ABSTRACT

Background: Kidney fibrosis is the final pathway for chronic kidney disease (CKD). This manifestation usually involved inflammation and also cell senescence with increased level of those respective cell markers. However, the correlation between senescence and pro fibrotic or inflammatory factors have not yet been elucidated.
Objective: The aim of this study to see if there is a correlation between p16, TGF-β and NF-κB mRNA expression and tubular injury in kidney fibrosis model in mice
Methods: This was a quasi-experimental study with posttest only group design. Kidney fibrosis is induced in Swiss background mice (n=24, 2-3 months) using unilateral ureter obstruction process. The mice was then terminated on day 3 (UUO3 group), day 7(UUO7 group), and day 14 (UUO14group). Sham operation was performed for control. Reverse Transcriptase, or PCR (RT-PCR) was performed for assessing mRNA expression of cell senescence, inflammation and fibrosis markers. P16 was used as a senescence marker, TGF-β was for fibrosis markers and NF-κB mRNA was used as inflammatory markers.
Results: UUO groups had higher mRNA expression of p16, NF-κB and TGF-β ($p<0.05$) compared with SO. There was a significant correlation between p16 expression and TGF- β ($p<0.05$). There was also a significant correlation between p16 expression and NF-κB ($p<0.05$). With both correlation showing a moderate positive correlation.
Conclusion: Kidney fibrosis induced mice caused the increase expression of cell fibrosis, senescence and inflammation markers. There was a significant positive correlation between cell fibrosis markers and cell senescence markers, as well as between cell inflammation markers and cell senescence markers.
Keyword: Unilateral Ureter Obstruction, p16, Renal Tubular Injury, Cellular Senescence, Kidney Fibrosis

INTISARI

Latar Belakang: Fibrosis ginjal adalah jalur akhir untuk penyakit ginjal kronis (CKD). Manifestasi ini biasanya terlibat dalam peradangan dan juga penuaan sel dengan peningkatan kadar masing-masing penanda sel. Namun, korelasi antara penuaan dan faktor pro-fibrotik atau inflamasi belum dijelaskan.
Tujuan: Tujuan dari penelitian ini untuk melihat apakah ada korelasi antara ekspresi mRNA p16, TGF-β dan NF-κB dan cedera tubular pada Model fibrosis ginjal pada tikus.
Metode Ini adalah penelitian eksperimental semu dengan desain kelompok post test. Fibrosis ginjal diinduksi pada tikus latar belakang Swiss (n = 24, 2-3 bulan) menggunakan proses obstruksi ureter unilateral. Tikus akan diberhentikan pada hari ke 3 (grup UUO3), hari ke 7 (grup UUO7), dan hari ke 14 (grup UUO14). Operasi palsu akan dilakukan untuk kontrol. *Reverse Transcriptase*, atau PCR (RT-PCR) akan dilakukan untuk menilai ekspresi mRNA sebagai penanda penuaan sel.
Hasil: : Tikus obstruksi ureter unilateral meningkatkan ekspresi p16, NF-κB dan TGF-β ($p < 0,05$) dibandingkan dengan kelompok kontrol. Ada korelasi yang signifikan antara ekspresi p16 dan TGF-β ($p < 0,05$). Ada juga korelasi yang signifikan antara ekspresi p16 dan NF-κB ($p < 0,05$). Dengan kedua korelasi menunjukkan korelasi positif sedang.
Kesimpulan: Tikus yang diinduksi fibrosis ginjal menyebabkan peningkatan ekspresi fibrosis sel, penuaan dan penanda peradangan. Ada korelasi positif yang signifikan antara penanda fibrosis sel dan penanda penuaan sel serta antara penanda inflamasi sel dan juga penanda penuaan sel.
Kata Kunci: *Unilateral Ureter Obstruction, p16*, Cedera tubulus, *Cellular Senescence*, Fibrosis ginjal