Apakah inflamasi kronik berperan dalam restenosis katup mitral pasca komisurotomi mitral transvena perkutan? = Does chronic inflammation play a role in rheumatic mitral valve restenosis after percutaneous transvenous mitral commissurotomy?

Butarbutar, Maruli Wisnu Wardhana, author

Abstrak

Latar Belakang: Restenosis katup mitral didefinisikan sebagai penurunan mitral valve area (MVA) <1,5 cm2 atau penurunan MVA >50% pasca KMTP. Restenosis katup mitral bersifat time-dependent dan dikaitkan dengan major adverse cardiovascular events (MACE), seperti gagal jantung kongestif, kematian, operasi penggantian katup dan KMTP ulangan. Mekanisme penyebab restenosis katup mitral belum diketahui secara pasti tetapi diduga berkaitan dengan proses inflamasi kronik.
Tujuan: Mengetahui hubungan inflamasi kronik dengan restenosis katup mitral pasca KMTP.
Metode: Total 40 pasien stenosis katup mitral yang telah menjalani tindakan KMTP dikelompokkan menjadi kelompok kasus (n=20) dan kelompok kontrol (n=20) berdasarkan matching. Diambil data sekunder dari rekam medis berupa karakteristik pasien (jenis kelamin, usia dan profilaksis sekunder), data ekokardiografi pre KMTP (Skor Wilkins dan MVA pre KMTP), dan data ekokardiografi post KMTP (MVA pasca KTMP). Dilakukan pemeriksaan ekokardiografi (MVA follow-up) dan pemeriksaan lab (kadar IL-6). Kemudian dilakukan analisis statistik untuk mencari hubungan antara kadar penanda inflamasi kronik serta variabel bebas lainnya dengan restenosis katup mitral.
Hasil: Median konsentrasi IL-6 adalah 2,39 (0,03 - 11,4) pg/mL. Tidak terdapat perbedaan statistik yang bermakna kadar IL-6 pada kedua kelompok (nilai p >0,05). Penurunan MVA adalah 0,13 (0 - 0,62) cm2/tahun dengan laju penurunan MVA 0,155 cm2/tahun merupakan prediktor kejadian restenosis katup mitral (nilai p <0.001, OR = 46,72, 95% CI 6,69 - 326,19).
Simpulan: Inflamasi kronik yang dinilai dengan IL-6 tidak berhubungan dengan restenosis katup mitral.
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Background: Mitral valve restenosis is defined as decreased mitral valve area (MVA) <1.5 cm2 or decreased MVA >50% after PTMC. It is time-dependent and associated with major adverse cardiovascular events (MACE), such as congestive heart failure, cardiac death, mitral valve replacement, and redo PTMC. The mechanism is not yet known; however, chronic inflammation may have a role.
Objective: To know the association between chronic inflammation and mitral valve restenosis after PTMC.
Methods: A total of 40 patients with mitral valve stenosis who underwent successful PTMC were matched and classified into restenosis/case group (n=20) and no restenosis/control group (n=20). Secondary data was taken from electronic medical records such as patient characteristics (gender, age & 2nd prophylaxis), echocardiography data before PTMC (Wilkins' score and MVA before PTMC), and echocardiography data after PTMC (MVA after PTMC). Follow-up echocardiography examination (follow-up MVA) and laboratory assessment of chronic inflammation marker (IL-6) were done on all patients. Statistical analyses were done to look for an association between the level of chronic inflammation marker & other independent variables with mitral valve restenosis.
Results: Median IL-6 concentration was 2.39 (0.03 - 11.4) pg/mL. There was no statistically significant

difference in IL-6 levels between both groups (p-value >0.05). MVA decrement was 0.13 (0 - 0.62) cm2/year with rate of MVA decrement 0.155 cm2/year was predictor of mitral valve restenosis (p-value <0.001, OR = 46.72, 95% CI 6.69 - 326.19).
Conclusion: Chronic inflammation assessed by IL-6 was not associated with mitral valve restenosis