Headache, Blurred Vision and Seizure in Hemodialysis Patient

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A 53-year-old man with severe headache, blurred vision, and single generalized tonic-clonic seizure during hemodialysis presented to the emergency department (ED). On admission to the ED, the patient was in postictal state and measured at the following levels: blood pressure 203/113 mmHg, pulse rate 80/min, respiratory rate of 17/min, body temperature of 36,5 °C, peripheral O2 saturation of 96%, and GCS of 9. Neurologic examination revealed no focal deficit. Bedside finger-stick glucose test was detected at 59 mg/dl. ECG of the patient was in normal sinus rhythm and revealed no ischemic sign. Intravenous 25 g glucose was administered to the patient and control of finger stick glucose test was detected at 281 mg/dl. However, the mental status of the patient did not improve. The patient had complaints of headache and blurred vision for at least one day according to family members. The patient had chronic renal failure and hemodialysis for 1.5 years and was on eye drops for glaucoma. On fundoscopic examination, acute pathology was not detected. Noncontrast enhanced cranial computerized tomography (CT) was performed and CT revealed no acute pathology. Laboratory tests of the patient showed creatinin 5.8 and urea 79 mg/dl and no electrolyte imbalance, leucositosis, or thrombocytopenia. GCS of the patient was detected at 14 without focal deficits at two hours after admission to the ED. Contrast enhanced magnetic resonance imaging (MRI) of cranial and diffusion MRI was also performed (Figure 1). **[see page 82 for diagnosis]**

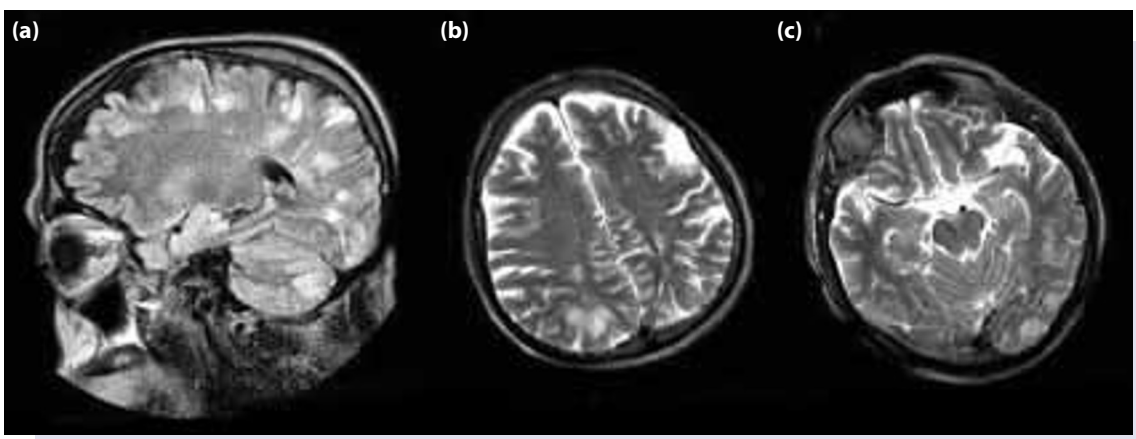

Figure 1. The patient's flair **(a)** and T2-weighted **(b, c)** cranial MRI images.

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DIAGNOSIS: Posterior Reversible Encephalopathy Syndrome

MRI revealed hyperintensity compatible with vasogenic edema on FLAIR and t2 weighted sequences in the bilateral occipital, parietal, and frontal region (Figure 2). After magnetic resonance imaging, the patient had generalized tonic-clonic seizures. Reduction of the patient's blood pressure from 203/113 mmHg to 179/95 mmHg did not cause any clinical improvement. Loading dose of valproic acid was given intravenously to the patient. The patient was intubated due to uncontrolled seizures and shallow breathing and was admitted to intensive care unit. The patient died after 6 days due to intracranial hemorrhage.

Posterior reversible encephalopathy syndrome (PRES), initially described in 1996 by Hinchey et al., is a clinical-neuroradiological entity characterized by headache, vomiting, altered mental status, blurred vision, and seizures.[1] This syndrome is most commonly encountered in association with acute hypertension, preeclampsia or eclampsia, autoimmune diseases, renal failure, post-transplantation, sepsis, shock, and exposure to immunosuppressants.[2-4] Although the lesions in PRES are due to vasogenic edema, the mechanism responsible for the imaging appearance remains unclear and controversial.[1,2] There are two main hypotheses: 1) Cerebral hypoperfusion related to disruption of the blood-brain barrier results in vasogenic edema (e.g. eclampsia/preeclampsia, cyclosporine toxicity, and infection/sepsis/septic shock), and 2) Cerebral hyperperfusion results in vasogenic edema by exceeding the capacity for autoregulation of perfusion pressure (e.g. acute hypertension).[5] In our patient, we considered that hypertension induced by renal failure led to regional dysautoregulation, consequently causing hypoperfusion.

Radiologic findings of PRES are best seen on MRI of the brain. The typical imaging findings of PRES are most apparent as hyperintensity on FLAIR and t2 weighted sequences of cranial MRI in the parietooccipital and posterior, frontal, cortical, and subcortical white matter and are reversible with appropriate management.[4,5]

The treatment is based in the management or withdrawal of the triggering factor. Hypoglycemia should be looked for routinely and corrected. Antiepileptic treatment should be initiated at the emergency department. Control of hypertensive emergency is an important part of the symptomatic management.[1,5] Mortality has been reported in 15% of patients. Cause of death in PRES may be due to the underlying disease, increased cerebral edema, and intracerebral hemorrhage.[6]

The early recognition and treatment of PRES is important to prevent permanent neurological sequelae. Awareness of the clinical and radiographic findings of acute PRES is essential to avoid misdiagnosis and treatment delay.

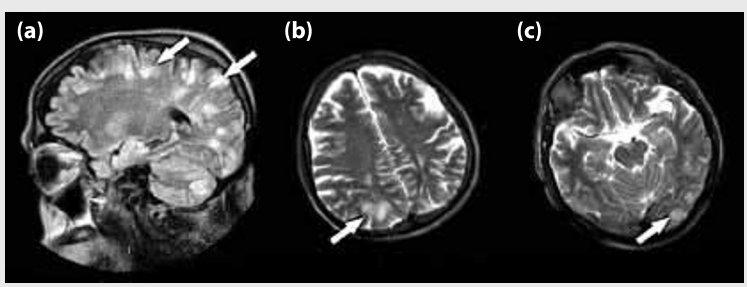

Figure 2. MRI of cranial showed hyperintensity on FLAIR **(a)** and t2 weighted sequences **(b, c)** in the posterior, frontal, occipital ve parietal region.

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