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Letter to the Editor

Coronary artery disease in high risk South Asian immigrants: Role of dysfunctional HDL in risk prediction

South Asian Immigrants (SAIs) carry excessive burden of coronary artery disease (CAD) that may not be fully explained by the traditional CAD risk factors [1]. Among dyslipidemias, low levels of high density lipoprotein (HDL) are commonly seen in SAIs [2]. Previously, we have shown the presence of a non-functioning or Pro-inflammatory HDL called dysfunctional HDL (Dys-HDL) in up to 50% SAIs [3,4]. Knowing that HDL prevents future risk of CAD by its anti-oxidant, anti-inflammatory and anti-thrombotic functions, it is imperative to understand the impact of Dys-HDL on CAD and if it can help identify high-risk groups early for timely management. A pilot study was conducted to determine the association of Dys-HDL with common carotid intima media thickness (CIMT) and coronary artery calcium scores (CACS), the surrogate markers of sub-clinical CAD in high-risk SAIs. After receiving Institutional Review Board (IRB) approval and using a cross-sectional study design, convenient but diverse ethnic sample of 1st and 2nd generation SAIs (mainly Hindus) between the ages of 35 and 70 years without known history of CAD were recruited from the main Hindu temple in Jacksonville, Florida. Information was obtained on socio-demographic and clinical parameters. Carotid ultrasound Doppler and multi-detector CT was performed for CIMT and CACS respectively. Dys-HDL was done using a cell-free assay and the HDL inflammatory index was assessed [5]. Though 40 eligible participants were recruited, only 28 provided complete information on the outcome parameters. Metabolic syndrome (MS) was seen in 35.7%. Dys-HDL was found in 37.1%, positive CIMT of $\geq$0.8 mm was seen in 14.3% and abnormal CACS (>0) was present in 21.43% of the participants. On univariate analysis, family history of heart disease ($p = 0.029$), low LDL levels ($P = 0.047$) and low total cholesterol levels ($P = 0.053$) were found to be the predictors of Dys-HDL. No association of Dys-HDL was seen with CIMT ($P = 0.273$), MS ($P = 0.417$) or with CACS ($P = 0.629$).

1. Conclusion

For over 50 years, a low HDL level is considered as an independent marker of CAD risk, however it has not yet been successfully targeted for the prevention or treatment of CAD. Successful exploitation of HDL for CAD management will depend on improved understanding of the structure-function relationships of HDL in at-risk populations. This is important for the development of biomarkers that reflect the functionality of HDL than the plasma levels of HDL or Apo A-I and can guide both the development of anti-atherogenic drugs and the clinical management of patients. SAIs, though are the fastest growing immigrant population in the US with highest CAD morbidity and mortality, recruiting them in studies has remained a challenge. This concern can be addressed by improving and increasing the awareness within SAI community of CAD risk and methods to prevent early CAD through education. Additionally, collaborative efforts and partnerships with South Asian community organizations and academic institutions will be fruitful in engaging substantial portion of at-risk groups in research trials so that the needed information on the causes of CAD risk can be evaluated. Further research is required to support the current study findings.

Conflict of interest

None.

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