Cerebral Glucose Utilization in Normal and Demented Subjects - A Quantitative Measurement using 18F-2-Fluoro-Deoxy-Glucose and Positron Emission Tomography

IV. 4 Cerebral Glucose Utilization in Normal and Demented Subjects — A Quantitative Measurement using ^{18}F-2-Fluoro-Deoxy-Glucose and Positron Emission Tomography

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The ^{18}F-fluorodeoxyglucose method and positron CT was used to determine regional cerebral glucose utilization (rCMRGLC) in four neurologically normal subjects, in two patients with Alzheimer's disease and in two with vascular dementia. Morphological changes of brain matter during aging had been studied in enormously large population using conventional X-ray CT. This study is the first step to explore the specificity of cerebral metabolic patterns in aged and/or demented brain.

Subjects and Methods

The routine ECAT examination for brain study using FDG were performed. 5 to 10 mCi of FDG was infused from forearm vein and arterial blood was sequentially taken from radial artery to measure input function of FDG to brain. Brain tissue distribution of FDG were measured using ECAT with medium shadow shield, medium resolution mode for head, and medium resolution of convolution factor. Twelve sequential scan from infusion time (=0) to 60 minutes were normally performed at the level of 50 mm above orbito-meatal line. Then, time dependent changes of tissue distribution can be measured. Finally, tissue distribution of FDG were measured at the level of 30 mm, 50 mm and 70 mm.

Absolute values of glucose utilization were calculated according to the Sokoloff's model[1] and simplified equation by Rhodes et al.[2] For the practical calculation, we needed the integrated value of plasma FDG activity from time 0 to mid time of scanning, blood sugar, data aquisition time of ECAT, filter scale factor, cross calibration factors between ECAT and well counter used. 0.42 of lumped constant was used.[3]

Four subjects, age ranged from thirty three to seventy three, had no neorological abnormality and no organic lesions on XCT examination. Mental activity was well preserved in aged subjects. Three patients suffering from Alzheimer's disease, fifty eight, fifty nine and sixty two years old, were also examined. Duration from the onset was two years, six years and four years,

respectively. Two vascular dementias examined had clinical history of transient ischemic attack of hemiparesis but not at the ECAT examination. Recent memory and preservation of memory was mainly disturbed.

Results and Discussion

Table 1 showed values of cerebral glucose utilization in four demented patients (No. 5, No. 6; Alzheimer's disease, No. 7, No. 8; vascular dementia). Region of interest was set on frontal, temporal, occipital cortex and basal ganglion of both hemisphers. Mean values of cortical gray matter was also calculated.

In normal subjects, glucose utilization of frontal cortex in both hemisphers was prominent, indicating hyperfrontality of "working brain". No difference was observed between right and left hemispher in comparable regions.

Two aged subjects showed decreased cerebral glucose utilization in all the regions of interest compared with thirty three and fifty eight years old subjects. Kuhl et al.[4] studied healthy aged volunteers and observed significant decrease of cerebral glucose consumption with aging, but not so remarkable compared with our results. It is generally accepted that individual difference among aged subjects is increasing through aging. Critical problems of aging study might be the selection of subjects.

Characteristic findings of demented brain in both types was reduced glucose utilization of frontal and temporal cortex, indicating loss of hyperfrontality. Absolute values of severely demented patients with Alzheimer's disease (No. 5) was still higher than that of non-demented aged subjects (No. 3, No. 4). This means that decreased glucose utilization doesn't necessarily induce mental deteriorations.

In vascular dementias, affected regions observed on XCT and frontal cortex of that hemispher utilized less glucose.

In the combined study with cerebral blood flow and volume, and oxygen metabolism, in vivo observation using these methods could contribute to understanding of pathophysiology of demented brain.

References

1) Sokoloff L. et al., J. Neurochem. 28.
2) Rhodes C. G. et al., Proc. of the Third World Cong. of Nucl. Med. and Biol.
3) Kuhl D. E. et al., J. Cereb. Blood Flow Metabolism (1982).

Table 1. Regional cerebral glucose utilization (mg/100 min)

		Right hemisphere				Left hemisphere			
		F	T	O	B	F	T	O	B
No. 1	normal, 33,	6.5	6.3	5.3	6.8	6.3	6.4	5.7	6.8
No. 2	normal, 58,	6.1	5.8	5.4	5.4	6.0	6.1	5.9	5.6
No. 3	normal, 70,	4.0	3.9	3.2	3.8	3.4	3.4	3.5	3.6
No. 4	normal, 73,	3.5	3.4	3.2	3.2	3.4	3.5	3.0	3.3
No. 5	AD, 58,	3.8	3.3	4.6	4.4	4.6	4.4	4.9	4.7
No. 6	AD, 59,	1.6	1.9	3.7	2.3	1.9	2.2	3.4	2.4
No. 7	AD, 62	3.1	2.3	3.3	3.4	3.1	2.2	3.2	3.1
No. 8	VD, 73,	3.4	3.9	4.8	3.7	3.5	3.7	4.7	3.4
No. 9	VD, 76,	2.9	2.7	3.8	2.0	3.1	3.4	3.8	3.3

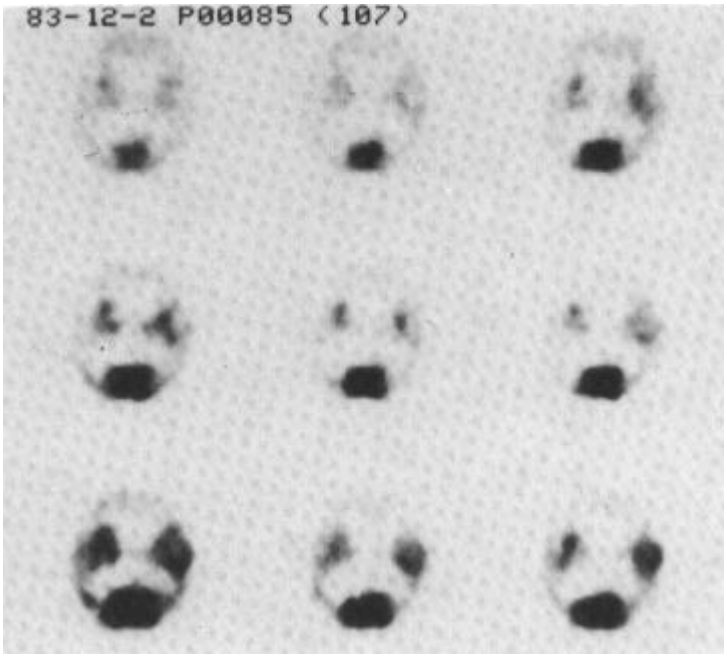

Fig. 1(a) Sequential images of ^{18}FDG distribution in Alzheimer's disease

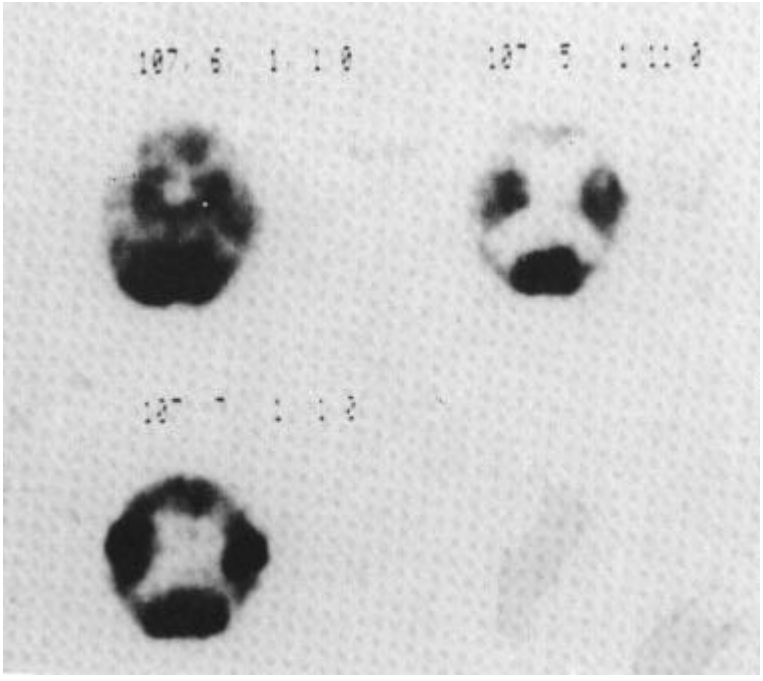

Fig. 1(b) Regional cerebral glucose utilization in Alzheimer's disease

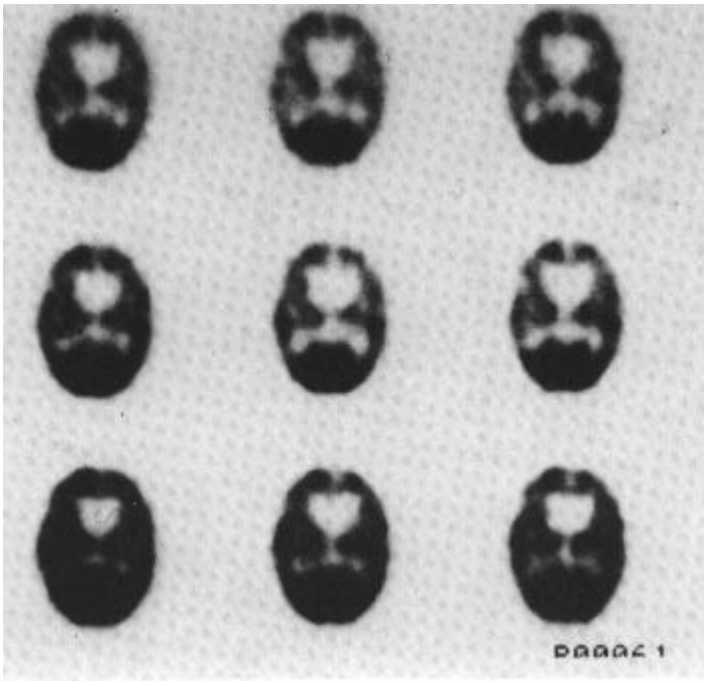

Fig. 2(a) Sequential images of ^{18}FDG distribution in vascular dementia

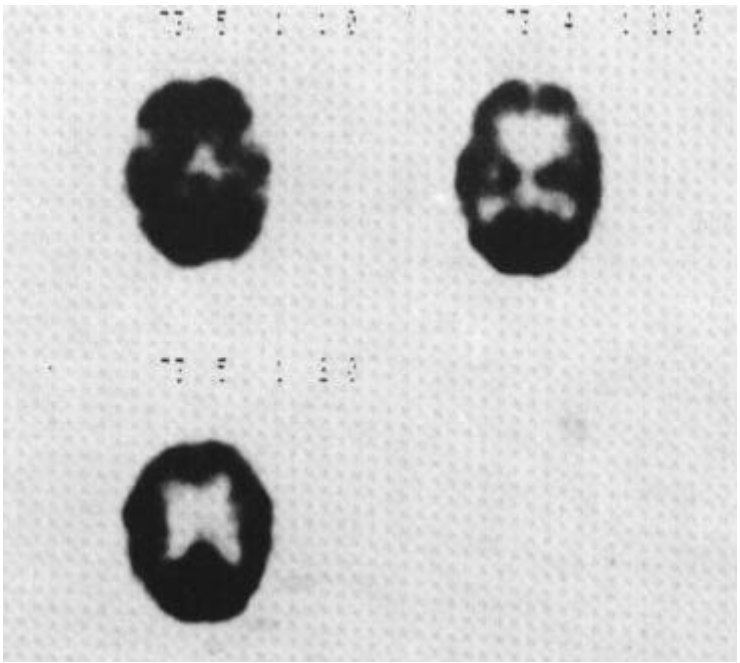

Fig. 2(b) Regional cerebral glucose utilization in vascular dementia