INTISARI

Pengaruh Induksi Hiperurisemia Dengan dan Tanpa Pemberian Allopurinol Terhadap Ekspresi Gen Wnt5a-Ror2, Proses Transisi Sel Epitel Menjadi Sel Mesenkim (EMT) Dan Cedera Tubulus Ginjal Pada Mencit

Latar Belakang : Kondisi hiperurisemia berhubungan dengan beberapa penyakit metabolik, penyakit jantung, disfungsi endotel dan cedera ginjal. Cedera tubulus ditandai dengan terjadinya proses transisi sel epitel menjadi sel mesenkim. Pemberian alopurinol dapat menurunkan kadar asam urat dan menurunkan kerusakan ginjal dengan cara menginduksi perubahan E-Cadherin dan α-SMA.
Tujuan : Penelitian ini bertujuan untuk mengetahui pengaruh induksi hiperurisemia dengan dan tanpa pemberian alopurinol pada ginjal mencit.
Metode : Penelitian ini menggunakan rancangan penelitian *post-test only with control group design* dengan subjek penelitian 25 ekor mencit jantan usia 3 bulan yang dibagi menjadi 5 kelompok perlakuan. Setelah dilakukan induksi hiperurisemia dengan dan tanpa pemberian alopurinol selama 7 dan 14 hari, dilakukan pemeriksaan kadar serum asam urat, penghitungan skor cedera tubulus ginjal dan pemeriksaan ekspresi gen Wnt5a, Ror2, E-Cadherin dan vimentin menggunakan RT-PCR.
Hasil Penelitian : Pada kelompok yang diinduksi hiperurisemia tanpa pemberian alopurinol menunjukkan terjadinya cedera tubulus, ekspresi gen E-Cadherin, vimentin, Wnt5a dan Ror2 dengan nilai $p<0,05$. Kelompok yang diinduksi hiperurisemia dan diberi allopurinol menunjukkan terjadinya penurunan cedera tubulus, ekspresi gen E-Cadherin, vimentin dan Wnt5a, namun tidak ditemukan perbedaan bermakna ekspresi gen Ror2 antar kelompok. Ekspresi gen Wnt5a-Ror2 berkorelasi positif terhadap ekspresi gen vimentin dan cedera tubulus ginjal tetapi berkorelasi negatif terhadap ekspresi gen E-Cadherin.
Kesimpulan : Induksi hiperurisemia menimbulkan cedera tubulus, meningkatkan ekspresi gen Wnt5a-Ror2, vimentin dan menurunkan ekspresi gen E-Cadherin, pemberian alopurinol memberikan efek sebaliknya.

Kata Kunci : hiperurisemia, ekspresi gen Wnt5a-Ror2, ekspresi gen E-Cadherin, ekspresi gen vimentin, cedera tubulus.

ABSTRACT

Hyperuricemia With or Without Allopurinol Treatment Induced Wnt5a-Ror2 Gene Expressions, Epithelial To Mesenchymal Transition (EMT) and Kidney Tubular Injury in Mice

Background : Hiperuricemia has been attributed to metabolic diseases, cardiovascular disease, endothelial dysfunction and tubular injury. Tubular injury marked by epithelial to mesenchymal transition process. Allopurinol treatment can ameliorated serum uric acid level and tubular injury by inducing E-Cadherin and α-SMA alterations.
Objective : The aims of this study is to evaluate the influence of hyperuricemia induction with or without allopurinol on mice kidney.
Methods : This was an experimental research with post-test only with control group design. The research subject is 25 male mice (3 month old) divided into 5 groups. After hyperuricemia induction with or without allopurinol treatment for 7 and 14 days, then we conducted serum uric acid level tested, counting kidney tubular injury score and gene expression of Wnt5a, Ror2, E-Cadherin and vimentin was tested by RT-PCR.
Results : Hyperuricemia group without allopurinol showed increasing of tubular injury, Wnt5a, Ror2, E-Cadherin and vimentin gene expression ($p<0,05$). While hyperuricemia with allopurinol group showed decreasing of tubular injury, Wnt5a, E-Cadherin and vimentin gene expression, but the gene expression of Ror2 is not significant. Wnt5a-Ror2 gene expression positively correlate with EMT gene expression and tubular injury, but negatively correlate with E-Cadherin gene expression.
Conclusion : Hyperuricemia treatment induced tubular injury by increasing Wnt5a-Ror2, vimentin gene expression and decreasing E-Cadherin gene expression, but group with hyperuricemia and allopurinol treatment has the opposite effect.

Keywords : hyperuricemia, Wnt5a-Ror2 gene expressions, E-Cadherin gene expression, vimentin gene expression, tubular injury