INTISARI

PENGARUH ALLOPURINOL TERHADAP KADAR SGOT, EKSPRESI *HEPATOCYTE GROWTH FACTOR* (HGF), DAN MARKER INFLAMASI CD68 PADA MENCIT YANG DIINDUKSI ASAM URAT

Latar Belakang: Hiperurisemia mengaktivasi jalur *damage associated molecular pattern* (DAMPs) dan menyebabkan inflamasi serta kerusakan organ, termasuk hepar. Inflamasi hepar menginduksi peurunan fungsi hepar, sehingga menurunkan ekspresi HGF sebagai substansi anti fibrosis. Allopurinol hingga saat ini masih digunakan sebagai obat lini pertama untuk mengatasi kondisi hiperurisemia. Namun belum banyak penelitian yang dilakukan untuk mengkaji pengaruh allopurinol terhadap kadar SGOT, gen HGF, dan marker inflamasi CD68.
Tujuan Penelitian: Penelitian ini bertujuan untuk mengkaji pengaruh allopurinol terhadap kadar SGOT, ekspresi mRNA gen *Hepatocyte Growth Factor* (HGF), dan infiltrasi makrofag dengan marker inflamasi CD68 pada mencit yang diinduksi asam urat.
Metode: 25 ekor mencit jantan galur Swiss berumur 3 bulan diinduksi asam urat 125/mg/kgBB dan diberikan allopurinol. Hewan coba dibagi dalam 5 kelompok perlakuan, yaitu kelompok Kontrol (n=5), AU7 (n=5), AU14 (n=5), AU7AL7 (n=5), dan AU14AL7 (n=5). Pada hari yang telah ditentukan diterminasi dan diambil organ hepar. Kadar SGOT diperiksa dari darah vena retroorbital. Pemeriksaan ekspresi mRNA gen HGF menggunakan RT-PCR. Pemeriksaan histopatologi digunakan untuk melihat marker inflamasi CD68.
Hasil Penelitian: Kelompok terinduksi asam urat memperlihatkan kadar SGOT lebih tinggi dan eskpresi mRNA gen HGF lebih rendah dibanding kelompok kontrol. Kadar SGOT ($p=0,000$) yang lebih rendah, ekspresi mRNA gen HGF ($p=0,020$) yang lebih tinggi, serta tren penurunan infiltrasi makrofag ditemukan pada kelompok perlakuan allopurinol dibandingkan dengan kelompok asam urat.
Kadar SGOT ($p<0,05$) dan ekspresi mRNA gen HGF ($p<0,05$), menunjukkan perbedaan bermakna, serta terdapat penurunan trend inflitrasi markrofag pada kelompok perlakuan allopurinol dibandingkan kelompok perlakuan asam urat.
Kesimpulan: Allopurinol berpengaruh dalam memperbaiki kondisi hiperurisemia dilihat dari kadar SGOT, ekspresi mRNA gen HGF, dan infiltrasi makrofag dengan marker inflamasi CD68.

Kata kunci: asam urat, hepar, SGOT, HGF, marker inflamasi CD68

ABSTRACT

THE EFFECT OF ALLOPURINOL TO SGOT LEVEL, *HEPATOCYTE GROWTH FACTOR* (HGF) EXPRESSION, AND INFLAMMATION MARKER CD68 ON URIC ACID INDUCED MICE

Background: Hyperuricemia activate Damage Associated Molecular Pattern (DAMP) pathways and leads to inflammation and organ injury, including liver. Hepatic inflammation induces liver function deterioration, thus reducing HGF expression as anti-fibrotic substance. Allopurinol still uses as the first line drug to treat hyperuricemia. But there are few researches conducted to analyze the effect of allopurinol to SGOT level, HGF gene expression, and inflammation marker CD68.
Objective: This study aimed to analyze the effect of allopurinol to SGOT level, *Hepatocyte Growth Factor* (HGF) mRNA expression, and macrophage infiltration in liver after uric acid and allopurinol treatments.
Method: We treated 25 Swiss male mices age 3 months with uric acid 125/mg/kg Body Weight and given allopurinol 50/mg/kg Body Weight. The mice were divided into 5 groups, there are control group (n=5), AU7 group (n=5), AU14 group (n=5), AU7AL7 group (n=5), and AU14AL7 group (n=5). In last day, mice were sacrificed and the hepar was taken. SGOT level was examined from vena retroorbital. mRNA HGF gene expression examined with RT-PCR. Histopathology examination was done to see inflammation marker CD68.
Result: Uric acid treated groups demonstrated higher SGOT level and lower HGF mRNA expression compared to Control group. SGOT level ($p=0.000$) is significantly lower and mRNA HGF gene expression ($p=0.020$) is significantly higher, also there is lower trend of macrophage infiltration in allopurinol groups than in uric acid groups.
Conclusion: Allopurinol could treat hyperuricemia condition seen through the effect on SGOT level, mRNA HGF gene expression, and macrophage infiltration with inflammation marker CD68.

Keyword: uric acid, hepar, SGOT, HGF, inflammation marker CD68