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A patient with type I CD36 deficiency whose myocardium accumulated ^{123}I-BMIPP after 4 years

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A 73-year-old man with aortic regurgitation was examined by ^{123}I-α-methyl-*p*-iodophenyl-pentadecanoic acid (BMIPP) myocardial single photon emission computed tomography (SPECT) in 1995. Myocardial accumulation was not evident on either the early or the delayed image obtained 15 minutes and 3 hours, respectively, after injecting ^{123}I-BMIPP. Flow cytometric analysis of CD36 expression in monocytes and platelets identified a type I CD36 deficiency. The patient was hospitalized for severe heart failure in 1999. Upon admission, the cardiothoracic ratio on chest X-rays was 73%, and the left ventricular end-diastolic diameter on echocardiograms was enlarged to 77 mm. On the second day, we performed ^{123}I-BMIPP myocardial SPECT. Myocardial accumulation was evident in the delayed, but not in the early image. We repeated ^{123}I-BMIPP myocardial SPECT on the 10th day after admission. Myocardial accumulation was evident on both early and delayed images. ^{99m}Tc-tetrofosmin myocardial SPECT was immediately performed after ^{123}I-BMIPP myocardial SPECT to distinguish myocardial from pooling images in the left ventricle, but, because the images from both ^{99m}Tc-tetrofosmin and ^{123}I-BMIPP myocardial SPECT were idential, we considered that the ^{123}I-BMIPP myocardial SPECT images reflected the actual myocardial condition.

The CD36 molecule transports long-chain fatty acid (LCFA) on the myocardial membrane, but ^{123}I-BMIPP scintigraphy does not show any myocardial accumulation in patients with type I CD36 deficiency, indicating that myocardial LCFA uptake occurs through CD36 on the human myocardial membrane. Even though our patient had type I CD36 deficiency, BMIPP was uptaken by the myocardium during heart failure, suggesting a variant pathway on the human myocardial membrane for LCFA uptake.

Key words: BMIPP, type I CD36 deficiency, myocardial uptake