Markers of Carotid Atherosclerosis in Patients with Type 2 Diabetes Mellitus

Daniel Petrovič

Institute of Histology and Embryology, University of Ljubljana, Korytkova 2, 1105 Ljubljana, Slovenia

Corresponding author: Daniel Petrovič, Faculty of Medicine, MD, PhD, FESC, Institute of Histology and Embryology, University of Ljubljana, Korytkova 2, 1105 Ljubljana, Slovenia, Tel: +38615437467; Fax: +38615437361; E-mail: daniel.petrovic@mf.uni-lj.si

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Editorial

It is generally accepted that beside cholesterol accumulation, chronic inflammation and immune system are very much involved in the pathogenesis of atherosclerosis [1]. A genetic variability of the candidate genes implicated in atherosclerosis (i.e. candidate genes of inflammation, oxidative stress, growth factors, renin-angiotensin aldosterone system, and so forth) has so far been demonstrated in several reports to affect the development of atherosclerosis [2]. Genetic variability in the candidate genes implicated in atherosclerosis may alter their transcriptional activity and contribute to susceptibility to cardiovascular disease [3-11]. In the pathogenesis of atherosclerosis genetic, environmental, and epigenetic factors are involved [4-5].

Genetic and epigenetic factors may be studied with two main approaches, candidate gene approach and genome wide association study (GWAS) approach [4-9,11]. Candidate gene approach is hypothesis-driven approach, whereas in GWAS approach appropriate subset of patients (i.e. patients with coronary artery disease, myocardial infarction, T2DM, bronchial asthma, Crohn disease and so forth) are genetically analyzed and distribution of genotypes/alleles compared with appropriate control subjects [4-8].

In cardiovascular research, s great potential of systems-based methodologies has been demonstrated in several phenotypes, including atherosclerosis [4,5]. Omics data (genomics, proteomics, metabolomics, transcriptomics, epigenomics, nutriepigenomics) are becoming widely available by progress in high-throughput molecular profiling. However, integration between the omics data, biological sciences, clinical data and other clinical diagnostic modalities are going to be essential for the success of translation of integrative analysis of biological biomarkers into the clinical practice.

There are two non-invasive markers of carotid atherosclerosis available, carotid intima-media thickness (CIMT) and carotid atherosclerosis (demonstrated as number of affected segments of carotid arteries, the sum of plaques thickness). In last decade it has been accepted that CIMT is biologically distinct from atherosclerotic plaques, not really atherosclerosis, but both represent an indicator for cardiovascular risk [12]. In contrast, carotid plaques are a characteristic phenotype of atherosclerosis, not a simple continuum of CIMT progression, and predict the cardiovascular disease better than CIMT [12,13].

So far, several studies using either candidate gene approach or GWAS approach have been reported several genetic markers for either CIMT or subclinical carotid atherosclerosis, however most reports studied patients in general population, and only few enrolled subjects with type 2 diabetes mellitus (T2DM) [4-9,11]. It should be emphasized, however, that whatever approach (candidate gene or GWAS approach) may be used, crucial is well-designed and appropriately selected study group. Moreover, according to expected great increase in the incidence of T2DM in coming years, patients with T2DM should be studied more often.

In patients with T2DM, cardiovascular complications are reported about 15 years earlier than in the population without T2DM, and they have worse prognosis as well [14,15]. Beside traditional risk factors identified in the general population, such as arterial hypertension, dyslipidemia, cigarette smoking and parental history of cardiovascular diseases, T2DM and different genetic factors also contribute to the high prevalence of cardiovascular disease in patients with diabetes mellitus [14-16].

To conclude, good cooperation between clinicians, geneticists, biologists, epidemiologists, statisticians, and computing scientists are essential for advance of carotid artery disease and cardiovascular diseases in general [14-16]. Secondly, well-designed and appropriately powered studies are needed to enroll different subset of patients with different environmental and genetical background.

References


