

Ameliorative effects of metformin on renal histologic and biochemical alterations of gentamicin-induced renal toxicity in Wistar rats

Sir,

Recently, we published an article in *J Res Med Sci*, entitled "Ameliorative effects of metformin on renal histologic and biochemical alterations of gentamicin-induced renal toxicity in Wistar rats."^[1] In this article, it is concluded that metformin (MF) may prevent or ameliorate GM-induced acute renal failure, and therefore, it might be beneficial in patients under treatment with this medicine.^[1] In this letter, we would like to point out a few points about the mentioned conclusion. In an agreement with our findings, Taheri *et al.* recently conducted a study on the effects of MF on renal function and structure after unilateral ischemia–reperfusion in rat. They found that MF provided some renal protection against ischemia and reperfusion (I/R) induced injury to the rats kidney. They concluded that MF with activation of adenosine monophosphate-activated protein kinase (AMPK) and endothelial nitric oxide synthase have tissue protective effects.^[2] More recently, Kim *et al.* performed a study using MF (350 mg/kg/day) for spontaneously diabetic Torii (SDT) rats for 17 weeks. They examined blood glucose, glycated hemoglobin and albuminuria, kidney histopathology, renal 8-hydroxydeoxyguanosine levels, and also apoptosis. They found that treatment of SDT rats with MF restored podocyte loss. They suggested that diabetes-induced podocyte loss in diabetic nephropathy could be suppressed by MF, through the repression of oxidative injury.^[3]

Diabetic nephropathy is one of the most important complication of diabetes mellitus^[4,5] and MF has been widely used for treatment of type 2 diabetes.^[3,4] Thus according to our results and those published by Taheri *et al.*, MF protects against tubular injury by restoring the biochemical alterations and modulation of oxidative stress on the tubules.^[1,2] Moreover, according to the study by Kim *et al.*, MF protects podocytes in diabetic nephropathy,^[3] while in diabetic nephropathy, there is also tubular cell injury due to glucosuria.^[3-7] These findings can more potentiate the clinical use of MF in the prevention of diabetic nephropathy. In this regard, to understand the MF-nephroprotective properties better, more experimental rat model or clinical studies are suggested.

Hamid Nasri

Department of Nephrology, Division of Nephropathology,
Isfahan University of Medical Sciences, Isfahan, Iran

Address for correspondence: Prof. Hamid Nasri,
Department of Nephrology, Division of Nephropathology,
Isfahan University of Medical Sciences, Isfahan, Iran.
E-mail: hamidnasri@med.mui.ac.ir

REFERENCES

1. Amini FG, Rafieian-Kopaei M, Nematbakhsh M, Baradaran A, Nasri H. Ameliorative effects of metformin on renal histologic and biochemical alterations of gentamicin-induced renal toxicity in Wistar rats. *J Res Med Sci* 2012;17:621-5.
2. Taheri N, Azarmi Y, Neshat M, Garjani A, Doustar Y. Study the effects of metformin on renal function and structure after unilateral ischemia-reperfusion in rat. *Res Pharm Sci* 2012;7(5).
3. Kim J, Shon E, Kim CS, Kim JS. Renal podocyte injury in a rat model of type 2 diabetes is prevented by metformin. *Exp Diabetes Res* 2012;2012:210821.
4. Baradaran A. Lipoprotein (a), type 2 diabetes and nephropathy; the mystery continues. *J Nephropathology* 2012;1:126-9.
5. Rahimi Z. ACE insertion/deletion (I/D) polymorphism and diabetic nephropathy. *J Nephropathology* 2012;1:143-51.
6. Tolouian R, Hernandez GT. Prediction of diabetic nephropathy: The need for a sweet biomarker. *J Nephropathology*. 2013; 2:4-5.
7. Tavafi M. Complexity of diabetic nephropathy pathogenesis and design of investigations. *J Ren Inj Prev* 2013;2(2):61-5.