INTISARI

Korelasi Antara *Endothelial Nitric Oxide Synthase* (eNOS) dengan Cedera Tubulus pada Model Fibrosis Ginjal Mencit *(Mus Musculus)* dengan *Unilateral Ureteral Obstruction*

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Latar belakang: Gagal ginjal kronis merupakan masalah kesehatan yang disebabkan fibrosis ginjal. Fibrosis ginjal ditandai dengan cedera tubulus dan atrofi dengan fibrosis interstisial. Endothelial Nitric Oxide Synthase (eNOS) diketahui sebagai substansi protektif di fibrosis ginjal karena efek vasodilatasinya.
Tujuan: mengetahui ekspresi eNOS setelah terjadinya fibrosis ginjal dan menentukan korelasinya dengan cedera tubulus.
Metode: Dilakukan *Unilateral Ureteral Obstruction* (UUO) pada ureter kiri untuk induksi fibrosis ginjal. 15 mencit jantan *background* swiss diterminasi pada hari 7 dan 14 setelah UUO. Sham digunakan sebagai control. Pada hari 7 dan 14 setelah UUO, mencit diterminasi kemudian ginjal diambil dan disimpan pada formalin untuk pembuatan paraffin. Slide paraffin diwarna dengan Periodic Acid Schiff (PAS) untuk perhitungan skor cedera tubulus. cDNA digunakan untuk perhitungan ekspresi eNOS menggunakan Reverse Transcriptase Polymerase chain Reaction (RTPCR)
Hasil: UUO menginduksi cedera tubulus dengan peningkatan skor cedera tubulus pada hari 7 dan 14 setelah UUO ($p<0.05$). Penurunan ekspresi eNOS didasarkan pada analisis *densitometry* dari eNOS RT PCR di hari 14 ($p<0.05$). Korelasi negatif signifikan antara ekspresi eNOS dan cedera tubulus ($r=-0.874$, $p<0.05$).
Kesimpulan: Penurunan eNOS berperan dalam perburukan cedera tubulus di fibrosis ginjal.
Kata kunci: Unilateral Ureteral Obstruction, Cedera tubulus,fibrosis ginjal, endothelial Nitric Oxide Synthase, Gagal ginjal kronis

ABSTRACT

Correlation Between Endothelial Nitric Oxide Synthase (eNOS) with tubular injury in mice (Mus musculus) kidney fibrosis model with Unilateral Ureteral Obstruction

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Background: Chronic renal failure is a health problem that lead to kidney fibrosis. Kidney fibrosis is characterized by tubular injury and atrophy with interstitial fibrosis. Endhotelial Nitric Oxide Synthase (eNOS) is knwon as a protective substance in kidney fibrosis due to its vasodilatation effects.
Objectives: This study is to examine eNOS expression after kidney fibrosis and elucidate its correlation with tubular injury
Methods: We performed Unilateral Ureteral Obstruction in left ureter to induce kidney fibrosis. We used 15 background swiss male mice that were terminated in day 7 and day 14 after UUO. Sham operated mice were used as control. In day 7 and 14 of UUO, mice were sacrificed, then kidneys were harvested and kept in formalin for paraffin sction. The paraffin slides were stained for PAS to quantify tubular injury score. cDNA were made and used to quantify eNOS expression using Reverse Transcriptase Polymerase chain Reaction (RTPCR).
Results: UUO induced tubular injury with significant increase of tubular injury acore in day 7 and 14 after UUO($p<0.05$).We confirmed reduction of eNOS expression based on densitomerty analysis of eNOS RT PCR in day14 of UUO ($p<0.05$). Significant inverse correlation between tubular injury and eNOS expression ($r-0.874;p<0,05$)
Conclusion: eNOS reduction might play role in worsening tubular injury in kidney fibosis.
Keywords: Unilateral Ureteral Obstruction, Tubular injury, kidney fibrosis, endothelial Nitric Oxide Synthase, Chronic renal failure