

An ex-intravenous drug addict with confusion and apathy

Koon H Chan, Raymond TF Cheung, Gardian CY Fong, Shu L Ho

INTRODUCTION

Wernicke's encephalopathy is an acute neurological disease, which is the result of thiamine deficiency. Cases in people who are not alcoholics are increasingly recognized. This case reports a Chinese man who was probably poorly nourished as a result of his intravenous drug addiction. During his initial admission to the intensive care unit, he was given parenteral nutrition containing high carbohydrate content without vitamin supplementation. Three weeks after discharge, he was readmitted because he had been suffering confusion for 1 week. Although classical clinical and radiological features are absent, Wernicke's encephalopathy was confirmed by biochemical tests. He responded to thiamine treatment with dramatic clinical and radiological improvements. Physicians should be

Dr Koon H Chan is Medical Officer, **Dr Raymond TF Cheung** is Associate Professor, **Dr Gardian CY Fong** is Medical Officer and **Dr Shu L Ho** is Associate Professor in the Division of Neurology, University Department of Medicine, University of Hong Kong, Queen Mary Hospital, Pokfulam, Hong Kong

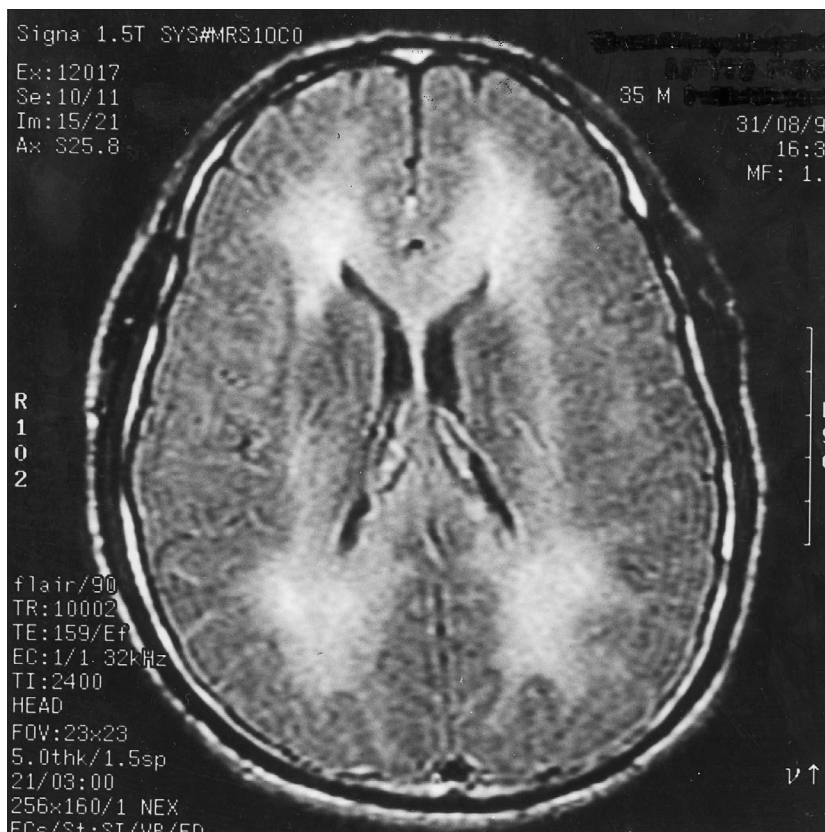


Figure 1. A fluid attenuation inversion recovery (FLAIR) magnetic resonance image of the brain showing diffuse, symmetrical periventricular hyperintensities.

Correspondence to: Dr RTF Cheung

CASE REPORT

In August 1997, a 35-year-old Chinese man, who was an intravenous drug addict and a chronic smoker, was admitted because of aspiration pneumonia complicating intravenous abuse of heroin. He was mechanically ventilated in the intensive care unit (ICU) for 5 days. He was well upon discharge on day 12, and his wife and sister kept him away from all illicit drugs. Three weeks after discharge, he was readmitted because of impaired short-term memory for 1 week together with disorientation in time, inability to perform activities of daily living, urinary incontinence and reduced mobility for 2 days. Physical examination revealed disorientation, inattention and apathy. He spoke a few simple words and obeyed very simple commands. He was afebrile, his neck was supple and extraocular movements were normal with no nystagmus. There was no focal neurological sign. Ataxia was not observed, but finger-to-nose and heel-to-shin tests could not be performed. All initial blood tests were normal or negative. Toxicology screening of blood and urine was negative. Urgent computed tomography of the brain was normal, and electroencephalogram revealed mild slowing of background activity only. Lumbar puncture revealed normal findings in the CSF. The initial diagnosis was viral encephalitis, but he did not respond to the empirical use of intravenous acyclovir.

A low red cell transketolase activity of 23 mmol/litre/min (normal: 45–90 mmol/litre/min) was reported later. Addition of thiamine pyrophosphate increased the transketolase activity by 163% (normal=4–40%). A magnetic resonance image (MRI) of the brain performed before commencement of thiamine treatment revealed diffuse, symmetrical hyperintensity in the deep periventricular white matter on T2-weighted and fluid attenuation inversion recovery (FLAIR) images (Figure 1), indicating diffuse demyelination. No abnormality was seen in the brainstem and mammillary bodies. Polymerase chain reaction for herpes simplex virus DNA was negative, and acyclovir was stopped. The patient responded to intravenous thiamine 100 mg daily. A follow-up MRI of the brain revealed marked resolution of the white matter changes (Figure 2). He recovered completely after 12 weeks of thiamine therapy. A record review of his first admission showed that the patient was given intravenous parenteral nutrition containing high carbohydrate content without vitamin supplementation during his ICU stay.

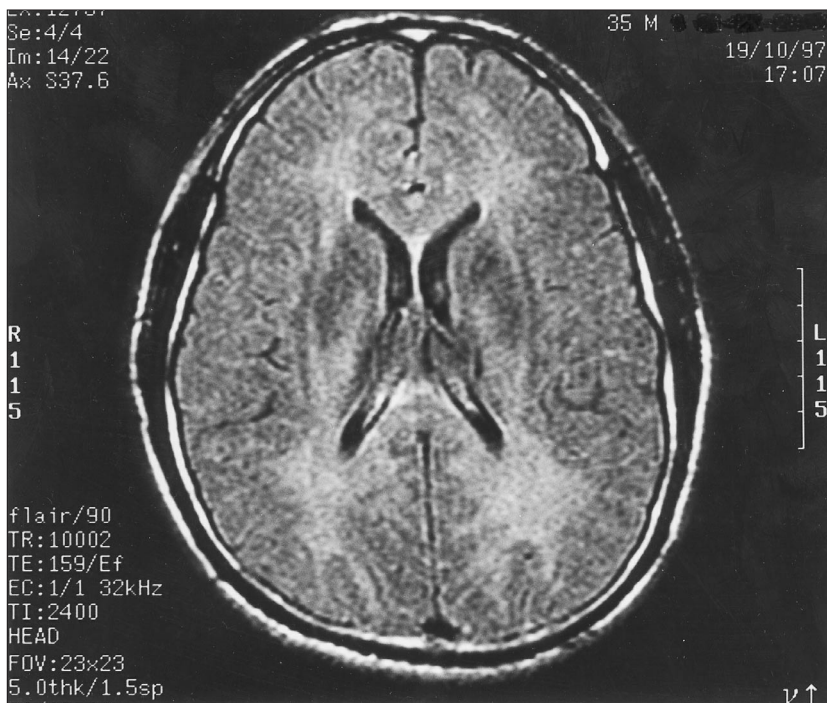


Figure 2. A follow-up fluid attenuation inversion recovery (FLAIR) magnetic resonance image (MRI) of the brain 7 weeks after the first MRI study showing marked reduction in the white matter hyperintensities.

aware of this treatable condition among patients who are not alcoholics and who have poor nutritional status.

DISCUSSION

Wernicke's encephalopathy as a result of thiamine deficiency is a well-known complication of chronic alcoholism and is underdiagnosed in clinical practice. The cardinal triad of ataxia, confusion and ocular signs may not be present, as illustrated by this patient. As well as being a result of alcoholism, thiamine deficiency can be caused by poor intake of thiamine plus increased demand on thiamine and/or excessive loss of thiamine. Clinical scenarios include hyperemesis gravidarum (Larvin et al, 1983), other causes of severe protracted vomiting, anorexia

nervosa, starvation (Gropman et al, 1998), gastrointestinal diseases such as gastric cancer and post gastric plication (Reuler et al, 1985), chronic renal failure on dialysis (Toro et al, 1997), and thyrotoxicosis (Otsuka et al, 1997).

As a co-enzyme for transketolase, pyruvate dehydrogenase and α -ketoglutarate dehydrogenase, thiamine is essential in carbohydrate metabolism. In this patient, a high intake of carbohydrate without vitamin supplementation increased the demand for thiamine and, in turn, precipitated Wernicke's encephalopathy. The patient's poor nutritional status makes it likely that he had low body reserves of thiamine.

Classical magnetic resonance imaging (MRI) findings are symmetrical white matter lesions in the periventricu-

lar regions of the thalamus, periaqueductal region and floor of the third ventricle (Gallucci et al, 1990). MRI of the brain may also reveal atrophy, contrast enhancement and haemorrhage in the mamillary bodies. Freiesleben et al (1997) suggested reversible impairment of axonal transport leading to accumulation of phosphorylated neurofilaments and subsequent ballooning of mamillary neurons as a pathogenic mechanism in Wernicke's encephalopathy.

CONCLUSIONS

Wernicke's encephalopathy should be suspected in all patients presenting with acute or subacute confusion, apathy and encephalopathy, irrespective of the history of alcohol abuse. A poor nutritional status is a major risk factor. A high carbohydrate diet without vitamin supplementation can lead to Wernicke's encephalopathy. In addition, absence of ocular features and ataxia does not exclude the diagnosis. Finally, typical MRI findings may not be found, and symmetrical periventricular demyelination may be the MRI abnormalities in the early stage. **HM**

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Correspondence

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