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Wernicke's encephalopathy induced by total parenteral nutrition in patient with acute leukaemia: unusual involvement of caudate nuclei and cerebral cortex on MRI

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Abstract We report a 13-year-old girl with leukaemia and Wernicke's encephalopathy induced by total parenteral nutrition. MRI showed unusual bilateral lesions of the caudate nuclei and cerebral cortex, as well as typical lesions surrounding the third ventricle and aqueduct. After intravenous thiamine, the patient improved, and the abnormalities on MRI disappeared.

Key words Encephalopathy, Wernicke's · Magnetic resonance imaging

Introduction

Wernicke's encephalopathy (WE) is a metabolic disorder caused by thiamine deficiency. Thiamine plays an important role as a coenzyme in the Krebs and pentosephosphate cycles. The disease is most frequently associated with chronic alcoholism, but can occur in other forms of malnutrition or malabsorption, such as prolonged parenteral nutrition without addition of thiamine, total gastrectomy, gastrojejunostomy, severe anorexia or hyperemesis gravidarum [1–8].

Clinically, WE is characterised by the classical triad of ophthalmoplegia, ataxia and abnormal mental state. The typical MRI findings are symmetrical high-signal lesions surrounding the third ventricle and aqueduct on T2- or proton density-weighted images. Neuropathologically, the acute stage is characterised by marked vascular dilatation, endothelial swelling and neuronal damage. In chronic lesions there is loss of neuropil with fibrillary astrocytosis and atrophy of the region involved [1].

We describe a case of acute WE due to total parenteral nutrition and protracted vomiting. MRI showed unusual caudate nucleus lesions and cortical involvement, as well as typical diencephalic and mesencephalic abnormalities. These abnormalities disappeared following thiamine administration.

Case report

A 13-year-old girl was admitted to hospital with acute myeloid leukaemia (type FAB M5b). She received the first cycle of chemotherapy (AIEOP "LMA '92", Idarubicin, Etoposide, Cytarabine). A central venous catheter was previously positioned to supply fluids and total parenteral nutrition, and to administer drugs. After a month she achieved a complete remission.

The patient underwent the second cycle of chemotherapy 40 days later. Over the following days she developed persistent nausea and vomiting. Examination disclosed nystagmus and ophthalmoplegia; deep tendon reflexes were brisk on the left. Her state of consciousness was progressively decreasing.

CT before and after contrast medium showed no abnormality MRI at 1.5 T revealed bilateral, symmetrical lesions on long TR images in the thalamus, periaqueductal grey matter, caudate nucleus, putamen and frontal and parietal cortex (Fig. 1); none of the lesions showed contrast enhancement. The chemotherapy was in-

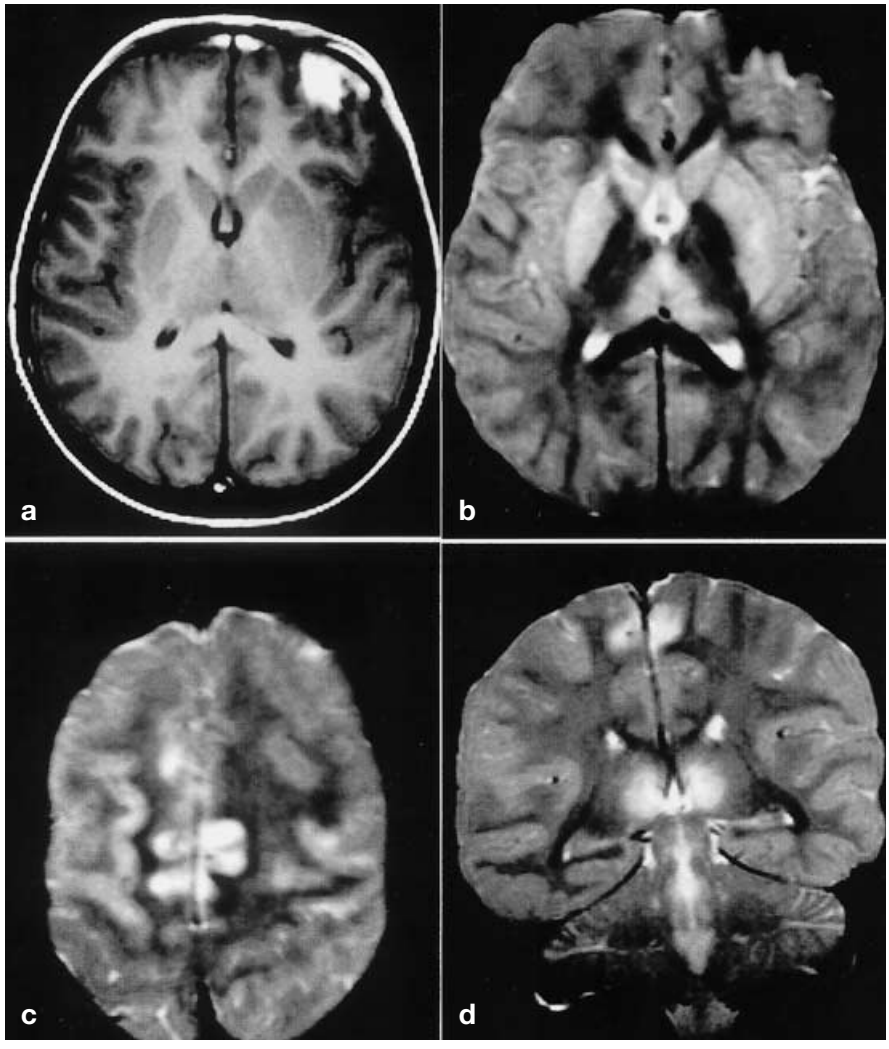

Fig. 1 a–d Acute phase. **a** Axial T1-weighted image showing mildly low-signal areas bilaterally in the thalamus. **b, c** Axial T2-weighted images showing high-signal lesions bilaterally and symmetrically in the thalamus, putamen and caudate nucleus **b** and frontal and parietal cortex **c. d** Coronal T2-weighted image shows the same lesions

terrupted. The patient was treated with mannitol and dexamethasone, but her condition did not improve. On the basis of the neurological signs and MRI findings, WE was suggested. The patient was immediately given intravenous thiamine and her symptoms and signs gradually resolved.

MRI 1 month later showed almost complete resolution of the lesions. On T2-weighted images, there was only a small area of high signal in the right caudate nucleus (Fig. 2).

The history, imaging findings and response to thiamine were taken as confirming the diagnosis of WE.

Discussion

WE is a potentially fatal acute or chronic metabolic disorder, amenable to treatment with thiamine. Early diagnosis is essential to avoid irreversible neuronal damage. In our case, thiamine deficiency was caused by vomiting and parenteral nutrition without addition of thiamine.

Traditionally, the diagnosis of WE has rested primarily on clinical findings and response to treatment; measurements of thiamine levels may be misleading [7]. MRI plays a role in identification of the disease, by showing bilateral, symmetrical high-signal lesions on T2-weighted images in the periventricular regions of the third ventricle, periaqueductal grey matter and floor of the fourth ventricle. Contrast enhancement of the mamillary bodies may in some cases lead to earlier diagnosis [2, 5]. The unusual findings in our case were the additional involvement of frontal and parietal cortex and the caudate nuclei, rarely been reported in the literature. Yamashita and Yamamoto [8] reported a case of we showing symmetrical high signal along the central and precentral sulci and Ohkoshi et al. [3] a case with bilateral caudate nucleus lesions [3]. Our case confirms that these areas may be vulnerable in WE.

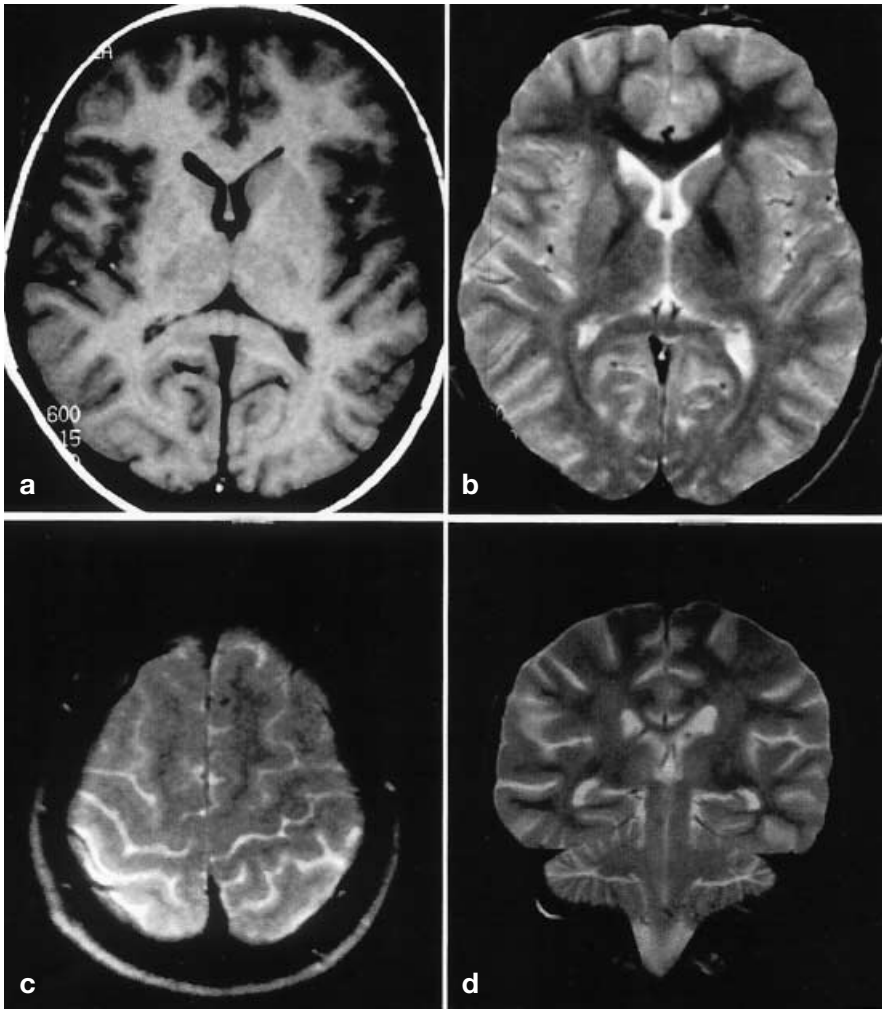

Fig. 2 a–d Images after one month show almost complete resolution of the lesions. **a** Axial T1-weighted image **b** Axial T2-weighted image showing a small residual area of high signal in the right caudate nucleus. **c,d** Axial and coronal T2-weighted images. The lesions in the cerebral cortex and thalamus have disappeared

References

1. Brody BA (1996) The Wernicke-Korsakoff syndrome. Neuropathology and pathogenic basis. Int J Neuroradiol 2: 216–230
2. D'Aprile P, Gentile MA, Carella A (1994) Enhanced MR in the acute phase of Wernicke encephalopathy. AJNR 15: 591–593
3. Ohkoshi N, Ishii A, Shoji S (1994) Wernicke's encephalopathy induced by hyperemesis gravidarum, associated with bilateral caudate lesions on computed tomography and magnetic resonance imaging. Eur Neurol 34: 177–180
4. Pagnan L, Berlot G, Pozzi-Mucelli RS (1998) Magnetic resonance imaging in a case of Wernicke's encephalopathy. Eur Radiol 8: 977–980
5. Shogry MEC, Curnes JT (1994) Mamillary body enhancement on MR as the only sign of acute Wernicke encephalopathy. AJNR 15: 172–174
6. Schroth G, Wichmann W, Valavanis A (1991) Blood-brain-barrier disruption in acute Wernicke encephalopathy: MR findings. J Comput Assist Tomogr 15: 1059–1061
7. Tallaksen CM, Bell H, Bohmer T (1993) Thiamine and thiamine phosphate ester deficiency assessed by high performance liquid chromatography in four clinical cases of Wernicke encephalopathy. Alcoholism Clin Exp Res 17: 712–716
8. Yamashita M, Yamamoto T (1995) Wernicke encephalopathy with symmetric pericentral involvement: MR findings. J Comput Assist Tomogr 19: 306–308