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COMMENT

Parental history as a independent risk factor of coronary heart disease in patients under 60 years of age

La storia familiare come fattore di rischio indipendente di cardiopatia ischemica in pazienti di età inferiore a 60 anni

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A comment to the paper of Hoseini K. et al., Monaldi Arch Chest Dis 2008; 70: 84-87

Since the completion of the human genomic sequence, more and more expectations suggest that a detailed catalogue of all important common genetic susceptibility variants for human diseases, including coronary heart disease (CHD) and related atherosclerotic cardiovascular disease (CVD), will soon be available.

A reported "family history" of premature cardiovascular disease (CVD) is an independent risk factor for CVD and has been a recommended risk stratification marker in the US-Framingham risk score [1] and in the European SCORE risk tools [2].

In the paper by Hoseini *et al.* in this issue of the Monaldi Archives [3], despite the apparent limits of a retrospective study, the authors clearly demonstrate the powerful relationship between CHD family history and presence and severity of CHD in relatives, independently from other traditional risk factors, particularly in younger middle-aged adults, suggesting a possible age-dependent effect of familial premature CHD. The study also demonstrates a stronger relationship between CVD and maternal premature CVD. This link underlines the utility of including information of family history of premature CHD in the current global risk assessment methods and practice guidelines.

Several limitations should be acknowledged as well. The study sample is limited both geographically and ethnically. Its results remain to be confirmed in different populations. The potential hazards of inaccurate reporting of family history have been well acknowledged by Nasir *et al.* [4].

Although the data have been adjusted for the most common CVD risk, confounding factors still remain derived from covariates not measured in the study.

Additionally, the study lacked of power to detect modest effect sizes for maternal and paternal premature CVD.

In the Framingham Offspring Cohort, the positive predictive value for a reported myocardial infarction in a father before age 55 years was only 28% when participant report was validated with medical records [5]. Hosseini *et al*. [6] provided the strongest level of evidence for the association between family history and CAD (when in clinical practice the family history is usually elicited by report and not by use of primary medical records); although non demonstrated, family history still results as an independent risk factor of premature CHD within families. In addition, no CVD risk factor adjustment was made.

Nevertheless, the fact that validated parental CVD in younger subjects was found significantly associated with CAD substantially extends prior evidence.

Moreover, the current study's finding that maternal premature CVD was associated with CHD is consistent with previous data linking maternal CHD with offspring CHD [7], including some studies that suggest a more powerful risk conferred by history of maternal versus paternal CVD [8].

Even when genome-wide genetic tests eventually will become available, an accurate family history will likely still have an important role in effective clinical practice and public health decision-making. These findings may have clinical implications as further evidence accumulates regarding the appropriate use of family history information together with subclinical atherosclerosis screening (coronary artery calcium screening, carotid intima-media thickness, C-reactive protein etc), even above established risk factors.

Thus, family screening of patients with premature coronary artery disease emerges both as an opportunity and a challenge of cardiovascular health in the 21st century [9].

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