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SPHINGOMYELIN CONTENT OF ERYTHROCYTE MEMBRANES IS INCREASED IN PATIENTS WITH ACUTE CORONARY SYNDROME

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Objective Intraplaque haemorrhage-mediated accumulation of erythrocytes has been recognised as a novel contributor in lipid core formation and unstable atherosclerotic plaques. We hypothesized that sphingomyelin content is increased in the erythrocytes from patients with acute coronary syndrome (ACS) and may thus represent a marker of clinical instability or even contribute to the formation of unstable atherosclerotic plaques.

Methods Sphingomyelin content of erythrocyte membranes (SEM) was measured from 327 Chinese subjects undergoing coronary artery angiography in three different groups, including ACS (n=215), stable angina pectoris (SAP, n=57) and control (n=55). Sphingomyelin was measured from RBC membrane lipid extracts and plasma following Mohammad Reza Hojjati's method. Results of SEM are expressed as micrograms of total membrane sphingomyelin per milligram of membrane protein, and sphingomyelin as micrograms of total sphingomyelin per liter plasma.

Results SEM levels in ACS group (121.68, 96.72–171.07 µg/mg) were significantly higher than those with SAP group (96.97, 75.65–106.84 µg/mg, $p<0.001$). As SEM has not been adequately assessed in the general population, we measured SEM levels in a group of individuals with normal coronary arteries for comparison as control group. There was no significant difference between SAP and control groups (90.77, 70.70–109.63 µg/mg). In the present study, plasma sphingomyelin levels were also determined. It was found that plasma sphingomyelin levels in ACS group (14, 9.83–17.54 µg/l) were significantly higher than those with SAP group (8.58, 6.29–13.17 µg/l, $p<0.001$). There was no significant difference between SAP and Control groups (8.17, 5.67–13.58 µg/l). Multivariable logistic regression was used to analysis the results of SEM levels regarding ACS status according to different co-variables (age, sex cardiovascular risk factors, plasma lipids, SEM and plasma sphingomyelin levels). When the above factors were the covariates, SEM levels (OR 8.204; 95% CI 3.521 to 19.114; $p<0.001$) were the positive predictors of ACS. And it also found that increased plasma sphingomyelin levels (OR 5.284; 95% CI 2.237 to 12.481; $p<0.001$) were positive but Apo A-I levels (OR 0.337; 95% CI 0.131 to 0.863; $p=0.023$) were negative predictors for ACS status. The association between SEM levels and plasma lipids was also assessed. Correlation analysis showed no linear association between SEM levels and plasma T-ch ($r=-0.021$; $p=0.729$), TG ($r=0.055$; $p=0.370$), LDL-C ($r=-0.093$; $p=0.127$), HDL-C ($r=-0.106$; $p=0.080$), Apo A-I ($r=0.015$; $p=0.810$), Apo B ($r=0.067$; $p=0.271$), lipoprotein (a) levels ($r=0.105$; $p=0.083$) and plasma SM levels ($r=-0.013$; $p=0.835$).

Conclusion Elevated SEM levels showed a good association with the presence of ACS. These observations suggest SEM could be a contributing factor in the development of ACS, and hence a potential new target for modulating plaque vulnerability of ACS.