A common heart problem caused by cancer therapy avoided blood vessel treatment

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Electromicroscopic image of damaged endothelial cells in the heart of a doxorubicin-treated mouse. The researchers found that in mouse heart, doxorubicin leads to blood vessel rarefaction, which was prevented by treatment with gene therapy using the VEGF-B growth factor. Credit: Ilkka Miinalainen, University of Oulu.
Researchers at the Wihuri Research Institute and the University of Helsinki, Finland, have found that some of the harmful effects of a commonly used cancer drug can be alleviated by using gene therapy that stimulates blood vessel growth in the heart.

Doxorubicin treatment, which is commonly used in a variety of cancers, leads to cardiac atrophy and body wasting. The researchers found that in mouse heart, doxorubicin leads to blood vessel rarefaction, which was prevented by treatment with gene therapy using the VEGF-B growth factor.

As advances in cancer treatment have decreased deaths from cancer, doxorubicin-induced heart problems have become an increasing problem. The new findings give hope that in future the heart could be protected by gene therapy, allowing more thorough cytostatic cancer treatment. "Thus, the cancer itself would be treated more effectively and the adverse effects could be avoided", explains Markus Räsänen, MD, who made the discovery during his thesis studies.

"Doxorubicin, a cytostatic agent of the anthracycline class, that was used in this study has been a target of intensive research in the scientific world for a long time, and its role has been described in thousands of research articles. This research article is the first one, where blood vessel-directed therapy has a clear protective effect against the doxorubicin toxicity", says Dr. Riikka Kivelä, who supervised the study.

"Our findings show, that especially the endothelial cells, which form the inner surface of the vessels in the heart, have an essential role in the protection against the cardiotoxicity. More preclinical studies are needed though for the development of VEGF-B gene therapy for cardiac protection in patients" elaborates Räsänen.

The results were published in the Proceedings of the National Academy
of Sciences (PNAS).


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