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Analysis of the leukocytes proteome in atherosclerosis related and non-related to chronic kidney disease

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The major cause of mortality in patients with chronic kidney disease (CKD) is atherosclerosis related to traditional and I non-traditional risk factors. However, the understanding of the molecular specificity that distinguishes the risk factors for classical cardiovascular disease (CVD) and CKD-related atherosclerosis (CKD-A) is far from complete. Although dyslipidemia is common in CKD patients, epidemiological data show that in CKD the link between cholesterol and its fractions is not as straightforward like in the general population. CKD is frequently accompanied by reduced of plasma HDL concentrations and normal or even low serum total cholesterol and LDL concentrations. Normal HDL function is reverse cholesterol transport from peripheral cells and its transport to the liver. HDL protects of LDL against oxidation and suppress of systemic inflammation. Therefore, HDL deficiency is the key in perpetuating chronic inflammation and oxidative stress leading to atherosclerosis. On the other hand, it is suggested that lipid abnormalities in CKD are characterized by more qualitative abnormalities and may be related to HDL function rather than HDL deficiency. Mass spectrometry-based proteomic analysis is excellent, powerful tool for tracking molecular changes during progression of CKD. In this study we investigated the alterations in leukocytes protein accumulation in patients with CKD and classical cardiovascular disease (CVD) without CKD. Cells collected from patients in various stages of CKD, CVD patients without symptoms of kidney dysfunction and healthy volunteers (HVs), were analyzed by a label-free proteomic approach. Obtained cells were also analyzed in term of inflammation modulators and the oxidative status enzymes. Label-free quantitation analysis revealed characteristic proteins of particular stage of atherosclerotic plaque formation process and CKD progression. All proteomic data were subjected to bioinformatic analysis for identification of specific for CKD and CVD pathways. Further research should focus on precise profiling of metabolites and/or lipids present in cells during the atherosclerosis development.

Biography

Joanna Tracz has received his/her Bachelor's degree in 2015 and completed his/her Master's degree in Chemical Science in 2016 and Biological Science in 2017. Presently, he/she is a PhD student in Laboratory of Mass Spectrometry at the Polish Academy of Sciences. He/she is involved in project concerning the molecular mechanism of atherosclerosis progression in chronic kidney disease.

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