

Hubungan antara variasi sirkadian aritmia ventrikular idiopatik dengan fungsi sistolik intrinsik ventrikel kiri melalui speckle tracking ekokardiografi = Correlation between circadian variation of idiopathic ventricular arrhythmia and left ventricular intrinsic systolic function assessed by speckle tracking echocardiography

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Deskripsi Lengkap: <https://lib.ui.ac.id/detail?id=20517262&lokasi=lokal>

Abstrak

Latar belakang: Aritmia ventrikular idiopatik, baik kompleks ventrikel prematur (KVP) maupun takikardia ventrikel (TV), dapat menyebabkan terjadinya penurunan fungsi sistolik ventrikel kiri (VKi) yang akan menimbulkan kardiomiopati dan meningkatkan mortalitas. Banyak faktor yang berkontribusi menyebabkan terjadinya kardiomiopati akibat KVP (KA-KVP) meskipun mekanisme terjadinya belum sepenuhnya dipahami. Variasi sirkadian KVP dilaporkan berhubungan dengan terjadinya penurunan fraksi ejeksi VKi. Deteksi dini adanya disfungsi sistolik intrinsik VKi dapat dilakukan melalui pemeriksaan speckle tracking ekokardiografi dengan mengukur nilai global longitudinal strain (GLS). Sampai saat ini belum diketahui apakah variasi sirkadian KVP berhubungan dengan penurunan fungsi sistolik intrinsik ventrikel kiri.

Tujuan: Penelitian ini bertujuan untuk mengetahui hubungan antara variasi sirkadian aritmia ventrikular idiopatik dengan fungsi sistolik intrinsik ventrikel kiri melalui speckle tracking ekokardiografi.

Metode: Penelitian ini adalah studi potong lintang dengan total subjek 67 pasien (17 laki-laki [25,4%]; usia rata-rata 46.5 + 9.8 tahun; fraksi ejeksi ventrikel kiri 63,2% + 7,5%) dengan KVP yang berasal dari jalur keluar ventrikel dari pemeriksaan elektrokardiogram 12 sadapan. Semua pasien menjalani pemeriksaan Holter monitoring 24 jam dan speckle tracking ekokardiografi. Dilakukan perhitungan variasi sirkadian beban KVP dan nilai global longitudinal strain (GLS) kemudian dilakukan analisis statistik untuk menilai hubungan kedua variabel tersebut.

Hasil: Sebanyak 31 pasien (46.3%) mengalami gangguan fungsi sistolik VKi (GLS lebih buruk dari -18%). Pasien dengan gangguan fungsi sistolik VKi memiliki GLS yang kurang negatif (-15.1% + 1.8% vs -21.3% + 2.0%; $p < 0,001$), beban KVP yang lebih tinggi (22.2% + 11.1% vs 13.9% + 8.3; $p = 0,001$), variasi sirkadian beban KVP yang rendah (koefisien variasi beban KVP per 6 jam 26.8% + 15.6 vs 52.0 % + 28.2%; $p < 0,001$), dan episode TV non-sustained yang lebih sering (10 pasien [76.9%] vs 3 pasien [23.1%]; $p = 0,019$). Sebanyak 70.6% pasien dengan jenis kelamin laki-laki mengalami gangguan disfungsi sistolik VKi ($p = 0,002$). Pada analisis multivariat didapatkan beberapa prediktor terhadap gangguan fungsi sistolik VKi antara lain variasi sirkadian beban KVP yang rendah dengan [(koefisien variasi beban KVP per 6 jam $< 35\%$), odds ratio (OR)=3.89 interval kepercayaan (IK)95%=1.09-13.80 $p = 0.036$], episode TV non-sustained (OR=14.4, IK 95%=2.36-88.55, $p = 0.008$), beban KVP $> 9\%$ (OR=6.81, IK 95%=1.35-34. Kesimpulan: Variasi sirkadian aritmia ventrikular idiopatik yang rendah berhubungan dengan penurunan fungsi sistolik intrinsik ventrikel kiri melalui speckle tracking ekokardiografi. Variasi sirkadian beban KVP per 6 jam $< 35\%$ memiliki risiko 3.89 kali lebih tinggi untuk terjadinya disfungsi sistolik ventrikel kiri

.....Background: Idiopathic ventricular arrhythmias (AVI) including premature ventricular complex (PVC) or ventricular tachycardia (VT) can cause left ventricular (LV) dysfunction which may lead to cardiomyopathy. The mechanisms of this cardiomyopathy remain elusive, many factors are believed to

contribute. PVC burden is influenced by circadian rhythmicity and lack of PVC circadian variability was proposed as one mechanism of LV dysfunction. Since early detection of LV systolic dysfunction can be done by speckle tracking echocardiography examination, further studies are needed to assess intrinsic left ventricular systolic function and its correlation with PVC circadian variation in patients with idiopathic ventricular arrhythmias.

Objective: This study aimed to investigate the correlation between circadian variation of IVA and left ventricular intrinsic systolic function assessed by speckle tracking echocardiography.

Methods: The subjects of this cross sectional study were 67 consecutive patients (17 men [25.4%]; mean age 46.5 ± 9.8 years; left ventricular ejection fraction $63.2\% \pm 7.5\%$) with PVC originated from ventricular outflow tract based on 12 lead electrocardiogram. All patients underwent 24-hour Holter monitoring and speckle tracking echocardiography examinations. The circadian variation of PVC burden and global longitudinal strain (GLS) were determined and statistical analysis was conducted to evaluate their correlation. **Results:** A total 31 patients (46.3%) had impaired LV systolic function by GLS (worse than -18%). Patients with impaired LV systolic function had a less negative GLS ($-15.1\% \pm 1.8\%$ vs $-21.3\% \pm 2.0\%$; $p < 0.001$), a higher PVC burden ($22.2\% \pm 11.1\%$ vs $13.9\% \pm 8.3$; $p = 0.001$), less variation in circadian PVC distribution (coefficient of variation 6 hourly $26.8\% \pm 15.6$ vs $52.0\% \pm 28.2\%$; $p < 0.001$), and more frequent episode of non-sustained VT (10 patients [76.9%] vs 3 patients [23.1%]; $p = 0.019$). Total 70.6% patient with male gender experienced impaired LV systolic function ($p = 0.002$). Independent predictors for impaired systolic LV function were less variation in circadian PVC distribution [(coefficient of variation $< 35\%$), odds ratio (OR)=3.89, 95% confidence interval (CI)= 1.09-13.80, $p = 0.036$], episode of non-sustained VT (OR=14.4, 95%CI=2.36-88.55, $p = 0.008$), PVC burden $> 9\%$ (OR=6.81, CI 95%=1.35-34.41, $p = 0.020$), and male gender (OR=14.4, CI 95%=2.02-101.1, $p = 0.004$).

Conclusion: Lack of circadian variation of IVA is associated with impaired LV systolic function by GLS. Coefficient of variation PVC burden $< 35\%$ has 3.89 times higher risk for development of left ventricular systolic dysfunction.