ADVANCED CMR-BASED STRATEGY FOR FINITE ELEMENT ANALYSIS OF MITRAL VALVE PROLAPSE BIOMECHANICS

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Introduction

Degenerative mitral valve prolapse (MVP) affects up to 3% of the population and carries the risk of serious secondary complications [1]. MVP biomechanics has already been investigated through finite element (FE) models based on cardiovascular magnetic resonance (CMR) imaging [2]. However, a dedicated approach to reconstruct 3D MV geometry is required since CMR data consist in stacks of 2D images. Also, chordae tendineae cannot be clearly identified and uncertainty in their definition in the FE model may heavily impact on the reliability of numerical analysis. Based on cardiovascular magnetic resonance (CMR) imaging, we aimed to provide additional insight into MVP biomechanics by advancing our FE modeling strategy.

Methods

A 45-year-old male subject with healthy MV and a 57-year-old male patient with severe MVP-induced regurgitation underwent CMR exam on a Philips Ingenia 1.5T scanner (Philips Medical System). CMR images were acquired on 18 evenly distributed radial planes [2], and on a stack of short-axis planes encompassing the entire MV. Under the hypothesis of pixel-intensity similarity along their intersections, CMR planes were realigned by optimizing their normalized cross correlation [3]. After manual tracing of MV annulus, leaflets and papillary muscle (PMs) tips at end diastole, the stress-free MV leaflets geometry was reproduced using Fourier and NURBS fitting functions (Fig. 1.a) and discretized (Ω_{ED}^{GRID}). A functionally equivalent model of chordae tendineae was included with insertions uniformly distributed over the leaflet surface (15 insertions/cm^2); in MVP case, chordal density was locally reduced in the prolapse region based on a Gaussian function. To calibrate initial chordal length, Ω_{ED}^{GRID} was morphed onto the mid-systolic leaflet surface reconstructed from CMR data (Ω_{MS}^{CMR}): i) MV annulus and free edge were displacement-controlled to match the corresponding Ω_{MS}^{CMR} profiles, ii) Ω_{ED}^{GRID} was driven to contact Ω_{MS}^{CMR} by a 5 mmHg pressure load, yielding the new position of chordae insertion, iii) simulating the chordae as inextensible, the MV model was pressurized to 120 mmHg, extracting the true force distribution in the chordae to infer their initial length at ED [4]. MV closure was finally simulated from ED to MS in Abaqus/Explicit (Dassault Systèmes) applying a 120 mmHg pressure load; anisotropic and hyperelastic mechanical response of MV tissues [4] as well as annular and PMs motion from CMR data were included.

Results

As tested on both the models, chordal length calibration allowed for consistency between the MV configuration simulated at MS and ground-truth CMR data (Fig. 1.b-c), reporting a mean Euclidean nodal distance from Ω_{MS}^{CMR} equal to 0.96 mm and 1.50 mm in normal and MVP model, respectively. As compared to ED design, in the healthy MV, calibration made 95% of chordae longer by 4.2 mm (+12.3%) and 5% shorter by 0.6 mm (-1.4%), on average. In the MVP model, 19% of chordae were extended by 3.1 mm (+13.4%) while 81% were shortened by 4.0 mm (-12.5%), on average.

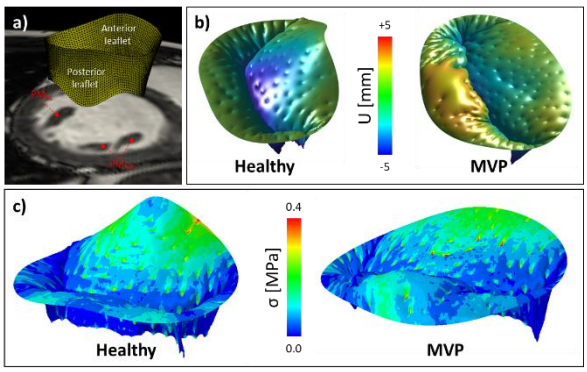

Figure 1: a) MV model at ED, b) relative leaflets displacement above the annulus, c) σ_I on MV leaflets

At MS, maximum principal stress (σ_I) markedly increased on a large portion of the prolapsing posterior MV leaflet if compared to healthy MV model (Fig. 1.c). Force transferred by chordae to PMs reached 20.7 N and 25.4 N in the normal and MVP model, respectively.

Discussion

Advances to our CMR-based MV modeling strategy effectively improved MV model reliability on a patient-specific basis, thus offering a deeper insight into biomechanical derangements associated with degenerative MVP.

References

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