Association of lipoprotein(a) with CT coronary calcium score in a multiethnic, asymptomatic cohort of patients referred to a tertiary lipid clinic

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Background and Aims

The association between lipoprotein(a) (Lp(a)) and subclinical coronary atherosclerosis remains unclear and the influence of ethnicity on this relationship is unestablished.

Our objective was to evaluate the association between serum Lp(a) concentration and CT coronary artery calcium (CAC) score within a multi-ethnic cohort of asymptomatic patients.

Methods

We performed a retrospective analysis of asymptomatic patients with dyslipidaemia referred to the Lipid clinic at Imperial College Healthcare NHS Trust. All patients underwent CT coronary artery calcium (CAC) scoring and serum Lp(a) testing between December 2015 and March 2019. Patients aged <40 and >75 years and those with a personal history of atherosclerotic cardiovascular disease were excluded.

Patients were stratified as follows: CAC score =0 or >0; and Lp(a) <40mg/dL and ≥40mg/dL (corresponding to the 80th percentile in population studies¹). The association of Lp(a) and CAC score was assessed using multivariate logistic regression adjusting for age, sex, statin use and traditional cardiovascular risk factors.

Results

510 patients were included in the analysis. Baseline Lp(a) concentration varied significantly by ethnicity (median 72.5mg/dL, 34.8mg/dL, and 22.9mg/dL in Blacks, South-Asians, and Caucasians respectively).

Univariate logistic regression (scan QR code):

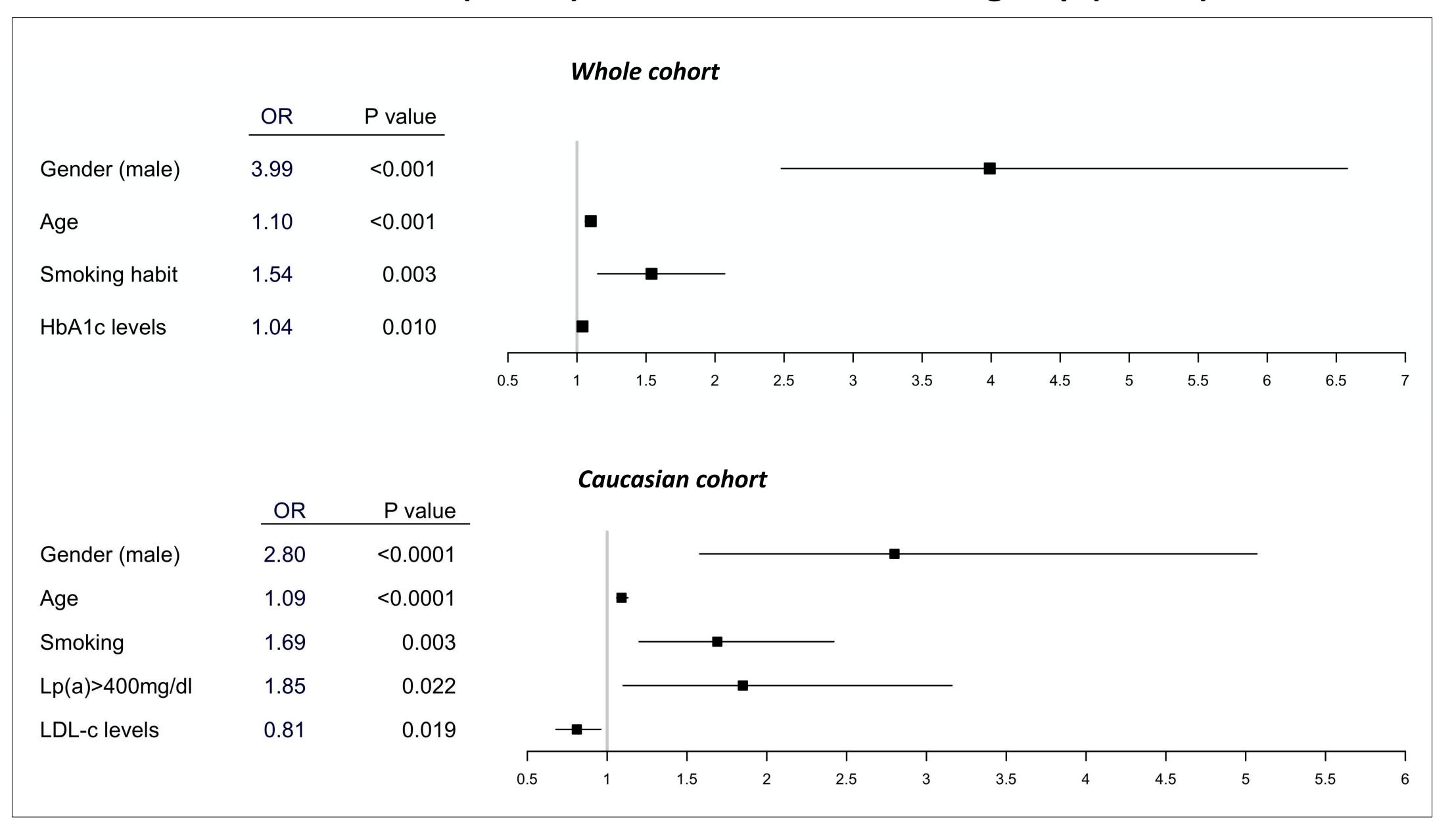
<u>Caucasian</u>: Elevated CAC associated with older age, male gender, smoking history, elevated Lp(a) ≥40mg/dL, total cholesterol and statin use.

South Asian: Elevated CAC associated with older age, total cholesterol and hypertension.

Multivariate logistic regression: Within the whole study population, elevated CAC was associated with older age, male gender, HbA1c levels and smoking, but not Lp(a).

Subgroup analysis by ethnicity identified positive correlation between Lp(a) and CAC within the Caucasian cohort, which remained significant (p<0.05) after adjusting for traditional cardiovascular risk factors – Figure 1. Lp(a) did not correlate with CAC within other ethnic groups.

Figure 1. The association of CAC > 0 with lipoprotein(a) and other cardiovascular risk factors in the whole cohort (n=510) and in the Caucasian subgroup (n=306).



Conclusions

Positive association between elevated Lp(a) concentration and CAC score was identified in asymptomatic Caucasians, however, not within other ethnic groups.

Our data implies that Lp(a), at least in Caucasians, accelerates the development of subclinical coronary atherosclerosis.

Further studies are needed to evaluate the influence of other ethnicities on this association.

Reference

1. Kamstrup PR, Tybjaerg-Hansen A, Steffensen R, Nordestgaard BG. Genetically elevated lipoprotein(a) and increased risk of myocardial infarction. JAMA. 2009 Jun 10;301(22):2331–9.









