

Paradoxical uptake of F-18 fluorodeoxyglucose by successfully reperfused myocardium during the sub-acute phase in patients with acute myocardial infarction

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The myocardial uptake of F-18 fluorodeoxyglucose (FDG) has been shown to indicate ischemia. To elucidate whether this is applicable to reperfused myocardium in patients with acute myocardial infarction (AMI), TI-201 SPECT and F-18 FDG PET were performed in 10 patients with successfully recanalized AMI (male : female = 8 : 2; mean age = 62 ± 9 years old). Regional myocardial perfusion of the infarct-related area was classified, on the basis of TI-201 images, into 2 groups (normal and defect) during the sub-acute phase, and into 3 grades (normal, redistribution (RD), and persistent defect) during the chronic phase (1 and 3 months after onset). Regional FDG uptake was calculated as FDG uptake in the region of interest normalized relative to that in a normal area. During the chronic phase, FDG accumulated only in the region of RD, indicating ischemia, but during the sub-acute phase, FDG accumulated mainly in the peri-infarct area. To elucidate whether the reperfused myocardium itself shows signs of accelerated glucose uptake, an experimental study was performed in rats. Glucose uptake in the isolated heart was measured by deoxyglucose and ^{31}P -NMR spectroscopy, and was significantly increased after reperfusion compared with the pre-ischemic level. In conclusion, the enhancement of FDG uptake during the sub-acute phase was observed in successfully reperfused myocardium of patients with acute myocardial infarction. Such augmentation disappeared during the chronic phase. An experimental study in rats indicated that ischemia and reperfusion themselves augment glucose uptake. This mechanism may be responsible for the increase in FDG uptake of reperfused myocardium observed clinically.

Key words: F-18 FDG PET, acute myocardial infarction, TI-201 SPECT, P-31 NMR spectroscopy