18F fluoromisonidazole positron emission tomography

18 F-fluoromisonidazole (FMISO) has been suggested as an effective tracer to distinguish glioblastoma multiforme from others.

In the present clinical practice for glioma, (18)F-fluorodeoxyglucose (FDG) PET is the most common tracer for predicting prognosis and differentiating other malignant brain tumors. Amino acid tracers such as (11)C-methionine (MET) are the most useful for detecting distribution of glioma, including low-grade. Tracers to image hypoxia are under investigation for potential clinical use, and recently, (18)F-fluoromisonidazole (FMISO) has been suggested as an effective tracer to distinguish glioblastoma multiforme from others [1].

Glioblastoma recurrences with decreasing 18F fluoromisonidazole positron emission tomography (FMISO) accumulation after short-term Bevacizumab (BEV) application could derive a survival benefit from BEV treatment. Change in FMISO PET appearance can identify BEV-resistant gliomas in early-stage regardless of MRI findings in a comprehensible way [2].

Two glioblastoma multiforme patients underwent (18)F-FMISO (fluoromisonidazole) positron emission tomography study to access the tumor oxygenation status before and immediately after fractionated radiotherapy concomitant with temozolomide chemotherapy. In both cases, a prominent (18)F-FMISO tumor accumulation observed in the first study was notably decreased in the second study, which was supposed to be a reoxygenation of the tumor. As far as they investigated, this is the first report of the changes of oxygenation status in glioblastoma multiforme treated through radiation therapy with temozolomide [3].

A study aimed to characterize hypoxic, but salvageable, tissue imaged by (18)F-fluoromisonidazole (18)F-FMISO), combining with perfusion computed tomography (PCT) for regional cerebral blood flow (rCBF) measurement and metabolism by microdialysis (MD) in aneurysmal subarachnoid hemorrhage (SAH) patients. (18)F-FMISO positron-emission tomography (PET)/CT was performed within the period of possible vasospasm (day 6.8+/-3 after SAH) in seven SAH patients. In parallel, rCBF was determined within the MD region of interest (MD-ROI) (n=5). The MD catheter was inserted into the brain parenchyma with highest risk for ischemia; extracellular levels of glutamate and energy metabolites were registered at time of PET and hourly for 10 days. Twelve-month outcome was evaluated. In asymptomatic patients (n=3) no hypoxia was detected and glutamate levels were low (<10 mmol/L), whereas symptomatic patients had higher glutamate concentrations (P<0.001). Increased (18)F-FMISO uptake within the MD-ROI (n=3) was related to higher glutamate levels, while rCBF was above the ischemic range. Hypoxia (increased (18)F-FMISO uptake) was present in symptomatic patients and associated with relevant metabolic derangement of extracellular glutamate levels, whereas energy metabolism and rCBF were preserved. This technique has the potential to improve our understanding of the role of cellular hypoxia in aneurysmal SAH [4].

1)
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