Abstract: P3423

The contribution of genetics to premature CAD through different degrees of lifestyle factors: a matter of relative significance?

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Topic(s):
Risk Factors and Prevention – Epidemiology

Citation:

Introduction: Coronary artery disease (CAD) is a multifactorial process with substantial genetic contribution. However, genetic predisposition among patients with a different number of lifestyle factors and premature CAD, remains a complex and thoroughly unexplored topic.

Objective: To evaluate, in a young population, the importance of conventional risk factors as well as of a genetic risk score in the appearance of CAD.

Methods: A case-control study was conducted with 1075 patients from the GENEMACOR study population, under 50 years-old (555 cases, 86.8% male, mean age 44.1±4.9 years and 520 controls, 86.2% male, mean age 44.3±4.8 years). Univariate analysis addressed the association of different modifiable risk factors with premature CAD. Genetic risk score (GRS) was computed comprising 33 genetic risk variants in a multiplicative method. GRS was evaluated according to the number of traditional risk factors and risk for premature CAD was estimated and its independent predictive value estimated by logistic regression.

Results: 72.6% of patients had ≥3 risk factors vs 31.2% of controls (p<0.0001). In comparison with having no risk factors (rf), patients with 1 rf had an OR of 2.79 (1.19-6.53; p=0.015), patients with 2 risk factors had an OR of 6.87 (3.03-15-57, p<0.0001) and patients with 3 modifiable risk factors had a OR of 24.17 (10.87-53.73, p<0.0001) – graph 1. In this young population, mean GRS level was consistently higher among patients with coronary artery disease comparing with a healthy population (0.6±0.6 vs 0.4±0.4, p<0.0001, respectively) – graph 2. GRS in multivariate analysis, proved to be an independent predictor for premature CAD (OR 1.71, CI95% 1.25-2.34, p=0.001). Conclusion: In our population, GRS was an independent predictor for premature CAD. In young patients with ≥3 risk factors, genetics play a less decisive role in the development of CAD. Even in young patients, modifiable risk factors should be addressed aggressively as they may represent a higher burden than genetic predisposition itself.
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