

Blindness, confusion and seizures in a cancer patient

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CASE REPORT

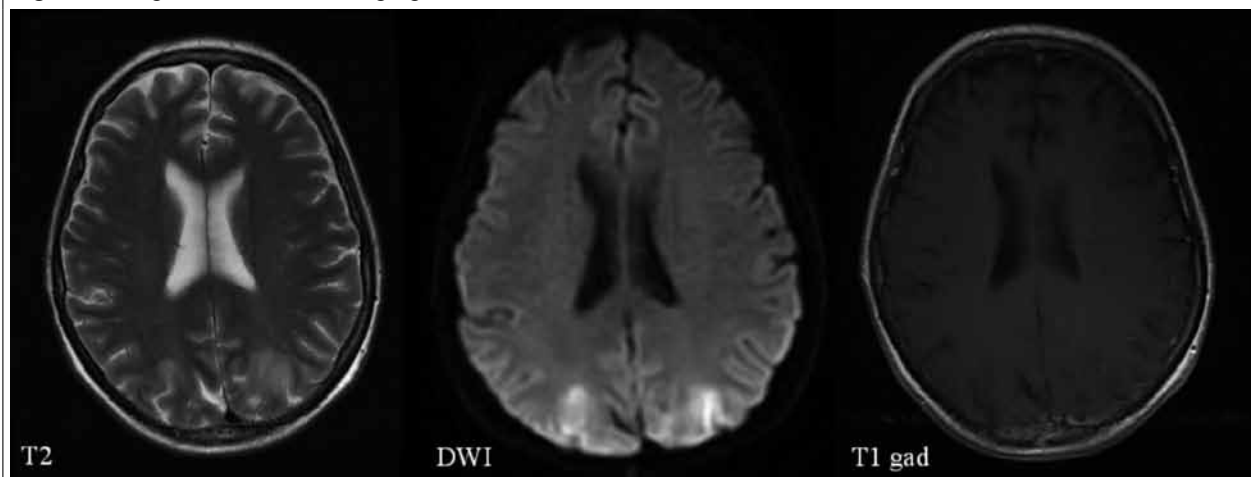
A 58-year-old woman with metastatic adenocarcinoma of unknown primary developed headache, confusion and bilateral cortical blindness of acute onset. This was followed shortly by two episodes of generalised seizures. She last received chemotherapeutic treatment with epirubicin, oxaliplatin and capecitabine two months previously. She had no other significant past medical history and was not on any medications. Blood pressure

was within normal limits and her laboratory tests were unremarkable. A magnetic resonance imaging of her brain was performed (*figure 1*).

WHAT IS YOUR DIAGNOSIS ?

See page 96 for the answer to this photo quiz.

Figure 1. Magnetic resonance imaging



DIAGNOSIS

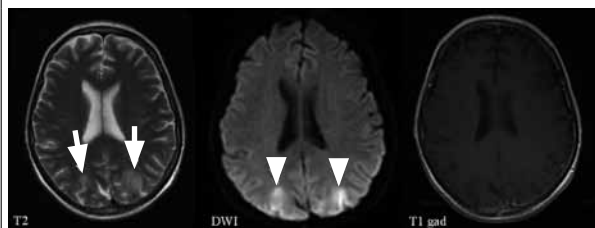
From the clinical presentation, the differential diagnosis includes cerebral metastases, malignant leptomeningeal involvement and posterior reversible encephalopathy syndrome (PRES). The magnetic resonance imaging showed high signal intensity on T2-weighted images (T2) affecting the cortex and subcortical white matter of both occipital lobes symmetrically (*figure 2*, white arrows). Corresponding hyperintensities were seen on diffusion-weighted images (DWI) suspicious for cerebral ischaemia (*figure 2*, white arrowheads). There was no enhancement on gadolinium-enhanced T1-weighted images (T1 gad) to suggest metastatic disease. The clinical and imaging findings were highly suggestive of PRES with possible cerebral ischaemia. She was treated with supportive care with anticonvulsants and her symptoms completely resolved in 12 days.

PRES can occur in patients with eclampsia, uncontrolled hypertension with resultant encephalopathy, renal impairment and those receiving immunosuppressive treatments such as ciclosporin and tacrolimus. Previous exposure to chemotherapeutic agents has also been implicated.¹ The pathophysiology of PRES is not well understood but is believed to be due to a paucity of sympathetic innervation within the posterior circulation,

making it more prone to interruption of autoregulation with disruption of the blood brain barrier. This results in vasogenic oedema with hydrostatic leakage and interstitial fluid accumulation in the cortex and subcortical white matter. The clinical presentation of PRES is similar to that experienced by our patient.

PRES is commonly diagnosed on magnetic resonance imaging of the brain with cortical/subcortical vasogenic oedema affecting mainly the occipital and parietal lobes in a bilateral symmetrical fashion. This manifests as areas of high signal intensity on T2-weighted images. There is no restricted diffusion (no high signal intensity) on DWI with vasogenic oedema. DWI is thus helpful in distinguishing PRES from other conditions such as cerebral ischaemia and infarction, which result in cytotoxic oedema that manifests as restricted diffusion (high signal intensity).² Occasionally, as in our patient, concurrent ischaemia may complicate PRES with resultant cytotoxic oedema and hence restricted diffusion on DWI.³ Despite this, our patient experienced complete resolution of symptoms. This reversibility together with the clinical presentation and imaging findings of lesions in a bilateral symmetrical posterior cerebral distribution is typical of PRES. Clinical recovery usually occurs in most patients within days.⁴ Early recognition is essential as delayed diagnosis can result in complications such as infarction. Repeat imaging is recommended to document resolution of abnormalities.

Figure 2. Magnetic resonance imaging



There were areas of high signal intensity on the T2-weighted images affecting the cortex and subcortical white matter of both occipital lobes symmetrically (white arrows). Corresponding hyperintensities were seen on diffusion-weighted images suspicious for cerebral ischaemia (white arrowheads). There was no enhancement on gadolinium-enhanced T1-weighted images to suggest metastatic disease.

REFERENCES

1. Bhatt A, Farooq MU, Majid A, Kassab M. Chemotherapy-related posterior reversible leukoencephalopathy syndrome. *Nat Clin Pract Neurol.* 2009;5:163-9.
2. Kinoshita T, Moritani T, Shrier DA, et al. Diffusion-weighted MR imaging of posterior reversible leukoencephalopathy syndrome: a pictorial essay. *Clin Imag.* 2003;27:307-15.
3. McKinney AM, Short J, Truwit CL, et al. Posterior reversible encephalopathy syndrome: incidence of atypical regions of involvement and imaging findings. *Am J Roentgenol.* 2007;189:904-12.
4. Lee VH, Wijdicks EFM, Manno EM, Rabinstein AA. Clinical spectrum of reversible posterior leukoencephalopathy syndrome. *Arch Neurol.* 2008;65:205-10.