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Hubungan Fungsi Apendiks Atrium Kiri dengan Kadar Ekspresi Molekul Adhesi Endotel pada Pasien Stenosis Mitral Rematik = Relationship between Left Atrial Appendage Function and Cellular Adhesion Molecule Levels in Rheumatic Mitral Stenosis Patients

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Abstrak

[Latar belakang. Timbulnya stasis darah dan pembesaran atrium kiri menyebabkan peningkatan angka kejadian trombus di atrium kiri dan apendiks atrium kiri (Left Atrial Appendage-LAA) pada SM. Diameter atrium kiri yang membesar disebutkan sebagai faktor terjadinya pembentukan LASEC dan meningkatkan angka kejadian tromboemboli. Selain itu adanya stasis darah yang dibuktikan dengan penurunan ejeksi fraksi LAA dan kecepatan aliran darah LAA dapat mencetuskan timbulnya trombus. Perubahan moluker adhesi sICAM-1 dan sVCAM-1 berhubungan dengan kejadian tromboemboli. Tetapi apakah terdapat hubungan antara peran hemodinamik, terutama terhadap diameter, fraksi ejeksi dan kecepatan aliran darah pada LAA dengan kadar molekul adhesi seperti sICAM-1 dan sVCAM-1 belum terjawab.

Metode. Pasien SM derajat sedang-berat (tanpa adanya regurgitasi mitral signifikan) yang menjalani pemeriksan ekokardiografi transesofageal diikutsertakan secara konsekutif sejak Januari-April 2014. Penilaian fungsi apendiks atrium kiri dilakukan dengan pemeriksaan transesofageal ekokardiografi. Kadar sICAM-1 dan sVCAM-1 dari vena perifer diukur dengan teknik enzyme-linked immunosorbent assay.

Hasil. Sebanyak 26 subyek penelitian dengan rerata usia 38,92±11,93 tahun, 65,3% berjenis kelamin perempuan, dan 46,1% memiliki irama fibrilasi atrium. Dengan sampel tersebut, didapatkan tidak ada hubungan antara komponen fungsi apendiks atrium kiri, baik diameter, ejeksi fraksi, dan kecepatan aliran darah LAA dengan kadar sICAM-1. Tidak terdapat hubungan antara fungsi LAA pada variabel diameter dengan kadar sVCAM-1. Terdapat hubungan terbalik antara ejeksi fraksi LAA dengan kadar sVCAM-1 (-0,21, p=0.038, 95% KI -0,41- -0,01) dan hubungan terbalik antara kecepatan aliran darah LAA dengan kadar sVCAM-1 (-0,29, p=0,048, 95% KI -0,59- -0,003).

Kesimpulan. Semakin rendah ejeksi fraksi dan kecepatan aliran darah LAA maka semakin tinggi kadar sVCAM-1.

......Background: Blood stasis and left atrial enlargement increase the incidence of thrombus in the left atrium and left atrial appendage (LAA). Enlargement of left atrial diameter is a factor for LASEC formation and increase the incidence of thromboembolism. Blood stasis which evidenced by a decrease in ejection fraction and LAA blood flow velocity can trigger the presence of thrombus. Changes in soluble adhesion molecules sICAM-1 and sVCAM-1 associated with thromboembolic events. But relationship between the role of hemodynamics, especially the ejection fraction and blood flow velocity in the LAA with the levels of adhesion molecules such as sICAM-1 and sVCAM-1 is not well understood.

Methods: Patient with moderate-severe Mitral Stenosis (in the absence of significant mitral regurgitation) underwent transesophageal echocardiography from January to April 2014. Levels of sICAM-1 and sVCAM-1 from peripheral vein were measured by enzyme-linked immunosorbent technique assay.

Results: A total of 26 subjects with a mean age of 38.92 ± 11.93 years, 65.3% female, and 46.1% had atrial fibrillation. We found no association between components of left atrial appendage function: diameter, ejection fraction and blood flow velocity of LAA with sICAM-1 levels. There was no relationship between the LAA function in the variable diameter of LAA with sVCAM-1 levels. There is an inverse relationship between ejection fraction of LAA and levels of sVCAM-1 (-0.21, p = 0.038, 95% KI -0.41- -0.01) and an inverse association between LAA blood flow velocity and levels of sVCAM-1 (-0 , 29, p = 0.048, 95% KI -0.59 - 0.003).

Conclusion: Low ejection fraction of LAA is associated with higher the levels of sVCAM-1.

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