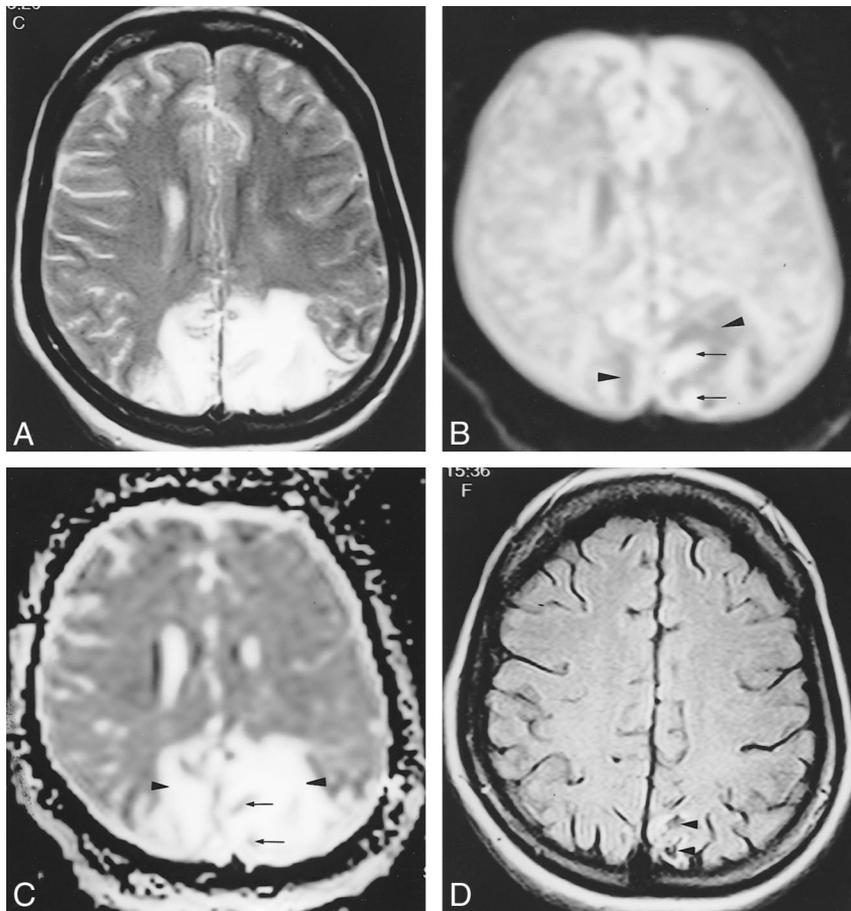


FIG 1. 24-year-old woman, 38 weeks' pregnant, with proteinuria, pedal edema, and a blood pressure of 160/110 mm Hg. A, T2-weighted MR image (4000/96/1) shows hyperintense abnormalities in the cortical and subcortical regions of the occipital lobes.

B, Diffusion-weighted MR image (8000/100/1) shows the T2 abnormalities in A to have a predominantly low signal, suggestive of vasogenic edema (arrowheads). However, the two hyperintense areas (arrows) are consistent with cytotoxic edema.

C, ADC map shows predominantly hyperintense areas of free motion of water, as seen in vasogenic edema (arrowheads). Two areas of restricted motion of water (arrows) correspond to the diffusion-weighted hyperintensities in B. These are suggestive of ischemic injury.

D, FLAIR sequence (6000/128/1) obtained 3 months later shows tissue loss in the areas that were hyperintense on the ADC map (arrowheads).

ment of hypertension during preeclampsia exceeds the upper limits of cerebral autoregulation and causes hyperperfusion. Subsequently, a breakdown of the blood-brain barrier ensues and vasogenic edema develops (8). This is supported by the radiologic findings of hyperperfusion on single-photon emission CT (1) and the reversibility of the subcortical lesions on brain CT and MR studies (2). The recently described diffusion-weighted abnormalities in eclampsia, suggestive of vasogenic edema, also seem to suggest such a mechanism (3–5). However, autopsy studies of women who have died of eclampsia have shown a clear predominance of cerebral ischemic microinfarcts and petechial hemorrhages, with associated fibrinoid necrosis of the microvasculature (9). It is unlikely that these pathologic findings represent the reversible abnormalities seen on neuroimaging studies in the majority of women with eclampsia. In fact, residual lesions, suggestive of ischemia, are only infrequently detected by MR imaging in women who survive eclampsia (2).

The discrepancy between the rather ominous pathologic findings and the predominantly benign radiologic picture is difficult to explain. It remains uncertain as to how autopsy results apply to the majority of eclamptic women who survive and whose clinical and radiologic features are reversible. The presence of hemorrhages appears to in-

dicade a poor prognosis (10). Similarly, the high prevalence of ischemic lesions found at autopsy seems to predict a poor outcome (9). Therefore, it may be that the spectrum of eclampsia ranges from an initially reversible phase of vasogenic edema formation to a later phase of ischemic damage and hemorrhage, which carries a worse prognosis. In fact, laboratory studies of hypertensive encephalopathy, a condition that may share clinical and neuroradiologic features with eclampsia, suggest that as vasogenic edema progresses, local tissue pressure increases. This causes a decrease in regional perfusion pressure and a reduction of blood flow to ischemic levels (11, 12). Subsequently, areas surrounding marked vasogenic edema may progress to infarction (11). A similar transition from early vasogenic edema to ischemia as detected by diffusion-weighted imaging has been reported clinically in one patient with posterior leukoencephalopathy syndrome, and seems to support this hypothesis (12).

The coexistence of these two phases and the transition from one to the other were also evident in our case. Diffusion-weighted images showed the presence of both vasogenic and cytotoxic edema. Our patient exhibited a focal neurologic deficit during the course of the disease and also had residual neuroimaging abnormalities, suggestive of a worse prognosis.

Conclusion

In the past, differentiating between cytotoxic and vasogenic edema has been difficult, as both appear hyperintense on T2-weighted MR images. The use of diffusion-weighted imaging and ADC maps allows an earlier and clearer differentiation of cytotoxic and vasogenic edema, which may have important prognostic implications in eclampsia. Earlier diagnosis of ischemia may identify women with eclampsia who are at high risk for an adverse outcome, which may, in turn, influence management decisions and lead to more careful control of blood pressure and an expedient delivery.

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