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Clinical Case

Does this patient have hypertensive encephalopathy?

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Abstract

A 63-year-old man was admitted to our hospital for further investigation and management of brain metastases. The patient was initially presented with a 4-day history of confusion. On the day of admission, the patient was confused, agitated, disorientated in place and time, and had visual disturbances. His blood pressure was repeatedly recorded high, with levels of systolic blood pressure between 170–210 mm Hg. A brain magnetic resonance imaging showed areas of high signal on T2 and fluid-attenuated inversion recovery images, located bilaterally in the white matter of the occipital regions and unilateral in the left frontal lobe, suggestive of posterior reversible encephalopathy syndrome. Aggressive treatment of hypertension resulted in complete resolution of the clinical and radiologic features of the syndrome. J Am Soc Hypertens 2016;10(5):399–403.

Keywords: Hypertension; encephalopathy; confusion; PRES.

Case Presentation

A 63-year-old man was admitted to our hospital for further investigation and management of possible brain metastases. The patient was initially presented with a 4-day history of confusion. The day previous to admission the patient was disoriented in place and experienced visual hallucinations. His family reported that during the last year he was suffering from headache but without any other symptom.

His past medical history was unremarkable except that he was infected with hepatitis B as it was evident from the positive serology for the surface antigen of the hepatitis B virus (HBsAg) but without antibodies for HBsAg. The patient did not have any clinical or laboratory evidence for cirrhosis and was not on any medication for hepatitis B. He did not measure his blood pressure regularly and was unaware of his blood pressure status. The patient consumed 2–3 units of alcohol per day and had stopped smoking 8 years ago. He was not taking any medications at all.

On the day the patient was referred to our hospital, he was afebrile and hemodynamically stable with a blood pressure of 150/90 mm Hg. The patient was confused, agitated, disorientated in place and time and had visual disturbances. His Glasgow coma scale was 14/15, but the clinical examination was unremarkable. The electrocardiogram and chest X-ray were normal. Before his referral from the district hospital, the patient had a computed tomography (CT) scan of the brain which showed multiple lesions that were interpreted as probable brain metastases. He was then started on steroids, haloperidol, and sodium valproate. On the day of admission to our hospital, the patient underwent a second brain CT scan due to worsening of his symptoms. The new CT scan confirmed the multiple lesions but without the typical signs for metastasis; magnetic resonance imaging (MRI) of the brain and a CT scan of the chest and abdomen were arranged. On the second day of admission, his systolic blood pressure was repeatedly

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recorded high, ranging between 170–210 mm Hg and amlodipine 10 mg was started. A brain MRI showed areas of high signal on T2 and fluid-attenuated inversion recovery images, located bilaterally in the white matter of the occipital regions and unilateral in the left frontal lobe, all suggestive of edema (Figure 1). Diffusion weighted imaging showed an increased signal on apparent diffusion coefficient maps and helped further to characterize the edema as vasogenic. The symmetrical appearance of vasogenic edema in the occipital regions was suspicious for posterior reversible encephalopathy syndrome (PRES).

A series of investigations were subsequently arranged for a thorough evaluation of his hypertension. Heart ultrasound revealed an enlarged ascending aorta with a diameter of 3.6 cm and mild aortic valve regurgitation. The patient had no detectable proteinuria or hematuria. An opthalmologic examination demonstrated bilateral cataracts and first-degree hypertensive retinopathy. These findings from the heart and eyes were suggestive of longstanding undiagnosed hypertension.

As the blood pressure target was not initially achieved with amlodipine monotherapy, hydrochlorothiazide, and irbesartan were subsequently added. This combination resulted in a significant reduction of his blood pressure measurements but caused a remarkable rise of more than 30% in his creatinine level (from 0.9 mg/dL to 1.5 mg/dL). We immediately suspected stenoses of the renal arteries which were confirmed by a duplex ultrasound showing bilateral renal arteries stenoses. Irbesartan was stopped and the patient started carvedilol titrated progressively to 12.5 mg twice daily and spironolactone 100 mg once daily. His blood pressure finally settled at a level of 135/85 mm Hg and the patient's neurologic symptoms progressively disappeared. The patient was discharged in a good health, free of symptoms and with normal creatinine levels (glomerular filtration rate [GFR] 100 mL/min/m^2). Ten days later, he returned to perform a CT angiography of the renal arteries, which confirmed the existence of bilateral renal arteries stenoses. A month later, a new MRI of the brain showed complete resolution of the lesions (Figure 2). At his regular follow-up, 2 months later, the patient was asymptomatic, with normal blood pressure measurements and normal creatinine levels (0.8 mg/dL). He continued his antihypertensive treatment without further changes.

Discussion

Reversible posterior leukoencephalopathy syndrome, also referred as PRES, is a clinical and radiographic syndrome of various etiologies. It has been initially described 30 years ago, but it was Hinchey who first codified it as reversible posterior leukoencephalopathy syndrome in 1996.[1] Hypertension has been implicated as the main risk factor for the presentation of PRES along with eclampsia, renal disease, as well as other etiologies unrelated to hypertension, such as immunosuppressive drugs, sepsis, and autoimmune diseases.[2–4] Its main clinical presentation includes headache, visual disturbances, seizures, and altered mental status.[2,5] The fact that these symptoms are not specific for PRES highlights the importance of a focused clinical approach along with advanced CT and MR

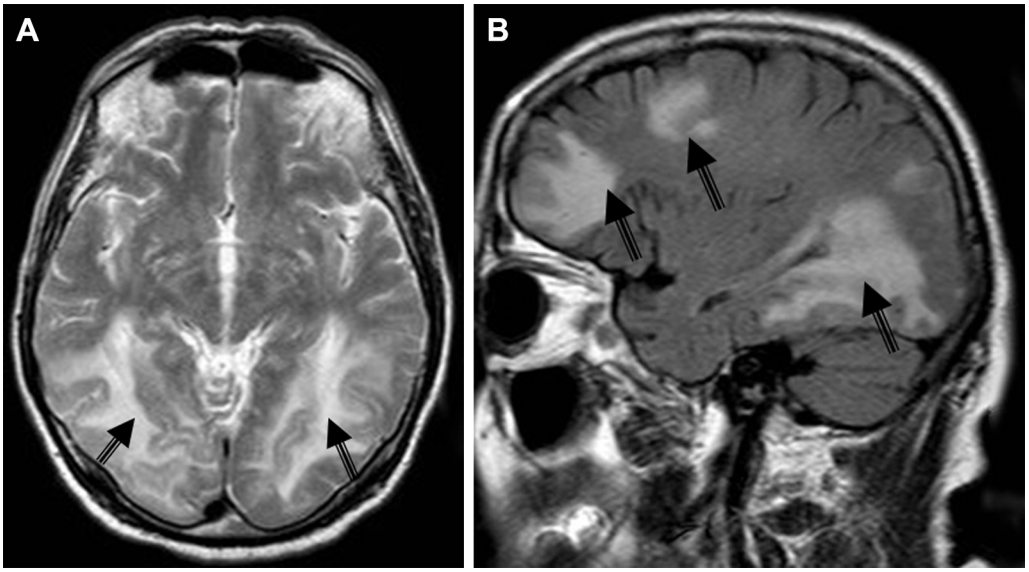

Figure 1. (A) Axial T2 magnetic resonance image at presentation shows extended areas of high signal in the white matter (vasogenic edema) symmetrically in the bilateral occipital lobes (indicated by arrows). (B) Sagittal fluid-attenuated inversion recovery (FLAIR) magnetic resonance image at presentation shows extended areas of high signal in the white matter (vasogenic edema) in left frontal and occipital lobe (indicated by arrows).