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Lipid metabolism in the heart —Contribution of BMIPP to the diseased heart—

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Lipid contributes greatly in cardiac metabolism to produce high energy ATPs, and is suggested to be related to the progression and deterioration of heart disease. It is fortunate that the I-123-betamethyliodophenylpentadecanoic acid (BMIPP) imaging technique is now available in determining heart condition, but we must be cautious about the interpretation of images obtained with this new tracer. From the uptake of BMIPP into the cell to breakdown and catabolism of it, there exist so many critical enzymatical pathways relating to the modification of BMIPP imaging. In clinical evaluation, the image will be translated as the integral effects of these pathways. In other words, we must be aware of these critical pathways regulating lipid metabolism and modifying factors in order to correctly understand BMIPP imaging.

Lipid transport is affected by the albumin/FFA ratio in the blood, and extraction with membrane transporter proteins. Fatty acid binding protein (FABP) in the cytosole will play an important role in regulating lipid flux and following metabolism. Lipid will be utilized either for oxidation, triglyceride or phospholipid formation. For oxidation, carnitine palmitoil transferase is the key enzyme for the entrance of lipid into mitochondria, and oxidative enzymes such as acyl CoA dehydrogenase (MCAD, LCAD, HAD) will determine lipid use for the TCA cycle. ATPs produced in the mitochondria again limit the TG store.

It is well known that BMIPP imaging completely changes in the ischemic condition, and is also shown that lipid metabolical regulation completely differs from normal in the very early phase of cardiac hypertrophy. In the process of deteriorating heart failure, metabolical switching of lipid with glucose will take place.

In such a different heart disease conditions, it is clear that lipid metabolical regulation, including many lipid enzymes, works differently from in the healthy condition. These lipid enzymes are regulated by nuclear factor peroxisome proliferator-activated receptors (PPAR) just like a conductor of an orchestra. Most of the regulating mechanisms of the PPAR are still unknown, but reduction of this nuclear factor is shown in the process of decompensated heart failure.

This review is based by mostly on our fundamental and Japanese clinical data. BMIPP has been used clinically in abundant cases in Japan. In such situations, further correct information on lipid metabolism, including BMIPP, will contribute to the understanding of deteriorating heart disease and its prognosis.

Key words: cardiac lipid metabolism, BMIPP, heart failure, hypertrophy, prognosis