

Permanent Visual Deficits Secondary to the HELLP Syndrome

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Abstract: A 34-year-old woman with eclampsia and the hemolysis, elevated liver enzymes, and low platelets (HELLP) syndrome developed encephalopathy, cardiomyopathy, pulmonary edema, liver failure, and disseminated intravascular coagulation (DIC), all of which resolved. She also had retinal hemorrhages in both eyes and a hemorrhagic infarct in the left occipital lobe that resulted in a permanent right homonymous hemianopia and a persistently depressed acuity of 20/100 OS. This case is unusual in demonstrating permanent visual deficits. In nearly all cases of pre-eclampsia or eclampsia, visual deficits are reversible. The superimposition of the HELLP syndrome may create more neurologic damage. Clinicians should be alert to patients at risk for HELLP syndrome and manage them aggressively.

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Hemolysis, elevated liver enzymes, and low platelet count (HELLP) may accompany hypertension and proteinuria (preeclampsia) and seizures (eclampsia) as part of a spectrum of hypertensive disorders of pregnancy (1,2). The HELLP syndrome may be considered a severe variant of preeclampsia/eclampsia (3), or a separate disorder with features that overlap preeclampsia/eclampsia (4–7).

The HELLP syndrome may cause hepatic rupture, disseminated intravascular coagulation (DIC), acute tubular necrosis, pulmonary edema, adult respiratory distress syndrome, and hemorrhage (8). The incidence of maternal mortality in the HELLP syndrome has been reported at 1% to 4% (5–8), whereas perinatal mortality has been estimated at 10% to 20% (5,9–11).

Although visual disturbances occur in 20% to 25% of patients with severe preeclampsia and eclampsia (12,13), most patients demonstrate complete resolution within several weeks. The few reported cases of ophthalmic mani-

festations in the HELLP syndrome do not describe major persistent visual loss (8,14–19). We report such a case.

CASE REPORT

A previously healthy 34-year-old white woman had preeclampsia at 33 weeks of gestation. Blood pressure was 158/94 with hemoglobin 11.7 g/dL, hematocrit 33.7%, platelet count 134,000/mL, creatine 0.8, and SGOT 28 U/L. She was treated with betamethasone 12 mg intramuscularly on admission and at 24 hours for preeclampsia and placed on bed rest. Over the next 48 hours, her blood pressure was 134 to 158 systolic, 68 to 80 diastolic.

On the third day after diagnosis, her blood pressure increased to 188/96 and she reported severe epigastric pain, nausea, and vomiting, followed by gross hematuria. Increased facial and extremity edema was noted. She then experienced a tonic–clonic seizure and was treated with magnesium sulfate 10 mg intramuscularly. At that point, she first reported blurred vision in both eyes. A laboratory workup revealed platelets 22,000/mL (150,000–400,000/mL), SGOT 2997 U/L (12–30 U/L), SGPT 1843 U/L (5–32 U/L), and lactate dehydrogenase (LDH) 2717 U/L (85–200 U/L). Based on these clinical and laboratory findings, eclampsia accompanied by the HELLP syndrome was diagnosed.

The patient was transfused with platelets, fresh-frozen plasma, and packed red blood cells before induction of labor. A healthy 4-lb 12-oz infant was delivered vaginally. On the first postpartum day, the patient had pulmonary edema, and blood gases were consistent with acidemia and hypoxia. She was treated with intravenous furosemide 20 mg intravenously and labetalol 10 mg intravenously with a peak blood pressure of 170/116. She was placed on a protocol of intravenous mannitol, labetalol, hydralazine, and sublingual nifedepine to maintain the systolic blood pressure at less than 150 mm Hg.

She was drowsy and confused but still reported persistent blurred vision in both eyes and an inability to recognize family members. A brain computed tomography (CT) showed a large left occipital hematoma with mass effect and a midline shift. These findings were confirmed on magnetic resonance imaging (MRI) 2 days later (Fig. 1), which also

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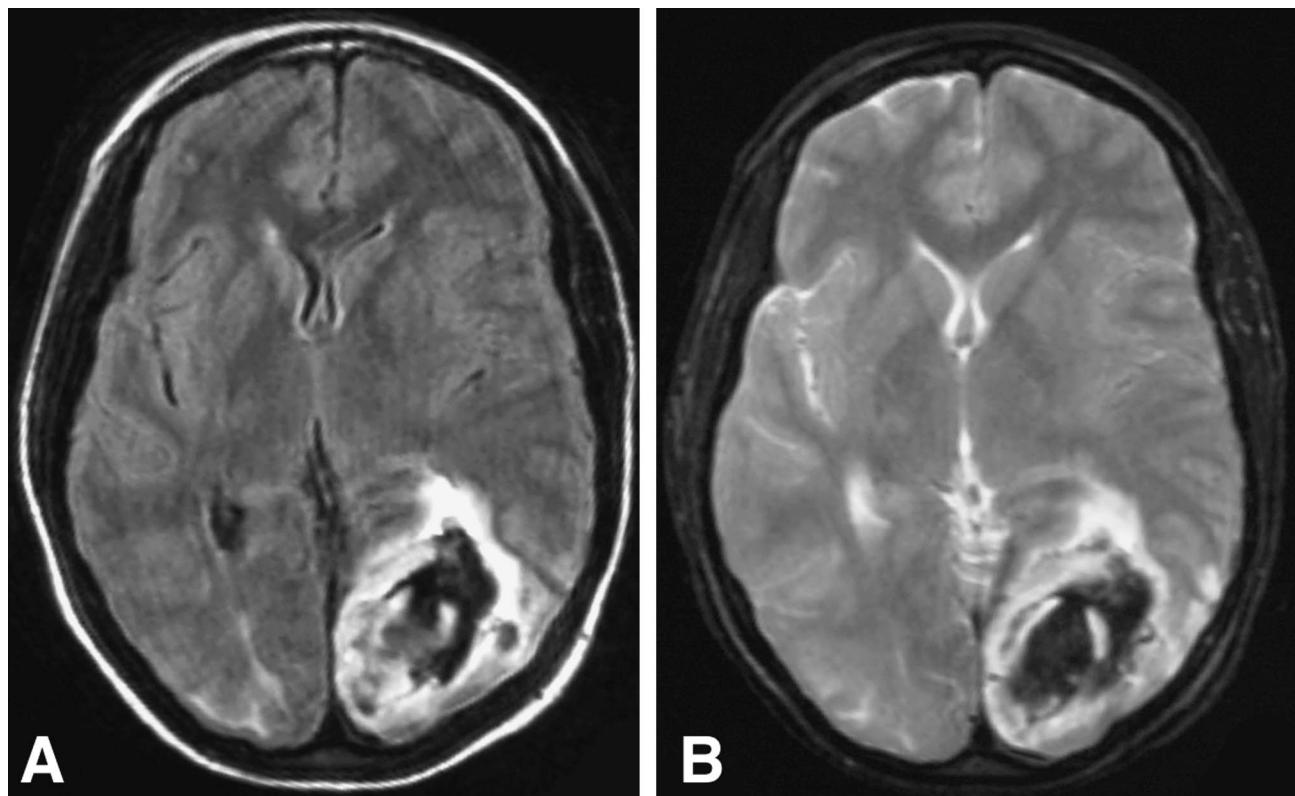


FIG. 1. FLAIR (A) and T2-weighted (B) MRI of the brain 3 days postpartum show a large hematoma in the left occipital region with midline shift and vasogenic edema in the right parieto-occipital region.

showed interstitial edema in the right parietal and occipital lobes, findings suggestive of the posterior reversible encephalopathy syndrome (PRES).

Based on laboratory findings, the patient had disseminated intravascular coagulation (DIC), with a prothrombin time (PT) 17.5 seconds (11.8–13.8 sec), partial thromboplastin time (PTT) 39.7 seconds (24–35 sec), fibrin degradation products (FDP) 40–80 $\mu\text{g}/\text{mL}$ ($<10 \mu\text{g}/\text{dL}$), and fibrinogen 94 mg/dL (194–400 mg/dL). A chest x-ray confirmed florid pulmonary edema, and an echocardiogram revealed a severe cardiomyopathy with an ejection fraction of 26%. Over the next several days, her laboratory findings, including platelet counts and liver enzymes, began to normalize. With aggressive diuresis and cardiopulmonary monitoring, her cardiac ejection fraction improved from 26% to 55% with normalization of blood pressure.

On the fifth postpartum day, visual acuity was counting fingers OD and hand movements OS. Confrontation visual fields revealed a right homonymous hemianopia and a central scotoma OS. The anterior segment examination was unremarkable.

Ophthalmoscopy OD showed a superior disc hemorrhage and diffuse intraretinal hemorrhages (Fig. 2). Ophthalmoscopy OS disclosed optic disc edema, diffuse

intraretinal hemorrhages, a preretinal hemorrhage inferonasal to the optic disc, and a large preretinal hemorrhage overlying the macula (Fig. 2). A subsequent Humphrey visual field 30–2 confirmed the right homonymous hemianopia with a central scotoma OS (Fig. 3).

Over the next 3 weeks, the patient's general condition gradually improved, with partial resolution of the left occipital hematoma by CT. A bedside neuropsychologic evaluation 3 weeks postpartum revealed inattention, perseveration, and uncertainty in making simple decisions, impaired visuospatial ability and memory, achromatopsia, and visual object agnosia. She could identify objects tactiley, although inconsistently. Her copying of a complex figure was impaired, with mild distortions and a significant number of missing elements.

Four months postpartum, the patient's visual acuity had improved to 20/25 OD and 10/180 OS. The large preretinal hemorrhage in the OS appeared less dense and was noted to extend into the vitreous. The patient had regained her ability to identify colors and was now able to read simple children's books but not newspapers. She continued to have difficulty with visual recognition of objects. She could not recognize familiar individuals on sight but could recognize them once they spoke (prosopagnosia). A brain MRI (Fig. 4) showed an evolving hematoma with

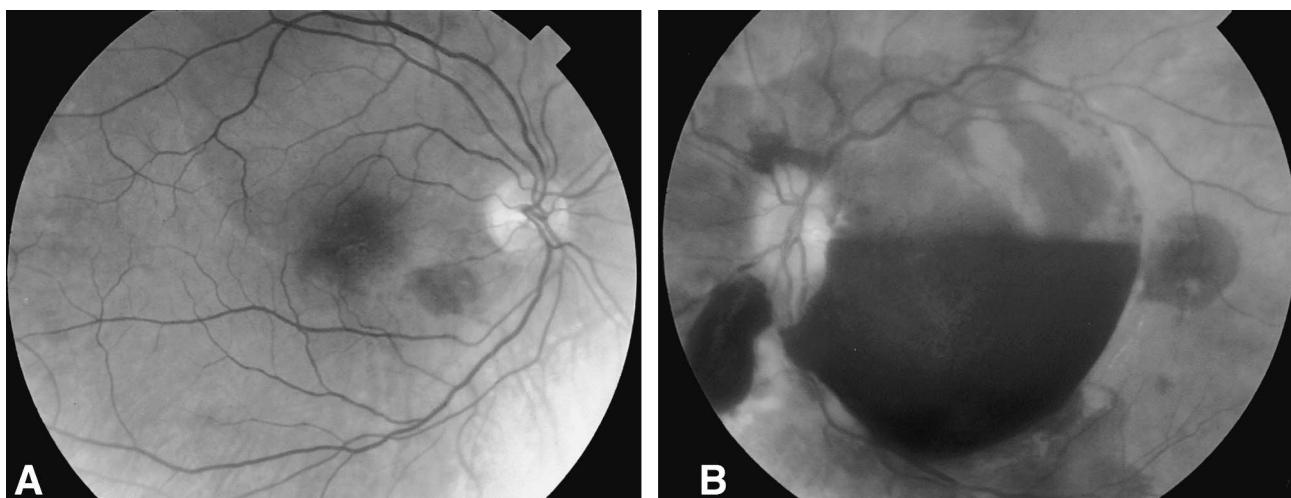


FIG. 2. Fundus photographs 3 weeks postpartum. There is an epiretinal membrane adjacent to the fovea with a small intraretinal hemorrhage OD. There are extensive intraretinal hemorrhages and a large preretinal hemorrhage overlying the macula OS.

encephalomalacia in the left occipital lobe and resolution of the vasogenic edema in the right parietal and occipital lobes consistent with PRES.

One year postpartum, the patient's visual acuity was 20/25 OD and 20/100 OS. Ophthalmoscopy OS showed diffuse pigment mottling in the macula in the area of the previous preretinal hemorrhage. A Humphrey visual field 30-2 showed a persistent right homonymous hemianopia, denser superiorly, and a central scotoma OS. Based on her neurovisual deficits, the patient remained permanently disabled from her previous job and continued to require assistance in caring for her family.

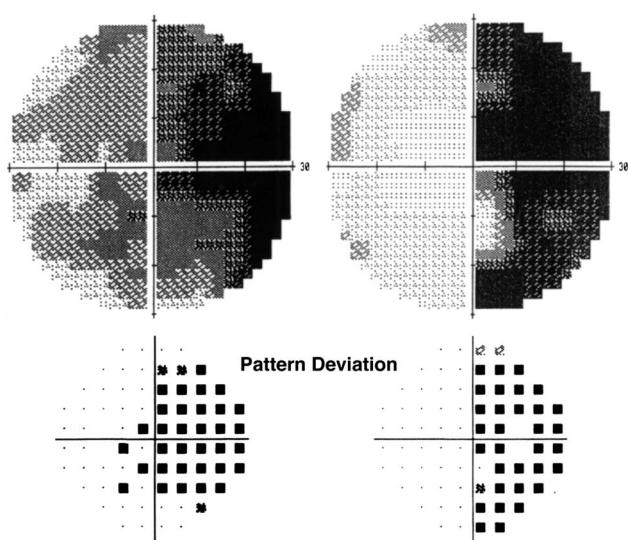


FIG. 3. Humphrey 30-2 visual fields 8 weeks postpartum show a right homonymous hemianopia with a central scotoma OS.

DISCUSSION

Our patient with eclampsia and the HELLP syndrome had a persistent right homonymous hemianopia from a hemorrhagic occipital lobe infarct, subnormal visual acuity OS caused by a retinal hemorrhage, and visual recognition deficits attributed to posterior bihemispheric cerebral ischemia.

Although visual scintillations, visual perceptual impairments, and visual loss are well-recognized manifestations of preeclampsia/eclampsia (20-22), they are usually transient. These ophthalmic manifestations are attributed to PRES, characterized by headaches, altered mental status, seizures, and visual loss associated with predominantly posterior cerebral hemispheric vasogenic edema involving both gray and white matter (22-24). Although PRES is most commonly associated with hypertension, eclampsia, and preeclampsia, it has also been reported after treatment with various drugs, including cyclosporine A and tacrolimus (22).

The main cause of PRES is acute elevation of blood pressure above the upper limit of cerebral blood flow autoregulation (23). Severe hypertension is not mandatory for PRES to develop, and previously normotensive individuals can have signs of encephalopathy at blood pressures as low as 160/100 (22). The pathophysiology of PRES is also thought to be related to endothelial dysfunction, especially in cases without severe hypertension (23,25). The relatively selective involvement of the posterior cerebral areas may reflect a major susceptibility of this region because of a lesser degree of adrenergic innervation supporting circulatory autoregulation (26,27).

The characteristic imaging pattern in PRES is edema involving the posterior portions of both cerebral hemispheres, especially the parieto-occipital regions, in a relatively

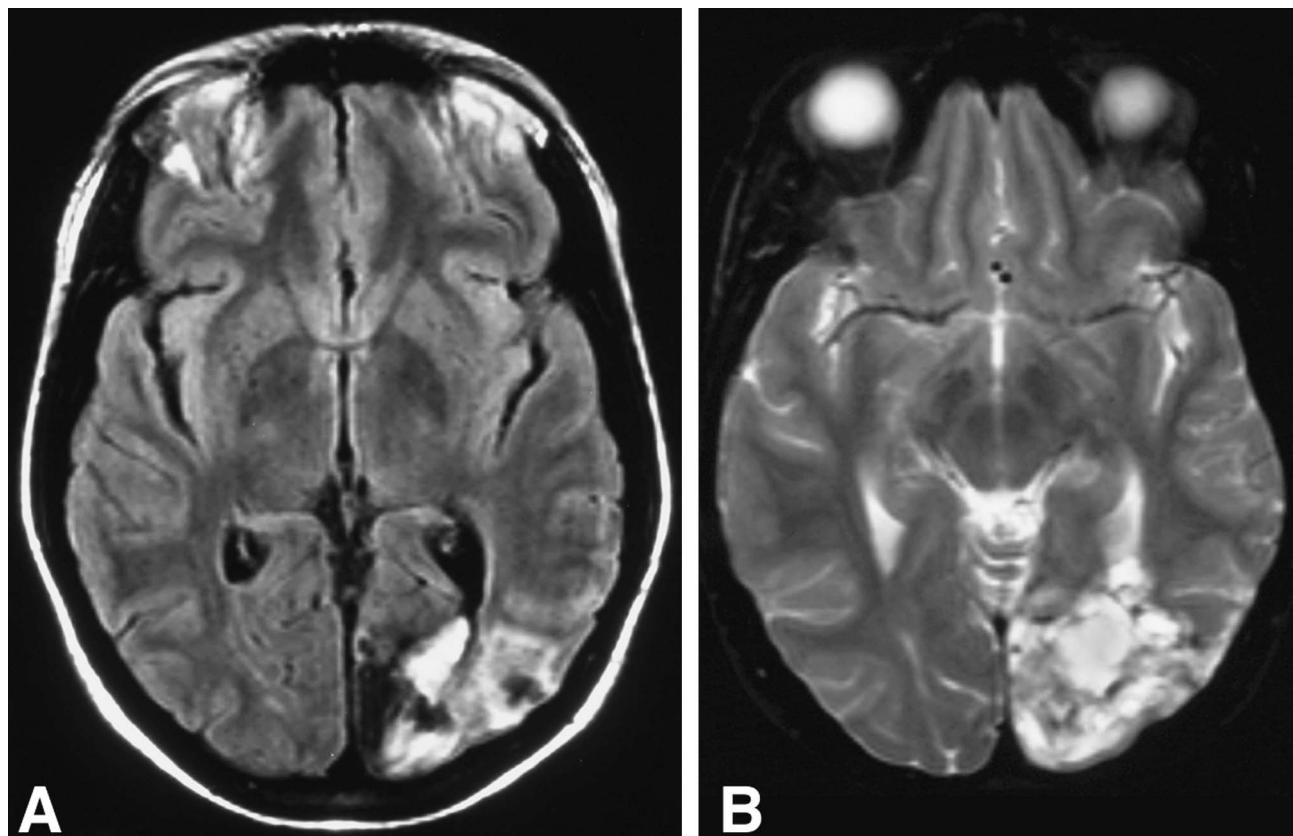


FIG. 4. FLAIR (A) and T2-weighted (B) MRIs of the brain 4 months postpartum show an evolving hematoma in the left occipital lobe with encephalomalacia and resolution of the vasogenic edema in the right parieto-occipital region.

symmetric pattern (22). Although the abnormalities primarily affect the subcortical white matter, the cortex and basal ganglia may also be involved (28,29). MRI shows punctate or confluent areas of increased signal on proton-density and T2-weighted images, and fluid-attenuated inversion recovery (FLAIR) sequences improve the ability to detect subtle signal changes (24). In most patients who have follow-up CT or MRI, these findings regress after appropriate therapy, suggesting transient edema rather than true infarction. However, initial differentiation between a reversible and permanent parenchymal lesion is not possible on the basis of conventional MRI or CT scans (22). Clinical signs and symptoms typically resolve before the imaging abnormalities disappear (28,30,31). Servillo et al (32) reported four cases of PRES in critically ill obstetric patients with eclampsia. In the three surviving patients, neurologic and neuroimaging abnormalities completely or almost completely resolved on follow-up MRI within 1 week. The fourth patient had a subarachnoid hemorrhage related to uncontrolled hypertension and died 2 weeks later.

From 4% to 12% of patients with preeclampsia or eclampsia have the HELLP syndrome (8,11,33). Patients may display one or more aspects of the syndrome: micro-

angiopathic hemolytic changes, elevated liver enzymes, and low platelets. Incomplete expression occurs in 50% of HELLP syndrome patients and does not carry the same risks as complete expression (34). The latter group tends to have a more severe disease process as manifested by an increased risk of postpartum hemorrhage and more complicated recovery (8,34). The diagnosis of HELLP syndrome in the 15% of patients without underlying preeclampsia is often delayed, leading to increased morbidity and mortality in this subgroup (5,8).

Although various investigators agree on the general definition of the HELLP syndrome, they disagree on the precise diagnostic criteria, making comparisons difficult between centers with regard to onset, diagnosis, management, and perinatal outcome (4–6). Patients with the HELLP syndrome are typically multiparous white women with a mean age of 25 years (5,8). Symptoms develop during the second or third trimester. HELLP occurs antepartum in 69% to 80% of cases, the remainder occurring between 48 hours to 7 days after delivery (5,8,34). The majority of cases initially worsen (22).

The presenting symptoms of HELLP syndrome include generalized malaise, nausea and vomiting, right upper

quadrant or epigastric pain, headache, visual changes, and shoulder or neck pain (10,34,35). Weight gain and edema are seen in approximately 50%, with hypertension present in two-thirds of patients (7).

The precise pathogenesis of the HELLP syndrome has not yet been determined. However, the prevailing theory (25,36) is that endothelial injury leads to fibrin deposition, causing activated platelets to release vasoconstrictive substances, leading to further platelet aggregation and consumption at sites of endothelial damage. This leads to the microangiopathic hemolytic changes seen with this syndrome.

Patients with HELLP syndrome are at significant risk for pulmonary edema, adult respiratory distress syndrome, placental abruption, DIC, ruptured liver hematomas, and acute renal failure (8). Stroke is the most common cause of death, with thrombocytopenia and cerebral vasospasm considered major risk factors (4,5,8,15). Given the multiple organ complications and the high maternal and perinatal morbidity and mortality associated with the HELLP syndrome, prompt diagnosis and treatment are essential (35). The reported risk of recurrent HELLP syndrome in future pregnancies is between 3% and 19%, with the risk of recurrent eclampsia or preeclampsia approximately 20% (37,38).

Whether the posterior hemispheric abnormalities in patients who have eclampsia and HELLP are more likely to lead to brain infarction is unclear. Feske et al (39) reported extensive reversible brain lesions in the posterior circulation on MRI in a patient with eclampsia and HELLP syndrome. That patient's neurologic examination was normal at discharge 4 days after admission, and follow-up MRI 1 week later demonstrated nearly complete resolution of the T2-weighted signal abnormalities in the occipital lobes, thalamus, midbrain, pons, and cerebellum.

Drislane et al (40) reported multifocal cerebral hemorrhages in a case review of four patients with preeclampsia or eclampsia, one of whom also had HELLP. All four patients had scans showing at least two sites of hemorrhage in the posterior cerebral areas; none had solely anterior hemorrhages. Two had bilateral occipital or parieto-occipital hemorrhages and two patients had normal scans initially, with hemorrhages visible on subsequent studies. The one patient who also had HELLP had the most prominent hemorrhage in the posterior cerebral hemispheres, similar to the findings in our patient. Of the three survivors in that study, two had mild residual neuro-ophthalmologic deficits (bilateral ptosis in one and a small, asymptomatic left visual field defect in another) at a 4-month follow-up examination. The third survivor had severe cognitive and visuospatial deficits at 3 months follow-up (40). No details regarding the ophthalmologic examination or visual fields were provided.

Our patient's striking hemorrhagic complications in both eyes and brain likely resulted from the combination of

acute severe hypertension and the marked thrombocytopenia, DIC, and hemolysis of the HELLP syndrome. Notably, the HELLP syndrome patient reported by Feske et al (39) had PRES but no brain hemorrhages. The difference in the outcome between their patient and ours might be attributed to our patient's significantly lower platelet count (21,000/mL versus 81,000/mL) and higher blood pressure (maximum systolic 190/diastolic 116 versus 172/90).

Among the few reported cases of visual complications in patients with the HELLP syndrome (8,14–19), none has had permanent visual deficits. Burke et al (14) described a case associated with bilateral serous retinal detachments, unilateral vitreous hemorrhage, and dural venous sinus thrombosis, but with complete recovery in all respects. In a series of 442 patients, Sibai et al (8) reported transient serous retinal detachment in 0.9%. There are two reported cases of acute cortical blindness in patients with preeclampsia and HELLP syndrome with full visual recovery 3 to 7 days postpartum (15,16). Some investigators report retinal edema with hemorrhages showing spontaneous resolution after delivery (17). Gonzalvo et al (18) described a patient with the HELLP syndrome who had a central retinal vein occlusion 10 days after delivery. The visual symptoms and ophthalmoscopic findings spontaneously resolved after 2 months. Hashiguchi et al (19) presented the only previously reported case of the HELLP syndrome with an intracerebral hematoma and associated diffuse cerebral edema. Fifteen days after evacuation of the hematoma, no focal neurologic deficits were noted. However, after experiencing a hypertensive crisis and a convulsive seizure, the patient entered an irreversible coma with fixed and dilated pupils.

Corticosteroids (41–44), plasmapheresis (6,45), and expectant management (46) are all therapeutic modalities undergoing investigation for treatment of the HELLP syndrome. To date, prompt delivery is the only intervention known to improve its clinical course (35,47). Given the poor maternal and fetal outcomes associated with the HELLP syndrome and the potentially devastating visual and systemic complications, early identification of women at risk is essential.

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