Posterior Reversible Encephalopathy Syndrome induced by Pazopanib: a case report

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Introduction

One of the major challenges that neurologists will increasingly face in the coming years is the management of neurological complications associated with novel cancer therapies. Innovative treatments such as monoclonal antibodies, small molecules, and CAR-T cells, while representing significant advances in oncology, can induce neurotoxic effects at various levels of the central and peripheral nervous systems, posing complex diagnostic and therapeutic challenges.

Case summary

A 72-year-old man with renal cancer on **pazopanib** (post-nephrectomy, locoregional recurrence) presented with acute confusion, mutism, and urinary incontinence. He rapidly developed impaired consciousness, right hemiparesis, and **severe hypertensive crisis requiring continuous IV therapy**. CT angiography was negative; CSF unremarkable; EEG showed right-hemisphere epileptiform discharges. Initial MRI was normal; a 36h MRI revealed bilateral cerebellar and pontine lesions (T2/FLAIR+, ADC+, DWI−, Gad−) consistent with vasogenic oedema. Both clinical status and MRI findings resolved within one week, consistent with **Posterior Reversible Encephalopathy Syndrome (PRES) related to pazopanib-induced hypertension**; the drug was discontinued.

Discussion

Initial presentation with expressive aphasia and confusion suggested a TIA. Rapid deterioration, typical MRI changes, and resolution after blood pressure control and pazopanib withdrawal confirmed PRES. Pazopanib, like other VEGF inhibitors, may trigger hypertension through endothelial dysfunction and reduced nitric oxide, predisposing to neurotoxicity.

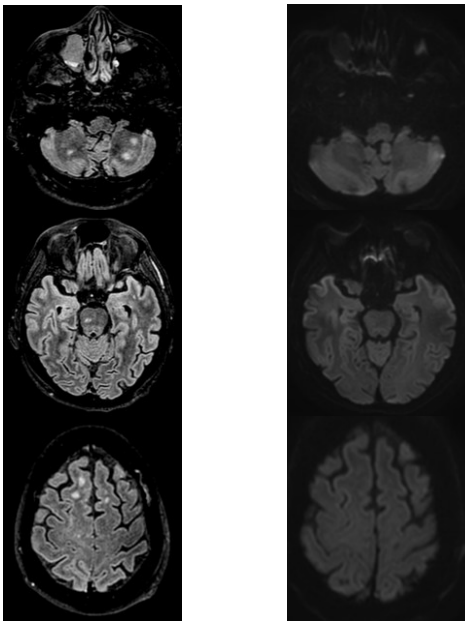

Fig. 1 FLAIR VS. DWI sequences. Vasogenic oedema is depicted.

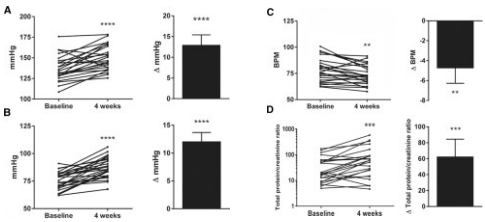

Fig. 2 Blood pressure changes during pazopanib treatment. (Robdrup Tinning A. et al. Hypertension, 2018)

Conclusions

With the growing use of VEGF-targeted therapies, neurologists should remain alert to treatment-related PRES, especially in patients with cerebrovascular risk factors.

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