## Myocardial CD36 expression and fatty acid accumulation in patients with type I and II CD36 deficiency

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Long-chain fatty acids (LCFA) are one of the major cardiac energy substrates, so understanding LCFA metabolism may help in elucidating the mechanisms of various heart diseases. CD36 is a multifunctional membrane glycoprotein that acts not only as a receptor for thrombospondin, collagen and oxidized low density lipoprotein but also as a receptor for LCFA. We investigated the relationship between CD36 expression in myocardial capillary endothelial cells and myocardial LCFA uptake in patients with CD36 deficiency. We analyzed CD36 expression in blood cells from 250 patients with heart diseases by means of a flow cytometer. In 218 patients, myocardial LCFA scintigraphy was performed with <sup>123</sup>I-β-methyl-p-iodophenyl pentadecanoic acid (BMIPP). In 5 patients, myocardial capillary endothelial cells were examined immunohistochemically for CD36 expression. Eleven patients (4%) showed signs of type I CD36 deficiency (neither platelets nor monocytes expressed CD36). Twenty patients (8%) had type II CD36 deficiency (monocytes expressed CD36 but platelets did not). In all 11 patients with type I CD36 deficiency, no BMIPP accumulation was observed in the heart, but in 13 patients with type II CD36 deficiency, BMIPP accumulation in the heart was focally reduced, but there were no patients without BMIPP accumulation in the heart. Although the myocardial capillary endothelial cells from two CD36positive patients expressed CD36, those from two patients with type I CD36 deficiency did not. In a patient with type II CD36 deficiency, some capillary endothelial cells displayed patchy CD36 expression.

CD36 deficiency was documented in 31 (12%) patients with heart diseases. Because CD36 was not expressed in the myocardial capillary endothelial cells in patients with type I CD36 deficiency, type I CD36 deficiency is closely related to lack of myocardial LCFA accumulation and metabolism in the myocardium.

Key words: CD36 deficiency, fatty acid metabolism, 123I-BMIPP, heart disease, cardiomyopathy