Re Metformin revisited: A critical review of the benefit–risk balance in at-risk patients with type 2 diabetes

Reviewed article: "La metformine revisitée : une revue critique de la balance bénéfice/risque chez les patients diabétiques de type 2 dits "à risque""

Keywords: Renoprotection; Metformin; Nephropathy

Mots clés : Metformine ; Néphropathie ; Protection rénale

Dear editor,

We read with great interest the published article by Scheen and Paquot in the esteemed journal of Diabetes & Metabolism entitled ‘Metformin revisited: A critical review of the benefit–risk balance in at-risk patients with type 2 diabetes’ [1]. I would like to raise a few points about metformin beyond its blood regulatory effects in diabetic patients. We conducted a study to find the ameliorative properties of metformin on renal biochemical and histological alterations in gentamicin-induced kidney damage in male Wistar rats [2]. We found that metformin could either prevent or mitigate gentamicin-induced acute kidney injury and, therefore, might be beneficial in patients receiving treatment with this drug [2]. In this letter, we would like to detail more information about this conclusion.

Recently, Taheri et al. [3] conducted a study on the effects of metformin on kidney function and structure after unilateral ischaemia–reperfusion in rats. They observed that metformin provided kidney protection against ischaemia and reperfusion-induced injury in these rats. They concluded that metformin together with activation of adenosine monophosphate-activated protein kinase and endothelial nitric oxide synthase have tissue-protective effects. Also, Kim et al. [4] conducted a study using metformin in spontaneously diabetic rats for 17 weeks and found that treatment of diabetic rats with metformin restored podocyte loss. They suggested that diabetes-induced podocyte loss in diabetic nephropathy could be suppressed by metformin through inhibition of oxidative injury.

Diabetic nephropathy is one of the most important complications of diabetes mellitus [5–11], and metformin has been widely used in the treatment of type 2 diabetes [12]. Thus, according to our results and those published by Taheri et al. [3], metformin can protect against tubular injury by restoring the biochemical alterations and modulation of oxidative stress on tubules. Furthermore, according to the study by Kim et al. [4], metformin protects podocytes in diabetic nephropathy whereas, in diabetic nephropathy, there is also tubular cell injury due to glycosuria [13–18]. Such findings further potentiate the clinical use of metformin for the prevention of diabetic nephropathy. In addition, Morales et al. [19] had previously shown that gentamicin-induced renal tubular damage was also attenuated by metformin. It was also evident that metformin treatment significantly attenuated the increase in malondialdehyde and generation of total reactive oxygen species while restoring the decrease in both enzymatic and non-enzymatic antioxidants [19]. These findings support the use of metformin in diabetes due to its kidney-protective efficacy beyond its blood regulatory effects. In this regard, to better understand metformin’s renoprotective properties, more experimental rat model or clinical studies are called for.

Disclosure of interest

The author declares that he has no conflicts of interest concerning this article.

References


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Received 27 March 2013
Accepted 30 April 2013