Hydrocephalus Associated with Malignant Hypertension and Renal Failure

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Dear Sir,

It has been reported that autopsy findings of patients who died of malignant hypertension (MHT) demonstrated increased brain weight, flattened gyri, and compressed ventricles [1]. Hypertensive encephalopathy sometimes develops during the course of renal diseases [1]. It is often difficult to decide upon the relative significance of uremia and hypertension per se in the genesis of symptoms of MHT associated with uremia [2]. The presented patient had hydrocephalus which was associated with MHT and renal failure and cured by antihypertensive therapy and dialysis treatment.

A 25-year-old male with a history of glomerulonephritis was admitted to the Fukuoka Red Cross Hospital on June 21, 1988, because of MHT, renal failure, and unconsciousness. On June 13, 1988, he noticed headache. On the next day, he developed blurred vision. On June 19, he was admitted to another hospital because of severe headache, nausea, and vomiting. The blood pressure was 244/178 mm Hg. On June 20, he became delirious. Computed tomography (CT) scanning of his brain showed no hemorrhage but flattened gyri and dilated ventricles (fig. 1A-C). Blood chemistry showed elevations in blood urea nitrogen and creatinine (101 and 13.6 mg/dl, respectively). On June 21, he became somnolent and was transferred to our institution. On admission he was drowsy. The blood pressure was 248/130 mm Hg. Ophthalmological investigation showed round pupils and retinal hemorrhage and papilledema bilaterally. Chest and abdomen were normal. The tendon reflexes of the extremities were symmetrical, and no abnormal reflexes were found.

The white blood cell count was 16,800/mm³, hemoglobin 12.2 g/dl, blood urea nitrogen 129 mg/dl, and creatinine 15 mg/dl.

Peritoneal dialysis was started on June 21, the day of admission to our institution. With 10 mg of nifedipine four times daily and peritoneal dialysis, the blood pressure fell, and the disturbance of consciousness improved; the patient became alert on the 3rd hospital day. On the 4th hospital day, nifedipine was decreased from 10 mg four times daily to 10 mg twice daily and 100 mg of atenolol was added. Following atenolol treatment, the blood pressure remained at 140–170/70–

90 mm Hg. On August 1, hemodialysis was started. The patient was discharged on August 9 and returned to work with regular dialysis treatment. As shown in figure 1, the CT scans performed on June 19 (A-C) and 22 (D-F) demonstrated diminished density in the white matter and flattened gyri as well as dilated ventricles. The CT scans performed on July 22 (G-I) showed normal findings.

Hydrocephalus may develop in adults after subarachnoid hemorrhage, cerebral trauma, meningitis, intracranial tumors, and intraspinal or intracranial operations. The mechanism for the development of hydrocephalus is thought to be associated with obstruction of the cerebrospinal fluid pathway [3]. Our patient had no history of cerebrospinal disease or operation. CT scanning of his brain did not demonstrate either hemorrhage or tumor. Although autopsy findings of patients who died of MHT demonstrated compressed ventricles [1], our patient had dilated ventricles which disappeared after antihypertensive therapy and peritoneal dialysis. In uremic patients, structural changes of the central nervous system have not been widely appreciated, although those of the peripheral nervous system have been demonstrated by many investigators [2]. Resistance to outflow of cerebral spinal fluid may be in normal-pressure hydrocephalus, benign intracranial hypertension, and protein-producing tumors [4], and the impaired cerebrospinal fluid outflow may cause dilation of the ventricular system [4]. In MHT, the intracranial pressure increases [1], and the increased vascular permeability causes cerebral edema [5]. Elevation of intracranial pressure and development of cerebral edema may cause an increase in the resistance to outflow of cerebrospinal fluid which may result in the dilatation of ventricles. Although Weingarten et al. [6] reported a 57-year-old man with hypertensive encephalopathy whose CT scans showed severe white-matter edema involving brain stem and cerebellum with compression of 4th ventricle, producing hydrocephalus, the first and second CT scans of the presented case (fig. 1) showed dilated lateral and fourth ventricles which disappeared following antihypertensive therapy and dialysis treatment. These findings suggest that MHT and/or renal failure might play a key role in the genesis of hydrocephalus in this case.

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JUN. 22

JUL. 22

Fig. 1. CT scans of the brain.

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