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Good outcome after posterior reversible encephalopathy syndrome (PRES) despite elevated cerebral lactate: a case report

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Abstract

Posterior reversible encephalopathy syndrome (PRES) may cause irreversible brain damage. The diagnosis is confirmed by magnetic resonance imaging (MRI), where vasogenic edema may be seen especially in the posterior parts of the brain. MR spectroscopy (MRS) may be included to help predict the outcome by measuring selected metabolites for instance lactate. Usually lactate is immeasurable in brain tissue, but elevates in cases of hypoxia, and it has been associated with poor outcome. We report a case of a patient with eclampsia and PRES, who had elevated lactate initially, but complete remission clinically and on MRI.

Keywords

Magnetic resonance spectroscopy, central nervous system, brain, brain stem

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Introduction

Posterior reversible encephalopathy syndrome (PRES) also referred to as reversible posterior leukoencephalopathy syndrome (RPLS) has been associated with accelerated hypertension, eclampsia, autoimmune disease, and various immunosuppressive or chemotherapeutic treatments, although the genesis is still unknown (1–4). The clinical symptoms are headache, visual disturbance, and seizures. Magnetic resonance imaging (MRI) confirms the diagnosis, illustrating vasogenic edema mainly in the posterior parts of the brain. MR spectroscopy (MRS) may be included to illustrate permanent tissue damage and predict the outcome (5). MRS depicts the amount of metabolites as lactate, N-acetylaspartate (NAA), total choline (Cho), myo-inositol (mI), and total creatine (Cr) in brain tissue, often in ratios to Cr (6). NAA is considered a marker of viable neurons, Cho is associated with degeneration of myelin and membranes or altered membrane turnover, mI is a glia marker and Cr is related to energy metabolism (6). Lactate is usually immeasurable in brain, but it elevates in cases of hypoxia, mitochondrial defects, anaerobic metabolism or accelerated glucose turnover. Elevated lactate may also be a transient finding caused by a reversible physiological condition such as hyper-ventilation or may be seen in patients with mitochondrial diseases or toxic encephalopaties.

We present a case with PRES in relation to eclampsia with elevated lactate on MRS initially, but with complete clinical and MRI remission. Minor MRS abnormalities in affected tissue remained.

Case presentation

A 26-year old woman, pregnant in week 33 + 2, with gestational diabetes and hypertension, was admitted to hospital after 3 days of progressively deteriorating consciousness. The patient had been treated with

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antihypertensive medication, but had stopped it on her own initiative. The morning of admittance she had experienced paresis of the left arm, cortical blindness and seizures. Glasgow Coma Score (GCS) was 10–14. Blood sugar was elevated to 10.1 mmol/L (reference interval, 4–7 mmol/L). Systolic blood pressure was higher than 200 mmHg, she had proteinuria and eclampsia was suspected. Blood samples showed elevated lactate dehydrogenase (LDH). Computed tomography (CT) of the brain showed cortical hypodensities bilaterally. No signs of intracranial bleeding. She received immediate treatment with an antihypertensive agent and magnesium. Acute Cesarean section was performed, followed by MRI and short echo time MRS at 3 Tesla with general anesthesia. MRI showed vasogenic edema cortically and subcortically in the occipital, parietal, and frontal lobes bilaterally and small petechial bleedings in the right occipital lobe. All in agreement with the diagnosis PRES. MRS was performed in three regions of interest: an occipital white matter location with vasogenic edema (VE), in mid-occipital grey matter (GM) partially affected by vasogenic edema, and in a contralateral occipito-parietal white matter (WM) location almost unaffected by vasogenic edema (Fig. 1). MRS showed significantly increased lactate in VE. Glucose was generally elevated due to the gestational diabetes. NAA/Cr, Cho/Cr, and mI/Cr were normal in all three locations. Three days later, the patient was awake and relevant, with a lower blood pressure, but still with partial paresis of the left arm. Eight days after hospital admittance, the patient's symptoms had disappeared and a follow-up MRI including MRS was performed. The vasogenic edema was regressing, but the radiological appearance had not normalized entirely. MRS in GM and WM was unremarkable and glucose was no longer detectable. The elevated lactate in VE had decreased and also NAA/Cr had decreased to a value 31% below the normal reference value. A follow-up MRI after 2 months was suggested. At this time all MRI images were normal,

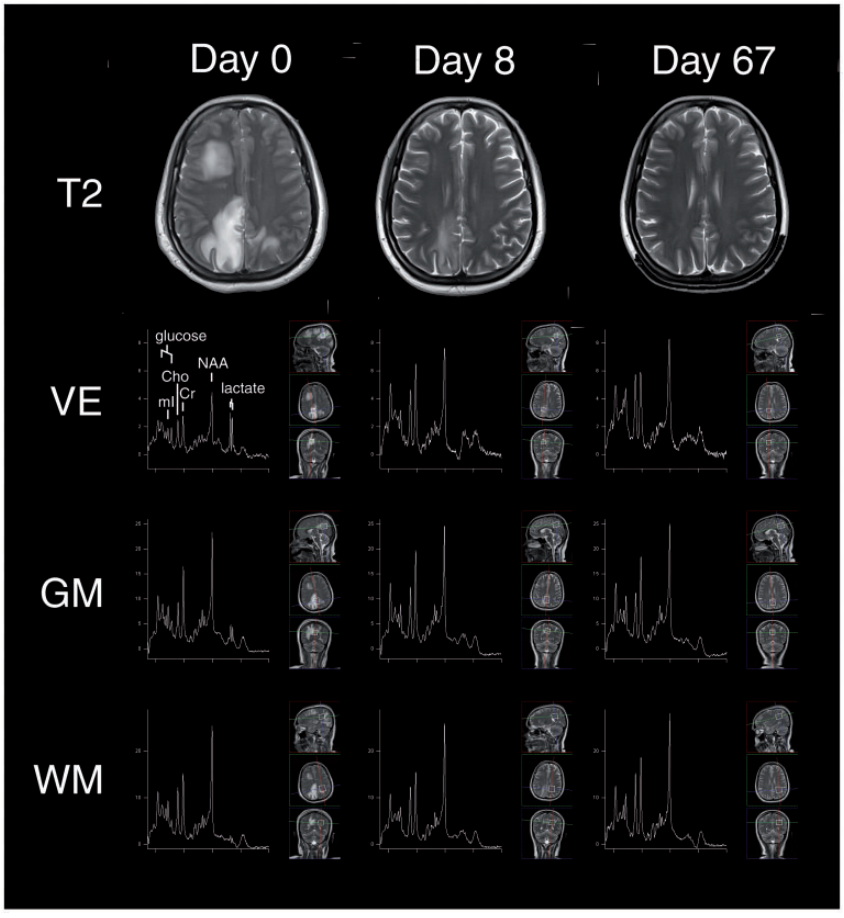

Fig. 1. T2-weighted MRI and MRS on Day 0, Day 8, and Day 67, where Day 0 is the day of hospital admittance. MRS region-of-interests were in an occipital location of vasogenic edema (VE), in a mid-occipital grey matter (GM) location partially affected by vasogenic edema, and in a contralateral occipito-parietal white matter (WM) location.

and the patient was feeling well. On MRS, lactate had disappeared from GM and initial VE, but NAA/Cr was still significantly reduced both in GM (−21%) and in initial VE (−23%). Furthermore increased Cho/Cr (+26%) and mI/Cr (+18%) were now detected in initial VE. MRS in WM remained normal.

Assuming that the Cr concentration in the almost unaffected WM did not change over time, a further analysis of Cr in VE compared to Cr in WM showed that Cr in VE was depressed by a factor 2.7 in the acute phase of vasogenic edema and returned to normal afterwards.

Discussion

We report a rare case of initially elevated lactate on MRS, which normalized completely after 2 months in a patient diagnosed with PRES. The initially elevated lactate value was probably due to acute, but reversible ischemia, which had not been present long enough to cause permanent damage. Elevated lactate has been associated with stroke and thus a poor outcome due to irreversible brain damage. Lactate has been reported to increase on *in vivo* proton MRS less than 24 h after the onset of stroke (4,7). In a retrospective paper by Kwon et al., four pediatric patients had MRS in the acute phase of PRES, and MRS revealed high lactate peak, normal NAA, and Cho. All patients had complete remission on follow-up MRS, and the transient derangement of energy metabolism suggested absence of neuronal damage and a favorable prognosis (2). Our patient had a 2.7-fold depression of Cr in VE in the acute phase, but NAA/Cr, Cho/Cr, mI/Cr were normal. This reflects that the volume concentration of all metabolites was 2.7-fold reduced. MRS measures intracellular metabolites, so most likely the 2.7-fold reduction reflects a reversible reduction of the cell volume due to vasogenic edema. Our patient had mildly increased Cho/Cr and mI/Cr in the occipital location with initial vasogenic edema (VE) after 2 months suggesting chronic membrane affection and gliosis.

NAA/Cr was slightly reduced in VE after 2 months consistent with minor but persistent neuro-axonal damage in the tissue previously affected by vasogenic edema.

Lee et al. reported decreased NAA/Cr and increased Cho/Cr at follow-up, but they did not see lactate in the acute phase (3). Eichler et al. did not report on lactate, but found decreased NAA and increased Cho in the acute phase of PRES in two patients, and at follow-up in one of the patients, metabolites were normal (1). Sengar et al. investigated 10 patients in the acute phase and after 2 weeks. They reported decreased NAA/Cr in all patients, and increased lactate in one patient, who never recovered. None of their findings were reversible (7). Russell et al. reported normal MRS in a case of PRES and Kwon et al. reported normal NAA/Cr, normal Cho/Cr, and presence of lactate in the acute phase in four patients, with reversal to normal after treatment in four PRES patients (2,5). The varying reports probably reflect varying degrees of neurological affection in combination with varying severity of the disease at the time of MRS, which was performed at different times in the papers.

The pathophysiology of PRES is still poorly understood, but it has been proposed that it may be due to a rise in blood pressure or due to endothelial dysfunction (8). Pregnancy may decrease the threshold for development of cerebral hyperperfusion and brain edema and treatment of hypertension in pregnancy may prevent progression from vasogenic to cytotoxic edema (8). Endothelial dysfuntion resulting in increased blood brain barrier permeability could permit passage of antivasogenic and antiendothelial proteins into the brain (9). Also changes in blood cell morphology and elevated LDH have been suggested as causes of PRES (10). Our patient also had elevated LDH in addition to hypertension. Treatment of the patient's eclampsia was initiated immediately upon hospital admittance with magnesium sulfate, which is a well established treatment option acting as a vasodilator, decreasing peripheral resistance and relieving vasoconstriction (9).

In conclusion, hypertension in pregnancy and eclampsia should be treated as both may lead to PRES. In unconscious patients MRS may help predicting the outcome. However, elevated lactate on initial MRS is not always a predictor of poor outcome, and the duration of symptoms must be borne in mind.

Conflict of interest

None declared.

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