

Characteristic Magnetic Resonance Imaging for Neurological Assessment in HELLP Syndrome with Eclampsia: A case report

Shinichi FUKUHARA, Keisuke MIGITA, Koji IIDA, Kiyoshi OKABAYASHI, Takeshi INOUE,
Yasumasa IWASAKI, Seishi WADA, Takao YAMANOUE and Minako OHTANI

*Department of Emergency and Critical Care Medicine, Hiroshima University, School of Medicine, 1-2-3
Kasumi, Minami-ku, Hiroshima 734-8551, Japan*

ABSTRACT

HELLP syndrome is a complication of pregnancy characterized by hemolytic anemia, elevated liver enzymes, and low platelets. We describe a patient who suffered a generalized tonic seizure as a clinical onset. Magnetic resonance imaging (MRI) of the brain showed high intensity lesions on T2-weighted images and low- and iso- intensity on T1-weighted images. The patient fully recovered and on follow-up MRI the abnormal lesions were almost totally resolved. The sensitivity and specificity of MRI is useful in distinguishing this complication of pregnancy from cerebral apoplexy, brain tumor, epilepsy and other causes.

Key words: HELLP syndrome, Magnetic resonance imaging

HELLP syndrome (hemolysis, elevated liver enzymes, low platelets) is an atypical form of preeclampsia. Its pathogenesis is unclear, but it is considered to be a thrombotic microangiopathy due to a disturbance of endothelial cell function. The incidence of this syndrome among patients with eclampsia is 30%⁸. Manifestations of preeclampsia/eclampsia in the central nervous system (CNS) are visual disturbance, blindness, seizures, headaches, and amnesia. Recently, Magnetic Resonance Imaging has been employed for assessment and it shows a high sensitivity relative to CT⁷.

Case reports previously described MRI findings in HELLP syndrome^{1,2,9}, and in this report we present the initial and follow-up MRI findings in a case with this syndrome.

CASE REPORT

A previously healthy 27-year-old woman, gravida III, para II, experienced epigastralgia and vomiting at 40 wk. of gestation. She was admitted to the local hospital and an emergency cesarean section was performed due to suspected HELLP syndrome, as evidenced by elevated liver enzymes on examination of blood sample. Before cesarean section, the patient had a generalized seizure. A live 2380g baby was delivered by cesarean section and the next day, the patient experienced a second generalized seizure. A head CT showed a low density area at the bilateral basal ganglia. Thrombocytopenia suddenly deteriorated, and the patient was transferred to our intensive care unit. On physical examination, her blood pressure was 147/94 mmHg. Neurologically, her Glasgow Coma

Scale was 7 (E2; M4; V1), but no marked cranial nerve symptoms or paralysis were observed. Laboratory analysis revealed elevated liver enzymes (AST, 858 IU/liter; ALT, 394 IU/liter), a low platelet count (2.9×10^4 /ml), a decreased hemoglobin concentration (7.9 g/dl), prolonged PT activity (52%), and an elevated fibrin/fibrinogen degradation products level (81.08 μ g/ml). Schistocytes were found in a blood smear. The above findings were consistent with HELLP syndrome.

A head MRI revealed high intensity lesions in the bilateral caudate nuclei, putamen, internal capsule, and gray matter on the T2-weighted images (T2W1), and low- and iso- intensity lesions on the T1-weighted images (T1W1) (Fig. 1. a-f).

On the day of admission, she was given midazolam, glycerol, steroids, and a Ca blocker. A plasma exchange was performed for two days. She recovered to normal neurological status 3 days after admission. On follow-up MRI 13 days after admission, the previous areas of abnormal signals were reduced (Fig. 2. a-d), and on follow-up MRI 48 days later, the abnormal lesions were virtually resolved.

DISCUSSION

The pathogenesis of neurological disorders related to HELLP syndrome and eclampsia remains unclear. Endothelial cell injury with subsequent vasospasm and platelet activation plays a central role in the pathogenesis³. Neuropathologic studies revealed small vessel wall damage with fibrinoid necrosis, hypoxic-ischemic brain lesions, focal or diffuse cerebral edema and hemorrhages of differ-

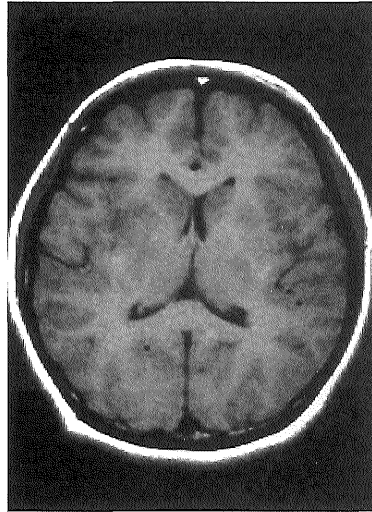


Fig. 1 (a)

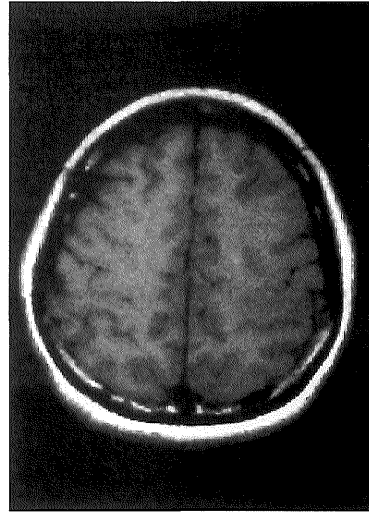


Fig. 1 (b)

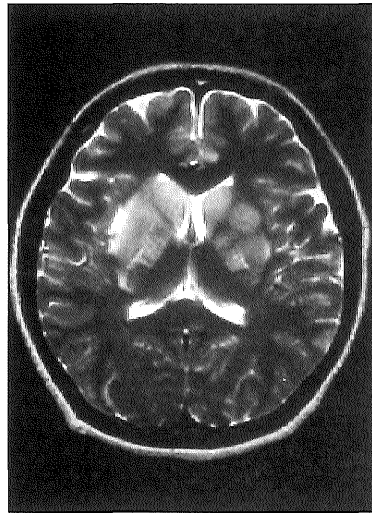


Fig. 1 (c)

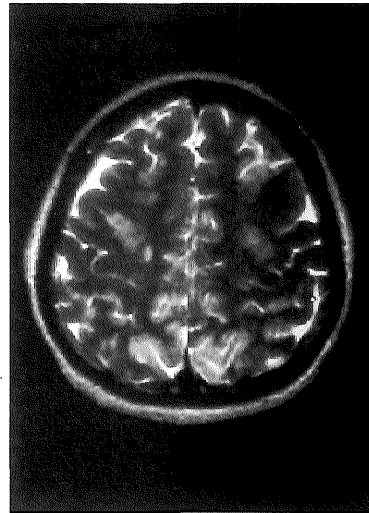


Fig. 1 (d)

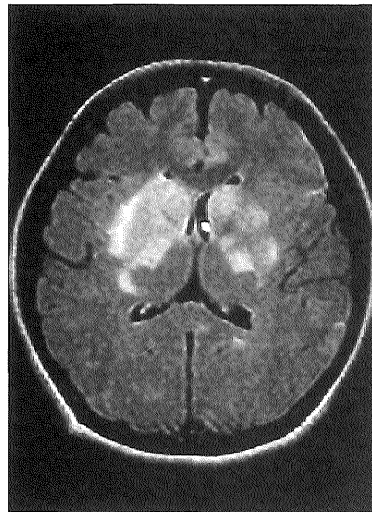


Fig. 1 (e)

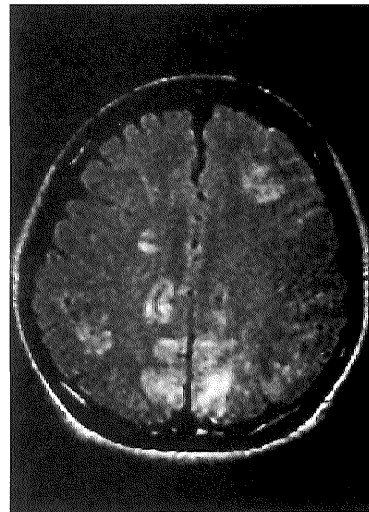


Fig. 1 (f)

Fig. 1. a-f. Initial MR examination performed on day 0.

Transverse T1 (a,b) -weighted images, T2 (c,d)-weighed images and FLAIR (e,f) images.

Hypointense lesions in basal ganglia (Rt.caudate nuclei and putamen) on the T1-weighted images (a,b).

Hyperintense lesions in basal ganglia (bilateral caudate nuclei, putamen and internal capsule) and parietoanterior cortex on the T2-weighted images (c,d) and FLAIR images (e,f).



Fig. 2 (a)

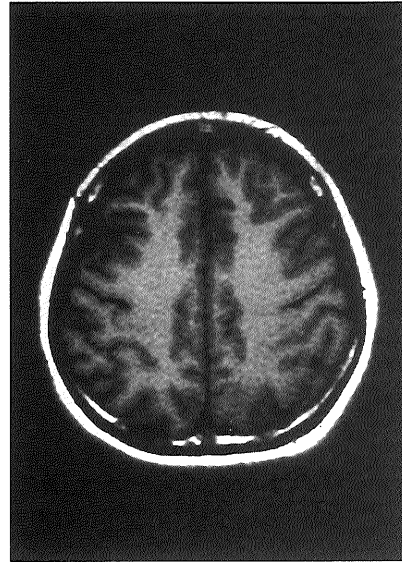


Fig. 2 (b)

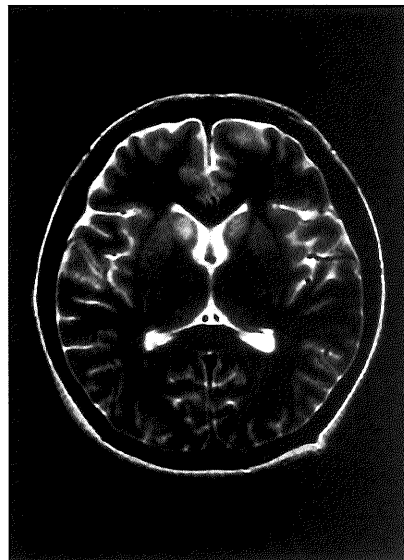


Fig. 2 (c)



Fig. 2 (d)

Fig.2. a-d. Follow-up MR examination performed on day 13. Same level as Fig.1 Transverse T1 (a,b)-weighted images, T2 (c,d)-weighed images. Abnormal lesions at initial MRI are reduced.

ent size and location⁶). Cerebral angiography disclosed cerebral vasospasm *in vivo*⁴.

In previous reports, MRI in the acute stage of HELLP syndrome with neurological symptoms or eclampsia has been reported to show high intensity areas on T2W1 and low- or iso-intensity areas on T1W1^{2,8,9}. Our MRI study revealed multiple areas with high intensity signals on T2 and low- and iso-signal-intense T1 signals consistent with ischemia or edema.

These MRI findings thus correlate well with clinical symptoms at the acute stage, and MRI is useful in evaluating those symptoms. The typical diagnosis of HELLP syndrome, based on lab findings of hemolysis, elevated liver enzymes and thrombocytopenia, could therefore be augmented by MRI findings.

Despite wide-spread brain abnormalities, follow-up MRI showed almost full resolution. Reversible ischemia or edema may be a contributing cause of cerebral dysfunction in eclampsia and HELLP syndrome. Previous reports have shown that abnormal areas disappeared at the chronic stage and, as in the case of our patient, each patient recovered.

The sensitivity and specificity of MRI may be of clinical value in distinguishing between eclampsia and other causes of seizure or other neurologic symptoms, such as apoplexy, brain tumor and epilepsy. MRI may thus be the technique of choice in diagnosing pregnant women with these symptoms.

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