ONLINE ISSN : 1880-313X
PRINT ISSN : 0388-6107

Biomedical Research

Vol. 30 (2009) , No. 4 August pp.251-258

[PDF (2240K)] [References]

Type 2a sodium-phosphate co-transporter serves as a histological predictor of renal dysfunction and tubular apical damage in the kidneys of septic mice

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(Received June 18, 2009)
(Accepted July 1, 2009)

ABSTRACT

Acute renal failure (ARF) occurs in septic patients and is histologically characterized by tubular apical damages, including brush border breakdown. Nevertheless, little information is available to identify the apical injury at a molecular level. Type 2a Na-phosphate (Pi) co-transporter (NaPiT2a) is constitutively expressed by brush borders of proximal tubules under a healthy condition. Therefore, we investigated if NaPiT2a could be used as a negative marker to predict the renal dysfunction, using an animal model of septic ARF. After the treatment of lipopolysaccharide (LPS), mice manifested the tubular apical injury and renal dysfunction, as evidenced by the increase in blood urea nitrogen (BUN) levels. Immunohistochemical examination revealed that the expression of NaPiT2a by renal proximal tubules became faint, being reciprocal to the development of tubular hypoxia during sepsis. Inversely, the loss in apical NaPiT2a was restored in a regenerating stage, associated with the recovery from renal hypoxia. Overall, there was a negative correlation between the NaPiT2a expression and BUN levels or tubular injury scores in septic mice. Our data indicate that the loss of NaPiT2a is a reliable marker for predicting the progression of septic ARF, while local hypoxia might be involved in the decrease of NaPiT2a expression.

doi:10.2220/biomedres.30.251

JOI JST.JSTAGE/biomedres/30.251