Abstract


Epicardial adipose tissue inflammation is related to vitamin D deficiency in patients affected by coronary artery disease.


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BACKGROUND AND AIMS: Alterations in epicardial adipose tissue (EAT) biology (i.e. increased fat thickness and inflammation) have been described in coronary artery disease (CAD) patients. In addition to its classic role in the regulation of calcium-phosphate homeostasis, vitamin D may exert immune-regulatory and anti-inflammatory effects. Whether EAT inflammation may be linked to vitamin D deficiency is still unknown. In the present study we evaluated plasma 25-hydroxycholecalciferol (25OHD) level in CAD patients and its relationship with EAT ability to locally metabolize vitamin D, EAT expression of inflammation-related molecules and EAT thickness.

METHODS AND RESULTS: Plasma 25OHD level was quantified by an immunoluminometric assay. EAT expression of inflammation-related molecules (MCP-1, PTX3, TNFα, IL-6, adiponectin), vitamin D receptor (VDR), CYP27B1 (25OHD-activating enzyme) and CYP24A1 (1,25-dihydroxycholecalciferol-metabolizing enzyme) was performed by microarray. EAT thickness was quantified by echocardiography. Median plasma 25OHD level was 10.85 ng/mL and 83% of CAD patients displayed 25OHD level below 20 ng/mL. At decreasing plasma 25OHD concentration, we observed a down-regulation in CYP27B1 and CYP24A1 level and an increased expression of VDR and pro-inflammatory cytokines (MCP-1, PTX3, TNFα, IL-6) at EAT level. No correlation was observed between plasma 25OHD level and EAT thickness.

CONCLUSION: Our data suggest an increased activation of inflammatory pathways at EAT level possibly related to systemic and local vitamin D deficiency in CAD patients. Whether maintaining an optimal vitamin D status may be helpful to reduce EAT inflammation and to prevent CAD and its progression needs further investigation.

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