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A Case of Central Pontine and Extrapontine Myelinolysis with Early Hypermetabolism on ^{18}FDG-PET Scan

We report a 63 year-old woman who developed central pontine and extrapontine myelinolysis after rapid correction of hyponatremia. Lesions on brain MRI showed hypermetabolism on ^{18}FDG-PET scan in the early stage of the disease and became hypometabolic on the follow-up scan. We suggest that active microglia and astrocytes are the main cause of the increased glucose metabolism.

Key Words : *CPM, EPM, ^{18}FDG-PET scan*

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INTRODUCTION

Central pontine and extrapontine myelinolysis (CPM and EPM) is a disorder which is caused by rapid correction of hyponatremia (1) or by extreme serum hyperosmolality (2). It reportedly occurs in various situations including chronic alcoholism (3), nutritional deficiency (3), pregnancy (4), organ transplantation (5), and burn (2). The lesion sites are also variable including the thalamus, subthalamic nucleus, striatum, internal capsule, amygdaloid nucleus, lateral geniculate body, cerebral and cerebellar white matters, and spinal cord as well as basis pontis (6). Pathologically, the lesion shows destruction of the medullated sheath with sparing of the axis cylinders and nerve cells. There are reactive phagocytes and glial cells, but signs of inflammation are absent (7). Currently, osmotically-damaged endothelial cells by rise in serum sodium level are believed to release some myelinotoxic factors or to disrupt the blood brain barrier (8). We recently experienced a case of CPM and EPM of which ^{18}FDG-PET scan showed unexpected hypermetabolism at lesion sites in the early stage of the disease which later turned into hypometabolism.

CASE REPORT

A 63-year-old woman was admitted because of decreased sensorium. She had been healthy until 11 days before admission when febrile sensation, myalgia, nausea, and vomiting developed. Nine days before admission, she had visited a local clinic because of persistent vomiting which culminated in hyponatremia 4 days before admission. Hyponatremia was corrected (Table) and, after that point, the frequency of vomiting decreased to 3 to 4 times a day. However, 2 days before admission, her level of consciousness decreased to a drowsy state and she became comatose on the day of admission. According to the department of internal medicine where she was first admitted, she was comatose without eye opening, verbal output, or motor response. When she was transferred to our department, 3 days after admission, she was still in a comatose state. Her pupils were small but isocoric and reactive. Doll's eye phenomenon could be elicited and corneal reflex was present bilaterally. She showed bilateral flexion to noxious stimuli. She stabilized thereafter to a vegetative state. We proceeded to our work-ups

Table. Serial serum Na^+ and K^+ levels

Day	$[Na^+]$ (meq/L)	$[K^+]$ (meq/L)	Remarks
−7	128	3.8	Vomiting
−4	98	2.3	N/S+2 MKCl 15cc IV, 2-3 L/d
−2	104	3.0	Onset of decreasing sensorium
−1	129	3.2	
0	127	4.0	Admission
+1	131	3.6	

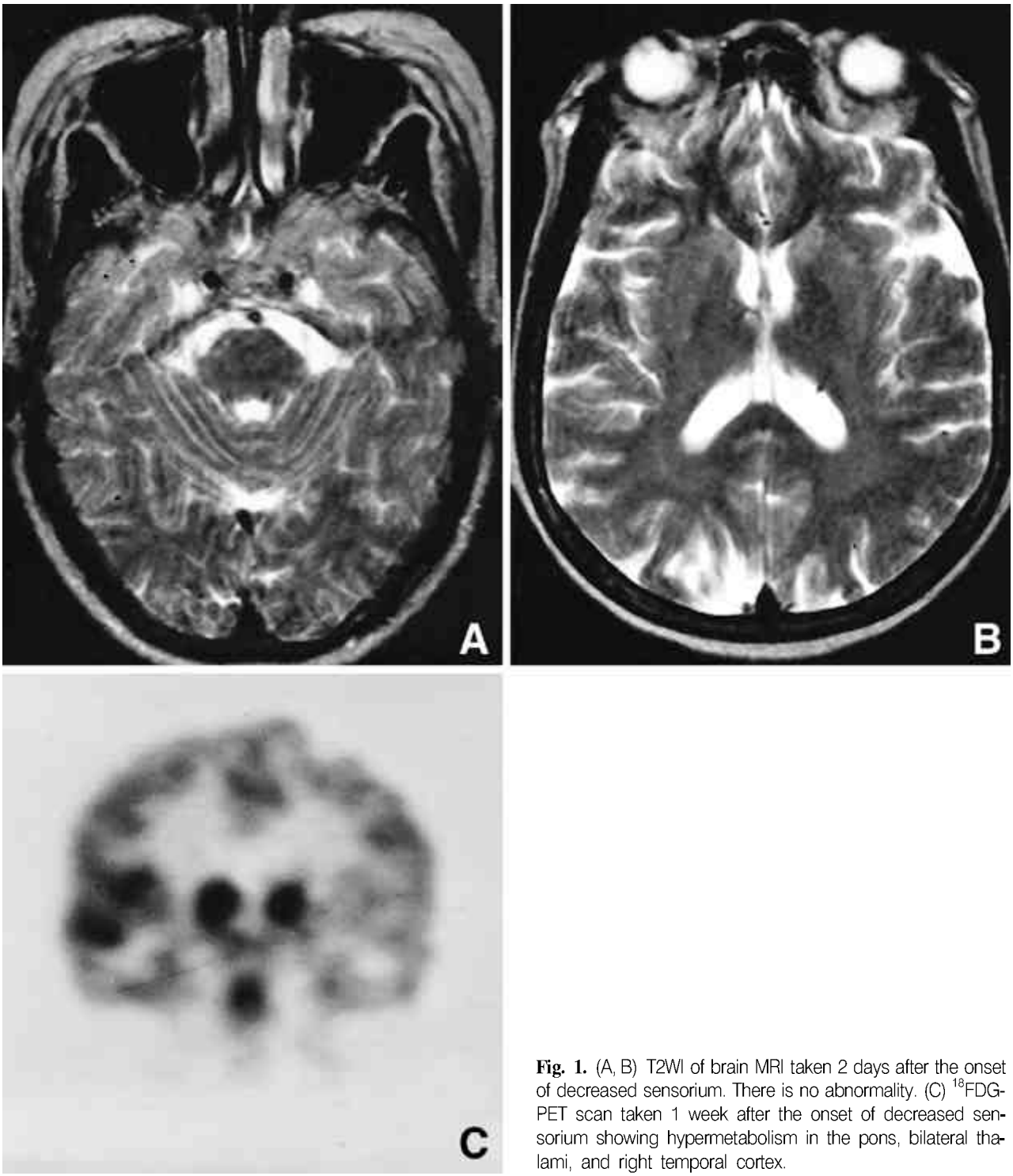

Fig. 1. (A, B) T2WI of brain MRI taken 2 days after the onset of decreased sensorium. There is no abnormality. (C) ^{18}FDG-PET scan taken 1 week after the onset of decreased sensorium showing hypermetabolism in the pons, bilateral thalami, and right temporal cortex.

under the impression of CPM and EPM.

Brainstem auditory evoked potential was normal but bilateral retrochiasmal lesions were suspected on visual evoked potential and right central conduction defect was detected on median nerve sensory evoked potential. EEG showed continuous high voltage generalized rhythmic synchronous triphasic waves. Initial brain MRI taken 2 days after the onset of decreased sensorium was normal (Fig. 1. A, B). Although ^{18}FDG-PET scan is not essential for the diagnosis of CPM itself, it happened to be taken for the differential diagnosis of coma, which showed hypermetabolism in the right temporal cortex, bilateral thalami, and pons (Fig. 1. C). There were high signal intensities on the follow-up T2-weighted image of brain

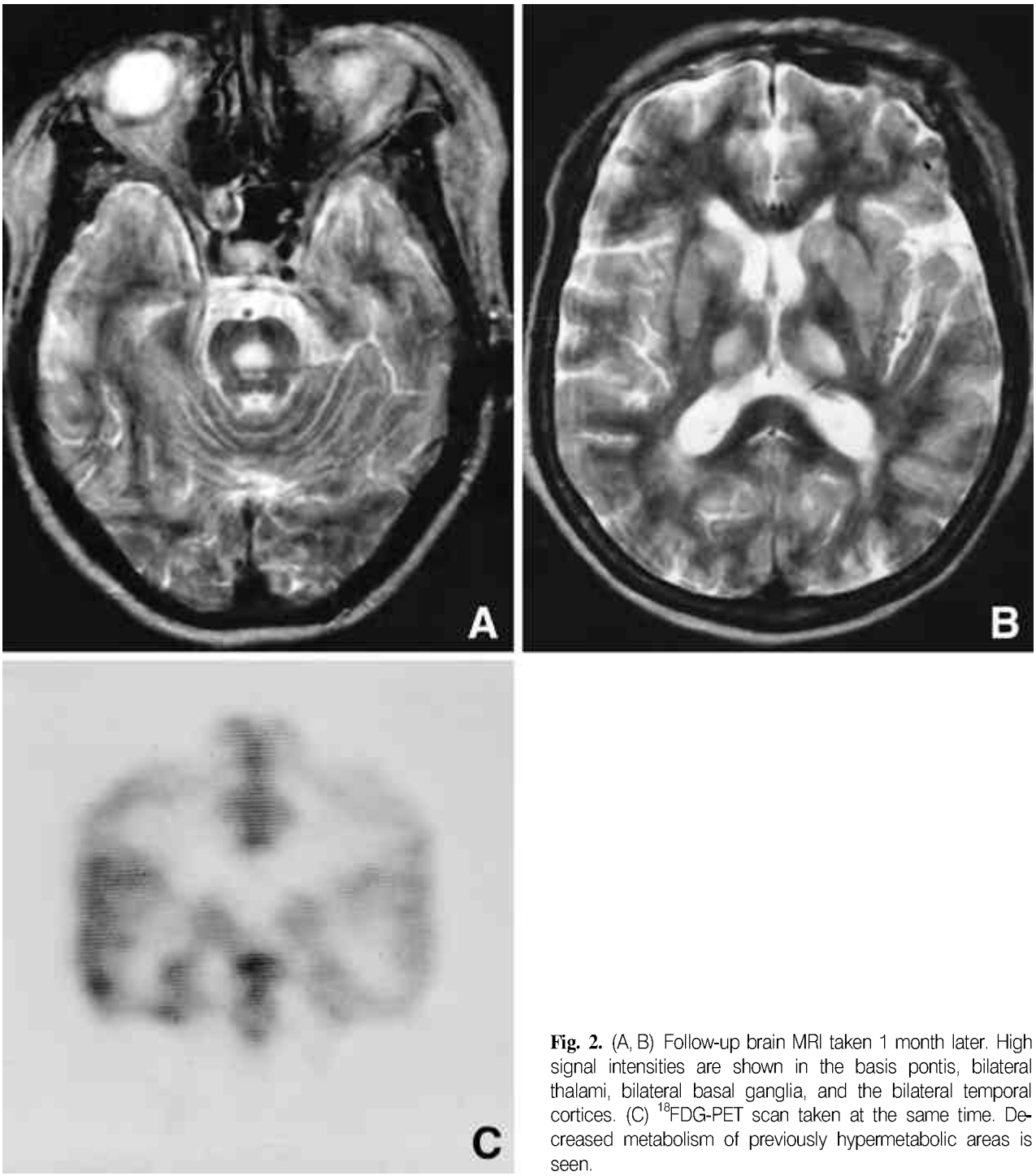

Fig. 2. (A, B) Follow-up brain MRI taken 1 month later. High signal intensities are shown in the basis pontis, bilateral thalami, bilateral basal ganglia, and the bilateral temporal cortices. (C) ^{18}FDG-PET scan taken at the same time. Decreased metabolism of previously hypermetabolic areas is seen.

MRI in the bilateral thalami, bilateral basal ganglia, periventricular white matter, and the basis pontis as well as a suspiciously high signal intensity in the bilateral temporal cortices, especially in the right (Fig. 2. A, B). The thalamic and pontine lesions turned hypometabolic but the right temporal lesion persisted as hypermetabolic on the follow-up ^{18}FDG-PET scan (Fig. 2. C). She remains in a vegetative state till now, at 1 year follow-up.

DISCUSSION

^{18}FDG-PET uses fluorodeoxyglucose (FDG) as a competitive substrate of glucose and indicates the level of glucose metabolism of a given site (9). Its utility is well established in various fields of neurology. The result of our case is quite unexpected since a destructive lesion without inflammation showed a hypermetabolism of

glucose. We propose that the increased glucose metabolism is caused by the activity of microglial cells and/or astrocytes. There have been no reports on the increased metabolism of either demyelinated nerve cells or oligodendrocytes which lost their myelin sheaths. Furthermore, the turnover rate of oligodendroglial cells is very low and only a small percentage of oligodendrocytes has a capacity to proliferate (7). On the other hand, microglial cells gather around the lesion at an early stage and act with phagocytic properties. As with astrocytes, in addition to hypertrophy, their proliferation may take place soon after injury and in an animal experiment, the mitotic index was maximal on the second or third day (7). The transient nature of hypermetabolism in our case is compatible with the above explanation. In multiple sclerosis and other demyelinating diseases, inflammation is present in addition to the previously-mentioned changes and ^{18}FDG-PET in their early stages is likely to show hypermetabolism of the lesions.

We do not know the exact nature of the right temporal lesion. It possibly represents the same abnormality as other lesions, but a definite characterization awaits confirmation by more reports.

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