NEUROLOGICAL PICTURE

Cerebral metastasis of renal carcinoma mimicking venous haemorrhagic infarction

Several months after nephrectomy for hypernephroma, a 52 year old woman developed acute demyelinating neuropathy. Paraneoplastic neuropathy was suspected and she was treated with intravenous immunoglobulins (IGs). On the first day of treatment, she had a secondarily generalised tonic-clonic seizure. Contrast enhanced CT revealed right parieto-occipital intracerebral haemorrhage (fig 1A–D). Angiography disclosed persisting cortical veins (fig 1E–F). Although IG therapy was stopped, the patient continued to deteriorate until her condition stabilized after two weeks. The preliminary diagnosis of immunoglobulin induced venous thrombosis with secondary haemorrhagic infarction was made and treatment with phenprocoumon and carbamazepine was initiated. MRI performed six months later showed a residual hyperintense lesion (fig 2A). Four years later, native CT revealed a small hyperdensity with hypodense rim in the same region (fig 2B). Although repetition with contrast enhancement was suggested, the patient was lost to follow up. Two years later, she was re-admitted for homonymous hemianopsia. MRI disclosed a contrast enhancing focal lesion suggestive of cerebral metastasis (fig 2C). Histology of the neurosurgically excised tissue confirmed hypernephroma. She has remained in remission since then.

Metastases may settle in infarcts in rare occasions.¹ However, the initial angiogram shows only subtle changes without clear evidence for venous thrombosis. Moreover, venous thrombosis following intravenous IG administration progresses over the duration of treatment and is reversible after cessation of treatment.² Therefore, we believe that it is implausible that the putative thrombosis arose on the first day of treatment, that small cortical veins were affected exclusively, and that symptoms worsened rather than improved after treatment was halted. Hence, although the

Figure 2 Axial follow up images at (A) six months (T2w MRI), (B) three years (CT), and (C) five years (contrast enhanced T1w MRI) after initiation of symptoms, disclosing a focal lesion in the right parieto-occipital region (arrows).

CT scan in 2000 provided the first unequivocal evidence of cerebral metastasis, we propose that the same metastasis may have caused the haemorrhage and seizure three years earlier. Retrospectively, a small hyperintensity suggestive of the metastasis is indeed visible in the right parieto-occipital region six months after the seizure (fig 2A, arrow).

It has been assumed that tumours account for about 5% of patients initially diagnosed with stroke, with about 30% of these cases having metastases and seizures being the leading symptom.¹ Several cases have been reported in which patients presenting with intracerebral haemorrhage were later diagnosed with metastases despite unsuggestive imaging.³ Intensely vascularised hypernephroma metastases are especially prone to haemorrhage.⁴ There are two reports on hypernephroma micro-metastases below the level of detection by imaging methods causing extensive intracerebral haemorrhage.^{3,5} Our case is remarkable in that diagnosis and treatment were delayed for five years. Furthermore, this case provides the opportunity to calculate the growth rate of an untreated intracerebral renal carcinoma metastasis. Based on the images in fig 2, an average growth rate of 0.9 cm/year can be estimated. A similar growth rate has been estimated for untreated extracerebral hypernephroma metastases.⁶

We conclude that special diagnostic care is warranted if the primary neoplasm is at high risk for haemorrhagic complications and when cerebral imaging studies are not suggestive of cerebral metastasis because microscopic metastases of these tumours can cause large intracerebral bleedings.

G C Petzold, J M Valdueza

Department of Neurology, Humboldt University, Berlin, Germany

C Zimmer

Department of Diagnostic Radiology, University of Leipzig, Germany

Correspondence to: Dr G C Petzold; Department of Neurology, Charité Hospital, Humboldt University, Schumannstr. 20/21, 10098 Berlin, Germany; gabor.petzold@charite.de

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Figure 1 A–D, axial CT images revealing intracerebral haemorrhage in the right parieto-occipital region. E–F, digital subtraction angiography showing long persisting cortical veins, leading to the initial suspicion of cerebral venous thrombosis.