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Clinical and radiological features of posterior reversible encephalopathy syndrome in patients with pre-eclampsia and eclampsia

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AIM: To analyse and summarise clinical and radiological features among patients with posterior reversible encephalopathy syndrome (PRES), to assess related factors with eclampsia and pre-eclampsia, and to compare the different factors between cytotoxic and vasogenic oedema among PRES patients.

MATERIALS AND METHODS: The clinical and radiological findings of 237 pre-eclamptic or eclamptic patients with neurological symptoms were evaluated retrospectively. Multiple logistic regression analyses were performed to compare the differences among these parameters.

RESULTS: Seventy-six patients (32.07%) were diagnosed with PRES. Multiple logistic regression indicated that seizure (odds ratio [OR], 2.760; 95% confidence interval [CI]: 1.087–7.011; p=0.033), visual disturbances (OR=2.062 95%CI, 1.033–4.115; p=0.004), multiple production history (OR=3.637; 95% CI: 1.068–8.228; p=0.002) were independent risk factors for PRES. PRES+ (OR=3.217; 95%CI, 1.346–7.686; p=0.009), Visual disturbances (OR=4.283; 95% CI: 1.843–9.953; p=0.001) had strong association with eclampsia. Visual disturbances (OR=7.200; 95% CI: 2.116–24.496; p=0.002) had strong correlation with eclampsia among PRES+ patients. Visual disturbances (OR=2.947; 95% CI: 1.135–7.648; p=0.026) were independently related to cytotoxic oedema.

CONCLUSIONS: Nearly one-third of pre-eclampsia or eclampsia patients with neurological symptoms have PRES. Visual disturbances, seizure, multiple production history are independent risk factors for PRES. Visual disturbances have a strong association with eclampsia whether patients have PRES or not. Visual disturbances are independently related to cytotoxic oedema among PRES+ patients.

Introduction

Posterior reversible encephalopathy syndrome (PRES), a condition first described by Hinchey *et al.* in 1996, is a clinicoradiological entity characterised by a collection of signs and symptoms: impaired consciousness, visual changes, acute-onset seizures, headache, nausea, and vomiting, and focal neurological signs.[1] Magnetic resonance

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imaging (MRI) evaluation of patients with PRES reveals bilateral and symmetrical vasogenic oedema involving the cortical and subcortical regions of the occipital and parietal lobes, although some patients present with atypical distributions including the anterior cerebral lobes, brain stem, cerebellum, and basal ganglia (BG).[2–6] The factors predisposing to PRES in patients with pre-eclampsia and eclampsia are still under debate, and the MRI findings of some patients demonstrate cytotoxic rather than the more typical vasogenic oedema.[7]

Pre-eclampsia and eclampsia, conditions that predispose to PRES, are characterised by clinical hypertension, peripheral oedema, proteinuria, and sometimes seizure during pregnancy; pre-eclampsia and eclampsia have a similar mechanism to PRES. The mechanism by which PRES occurs is still not clear, but there are three popular theories: hypertension-induced vasoconstriction, ischaemia and vasospasm, and endothelial injury.[2,8–12] Patients without hypertension can still develop PRES.[4] The reported prognosis of pre-eclampsia or eclampsia patients with PRES is relatively favourable. In the majority of patients, PRES is completely reversible with or without restricted cytotoxic oedema.[13]

The aim of the present study was to assess the incidence and risk factors of PRES in a large sample of patients with pre-eclampsia or eclampsia, and to evaluate clinical and radiological features among PRES patients. Related factors with eclampsia or pre-eclampsia were also assessed and the different factors between cytotoxic and vasogenic oedema were compared among PRES patients.

Materials and methods

Patients and study protocol

A hospital-based retrospective review of women with neurological symptoms who were diagnosed with pre-eclampsia or eclampsia between 2010 and 2015 was conducted. All participants provided written informed consent prior to inclusion in the study. The diagnostic criterion for pre-eclampsia was a first instance of blood pressure (BP) $\geq$140 mmHg or a diastolic BP (DBP) $\geq$90 mmHg occurring after 20 weeks of gestation together with proteinuria, >300 mg/24 h; eclampsia was defined as the occurrence of seizures that could not be attributed to other causes in women with pre-eclampsia.[14] The diagnostic criteria for PRES were: (1) acute or subacute onset of at least one neurological symptom such as headache, dizziness, seizure, visual disturbance, or impaired consciousness; (2) MRI showing focal bilateral oedema of the cortex and subcortical white matter; (3) complete resolution of most or all clinical symptoms and radiological findings during pregnancy or post-partum; (4) no other disease that could have similar manifestations such as acute stroke, cerebral venous-sinus thrombosis, viral encephalitis, and immune disorder associated encephalopathy.

The BP value was recorded, if documented, at the onset of neurological symptoms or at the time of admission for those patients who were not admitted to the hospital when their neurological symptoms began. Mean arterial pressure (MAP) was also investigated at: two-thirds of the DBP and one-third of the SBP. Data were collected on clinical parameters (patient age, SBP, DBP, mean blood pressure [MBP]), routine laboratory investigations (haemoglobin, platelet count, D-dimer level), neurological symptoms (headache, seizures, visual disturbances), and MRI findings.

Neuroimaging

MRI using a 3 T magnet was used in all patients. The standard protocol consisted of axial T1- and T2-weighted sequences, fluid-attenuated inversion recovery (FLAIR) sequences, and diffusion-weighted imaging (DWI). Apparent diffusion coefficient (ADC) maps were calculated on a pixel-by-pixel basis. The mean ADC values for each region of interest were calculated automatically.

All MRI results were evaluated by two neuroimaging professors blinded to the clinical information. Patients were divided into two groups according to MRI findings, the PRES+ group and the PRES− group, depending on the presence of high signal intensity on T2-weighted imaging and FLAIR. The PRES group was further divided into those patients with cytotoxic oedema and those with vasogenic oedema: a high DWI signal combined with a decreased ADC indicated the presence of cytotoxic oedema, while a low DWI signal with increased ADC indicated vasogenic oedema. Patients' clinical features, imaging lesions, and laboratory data were collected and compared to determine whether there were any differing factors related to these two different types of oedema. The involved lesions were summarised and compared between PRES patients with pre-eclampsia versus eclampsia.

Treatment and follow-up

All patients were given magnesium sulphate for seizure prophylaxis, as 20 ml of a 25% magnesium sulphate solution diluted in a 10% glucose solution, given intravenously over 5–10 minutes. Patients were monitored for signs of toxicity: absent patellar reflex, respiratory rate <16/min, or urine output <25 ml/h.[15] Patients with a SBP $\geq$160 mmHg, DBP $\geq$110 mmHg, or MAP $\geq$140 mmHg received intravenous labetalol: 20 mg as a first dose, followed by 40 mg if not effective within 10 minutes, then 80 mg every 10 minutes to a maximum total dose of 240 mg.[14] In patients with eclampsia, seizures were controlled with 10 mg diazepam; if this was ineffectual, they received phenytoin, 100 mg every 8 or 12 hours. Thirty patients underwent emergency termination of pregnancy for insufficient seizure control. All PRES patients were followed for 1 month for the persistence of neurological symptoms and for follow-up MRI findings.

Data analysis

Statistical analyses were conducted using Statistical Package for the Social Sciences, version 17.0. Continuous data were reported as the mean ± standard deviation;

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