Summary

Evaluation of ^{18}F-FDG PET in Acute Ischemic Stroke: Assessment of Hyper Accumulation Around the Lesion

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[**Purpose**] Although pathophysiology of cerebrovascular disease has been reported previously, few clinical studies of glucose metabolism in acute stroke have been published. Purpose of this study is to evaluate glucose metabolism in acute stroke patients by ^{18}F-FDG PET.

[**Subjects and Methods**] Twenty-four patients with acute ischemic stroke were involved in this study. All subjects underwent MRI (conventional T1- and T2-weighted images, diffusion-weighted imaging, and MR angiography), CT and ^{18}F-FDG PET. ^{18}F-FDG PET was performed within 1 to 7 days after the first episode. ^{18}F-FDG PET images were visually evaluated as well as MRI and CT images.

[**Results**] Four patients out of 24 showed no abnormal ^{18}F-FDG accumulation, while MRI demonstrated abnormal signal area and abnormal vascular findings that suggested acute stroke. Decreased ^{18}F-FDG accumulation corresponding with abnormal signal area on MR images was noted in 20 cases. In 7 cases among these 20 with decreased ^{18}F-FDG, hyper accumulation of ^{18}F-FDG was recognized around the decreased accumulation area.

[**Conclusion**] Increased ^{18}F-FDG accumulation (increased glucose metabolization) around the lesion may be due to: 1) acceleration of anaerobic glycolysis, 2) activated repair process of damaged brain tissue, i.e., phagocytosis and gliosis, and 3) neuronal excitation by excito-toxic amino acids which can be released after ischemia.

Key words: Acute cerebral infarction, FDG, Excito-toxic amino acids.