The best treatment of cardiovascular disease is to prevent the disease progression and predict future events.

SMART-er diagnostics in cardiology

The project "Simulation of coronary heart disease: a support tool for clinical decisions: SMARTool" (Grant Agreement No. 689068), funded by the European Commission under the Horizon 2020 Program and coordinated by the National Research Council - has just concluded. Institute of Clinical Physiology (CNR - IFC) of Pisa, developed a platform, based on artificial intelligence and cloud technology, for the management of patients with coronary heart disease by standardising and integrating heterogeneous clinical data. The platform includes a series of multiscale and multilevel models of coronary plaque characterisation and progression based on non-invasive coronary CT angiography (CCTA) [1-3]. Integrated with data of various patient-specific nature (symptoms, risk factors, lifestyle, blood tests, genetic data, lipid profile) and processed by AI algorithms.

Patient-specific CAD stratification
A machine learning based risk stratification model will be implemented by patient genotyping and phenotyping.

Patient-specific CAD diagnosis
CAD diagnosis is based on semi-automate 3D arterial reconstruction and non-invasive FFR measurement.

CAD prognosis decision support
Models of site specific plaque growth and prediction will be implemented for the prediction of regions prone to plaque growth.

Cloud based platform
All outcomes are integrated into a unified cloud based platform.

Treatment decision support
A virtual angioplasty tool will be developed.

Point-of-care testing
SMARTool will deliver a microfluidic device for on-chip blood analysis usable in CDSS.

Atherosclerosis is the pathology underlying coronary artery disease (CAD), a significant cause of morbidity and mortality, with a cost to the EU of almost 196 billion euros a year; its clinical and associated manifestations range from stable angina to acute events such as heart attacks and sudden coronary death. Local factors (high-risk plaques) and individual systemic biohumoral factors (inflammatory/thrombogenic/lipid profile and genetic profile) contribute to the development and progression of CAD.

The main reasons for the low efficacy of most of the strategies for preventing acute complications of CAD are linked both to the poor prognostic value of classic risk factors alone and to the difficulty of identifying the "high risk" plaques. The occlusive artery thrombosis and the onset of the acute ischemic coronary event could probably be triggered by the combination of local complications at plaque level (rupture, erosion, etc.) and systemic alterations with elevated blood levels of inflammatory molecules and/or thrombogenesis. The complexity of these processes and the number of factors involved explains why statistical risk models often fail in investigating and predicting the evolution of the disease.

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In a vision of e-Health 2.0, supported by advanced computer systems optimized on artificial intelligence (IA), a possible strategy is to create a predictive model of severity and progression of the plaque that integrates, in a single platform, all the local and systemic factors of the individual patient, from parameters with complex statistical models, valid in a population with different degrees of severity and progression of the disease and frequency of events. This platform could be used as a tool to support clinical decision-making for risk stratification, diagnosis, prognosis and treatment of patients with coronary artery disease.

The project confirmed that the concentration in the blood of inflammatory molecules such as Interleukin-6, equal to twice the value compared to healthy subjects, is associated with a higher risk of disease progression over time [4]. While a low plasma concentration of leptin, a hormone involved in the regulation of the energy balance, with a reduction up to 50% of the value compared to the control group, is directly linked to the severity of the disease [5]. The advanced analysis of the lipid profile in the blood also revealed a series of lipid classes associated with the presence and severity of the disease.

References


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