Impairment of Endothelial Function—A Possible Mechanism for Atherosclerosis of a High-fat Meal Intake

C K Ng,*, MBBS, MRCP (UK), A P K Chan,** MD, FACC, A Cheng,*** FAMS, MD, FRCP

Abstract

Introduction: Endothelial dysfunction is known to occur in patients with coronary artery disease. Flow-mediated dilation of the brachial artery using Doppler ultrasound is a non-invasive technique for the assessment of endothelial function. The objective of the study was to use the above method to evaluate the pathophysiology of high-fat (HF) intake on endothelial function in a local population. A popular local dish “nasi-lemak”, a source of high saturated fat content from coconut milk, was chosen to represent a local high-fat meal (LHF). In addition, the effects of a Western high-fat (WHF) (“McDonald’s”) meal and a low-fat (LF) meal control on endothelial function were studied. Materials and Methods: The study population consisted of 10 healthy male non-smoker (mean age 22 ± 2 years) with normal body mass index, normal fasting sugar and lipid profiles. Nitric oxide dependent flow-mediated dilation and nitric oxide independent (GTN) dilation was assessed by Doppler flow in the brachial artery before and 4 hours after each meal on separate occasions by 2 experienced sonographers blinded to the type of meals. Results: The baseline brachial artery size, baseline vessel flow and increase in flow after cuff deflation were similar for each of the six arterial studies. In response to reactive hyperaemia after cuff deflation, the endothelium-dependent dilation was significantly different between the meals. There was a marked decrease in endothelium-dependent dilation after the WHF meal compared to the LF meal (8.6 ± 2.2% vs. -0.8 ± 1.1%, P <0.006). There was also a marked decrease in endothelium-dependent dilation after the LHF meal compared to the LF meal (7.7 ± 2.1% vs. -0.8 ± 1.1%, P <0.001). When comparing between the two HF meals, the change in endothelium-dependent dilation was not significant (7.7 vs 8.6%, P = 0.678). GTN-induced dilation was not significantly different before and after the LF, WHF or LHF (0.1 ± 0.5% vs. 0.2 ± 0.9% vs. 1.3 ± 0.5%, P = 0.094). Conclusion: The results suggest that in a local population, impairment of endothelial function is a possible mechanism in the pathophysiology of atherosclerosis from HF intake, beyond just affecting lipid levels. This effect is observed after both a LHF and a WHF meal intake. This technique to study endothelial function may be a useful non-invasive screening tool in the study of other HF diet choices and provides further information for the education of the influence of dietary choices on atherosclerosis.


Key words: Coronary artery disease, Dietary intake, Endothelial dysfunction, Flow-mediated dilation, Risk factor

* Registrar
  Department of Cardiology, Tan Tock Seng Hospital

** Consultant Cardiologist, Private Practice

*** Consultant Cardiologist, Private Practice
  Visiting Consultant, National Neuroscience Institute

Address for Reprints: Dr Ng Chee Keong, Department of Cardiology, Tan Tock Seng Hospital, 11 Jalan Tan Tock Seng, Singapore 308433.
E-mail: ngchk@hotmail.com