



《特別講演 I》

Diabetes and Heart: Insight by Imaging

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It has long been known that diabetic patients have myocardial dysfunction and heart failure not attributable to any known cardiac disease. Abnormal intracellular calcium metabolism and coronary regulation, autonomic neuropathy, and defective glucose and fatty acid metabolism have been proposed as pathogenetic mechanisms for this diabetic heart disease.

As diabetes has significant effects on circulating substrate levels, it can be assumed that cardiac substrate metabolism is also altered in diabetes. In addition to potential changes in glucose and FFA metabolism, there are changes in concentrations of lactate and ketone bodies leading their increased uptake in uncontrolled diabetes.

In Type 1 diabetic patients, two studies have reported preserved myocardial glucose uptake despite peripheral insulin resistance and reduced glucose uptake in skeletal muscle. In Type 2 diabetic patients, the results are more controversial. In some studies reduced myocardial glucose uptake was observed in diabetic heart, while in some studies no difference was found. Most of those studies that were performed during standardized metabolic conditions with comparable insulin, glucose and FFA concentrations found similar myocardial glucose uptake in Type 2 diabetic and non-diabetic subjects.

A recent study applying PET technique demonstrated that FFA uptake in the femoral muscle was decreased by about 25 % in the glucose-intolerant group, whereas no differences could be observed in the myocardial FFA uptake between the two groups. Thus, in subjects with disturbed glucose tolerance, heart and skeletal muscle may differ with respect to the substrate utilization. The study also argued against the hypothesis that excessive FFA utilization per se is the key explanation for impaired glucose utilization.

Despite the limitations in the above-mentioned studies, one may summarize that during normal insulin, glucose and FFA concentrations there seems to be no major defect in myocardial substrate metabolism in diabetic subjects. However, there are no data about myocardial glucose uptake during different metabolic conditions, during ischemia or exercise. Thus, these studies do not exclude that in the conditions where circulating FFA concentrations are increased, FFA utilization could be enhanced and inhibiting glucose metabolism.

Diabetic heart has also been characterized by abnormal coronary vascular function. Although the exact mechanisms have remained unsolved, this may also contribute the cardiac disease associated with diabetes. The proposed mechanisms have been hyperglycaemia, hyperinsulinemia, vascular insulin resistance and whole body insulin resistance. Of these, the recent studies suggest that chronic hyperglycemia itself may be the most important factor.