

Efek kuersetin terhadap jantung tikus pasca nefrektomi 5/6 tinjauan pada fibrosis dan stres oksidatif = The effect of quercetin on heart tissue in rat with 5/6 nephrectomy overview on fibrosis and oxidative stress / Tri Yuliani

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Abstrak

[ABSTRAK

Latar Belakang: Kardiomiopati uremikum adalah kelainan jantung yang didasari oleh kelainan pada ginjal dan merupakan penyebab kematian tertinggi pada pasien penyakit ginjal kronik (PGK). Overload cairan dan stres oksidatif berperan dalam patogenesis penyakit ini. Kuersetin adalah antioksidan yang bersifat kardioprotektif, namun belum ada data tentang efeknya pada kardiomiopati uremikum. Penelitian ini bertujuan untuk mengetahui efek kuersetin pada kardiomiopati uremikum menggunakan model nefrektomi 5/6 pada tikus.

Metode: Uremia diinduksi pada 3 kelompok tikus jantan Sprague-Dawley dengan nefrektomi 5/6, satu kelompok kontrol tanpa nefrektomi 5/6, masing-masing 6 ekor/kelompok dan diamati selama 8 minggu. Kelompok SNX tidak diberi pengobatan. Kelompok SNX+Q mendapat kuersetin per oral dengan dosis 100 mg/kgBB/hari dan kelompok SNX+Cap mendapat kaptopril 10 mg/kgBB/hari. Hewan uji dikorbankan untuk diukur kadar malondialdehid (MDA) plasma dan jantung, aktivitas glutathion peroksidase (GPX) jantung, NT-proBNP plasma, dan fibrosis jantung. Data dianalisis dengan uji ANOVA.

Hasil: Nefrektomi 5/6 menimbulkan sedikit fibrosis jantung, tidak mempengaruhi NT-proBNP, tidak mempengaruhi MDA jantung dan plasma dan meningkatkan secara bermakna aktivitas GPX jantung ($p < 0.05$) sedangkan pemberian kuersetin dan kaptopril tidak mempengaruhi fibrosis jantung, tidak mempengaruhi NT-proBNP ($p > 0.05$), tidak mempengaruhi MDA jantung dan plasma ($p > 0.05$) dan tidak mempengaruhi aktivitas GPX jantung pada tikus uremia yang diinduksi dengan nefrektomi 5/6 ($p > 0.05$).

Kesimpulan: Kuersetin tidak mempengaruhi fibrosis jantung dan fungsi jantung tikus uremia pasca nefrektomi 5/6. Peningkatan secara bermakna aktivitas GPX jantung pada semua kelompok pasca nefrektomi 5/6 ($p < 0.05$) dibandingkan kelompok kontrol normal menunjukkan bahwa jantung tikus uremia masih berada pada fase kompensasi, yaitu mekanisme adaptasi jantung dan fungsi jantung belum terganggu meskipun terjadi sedikit fibrosis jantung.

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ABSTRACT

Background: Uremic cardiomyopathy is a heart disease because of abnormalities in the kidneys that is the leading cause of death in patients with chronic kidney disease (CKD). Fluid overload and oxidative stress play an important role in its pathogenesis. Quercetin, as an antioxidant, has cardioprotective effect. To the best of our knowledge, its effect on uremic cardiomyopathy has not been investigated yet. This study aims to determine the effect of quercetin on uremic cardiomyopathy using 5/6 nephrectomy model in rats. Methods: Uraemia was induced surgically in male Sprague-Dawley rats via 5/6 nephrectomy (SNX). Quercetin was administered per orally at a dose of 100 mg/kgBW/day for 8 weeks prior to sacrifice. Meanwhile captopril was administered per orally at a dose of 10 mg/kgBW/day. Oxidative stress was assessed using tiobarbituric

acid reactive substances reaction then glutathione peroxidase (GPX) activity was determined to study on antioxidant mechanism. Myocardial fibrosis was analyzed using Massons' Trichrome staining and NTproBNP was measured as a marker of cardiac function. Data was analyzed using ANOVA. Results: Nephrectomy 5/6 had no effects on plasma NT - proBNP, cardiac and plasma MDA, but induced mild myocardial fibrosis and increased cardiac GPX activity significantly ($p<0.05$). However, administration of quercetin and captopril had no effects on plasma NT - proBNP, cardiac and plasma MDA, myocardial fibrosis and GPX activity in uremic rats' heart induced by 5/6 nephrectomy.

Conclusion: Uremic rats' heart induced by 5/6 nephrectomy demonstrated mild myocardial fibrosis but preserved in vivo cardiac function. Increased GPX activity in uremic rats' heart compared to normal control ($p<0.05$) suggests induction of antioxidant defense mechanisms that might not be exhausted yet highlighting a compensatory phase which was unchanged following chronic either quercetin or captopril administration,

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