Although informative with regard to the precise MR imaging-pathology correlation, the case report appearing in the current issue (3) was unfortunate since the patient did not survive the disease. Indeed, the aim of the report was not at describing how to treat this disorder. However, the important lesson learnt through this case is that thiamine at a dose of 100 mg given on the day of admission was not sufficient in arresting the development of Wernicke’s disease, or to improve transketolase activity and blood level of thiamine to the normal range. Several neurology textbooks recommend giving parenteral thiamine in a dose of 100 mg per day until neurological deficits are cleared up or dietary intake becomes adequate.

Currently, there have been sporadic and yet sizable numbers of cases of Wernicke’s disease in non-alcoholic populations in Japanese medical literature. In a literature search of the 1995 issue of Japanese index journal, “Igaku Chuo Zasshi”, there were 8 reports related to intravenous fluid therapy for a variety of reasons and two in relation to hyperemesis gravidarum. Since June 1995 the remark sheet attached to high-caloric fluids states that thiamine should be considered to be added as serious acidosis may be encountered, without however, mentioning Wernicke’s encephalopathy.

There is also a single important medical-economical problem with this. The fear that the preventive use of vitamin B complex might not be covered by health (medical) insurance causes physicians to be reluctant to add it to IVH or intravenous dextrose solution. This is indeed unfortunate and might be a problem inherent only to our health care system. Japanese health insurance used to be generous in allowing physicians to prescribe multiple vitamins freely for many vitamin-unrelated conditions. However, increasingly stringent restriction of vitamin use for those patients who are not obviously deficient of them or at least who are orally fed has gradually made physicians hesitant to routinely add vitamin B complex for intravenous fluid therapy.

Dextrose solution when given consumes thiamine as it acts as a cofactor in two major enzyme systems (4), i.e.; one relates to glycolysis and involves the oxidative decarboxylation of pyruvic and alpha-ketoglutaric acid, and the other to two transketolation steps of the phosphoglyconate pathway (hexose monophosphate shunt), an alternate route of carbohydrate metabolism of importance. Victor et al (1) described two patients in the early stage of Wernicke’s disease who had been given only dextrose solutions or carbohydrate diet without thiamine. They had progressively deteriorated as to their neurological status until thiamine was supplemented.

Wernicke’s disease is clearly preventable and if treated early enough, one may recover with no residual or minimal neuro-
logical deficits. However, in cases untreated or delayed administration of thiamine, the outcome may be catastrophic as one may die or remain severely disabled chiefly due to profound amnesia, e.g.; he might have survived the illness but may have totally forgotten things for the past events (retrograde amnesia) and is unable to register new memory at all. I would urge readers that the treatment is to immediately give a large dose of thiamine (150–300 mg a day parenteral) for at least several days, whenever Wernicke’s disease is clinically suspected. One should not wait for neuroimaging studies or the results of blood thiamine level before the treatment, as the diagnosis can be made on clinical ground alone. Also, we will not be able to defend ourselves if it is the case of iatrogenic Wernicke’s disease by giving a large dose dextrose solution without thiamine supplementation.

References