REVIEW

Annals of Nuclear Medicine Vol. 17, No. 5, 341–350, 2003

Pathophysiology and diagnosis of hibernating myocardium in patients with post-ischemic heart failure: the contribution of PET

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Identification and treatment of hibernating myocardium (HM) lead to improvement in LV function and prognosis in patients with post-ischemic heart failure. Different techniques are used to diagnose HM: echocardiography, MRI, SPECT and PET and, in patients with moderate LV impairment, their predictive values are similar. There are few data on patients with severe LV dysfunction and heart failure in whom the greatest benefits are apparent after revascularization. Quantification of FDG uptake with PET during hyperinsulinemic euglycemic clamp is accurate in these patients with the greatest mortality risk in whom other techniques may give high false negative rates.

The debate on whether resting myocardial blood flow to HM is reduced or not has stimulated new research on heart failure in patients with coronary artery disease. PET with $H_2{}^{15}O$ or $^{13}NH_3$ has been used for the absolute quantification of regional blood flow in human HM. When HM is properly identified, resting blood flow is not different from that in healthy volunteers although a reduction of ~20% can be demonstrated in a minority of cases. PET studies have shown that the main feature of HM is a severe impairment of coronary vasodilator reserve that improves after revascularization in parallel with LV function. Thus, the pathophysiology of HM is more complex than initially postulated. The recent evidence that repetitive ischemia in patients can be cumulative and lead to more severe and prolonged stunning, lends further support to the hypothesis that, at least initially, stunning and HM are two facets of the same coin.

Key words: coronary artery disease, congestive heart failure, positron emission tomography, myocardial blood flow, myocardial metabolism