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Possible impact of Dapagliflozin on microRNAs regulation and cell apoptosis in Gentamicin induced nephrotoxicity in rats**Doaa Ibrahim¹, Eman Khairy², Sherin Shafik Tawfek¹, Eman K Habib¹ and Abdelhalim Shahat³**¹Ain Shams University, Egypt²Shams University, Egypt³Alazhar University, Egypt

Drug-Induced Kidney Injury (DIKI) is a serious complication associated with higher probabilities of developing progressive chronic kidney disease or end-stage renal diseases. Understanding the molecular disarrangement caused by DIKI would pave the way for a new class of therapeutics to relieve the damage. Yet, another approach to ameliorate DIKI is identifying sensitive and specific biomarkers that outperform the current diagnostic approach like serum creatinine and facilitate early diagnosis. MicroRNAs (miRNAs), a class of non-coding RNAs are increasingly being recognized to have a regulatory role in gene expression and signaling pathways thereby making them novel therapeutic targets. Kidney function, oxidative stress and apoptosis markers, miRNAs expression in serum and renal biopsies were examined in gentamicin induced nephrotoxicity. Dapagliflozin (DAPA) was found to improve kidney function, oxidative stress markers, decrease apoptosis of renal tubular cells and regulate certain miRNAs expression, indicating protective effect against Gentamicin (GNT) induced nephrotoxicity.

Biography

Doaa Ibrahim Mohamed has completed her M.D. degree in Pharmacology and therapeutics, from Ain Shams University, Cairo-Egypt. She is currently working as a Lecturer in Department of Pharmacology-Faculty of Medicine, at Ain Shams University. She has published several international publications.

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