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Mitral valve function following ischemic cardiomyopathy: a biomechanical perspective

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Abstract

Ischemic mitral valve (MV) is a common complication of pathologic remodeling of the left ventricle due to acute and chronic coronary artery diseases. It frequently represents the pathologic consequences of increased tethering forces and reduced coaptation of the MV leaflets. Ischemic MV function has been investigated from a biomechanical perspective using finite element-based computational MV evaluation techniques. A virtual 3D MV model was created utilizing 3D echocardiographic data in a patient with normal MV. Two types of ischemic MVs containing asymmetric medial-dominant or symmetric leaflet tenting were modeled by altering the configuration of the normal papillary muscle (PM) locations. Computational simulations of MV function were performed using dynamic finite element methods, and biomechanical information across the MV apparatus was evaluated. The ischemic MV with medial-dominant leaflet tenting demonstrated distinct large stress distributions in the posteromedial commissural region due to the medial PM displacement toward the apical-medial direction resulting in a lack of leaflet coaptation. In the ischemic MV with balanced leaflet tenting, mitral incompetency with incomplete leaflet coaptation was clearly identified all around the paracommissural regions. This computational MV evaluation strategy has the potential for improving diagnosis of ischemic mitral regurgitation and treatment of ischemic MVs.

Keywords

Mitral valve; ischemic mitral regurgitation; leaflet coaptation; echocardiography; finite element

1. Introduction

Ischemic mitral valve (MV) is a common complication of global or regional pathologic remodeling of the left ventricle due to acute and chronic coronary artery diseases [1]. It frequently represents the pathologic consequences of increased tethering forces and reduced coaptation of the MV leaflets leading to ischemic mitral regurgitation (IMR) [2]. Previous experimental and clinical studies have demonstrated that the primary mechanism of IMR is not simply caused by papillary muscle (PM) dysfunction, but by medial/lateral and apical displacement of the PMs resulting in leaflet tethering [3–5].

The MV apparatus has a complex three-dimensional (3D) anatomical structure consisting of two asymmetric leaflets, a saddle-shaped annulus, chordae tendineae, and PMs. Recent advances in imaging techniques such as 3D transesophageal echocardiography (TEE) provide excellent volumetric information of the MV apparatus along with conventional 2D image and Doppler ultrasound data, allowing us to better understand geometric changes of the MV apparatus [2]. Several studies have identified the mechanisms of abnormal MV leaflet tenting leading to MR in association with infarcted region in ischemic MV [2, 3, 5, 6]. However, there still remains a lack of conclusive investigations to support surgical interventions for IMR treatment. It is important to further investigate the pathophysiology of ischemic MV, develop diagnostic imaging protocols for improved evaluation of ischemic MV, and provide novel therapeutic approaches for IMR treatment [2].

Computational MV evaluation strategy using finite element (FE) analysis has been utilized to assess MV function [7–10]. FE simulations of the MV function can provide valuable additive information to better understand how closely localized mechanical stress concentration and large flexural deformation over the MV apparatus are related to tissue degeneration and failure [11–13].

The purpose of the present study is to utilize our solid computational protocol for evaluation of MV function [8, 9] as a tool to better understand the pathophysiologic mechanism of IMR. Two types of ischemic MV containing medial-dominant or balanced leaflet tenting have been investigated and compared to a normal MV.

2. Materials and Methods

2.1. Virtual MV modeling using patient 3D TEE Data

Geometric information of the MV apparatus including the anterior and posterior leaflets, annulus and PMs in a patient with normal MV was acquired utilizing an iE33 ultrasound unit (Philips Medical Systems, Bothell, WA) with a 3D TEE transducer (frame rate = 25–56 fps). This study was approved by the Committee for the Protection of Human Subjects at The University of Texas Health Science Center at Houston. The mitral annulus and anterior/posterior leaflets at end diastole were segmented and traced in eighteen cut-plane images in the cylindrical coordinate system using a custom-designed image processing algorithm [9, 14]. The 3D geometric data of the MV apparatus was transformed into the Cartesian coordinate system. The 3D MV leaflets and annulus were created using the non-uniform rational B-spline (NURBS) surface modeling technique, meshed, and imported to ABAQUS (SIMULIA, Providence, RI). A total of 24 chordae tendineae were created using discretized line elements connecting the two PM tips and the anterior/posterior leaflet free margin. Dynamic motion of the annulus and PM tips was incorporated using the geometric information of the MV apparatus at peak systole from the patient 3D TEE data [9, 14].

2.2. Modeling of ischemic MVs

Ischemic MVs can be classified by the leaflet tenting pattern caused by ischemic cardiomyopathy [3]. In this study, two types of ischemic MV model were created using the normal patient MV model. Schematics of the cross-sectional view of the normal and ischemic MV models at peak systole are demonstrated in Fig. 1. The medial/lateral and

apical displacement of the PM(s) with inferior myocardial infarction results in asymmetric medial-dominant or symmetric leaflet tenting [3, 4]. Therefore, ischemic MVs were modeled by altering the configuration of the normal PM locations (Fig. 2). In order to mimic the physiologic condition of the ventricular wall deformation due to ischemic cardiomyopathy, the PM tips were simulated shifting toward the medial/lateral and apical directions by 25% of the inter-PM distance and the vertical distance between the annulus and PM location, respectively [15].

2.3. Computational evaluation of MV function with IMR

A hyperelastic material using a Fung-type elastic constitutive model was utilized to model the anisotropic characteristics of the MV leaflets with respect to the circumferential and radial directions [9, 16, 17]. The thicknesses of the anterior and posterior leaflets were set to be 0.69 mm and 0.51 mm, respectively [18]. The chordae tendineae were assumed to be elastic (Young's modulus = 40.7 MPa) with a cross-sectional area of a 0.4 mm² [19]. The density and Poisson's ratio of the whole MV apparatus were set to be 1,100 kg/m³ and 0.48, respectively [10, 20, 21].

Time-varying transvalvular physiologic pressure gradient across the left ventricle and left atrium was applied on the virtual MV leaflets for dynamic finite element simulation. The general contact algorithm with the penalty constraint enforcement method was utilized for leaflet contact modeling to evaluate leaflet coaptation. The friction coefficient was assumed to be 0.05 [22]. Further details of our protocols of MV modeling and dynamic finite element simulation of MV function are described in the previous studies [8, 9].

3. Results

Deformed leaflet morphology of the normal and ischemic MVs at peak systole (i.e., fully closed position) is shown in Fig. 3. The cross-sectional edges of the anterior and posterior leaflets along the A2-P2 plane are displayed in red and blue, respectively. Both leaflets were pulled downward due to the restricted PM displacement, and the location of leaflet coaptation was shifted toward the apical direction in the ischemic MVs. Both ischemic MVs demonstrated seagull shaped deformation of the anterior leaflet affected by the traction of the strut chordae. In the ischemic MV with symmetric leaflet tenting, severely restricted mobility of both leaflets with markedly reduced leaflet coaptation was found.

In the normal MV, relatively large stress values (>1.0 MPa) appeared only around the mitral annulus-aorta junction (Fig. 4). The ischemic MV with medial-dominant leaflet tenting demonstrated excessive stress concentration along the radial direction in the posteromedial commissure where IMR occurred. In the ischemic MV with symmetric leaflet tenting, a similar degree of excessively large stress distribution was found not only in both commissural regions of the anterior leaflet and annulus but also in the wide range of the posterior leaflet and annulus. Both ischemic MVs demonstrated excessive stress concentration particularly in the upper region of the anterior leaflet above where the strut chordae was attached. The maximum stress values were comparable in the ischemic MVs with asymmetric medial-dominant (4.47 MPa) and symmetric (4.42 MPa) leaflet tenting.

The normal MV showed full contact between the leaflets at peak systole (Fig. 5). Incomplete leaflet coaptation was clearly demonstrated in the simulations of MV function in the ischemic MVs due to anomalous displacement of the PMs (Fig. 5). The ischemic MV with medial-dominant leaflet tenting revealed tapered leaflet morphology with a lack of coaptation in the posteromedial commissural region. An excessive stretching of the leaflets appeared in association with increased tethering forces of the chordae tendineae. Leaflet coaptation in the anterolateral commissural region sufficiently prevented mitral incompetency. In the ischemic MV with symmetric leaflet tenting, both leaflets stretched toward the apical direction at peak systole. This resulted in markedly reduced leaflet coaptation leading to a large degree of IMR.

4. Discussion

Although isolated PM dysfunction and local remodeling of the left ventricle with anomalous PM displacement following ischemic cardiomyopathy are attributed to the primary causes of IMR [23–25], the mechanisms of IMR have not been fully understood from a biomechanical perspective. In the present study, the feasibility of computational MV evaluation strategy was demonstrated to identify the mechanisms of MR in different types of ischemic MVs.

One of the most important biomechanical perspectives on MV function following ischemic cardiomyopathy is the degree and extent of leaflet coaptation. There was a clear difference in the contact mechanisms between the ischemic MVs containing medial-dominant and balanced leaflet tenting. The ischemic MV with medial-dominant leaflet tenting demonstrated distinct large stress distributions in the posteromedial commissural region due to the medial PM displacement toward the apical-medial direction resulting in a lack of leaflet coaptation. In the ischemic MV with balanced leaflet tenting, mitral incompetency (i.e., IMR) with incomplete leaflet coaptation was clearly identified all around the paracommissural regions. This indicates that anomalous remodeling of the left ventricular wall following ischemic injury triggers large displacement of the PM locations, elongates the chordae tendineae, induces excessive tethering of the MV leaflets, affects planar tensile forces in the leaflets, and leads to incomplete leaflet closure. Contact force between two surfaces can be determined by the normal and shear forces at a given point. Non-contact is, therefore, attributed to excessively large transfer of the contact forces to the tensile direction and the reduced normal force component.

Although we created the ischemic MV modes using a normal patient MV data, the effect of the PM displacement on the mechanisms of IMR in the ischemic MVs containing asymmetric and symmetric leaflet tenting was successfully evaluated. With further clinical investigation, computational MV simulation techniques combined with patient 3D TEE data can provide a novel evaluation tool to better understand functional and physiological abnormalities in patients with IMR from a biomechanical perspective. This computational MV evaluation strategy has the potential for improving diagnosis of IMR and treatment of ischemic MVs.

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Fig. 1.

Schematics of normal and ischemic MV function. (a) Normal MV, (b) Ischemic MV with asymmetric medial-dominant leaflet tenting, (c) Ischemic MV with symmetric leaflet tenting. Arrows indicate PM displacement.

Fig. 2. Modeling of ischemic MVs by altering the PM locations. (a) Normal MV, (b) Ischemic MV with asymmetric medial-dominant leaflet tenting, (c) Ischemic MV with symmetric leaflet tenting.

Fig. 3.

Deformed leaflet morphology at peak systole with the cross-sectional view along the A2-P2 plane. (a) Normal MV, (b) Ischemic MV with asymmetric medial-dominant leaflet tenting, (c) Ischemic MV with symmetric leaflet tenting.

Fig. 4. Stress distribution across the MV leaflets and annulus at peak systole. (a) Normal MV, (b) Ischemic MV with asymmetric medial-dominant leaflet tenting, (c) Ischemic MV with symmetric leaflet tenting.

Fig. 5. Leaflet contact stress distribution at peak systole. (a) Normal MV, (b) Ischemic MV with asymmetric medial-dominant leaflet tenting, (c) Ischemic MV with symmetric leaflet tenting