Penetapan Hewan Model Untuk Penyakit Ginjal Ischaemia-Reperfusion Injury Melalui Parameter Biokimia Ginjal = Animal Model Establishment for Kidney Disease of Ischaemia-Reperfusion Injury Through Renal Biochemistry Parameter

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Abstrak

ABSTRAK
Acute Kidney Injury (AKI) merupakan sindrom penurunan fungsi ginjal dalam mengatur keseimbangan cairan dan elektrolit tubuh, serta eksresi zat sisa metabolisme secara tiba-tiba, yang ditandai dengan penurunan fungsi ginjal dalam beberapa hari. Dalam patofisiologinya,terdapat 3 jenis AKI yaitu AKI pra-renal, intrinsik, dan post-renal. Salah satu penyebab AKI adalah kondisi Ischemia Reperfusion Injury (IRI). IRI merupakan kondisi kerusakan jaringan yang disebabkan aliran darah balik ke jaringan setelah terjadi iskemia (anoksia, hipoksia). Iskemia yang terjadi pada jaringan ginjal menyebabkan berbagai kondisi yang berakibat pada stres oksidatif dan inflamasi. Penelitian ini bertujuan untuk mengetahui profil BUN dan kreatinin pada tikus model Renal Ischemia-Reperfusion Injury. Sebanyak 24 ekor tikus jantan galur Sparague Dawley yang dibagi menjadi 4 kelompok yaitu kelompok normal (sham), iskemia 15 menit, 30, dan 45 menit. Setiap kelompok terdiri atas 6 tikus dengan berat badan antara 150-200gram. Induksi Renal Ischemia-Reperfusion Injury dilakukan dengan metode bilateral renal pedicle clamping. Pengamatan dilakukan sebelum dilaksanakan perlakuan atau jam ke 0 serta di jam ke 24, 48, dan minggu kedua setelah induksi melalui kadar kreatinin serum dan kadar BUN serum. Data diolah secara statistik secara SPSS dengan one way ANOVA method. Induksi Ischemia Reperfusion Injury selama 15 menit menyebabkan peningkatan kadar serum kreatinin dan BUN pada jam ke 24 ($p<0,05$), 48 ($p<0,05$) serta penurunan pada minggu ke 2 ($p>0,05$). Sedangkan pada Induksi Ischemia Reperfusion Injury selama 30 menit, peningkatan kadar serum kreatinin kadar BUN baru terjadi di jam ke 48 ($p<0,05$) serta penurunan di minggu ke dua ($p>0,05$). Berdasarkan hasil tersebut, induksi Ischemia Reperfusion Injury menyebabkan peningkatan kadar kreatinin dan BUN pada 24 jam setelah reperfusi serta penurunan pada 14 hari setelah reperfusi
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<i>ABSTRACT
Acute Kidney Injury (AKI) is a syndrome of decreased kidney function in regulating the body's fluid and electrolyte balance, as well as sudden excretion of metabolic waste, which is characterized by a decrease in kidney function within a few days. In its pathophysiology, there are 3 types of AKI namely pre-renal, intrinsic, and post-renal AKI. One of the causes of AKI is the condition of Ischemia Reperfusion Injury (IRI). IRI is a condition of tissue damage caused by blood flow back to the tissue after ischemia (anoxia, hypoxia). Ischemia that occurs in kidney tissue causes various conditions that result in oxidative stress and inflammation. This study aims to determine the profile of BUN and creatinine level as a biochemical marker for kidney disease in Renal Ischemia-Reperfusion Injury rat model. A total of 24 Sparague Dawley male rats were divided into 4 groups: normal (sham), ischemic 15 minutes, 30, and 45 minutes. Each group consists of 6 rats weighing between 150-200 gram. Induction of Renal Ischemia-Reperfusion Injury is performed using bilateral renal pedicle clamping method. Observations were made before the treatment or the 0th hour and at

24, 48, and the second week after induction through creatinine serum levels and BUN serum levels. The data is processed statistically by SPSS with the one way ANOVA method. Induction of Ischemia Injury Reperfusion for 15 minutes caused an increase in serum creatinine and BUN levels at 24 hours ($p < 0.05$), 48 ($p < 0.05$) and replacement at week 2 ($p > 0.05$). Whereas in the induction of Ischemic Reperfusion Injury for 30 minutes, the increase in serum BUN creatinine levels occurred at 48 hours ($p < 0.05$) and decreased in the second week ($p > 0.05$). Based on these results, the induction of Reperfusion Ischemia Injury caused an increase in creatinine and BUN levels 24 hours after reperfusion and decreased 14 days after reperfusion.<i/>