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Case Report

The effect of carotid occlusive disease on the distribution of brain lesions in patients with systemic conditions—an example of PRES

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ABSTRACT

Severe hypertension is a major cause, among a long list of recognized causative factors of posterior reversible encephalopathy (PRES). We present an interesting case of a middle-aged patient with hypertension and asymmetric distribution of PRES due to tumor-related encasement and narrowing of the right internal carotid artery and relative sparing of the ipsilateral right cerebral hemisphere. White matter lesions from PRES were mostly seen in the contralateral left cerebral hemisphere and bilateral posterior fossa. It is theorized that organs or tissues distal to a chronically constricted vessel are protected from hypertensive changes (such as PRES) as the arterial stenosis reduces the transmission of hypertension-related effects, presumably due to a combination of hypoperfusion and alterations in compensatory changes in vessel autoregulation. While protected from the effects of hypertension, the patient is, however, at risk for border-zone infarction from hypoperfusion.

We believe that this case report provides a better understanding of the mechanism underlying the etiology of PRES as primarily regulated by the important baroreceptors and autoregulation mechanism of the carotid artery.

Case presentation

A 63-year-old patient with past medical history of breast cancer and hypertension presented with right posterior parieto-occipital headaches, confusion, and word-finding difficulty. The patient's vital signs at the time of admission were remarkable for a blood pressure of 171/90 and a heart rate of 110. On physical exam, incidental soft tissue swelling was noted within the right pharynx. Neurologic exam was

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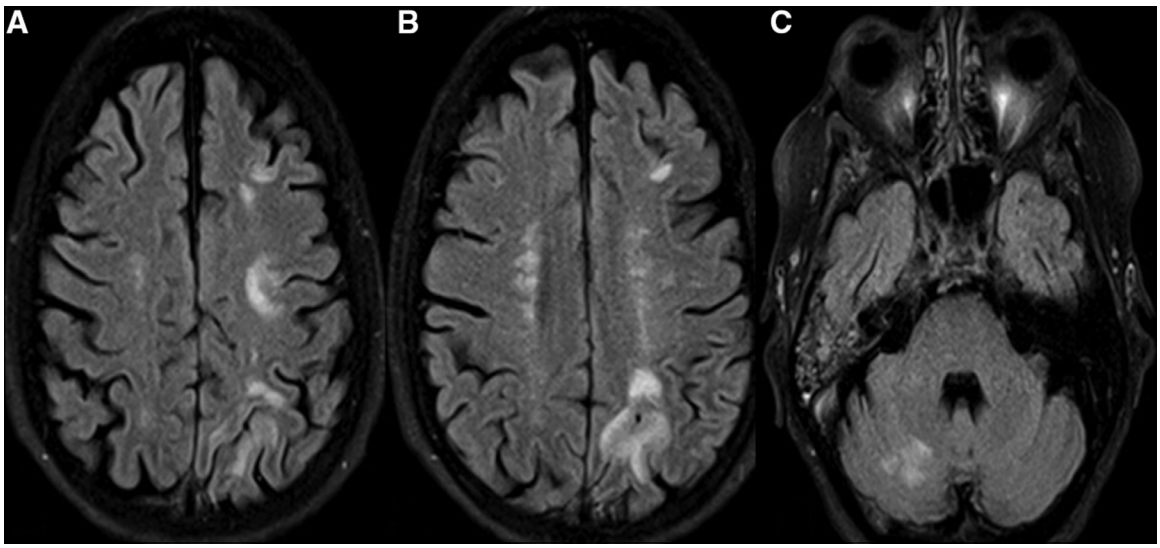

Fig. 1 – Axial FLAIR images on initial presentation at an outside institution demonstrate hyperintense FLAIR signal abnormality within the left frontal and parietal lobes (A and B) and right cerebellar hemisphere (C).

significant for speech latency, impaired naming but intact repetition, possible right visual neglect and impaired right-sided proprioception. Laboratory exam showed an elevated white blood cell count of 20,900. A lumbar puncture was unremarkable with normal opening pressure.

Magnetic resonance imaging (MRI) demonstrated asymmetric and unilateral hyperintense T2/FLAIR signal abnormalities involving the subcortical white matter of the left parietal and frontal lobes and right cerebellar hemisphere (Fig. 1).

At the level of the skull base, there was an incompletely visualized soft tissue mass within the right poststyloid parapharyngeal space (Fig. 2). The adjacent internal carotid artery flow void was encased and showed severe luminal narrowing. Biopsy of the right neck mass was later performed with pathology demonstrating squamous cell carcinoma.

Initially, there was concern that the brain findings could represent metastatic lesions, although this was thought to be less likely given absence of enhancement on postcontrast images. PRES was a differential consideration, but the presentation was thought to be atypical given the unilateral involvement of the left cerebral hemisphere.

After successful blood pressure management, a repeat brain MRI was performed 3 days following the initial MRI. There was substantial improvement of the previously seen white matter lesions, strongly supporting the diagnosis of PRES. A second follow-up brain MRI approximately 2 weeks later showed near complete resolution of the white matter lesions in the cerebral and cerebellar hemispheres (Fig. 3).

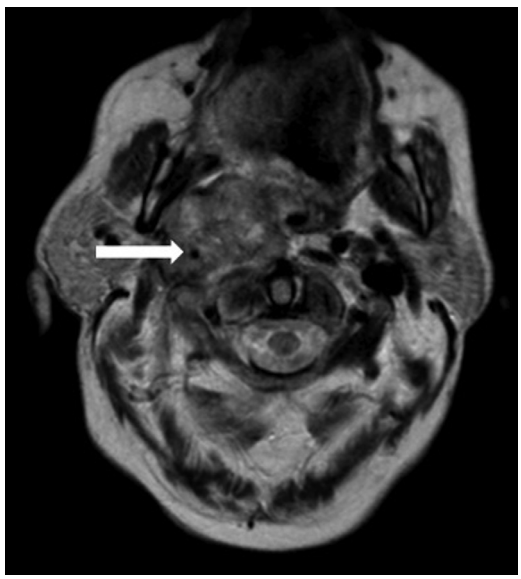

Fig. 2 – Axial T2 images at the level of the skullbase show the incidentally identified soft tissue mass in the right poststyloid parapharyngeal space with encasement and narrowing of the right internal carotid artery (arrow).

Discussion

Since its first recognition in 1996, many causative factors of PRES have been identified. Hypertension, acute renal disease, and immunotherapy are the most commonly recognized causes. The mechanism of the cerebral edema is endothelial damage or dysfunction that permits vasogenic edema to develop. The posterior portions of the cerebral hemispheres are typically involved, but some patients have frontal lobe emphasis, and in others the brainstem and cerebellum are preferentially involved.

The classic imaging features include vasogenic edema as a result of altered blood-brain barrier permeability with increased FLAIR and T2 signal abnormalities, hyperintense ADC

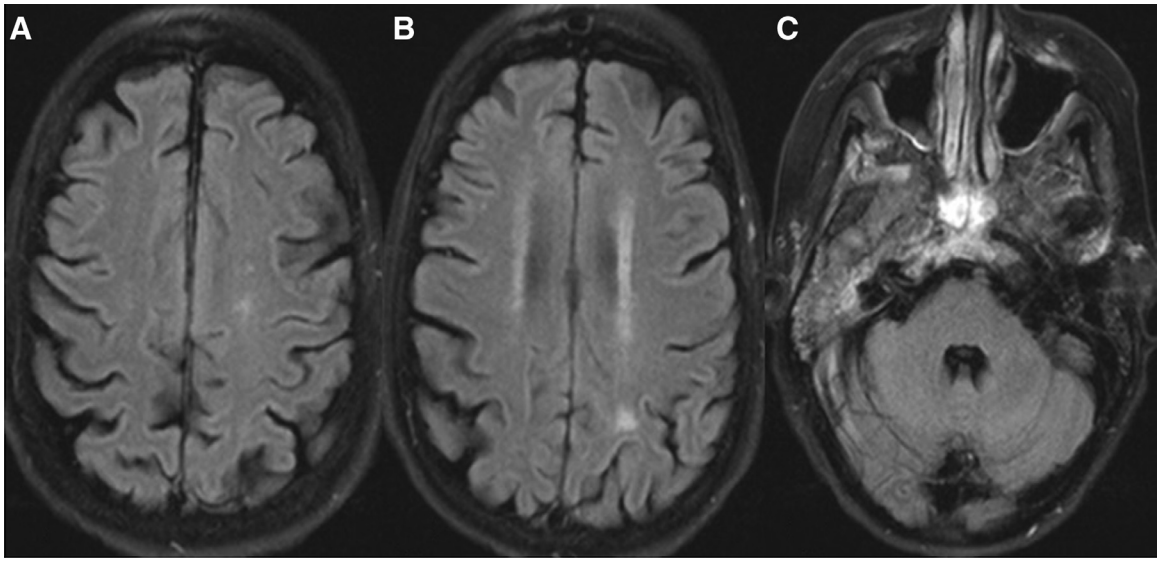

Fig. 3 – Follow-up brain MRI performed approximately 2 weeks after the initial exam demonstrates near complete resolution of previously seen white matter lesions in the left cerebral hemisphere (A and B) and the right cerebellum (C).

signal, and usually normal DWI. In our patient, the unique feature of PRES was the unilaterality of the hemispheric abnormalities which posed a diagnostic challenge. This case exemplifies the importance of hemodynamic factors in the distribution of lesions caused by PRES.

It has been experimentally shown that when a renal artery is constricted, the resulting hypertension related changes (from release of chemical mediators) affect other systemic organs, but the hypoperfused kidney remains unaffected [1,2]. A similar observation was made in the eyes in hypertensive patients who had unilaterally stenosed internal carotid arteries [3]. The retina on the side of the stenosis did not show hypertensive changes.

Thus, it is hypothesized that the brain distal to a constricted vessel is protected from hypertensive changes (such as PRES) as chronic arterial stenosis reduces the transmission of hypertension-related effects and results in alterations in vascular autoregulation. As a result, in our patient, the right internal carotid artery narrowing precluded development of hypertensive changes in the right cerebral hemisphere. However, the "unprotected" left cerebral hemisphere and posterior fossa were involved since the arteries supplying these structures were widely patent.

The stenosed vessel is associated with changes in volume flow and flow velocity that affects organ perfusion; while protected from the effects of hypertension, the patient is however at risk for border-zone infarction from hypoperfusion as well as from the effectiveness of the bloodstream in clearance (washout) of downstream emboli due to decreased flow [4,5].

A study in 2007 investigated the varying imaging appearances of PRES and presented an until then undescribed unilateral, "tumefacient" variant of PRES where the white matter lesions mimicked intracranial neoplasms [6]. In the following years, some other case reports have described the rare, unilateral occurrence of PRES in the setting of subarachnoid hemorrhage [7–9], as a stroke-mimick [10] and with use of a chemotherapy drug [11].

This case report provides an improved understanding of the mechanism underlying the etiology of PRES as primarily regulated by the important baroreceptors and autoregulation mechanism of the carotid artery.

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