A CASE OF BILATERAL HIPPOCAMPAL ATROPHY AND CORTICAL DISTURBANCE OF THE BRAIN AFTER CARDIOPULMONARY RESUSCITATION

MASAMI IMANISHI*, HISAYUKI TABUSE*, SEIJI MIYAMOTO*
TOSHISUKE SAKAKI** and SATORU IWASAKI***
*Department of Emergency and Critical Care Medicine, Nara Medical University
**Department of Neurosurgery, Nara Medical University
***Department of Radiology, Nara Medical University
Received February 15, 1999

Abstract: The brain is the most vulnerable organ to ischemia, and the basal ganglia, cerebral cortex, hippocampus, and cerebellum are the areas most vulnerable to whole brain ischemia due to sudden cardiopulmonary arrest. Moreover, delayed neuronal death has been reported to occur in the hippocampus. We report here a case of a 50-year-old man, who was transferred to our department in sudden cardiopulmonary arrest caused by acute heart failure hypertrophic cardiomyopathy. He received cardiopulmonary resuscitation, and spontaneous circulation was restored. However, he fell into a persistent vegetative state. Disturbance of the cerebral cortex and atrophy of the hippocampus bilaterally were observed on MR imaging. Hypoperfusion in the cerebral cortex was observed on SPECT. The atrophy of the hippocampus bilaterally was believed to be caused by delayed neuronal death after whole brain ischemia. We consider that the comprehensive disturbance of the cerebral cortex was caused not only by the whole brain ischemia but also by disturbance of the intracranial venous circulation due to chest compression and increased intracranial pressure (thoracic pump theory). (奈医誌. J. Nara Med. Ass. **50**, 149～154, 1999)

Key words: hippocampus, thoracic pump theory, cardiopulmonary resuscitation, cardiopulmonary arrest

INTRODUCTION

It has been well documented that even a short episode of hypoxia due to sudden cardiopulmonary arrest (CPA) can cause irreversible damage to neuronal cells. There are neuronal cells are especially vulnerable to ischemia in several regions of the brain. Previous experimental studies in animals have demonstrated the phenomenon of delayed neuronal death due to ischemia in the hippocampus.

We reported here a case of sudden CPA in which cardiac sinus rhythm was restored by cardiopulmonary resuscitation (CPR). After 30 days, MRI showed bilateral atrophic changes of the hippocampus and damage to the cerebral cortex. We reviewed causes of the bilateral hippocampal atrophy and the damage to the cerebral cortex.

CASE

A 50-year-old man complained of sudden dyspnea and lost consciousness after taking a bath

on March 18, 1995. He was transferred to our hospital by ambulance. He had been diagnosed with hypertrophic cardiomyopathy several years before. During transport, the emergency life -saving technicians performed external cardiac compression (basic cardiac life support). At the hospital, CPR (advanced cardiac life support) was continued and sinus rhythm was restored. The duration of cardiac arrest was about 28 minutes.

After restoration of a spontaneous heartbeat, a brain CT was performed. In addition, the patient received MR imaging and single-positron emission CT (SPECT) as follow-up care. On the brain CT (Fig. 1) taken immediately after restration of cardiac function, the basal ganglia, thalamus, and the corticomedullary junction of the cerebrum were indistinct, indicating the presence of mild cytotoxic edema. There were no other abnormal findings. MR imaging (Fig. 2) taken after 30 days at hospital showed enlarged lateral ventricles and suluci, and diffuse brain atrophy. However, T 2-weighted images (Fig. 2) showed hyperintensity of the cerebral cortex, suggestive of diffuse damage to the cerebral cortex. The basal ganglia was slightly hyperintense. In addition, a coronal view (Fig. 3) revealed bilateral atrophy of the

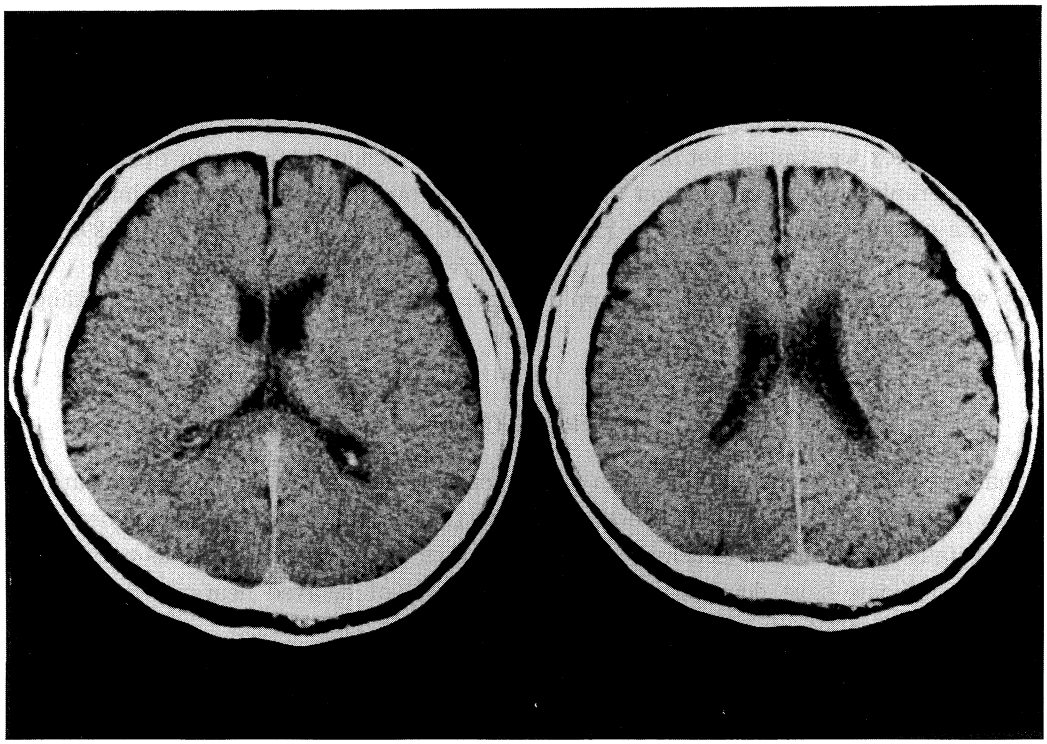

Fig. 1. Brain CT was carried out immediately after restoration of the patient's spontaneous circulation. The basal ganglia and the thalamus and corticomedullary junction of the cerebrum are indistinct areas, These findings indicate the presence of cytotoxic edema. No other abnormal findings were detected, including hemorrhage and infarction.

hippocampus. A brain SPECT (^{99m}Tc-HMPAO) (Fig. 4) taken simultaneously with blood flow to the basal ganglia and thalamus, but marked hypoperfusion of the cerebral cortex with MR imaging showed satisfactory blood flow to the basal ganglia and thalamus, but marked

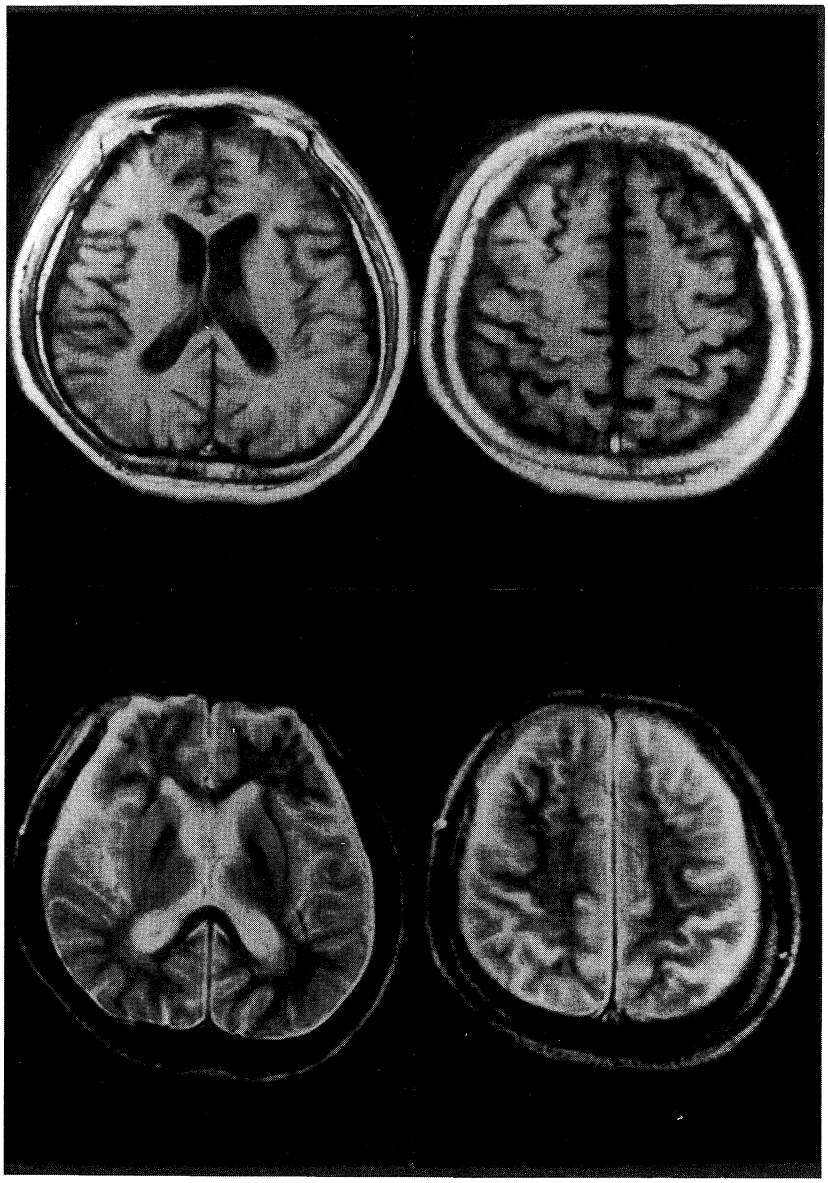

Fig. 2. MR imaging of the brain was carried out after 30 days at hospital. The MR imagings show enlarged lateral ventricles and cisterns, and diffuse atrophy of the brain. T2-weighted images (lower row) show hyperintensity of the cerebral cortex. The basal ganglia and thalamus were slightly hyperintense.

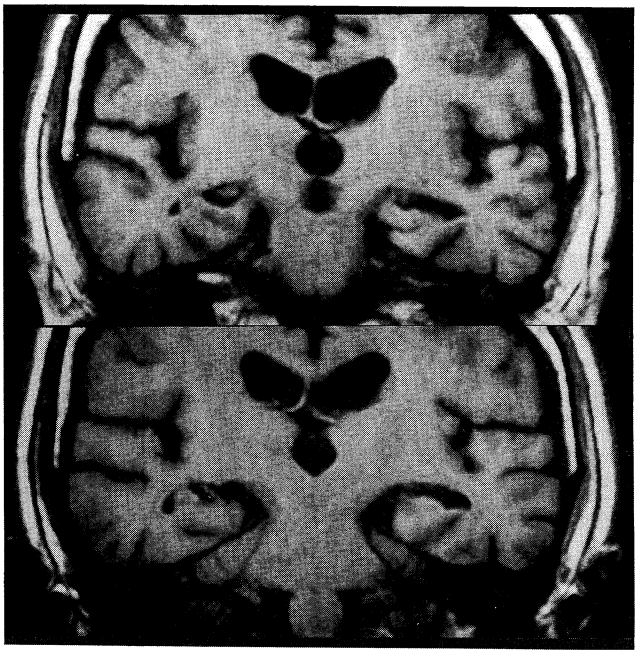

Fig. 3. Coronal view of MR imaging scan reveals bilateral atrophy of the hippocampus.

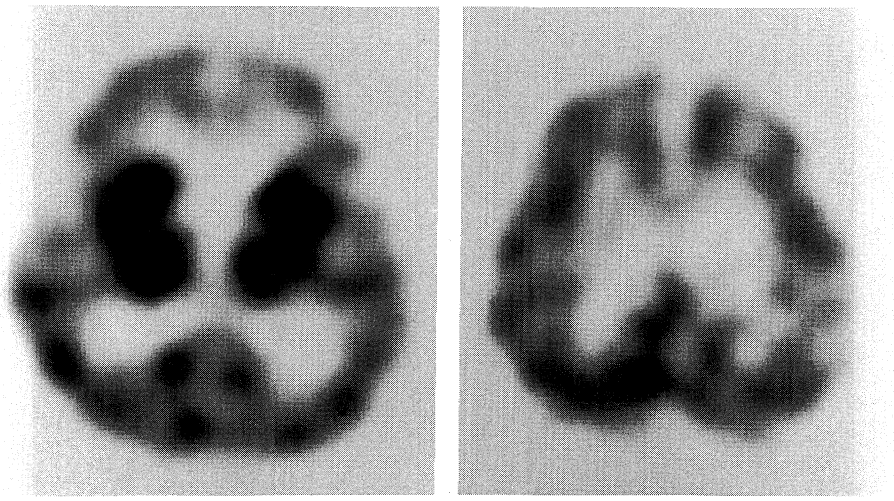

Fig. 4. A brain SPECT (^{99m}Tc-HMPAO) shows satisfactory blood flow to the basal ganglia and thalamus, but hypoperfusion of the cerebral cortex

hypoperfusion of the cerebral cortex. The patient was transferred on April 28, 1995, to another hospital for care of his persistent vegetative state.

DISCUSSION

Sudden cardiac arrest or hypoxia causes lethal damage to neuronal cells. Following a short episode of ischemia, neuronal cells vulnerable to ischemia undergo irreversible degeneration, thereby causing a specific pattern of neuronal cell loss[1-3]. These vulnerable neuronal cells include the pyramidal cells of the hypocampal CA 1 region and the Purkinje cells of the cerebellum. In addition, the moderate- to small-sized cells of the basal ganglia and the pyramidal cells of the 3 rd, 5 th, and 6 th layers of the cerebral cortex have been reported to be especially vulnerable to ischemia[1,4].

In this case of sudden CPA due to hypertrophic cardiomyopathy, immediate CPR restored self-circuration, but the patient fell into a persistent vegetative state. The duration of cardiac arrest was about 28 minutes. However, MR imaging and SPECT scans taken after the restoration of a spontaneous heartbeat showed irreversible brain damage in a wide region of the cerebral cortex and bilateral atrophy of the hippocampus.

Coronal MR imaging views are useful for the radiographic diagnosis of hippocampal lesions[5]. Atrophic changes of the hippocampus have also been demonstrated in Alzheimer's disease[6] and epilepsy[7-9]. However, there have been few reports describing hippocampal atrophy following brain ischemia due to cardiac arrest. The atrophic appearance of the hippocampus on MR imaging indicated the presence of neuronal cells that are vulnerable to ischemia. The radiographic changes observed due to ischemia are thought to reflect the "delayed neuronal death" phenomenon in hippocampus[2,10-12]. Several regions other than the hippocampus have been reported to be vulnerable to ischemia. Pulsinelli[1,12] demonstrated the ischemic vulnerability of the hippocampus, cerebellum, striate body, and cerebral cortex in animal models, in descending order of vulnerability. On the other hand, Kjos[3] and Jurgensen[13] showed that the cerebral cortex was more vulnerable than the basal ganglia. In our patient, SPECT revealed early damage in the cerebral cortex. The persistent vegetative state of the patient was believed to be irreversible damage to a wide region of the cerebral cortex.

One of the causes of damage in the cerebral cortex of this patient may have been reflux in the jugular veins by external cardiac compression, because external cardiac compression increases the pressure in the jugular vein and intracranial pressure. External cardiac compression increases intrathoracic pressure, and this pressure causes blood to flow the heart to the organs (thoracic pump theory[14,15]). Elevation of intrathoracic pressure could cause not only ejection of blood from the heart but also reflux or stagnation in the jugular veins. Circulatory dysfunction in this venous system may have caused damage in the cerebral cortex. It has also been reported that external cardiac compression elevates jugular pressure to the same level as the carotid pressure[15]. As we showed in a previous study on intracranial gas after cardiopulmonary resuscitation[16] external cardiac compression caused elevation of the jugular veins was considered to be one of the causes of damage in the cerebral cortex.

As described above, the patient's heartbeat was restored by cardiopulmonary resuscitation after ischemia (cardiopulmonary arrest), but he fell into a persistent vegetative state, and atrophy of the bilateral hippocampiand damage in a wide region of the cerebral cortex were

observed by SPECT and MR imaging. Elevation of venous pressure by external cardiac compression was considered to be one of causes of damage in the cerebral cortex.

REFERENCES

1) **Pulsinelli, W. A.**: Selective neuronal vulnerability: morphological and molecular characteristics. Brain Research **63**: 29-37, 1985.
2) **Kirino, T.** and **Sano, K.**: Selective vulnerability in the gerbile hippocampus following transient ischemia. Acta Neuropathol. **62**: 201-208, 1985.
3) **Kjos, B. O., Brant-Zwadzki, M.** and **Yong, R. G.**: Early CT findings of global central nervous system hypoperfusion. AJR **141**: 1227-1232, 1983.
4) **Brierley, J. B.** and **Graham, D. I.**: Hypoxia and vascular disorder of the central nervous system. In Greenfield's Neuropathology, 4 th ed., Admas, J. H., Corsellis, J. A. N., Duchen, L. W. EDS.; Edward Arnold, London, pp.125-156, 1984.
5) **Naidich, T. P., Daniels, D. L., Haughton, V. M., Williams, A., Pojunos, K.** and **Palacios, E.**: Hippocampal formation and related structures of the limbic lobe: Anatomic-MR correlation. part 1. Surface features and coronal sections. Radiology **162**: 747-754, 1987.
6) **Seab, J. P., Jagust, W. J., Wong, S. T. S., Roos, M. S., Reed, B. R.** and **Budinger, T. F.**: Quantitatic NMR measurements of hippocampal atrophy in Alzheimer disease. Mag Reson **8**: 200-208, 1988.
7) **Tien, R. D.** and **Felsberg, G. J.**: The hippocampus in status epilepticus: Demonstration of signal intensity and morphogic changes with sequential fast spin-echo MR imaging. Radiology **194**: 249-256, 1995.
8) **Iwasaki, S., Taoka, T., Nakagawa, H., Kichikawa, K., Fukuzumi, A., Ohishi, H., Uchida, H.** and **Kuru, Y.**: MRI in non-refractory temporal lobe epilepsy: hippocampal sclerosis and other temporal lesions. Neuroradiology **33**: 632-633, 1991.
9) **Achten, E., Boon. P., De Poorter, J., Calliauw, L., Van De Kerckhove, T., De Reuck. J.** and **Kunnen, M.**: An MR protocol for presurgical evaluation of patients with complex partial seizures of temporal lobe origin. AJNR **16**: 1201-1213, 1995.
10) **Petito, C. K., Feldmann, E., Pulsinelli, W. A.** and **Plum F.**: Delayed hippocampal damage in human following cardiorespiratory arrest. Neurology **37**: 1281-1286, 1987.
11) **Abe, K., Aoki, M., Kawagoe, J., Yoshida, T., Hattori, A., Kogure, K.** and **Itoyama, Y.**: Ischemic delayed neuronal death. Stroke **26**: 1478-1489, 1995.
12) **Pulsinelli, W. A., Brierley, J. B.** and **Plum, F.**: Temporal profile of neuronal damage in a model of transient forebrain ischemia. Ann. Neurol. **11**: 491-498, 1982.
13) **Jurgensen, J. C., Towfighi, J., Brennman, R. W.** and **Jeffreys, W. H.**: Symmetric brainstem necrosis in an adult following hypotension: An arterial end-zone infarct? Stroke **14**: 967-970, 1983.
14) **Niemann, J. T., Rosborough, J. P.** and **Hausknecht, M.**: Pressure-synchronized cuneangiography during experimental cardiopulmonary resuscitation. Circulation **64**: 958-991, 1981.
15) **Safer, P.** and **Bricher, N. G.**: Closed-chest cardiopulmonary resuscitation. In Cardiopulmonary Cerebral Resuscitation 3 rd ed., W. B. Saunders Company Ltd., London, Philadelphia, Toront, Sydney, Tokyo, pp.101-106, 1988.
16) **Imanishi, M., Nishimura, A., Tabuse, H., Miyamoto, S., Sakaki, T.** and **Iwasaki, S.**: Intracranial gas after cardio-pulmonary resuscitation.: 4 cases. Neuroradiology **40**: 154-157, 1998.