ABSTRACT

CORRELATION BETWEEN COLLAGEN I EXPRESSION AND FIBROBLAST CELL NUMBER IN HYPERURICEMIA MODEL IN MICE

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BACKGROUND: Chronic Kidney Disease (CKD) has been associated with hyperuricemia by increasing fibrotic response and affected 10% of the population worldwide. It is characterized by glomeruloschlerosis and tubulointerstitial fibrosis. Fibroblast is known as principal effector mediating kidney fibrosis. In recent studies, the resident interstitial fibroblasts proliferate and produce an excessive matrix components such as type I collagen leads to interstitial fibrosis which consider hallmark of CKD.
STUDY OBJECTIVE: To elucidate the correlation between Collagen I expression and Fibroblast cell number in hyperuricemia model in mice.
METHOD: We performed quasi experimental post-test only study in 15 male Swiss Background mice which were divided into 3 groups; Control, D7UA, D14UA. Hyperuricemia induced was perform by injecting uric acid 125 mg/kg/day intraperitoneally. The mice were sacrificed in day 7 (D7UA; n=5) and day 14 (D14UA; n=5). We observed Collagen I expression by using RT-PCR and fibroblast cell number by using immunohistochemical staining with PDGFR-β as primary antibody marker then was quantified with ImageJ software. Result is meaningful if the value was $p<0,05$.
RESULT: The manifestation of CKD, tubulointerstitial fibrosis was proven by significant increased of interstital fibroblast between each group ($p<0,05$). Increasing of Collagen I expression was significant in D7UA to D14UA and Control to D14UA group ($p<0,05$). Increase of Collagen I expression had positive correlation with Fibroblast cell number in hyperuricemia condition ($r=0,688$).
CONCLUSION: Hyperuricemia induced kidney injury represented by interstitial fibrosis, confirmed by increasing of fibroblast cell number and Collagen I expression.
KEYWORDS: collagen I, fibroblast, myofibroblast, hyperuricemia, tubulointerstitial fibrosis, chronic kidney disease

INTISARI

KORELASI ANTARA EKSPRESI COLLAGEN I DENGAN JUMLAH FIBROBLAST PADA MODEL HIPERURICEMIA PADA MENCIT.

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LATAR BELAKANG: Kondisi hiperuricemia berhubungan erat dengan penyakit ginjal kronis dengan peningkatkan respon terhadap fibrosis. Fibrosis ginjal merupakan kondisi akhir dari gagal ginjal dan penyakit ginjal kronis. Karakteristik yang muncul berupa glomeruloschlerosis dan fibrosis tubulointerstitial. Efektor utama yang memediasi fibrosis ini adalah fibroblast. Dari penelitian sebelumnya diketahui bahwa residen fibroblast berproliferasi dan memproduksi matriks ekstraselular. Kelebihan produksi dari matriks tersebut terutama pada komponen Collagen I dapat menyebabkan fibrosis interstitial yang merupakan sebuah tanda dari penyakit ginjal kronis.
STUDY OBJECTIVE: Mengamati ekspresi Collagen I dan jumlah fibroblast serta korelasi antara keduanya pada kondisi hiperuricemia.
METODE: Metode quasi experimental post-test menggunakan 15 mencit yang dibagi menjadi 3 kelompok yaitu Control, D7UA, dan D14UA. Induksi hiperuricemia dilakukan dengan menyuntikkan asam urat rutin secara intraperitoneal selama 7 dan 14 hari. Terminasi dilakukan pada hari ke 7 dan 14 setelah perlakuan. Ekspresi Collagen I diperoleh dengan RT-PCR dan jumlah fibroblast diamati melalui pewarnaan Immunohistochemistry dengan PDGFR-β sebagai marker antibodi primer dilanjutkan quantifikasi menggunakan software ImageJ. Hasil bermakna jika nilai $p<0,05$.
HASIL: Manifestasi CKD yaitu fibrosis tubulointerstitial diperoleh dari peningkatan jumlah fibroblast secara bermakna pada setiap grup ($p<0,05$). Peningkatan ekspresi Collagen I bermakna pada grup D7UA ke D14UA dan Control ke D14UA ($p<0,05$). Peningkatan ekspresi Collagen I berkorelasi dengan jumlah fibroblast ($r=0,688$).
KESIMPULAN: Terdapat korelasi positif antara ekspresi Collagen I dengan jumlah fibroblast pada model hiperuricemia pada mencit.
KATA KUNCI: Collagen I, Fibroblast, Myofibroblast, Hiperuricemia, Fibrosis Tubulointerstitial, Penyakit Ginjal Kronis.