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Case report

Wernicke's encephalopathy in early pregnancy complicated by disseminated intravascular coagulation

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Summary. A 29 year-old Japanese woman with hyperemesis gravidarum became comatose and died. The autopsy revealed a typical case of Wernicke's encephalopathy complicated by disseminated intravascular coagulation (DIC). Repeated vomiting and parenteral nutrition without vitamins led to Wernicke's encephalopathy and a spontaneous abortion 24 h before death triggered the induction of DIC.

Key words: Wernicke's encephalopathy – Hyperemesis gravidarum – Disseminated intravascular coagulation

Introduction

In 1881 Wernicke reported three cases of focal haemorrhages and proliferation of capillaries symmetrically located in the wall of the third ventricle, periaqueductal gray matter and the floor of the fourth ventricle. This form of encephalopathy has been frequently documented.

We now report a case of Wernicke's encephalopathy in early pregnancy complicated by DIC.

Case report

A 29 year-old paragravida, whose last menstrual period began on 9 July, 1978, was admitted to an obstetric hospital in her 10th week of pregnancy with complaints of vomiting for the previous 3 weeks. After admission, an intravenous drip and major tranquilizer therapy were prescribed. Vomiting was transiently relieved, but 3 weeks after admission her consciousness became clouded and she was disoriented. During admission for about 1 month no vitamins were administered. She was then transferred to the emergency unit of Kyushu University hospital. There was no history of alcoholism.

On admission, she was dehydrated and semicomatose. Her temperature was 38° C, pulse 150, respiration 30 and blood pressure 104/76. Neurological evaluation disclosed symmetrically diminished tendon reflexes and her muscles were flaccid. There were no pathological reflexes. The white-cell count was 24,100 (75% segmented neutrophils, 12% band forms, 2% monocytes and 11% lymphocytes). The calcium was 11.9 mg/dl, the glucose 145 mg/dl, the bilirubin

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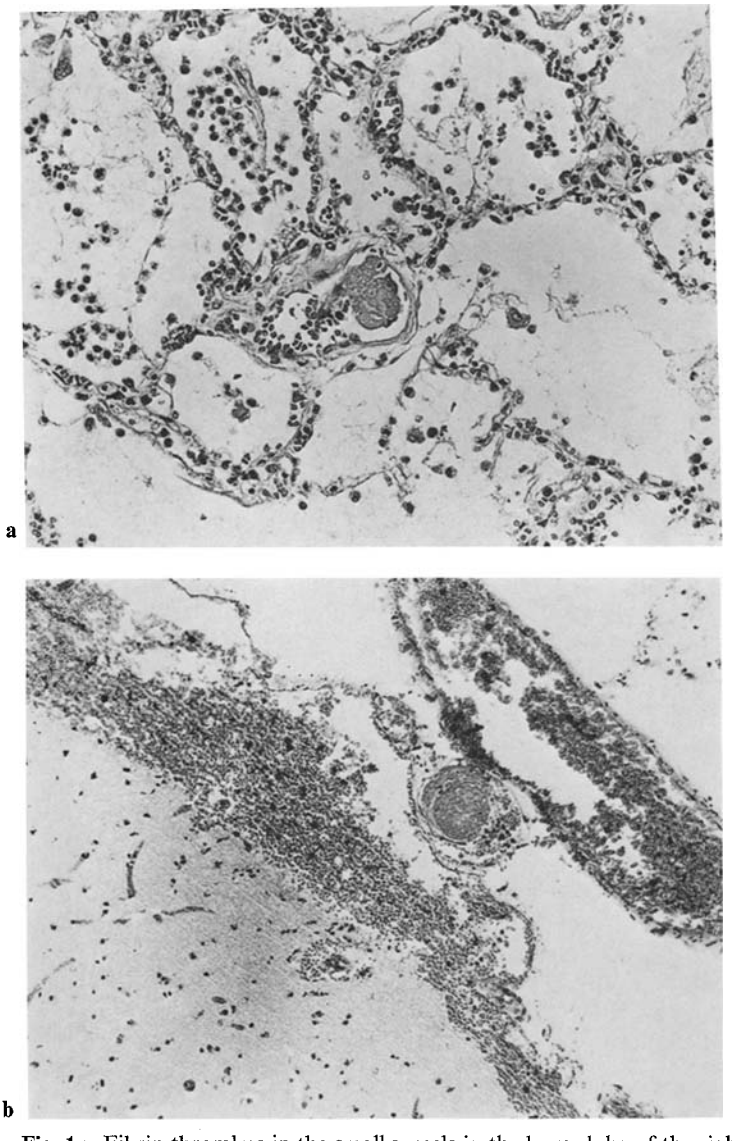
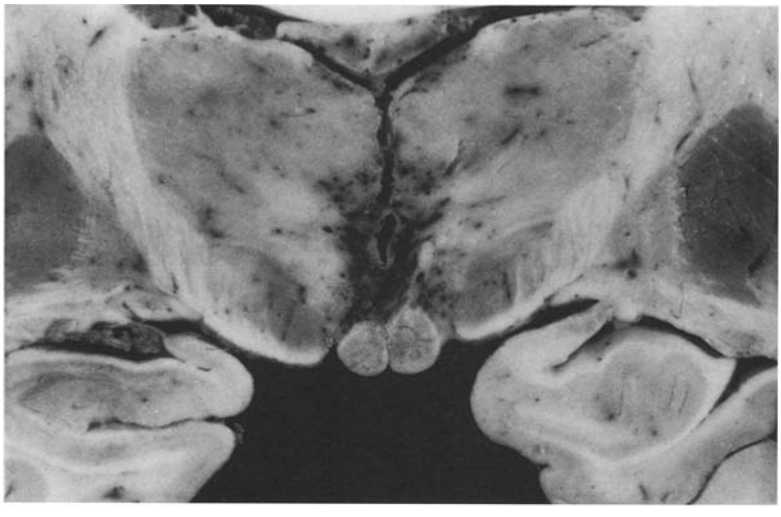


Fig. 1a. Fibrin thrombus in the small vessels in the lower lobe of the right lung. (H.E. ×200). (**b**) Fibrin thrombus in the arachnoid. Haemorrhage is also evident. (H.E. ×100)

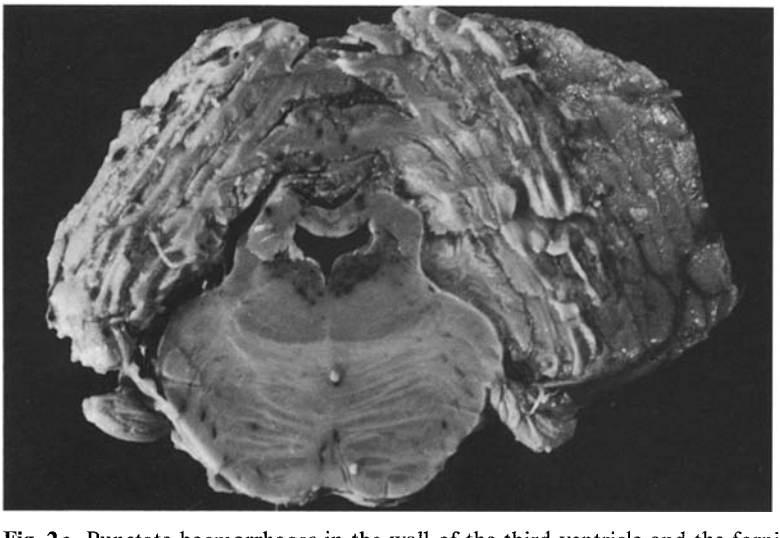
1.8 mg/dl, GOT 67 and GPT 78. The sodium was 160 mEq/L, the potassium 6.2 mEq/L, the cloride 125 mEq/L. The lactate was 11.49 mg/dl and the pyruvate 0.51 mg/dl. She was treated for dehydration, but she did not regain consciousness. Twenty-four hours before death, the fetus was spontaneously aborted. Four days after admission she died.

Postmortem examination

General pathological findings. There were multiple haemorrhages in both lungs, bronchi, pericardium, myocardium, oesophagus, stomach and intes-



a



b

Fig. 2a. Punctate haemorrhages in the wall of the third ventricle and the fornix. **(b)** Punctate haemorrhages mainly in the floor of the fourth ventricle

tine. Microscopically, there were multiple thrombi in small vessels of both lungs (Fig. 1a), right kidney, liver, arachnoid (Fig. 1b) and cerebral cortex, suggesting DIC. The lungs weighed 745 and 800 g. The microscopic examination revealed oedema, bronchopneumonia and oxygen pneumonia. The kidneys weighed 250 and 240 g. Follicular adenoma of the thyroid was present and the liver showed a fatty metamorphosis. Adenohyophysis was hyperplastic due to pregnancy.

Neuropathological findings. The brain weighed 1250 g. Macroscopically, petechial haemorrhages were found in the cerebral cortex, the fornix, the wall of the third ventricle (Fig. 2a), the periaqueductal gray matter and

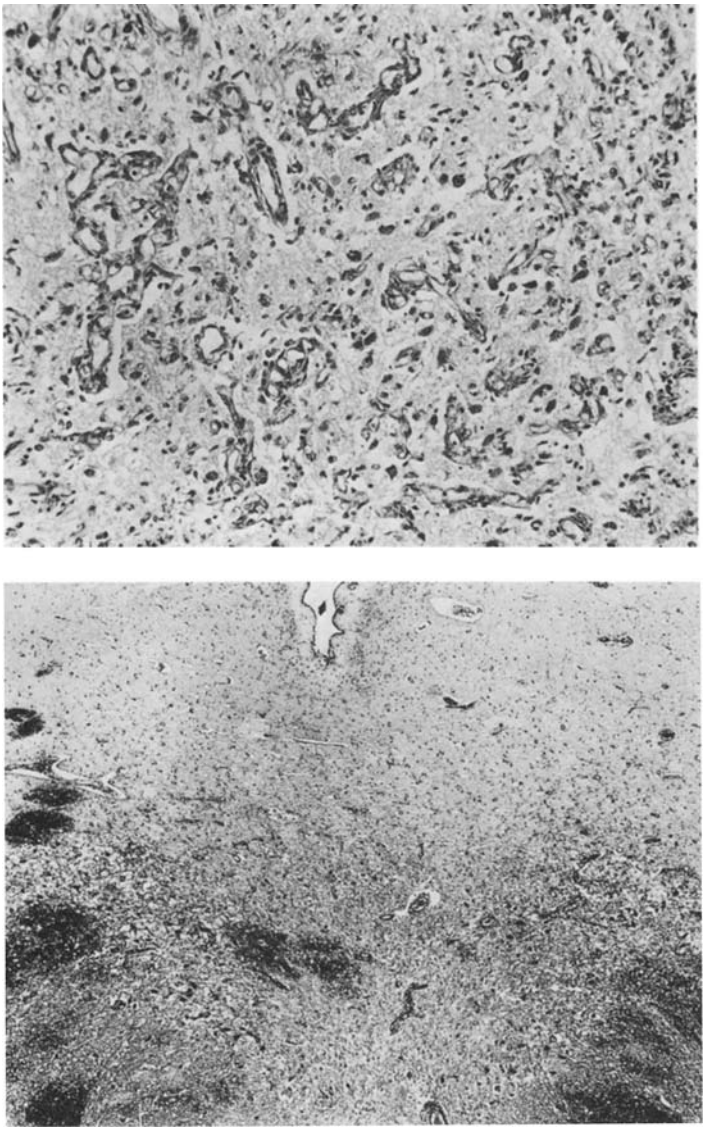


Fig. 3a. Mammillary body (*left*). Marked proliferation of capillaries with endothelial hyperplasia. Gliosis was associated and neurons were relatively diminished in number. (Nissl. ×100). (**b**) Midbrain. The glial matrix was spongy in the area of oculo-motor nuclei with haemorrhage and proliferation of capillaries. (H.E. ×23)

the floor of the fourth ventricle (Fig. 2b). Small foci of haemorrhages were also found in the cerebellum and the tegmentum pontis (Fig. 2b). Microscopic examination disclosed marked capillary proliferation with endothelial hyperplasia especially in the bilateral mammillary bodies (Fig. 3a). The capillary proliferation was also found in the wall of the third ventricle, the

periaqueductal gray matter (Fig. 3b) and the floor of the fourth ventricle with extravasated erythrocytes.

Discussion

Underlying factors in Wernicke's encephalopathy include chronic alcoholism, malignancy (Campbell and Biggart 1939; Ebels 1974), chronic gastritis (Neubürger 1938) and peptic ulcer (Ebels 1974). Our patient had a hyperemesis related to pregnancy. Cases of Wernicke's encephalopathy associated with excessive vomiting in early pregnancy have been reported (Campbell and Biggart 1939; Chaturachinda and McGregor 1968; Ebels 1974). Excessive vomiting and no vitamin ingestion no doubt leads to a thiamine deficiency and Wernicke's encephalopathy.

Ziporin et al. in 1965 found that deprivation of thiamine induced clinical symptoms in humans in 9–27 days. Studies on monkeys (Blank et al. 1975) revealed that neurological signs became evident within 7 to 10 weeks. During starvation for the treatment of obesity, the requirement for thiamine diminished and clinical signs such as disorientation and confusion did not appear until re-feeding, when the requirement for thiamine was increased (Drenick et al. 1966). Nausea and vomiting within 7 weeks of starvation seemed to be non-specific symptoms of an avitaminosis. Parenteral nutrition without vitamins accelerated the loss of the thiamine store.

In our case disorientation appeared within 7 weeks from the beginning of repeated vomiting. Clinical data, such as increased levels of serum glucose, pyruvate and lactate, electrolyte imbalance, dehydration and semicoma all helped in making a diagnosis.

The pathogenesis of this disease is uncertain. Scholz (1949) suggested that protein-rich exudate in Wernicke's encephalopathy induced glial and vascular proliferation.

Another important point is that this case was complicated by DIC, according to the criteria of Tanaka et al. (1978). There is apparently no case in the literature of Wernicke's encephalopathy complicated by DIC. During pregnancy, serum coagulation and fibrinolytic factors are increased (Nakabayashi et al. 1978) and the placenta contains much tissue thromboplastin (Gonmori and Takeda 1976). Therefore, the miscarriage may possibly have been related to DIC. Punctate haemorrhages in the cerebral cortex and cerebellum are atypical of Wernicke's encephalopathy. The main cause of haemorrhages in these areas may not be related to Wernicke's encephalopathy but rather to DIC, though the symmetrical periventricular haemorrhages are possibly due to the encephalopathy.

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