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CASE REPORT

Posterior reversible encephalopathy associated with nocturnal blood pressure non-dipping pattern

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Abstract

We report the case of a 70-year-old man who presented with a recent history of headache, altered mental status and sleepiness. He was known to have type II diabetes mellitus, and a mild, treated and apparently well controlled hypertension. Brain magnetic resonance imaging demonstrated extensive abnormalities in the parieto-occipital white matter, suggestive of posterior reversible encephalopathy syndrome (PRES). An extensive diagnostic evaluation did not allow the detection of any known cause of the syndrome. Twenty-four-hour non-invasive ambulatory blood pressure monitoring showed a mild to moderate hypertension, with non-dipping pattern. This case suggests that, in the context of an overnight blunted blood pressure profile, even a mild or moderate hypertension can result in cerebral vasogenic oedema, underlining the diagnostic importance of 24-h blood pressure monitoring in patients with PRES without severe hypertension or other commonly recognized causes of posterior reversible encephalopathy.

Key Words: *ambulatory blood pressure monitoring, blood pressure, posterior reversible encephalopathy syndrome*

Introduction

Posterior reversible encephalopathy syndrome (PRES) is a clinical–radiological syndrome mostly characterized by confusion, headache, altered consciousness, seizures and visual disturbance. Magnetic resonance imaging (MRI) shows vasogenic brain oedema typically predominating in the occipito-parietal regions (1), but often widespread (2). It is commonly associated with hypertensive encephalopathy, eclampsia, transplantation, autoimmune conditions, renal diseases, sepsis, immunosuppressive or chemotherapic therapy, or other drug use (1–5). The pathophysiology of brain vasogenic oedema formation is poorly understood: however, the ultimate underlying process seems to be an endothelial dysfunction triggered by different systemic conditions (2). We report a case with PRES associated with a mild masked hypertension (6) and a non-dipper pattern during night (7).

Case report

A 70-year-old Italian man presented with a recent progressive history of headache, altered mental status and sleepiness. The neurological examination at the first visit showed disinhibited, sometimes aggressive behaviour and attention deficit. There was no evidence of localizing neurological or meningeal signs. Brain MRI demonstrated bilateral subcortical parietal-occipital hyperintensities on T2-weighted and fluid-attenuated inversion recovery (FLAIR) images (Figure 1A). On the basis of this neuroimaging pattern, a diagnosis of PRES was made and confirmed by a marked reduction of MRI abnormalities 15 days later (Figure 1B.). One-month follow-up MRI revealed complete regression of vasogenic oedema.

The patient's personal history was remarkable for type II diabetes mellitus, and a mild, treated hypertension, apparently wellcontrolled based on casual office measurements. The complete blood count, serum urea and creatinine, glucose level and liver function tests were normal, as were erythrocyte sedimentation rate, C-reactive protein and laboratory immunological tests. Twenty-four-hour non-invasive ambulatory blood pressure monitoring (ABPM) showed a mild to moderate hypertension, with non-dipping profile (average $\pm$ standard deviation

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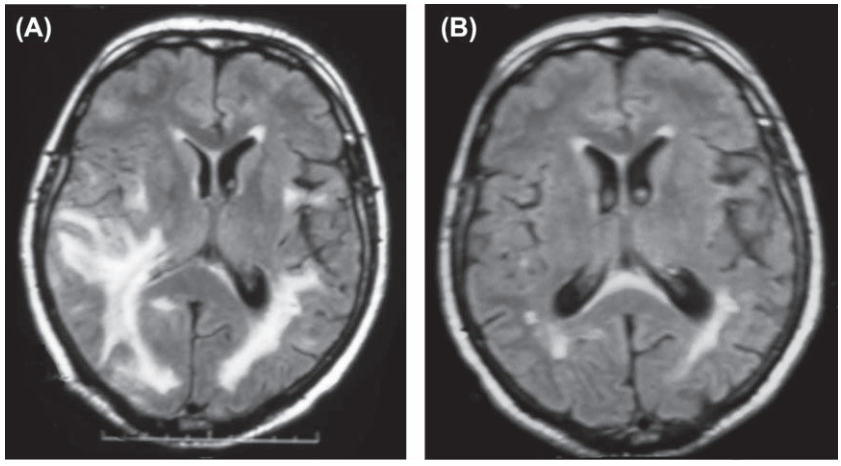

Figure 1. (A) Axial magnetic resonance imaging (MRI) obtained at admission shows bilateral predominantly parieto-occipital subcortical hyperintensities. (B) Follow-up MRI performed 15 days later showing marked reduction of MRI abnormalities.

daytime, awake systolic and diastolic pressure: 147.2 ± 7 and 78.8 ± 5 mmHg, respectively; mean sleep-time systolic and diastolic pressure: 138.8 ± 8 and 75.1 ± 6, respectively). The patient was treated for brain oedema with osmotic diuretics and sodium restriction. The antihypertensive therapy was modified and timed to target both daytime and night-time blood pressure (BP). He clinically improved after few days, and fully recovered in a month.

Discussion

PRES develops in patients suffering from different systemic conditions. One of the most frequently recognized cause of PRES is hypertension, particularly hypertensive crisis. Following the "hypertension theory" (2), severe increase in BP, can result in autoregolatory failure, vasoparalysis and fluid extravasation. However, about 20–30% of cases are normotensive or suffer from a mild to moderate hypertension (2), not challenging the upper limit of cerebral autoregulation. An endothelial dysfunction/damage could represent a common pathway in the pathogenesis of PRES, regardless of aetiology (2).

In this patient, we did not find any known cause of PRES, and the highest recorded systolic and diastolic values on ABPM were 180 and 110 mm/Hg, respectively. However, despite apparently controlled blood pressure values, he had an insidious condition of inadequately controlled hypertension over most the time, and above all a non-dipping pattern. Both are known to be associated with increased risk of target organs damage, cerebrovascular and cardiovascular complications (6,7). In particular, patients with non-dipper pattern have greater impairment of endothelial function than patients with normal nocturnal blood pressure fall (8), above all in the presence of diabetes or other factors causing endothelial damage, and this could be a mechanism linking non-dipping pattern and breakdown of the blood–brain barrier, leading to brain oedema (2,6,7).

PRES was reported to be associated with abnormal circadian blood pressure profile in four cases (9,10). However, two of them had malignant hypertension and "riser" nocturnal pattern (9), the others presented with hypertensive crisis, renal dysfunction and nocturnal non-dipper BP pattern (10).

Our case suggests that, in the context of an overnight blunted blood pressure profile, even a mild or moderate hypertension can result in cerebral fluid extravasation, leading to PRES.

Moreover, it underlines the diagnostic importance of ABPM in patients with PRES without severe hypertension or other commonly recognized causes of posterior reversible encephalopathy.

Declaration of interest: The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

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