MICRO-BEADS DECREASE THE RUPTURE THRESHOLD ON AN ATHEROMA CAP LABORATORY MODEL

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Introduction

Approximately half of all cardiovascular deaths associated with acute coronary syndrome occur when the atherosclerotic fibrous cap tissue in a coronary vessel breaks under the action of high blood pressure. Recent studies showed that the plaque's mechanical stability is compromised by the presence of micro-calcifications μCalcs inside the fibrous cap, as μCalcs (D = 5-65μm) act as tissue stress concentrators [1-3]. In this context, we designed a silicone-based laboratory model to investigate changes on the rupture threshold caused by μBeads embedded in a hyperelastic material mimicking arterial tissues.

Methods

We created silicone-based (Sylgard 184, PDMS, Dow Corning) dumbbell-shaped laboratory models (ASTM D412 standard geometries) of arterial tissues. Models were scaled down to 20% of original ASTM dimensions to correspond to those of human fibroatheroma caps. Samples were manufactured using 10:1 pre-polymer to cross-linking agent ratio into two groups, with (n=8) and without (n=8) μBeads. For samples containing μBeads, we added a 1% volume of glass beads (18μm diameter, 3M) to the mixture. The PDMS was thoroughly mixed, degassed for about 30 minutes, poured into sample molds cured at 30°C for two days, removed from the molds and then cured again at 100°C for one hour [4]. After curing, we tested the samples using a custom made micro material testing system equipped with real-time control and acquisition software (LabVIEW, v. 2018, National Instruments). Each sample was tested using a ramp waveform under displacement control at constant strain rate (1.5mm/s) up to rupture. Throughout the test, the reaction force was measured and the rupture recorded using a high resolution camera. The force-displacement curves were converted into engineering stress vs stretch curves, and the ultimate stress to rupture was determined. To obtain a constitutive material description of the samples, we carried out a material evaluation analysis in Abaqus/CAE 6.14-3.

Results

Samples with and without μBeads exhibited a distinct hyperelastic behaviour **(Figure1)**. The constitutive model that most closely represented the data was Ogden third order [5], where the strain energy function depends on the hyperelastic constants μ_i, α_i (**Table 1**) that describe shear moduli and power law coefficients. The ultimate stress (stress at rupture) in the PDMS with μBeads group was much lower than the PDMS only group. Comparison of the mean ultimate stress between these two groups was performed using a two tailed T-test, which demonstrated a significant effect (p=2.06E-5) of μBeads on the rupture threshold of this atheroma cap laboratory model **(Figure 1, top left).**

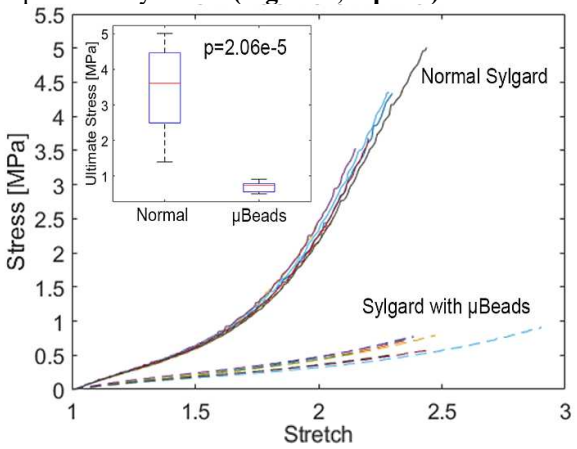

Figure 1: Stress vs stretch ratio for normal samples and samples with μBeads. Top left, Box Plot of the ultimate stresses for the two groups.

Group	μ_1	μ_2	μ_3	α_1	α_2	α_3	D
Norm.	-4.8	4.7	0.7	16.3	16.3	-16.5	0
μBeads	0.2	8.9	-7.4	14.8	14.7	0.9	0

Table 1: Constitutive coefficients values for the two samples groups. μ_i is reported in MPa.

Discussion

Our results clearly capture the influence of μBeads on the hyperelastic behavior and rupture threshold of a vascular tissue mimicking material, as samples with μBeads display significantly lower ultimate stresses. The plausible explanation for the observed change in rupture threshold is the increase in stress concentration around spherical μBeads, which we have previously shown in analytical and numerical studies [1-3] can range from 2 to 5 times the background stress in the material. These experimental observations support our previous studies suggesting that μCalcs located within the fibroatheroma cap may be responsible for significantly increasing the risk of cap rupture that precedes the formation of a thrombus, myocardial infarction and sudden death.

References

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