Impact of the age of plaque haemorrhage on plaque stress in patients with symptomatic carotid artery disease- a patient specific magnetic resonance imaging-based finite element method simulation study

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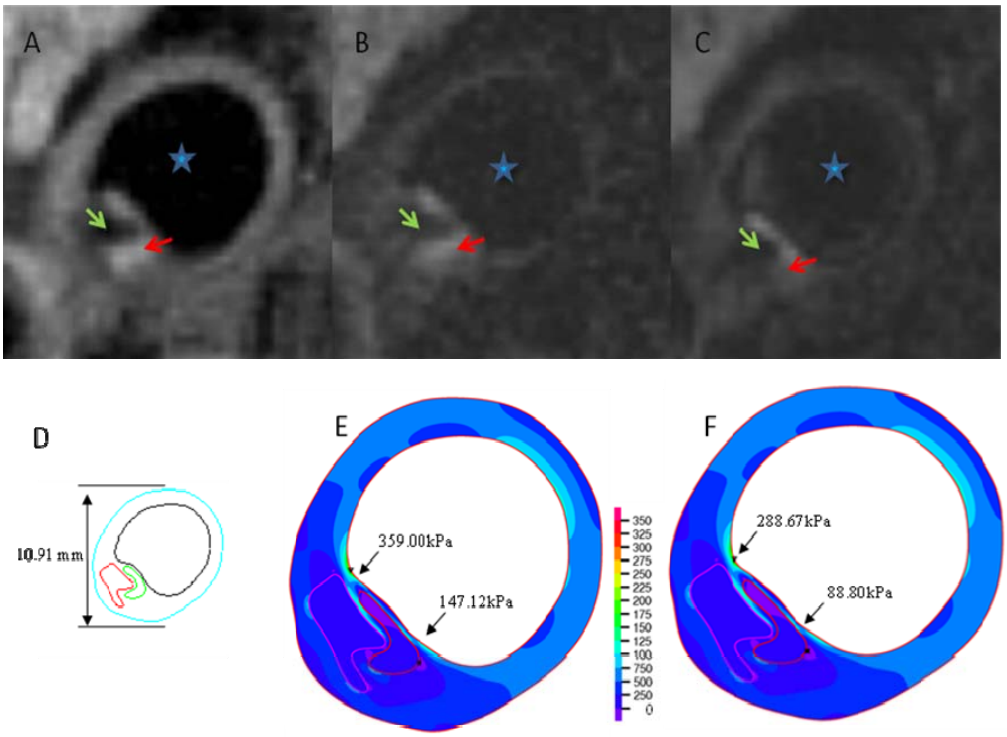
Introduction: Patients suffering from a transient ischemic attack (TIA) are at high risk of a full stroke/recurrent TIAs, particularly within the first 4 weeks (1). The important characteristics of a carotid plaque associated with recurrent TIAs, are: a thin fibrous cap (FC) with or without erosion/disruption and plaque haemorrhage (PH). In biomechanical terms, when the plaque stress exceeds the material strength, FC rupture occurs. The risk of recurrent thromboembolic events gradually decreases afterwards, presumably resulting from the changes in the carotid plaque components: repair of the FC through fibrosis and gradual resorption /organization of the PH. The United Kingdom National Stroke Strategy warrants emergency management of high-risk TIA patients within 24 hours and low-risk patients within 7 days of an acute event (1). High resolution magnetic resonance (MR) imaging can assist us to identify vulnerable plaques and assess their morphological changes. It can also be used to assess the plaque component-dependent biomechanical stresses using computational simulations. This can highlight the extent to which a plaque component can influence the stresses within a plaque, rendering it high-risk and vulnerable.

Aim: To assess the impact of age of plaque haemorrhage on plaque stresses in patients suffering from transient ischemic attacks.

Methods: Ten patients underwent black-blood high resolution MR imaging of their symptomatic carotid artery within 3 days of a TIA. Axial T_1, T_2-weighted, STIR and proton density-weighted images covering the entire carotid plaque were acquired. Manual segmentation of plaque components was done using previously published criteria, to identify FC, lipid pool and age of the PH (2). Patients with MR evidence of fresh haemorrhage were only included for this study. Maximum principle stress was generated using finite element method and solved in ADINA 8.5 (ADINA, Inc.). The plaque components were treated to be hyper elastic materials. The blood pressure of each patient before the MR imaging was used as the loading condition to perform the patient-specific simulation. To simulate the affect of ageing haemorrhage on plaque stress, the material properties for fresh PH were then replaced with that of old PH. The percentage stress difference was determined to quantify the plaque stress difference between the two states of PH: fresh and old.

Results: The maximum principle stress was found to be present at the plaque shoulders, the thinnest FC location and at the site of FC erosion/disruption. The median plaque stress with fresh haemorrhage was 196kPa (95%CI: 151-575) vs. 114kPa (95%CI: 36-316) for simulated old PH (p=0.0001). Plaques had 37% higher stress with fresh haemorrhage compared to when simulation was performed assuming haemorrhage to be chronic.

Conclusions: Age of the plaque haemorrhage significantly affects stresses within atherosclerotic plaques. Patients with fresh haemorrhage, identifiable by MR imaging, have significantly higher plaque stresses compared to plaques with simulated old haemorrhage. This indicates that with time, atherosclerotic plaques tend to stabilize. This highlights the importance of the initial emergency management of patients immediately after a TIA, when plaques are most unstable and extremely high-risk.



Figures Legend: Figure A, B and C showing T_1, T_2 and STIR weighted black-blood MR images- green arrow indicating an ulcer in the atherosclerotic plaque, red arrow indicating PH (fresh) [2]and blue star showing the lumen of the carotid artery. Figure E (fresh PH) and F (old PH simulation) showing the maximum principle stress distribution which is maximal at the plaque shoulder and the thinnest point of FC.

References: 1. http://www.dh.gov.uk/en/Publicationsandstatistics/Publications/PublicationsPolicyandGuidance/DH_081062

2. Chu B et al. Stroke. 2004; 35(5):1079-8