

CARDIOVASCULAR FLASHLIGHT

<https://doi.org/10.1093/eurheartj/ehac762>

Online publish-ahead-of-print 30 December 2022

Remarkable regression of diffuse coronary atherosclerosis in patients with triglyceride deposit cardiomyovasculopathy

Ken-ichi Hirano ^{1*}†, Masahiro Higashi ^{1,2†}, and Kenichi Nakajima ³

¹Department of Triglyceride Science, Graduate School of Medicine, Osaka University, 6-2-4, Furuedai, Suita, Osaka 565-0874, Japan; ²Department of Radiology, National Hospital Organization Osaka National Hospital, 2-1-14, Hoenzaka, Chuo-ku, Osaka 540-0006, Japan; and ³Department of Functional Imaging and Artificial Intelligence, Kanazawa University, 13-1 Takara-machi, Kanazawa 920-8640, Ishikawa, Japan

*Corresponding author. Tel/Fax: +81-6-6872-8215, Email: khirano@cnt-osaka.com

†The first two authors contributed equally to the study.

Triglyceride deposit cardiomyovasculopathy (TGCV) is an emerging cardiovascular disorder (ORPHA code: 565612), characterized by diffuse narrowing coronary atherosclerosis with triglyceride (TG) deposition caused by defective intracellular lipolysis. TGCV exists undiagnosed among patients resistant to standard medical and invasive therapies. Cases 1 (Panel A) and 2 (Panel B) concern patients in their 60s with refractory angina pectoris and diabetes mellitus (details described in [Supplementary material online](#)). After the TGCV diagnosis, they started dietary intake of tricaprin, which was recently proven to facilitate myocardial lipolysis in patients with TGCV. Their symptoms improved within a couple of months. Follow-up coronary computed tomography angiography with colour-coded display showed marked regression of atherosclerotic lesions with luminal dilatation (left anterior descending branch shown as red line in upper Panel A; right and left coronary arteries in upper Panel B). The low-attenuation area observed from the adventitial side (yellow with -25 to 0 and orange with 0 – 40 Hounsfield units, respectively, in the middle Panel A; see [Supplementary material online, Figures S1 and S2](#)) and its volume (yellow bars in the middle Panel B, see [Supplementary material online, Figure S3](#)) were reduced, indicating amelioration of coronary lipid involvement. These observations were associated with increased myocardial lipolysis on iodine-123- β -methyl-p-iodophenyl pentadecanoic acid scintigraphy (lower Panels A and B), but without any changes in serum lipid and HbA1c levels before and after tricaprin (see [Supplementary material online, Table S1](#)). While atherosclerosis regression following decreased serum lipid levels is well-described, the regression we observed was conceptually novel, highlighting the roles of intracellular TG lipolysis pathways in the pathogenesis and treatment of coronary atherosclerosis.

[Supplementary data](#) is available at [European Heart Journal online](#).

The investigation conformed with the principles outlined in the Declaration of Helsinki of 1964. Patients were instructed to continue other medical therapies. This study was approved by the ethical committee of Osaka University Hospital, and written informed consent was obtained from the patients.

The data underlying this article are available in the article and in its online [supplementary material](#). Any other information can be provided upon reasonable request to the corresponding author.

This study was partially supported by a research grant for rare and intractable diseases from the Ministry of Health, Labour, and Welfare (grant No. 20FC1008) to KH. This study was also supported by Nihon Medi-physics Co., Ltd. (Tokyo, Japan) in the form of donated supplies to KH.

K.H. conceived the idea, took care of patients, and wrote the manuscript. M.H. is responsible for coronary computed tomography angiography as a guarantor. K.N. is responsible for nuclear imaging of BMIPP. All authors provided critical feedback and contributed to the discussion.

K.H. holds the position of Joint Research Chair in collaboration with TOA EIYO LTD (Tokyo, Japan) since February 2021 and medical adviser for TOA EIYO LTD since December 2021. K.H. has licenced and pending patents (WO2013031729 and PCT/JP2021/008689, respectively). K.N. belongs to an endowed department that is partly funded by Nihon Medi-Physics (Tokyo, Japan). M.H. has no competing interests.

© The Author(s) 2022. Published by Oxford University Press on behalf of the European Society of Cardiology.

This is an Open Access article distributed under the terms of the Creative Commons Attribution-NonCommercial License (<https://creativecommons.org/licenses/by-nc/4.0/>), which permits non-commercial re-use, distribution, and reproduction in any medium, provided the original work is properly cited. For commercial re-use, please contact journals.permissions@oup.com

