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Keep the corners; adriamycin nephropathy

Mahnaz Momenzadeh^{1,2*}



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¹Department of Clinical Pharmacy and Pharmacy Practice, Isfahan University of Medical Sciences, Isfahan

²Nickan Research Institute, Isfahan, Iran

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Implication for health policy/practice/research/medical education:

Adriamycin is an anticancer agent with broad-spectrum efficacy against tumors. However, chemotherapy-induced renal toxicity constraints clinical efficacy of cancer treatment. The nephropathy of adriamycin is detected by podocyte damage and foot process effacement, which followed by glomerulosclerosis and inflammation in the tubulointerstitial area and fibrosis.

Keywords: Adriamycin, Focal segment glomerulosclerosis, Podocyte, Cardiotoxicity, Adriamycin nephropathy, Cancer

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Adriamycin is an anticancer agent with broad-spectrum efficacy against tumors (1). Cardiotoxicity is a main preventive parameter in the administration of adriamycin; however, the renal toxicity of this drug limits also its chemotherapeutic efficacy (2). However, adriamycin-related nephropathy is still ill-understood. The administration of this chemotherapeutic agent can be associated with a dose-dependent toxicity to the kidneys (1). Previous investigations showed, the nephropathy of adriamycin is detected by podocyte damage and foot process effacement, which followed by glomerulosclerosis and inflammation in the tubulointerstitial area and fibrosis (3). According to the experimental studies, adriamycin-administered mice demonstrated, reduction of glomerular cells followed by mesangial expansion, along with extensive effacements of foot process and thickening of capillary walls. This process will be continued by accumulation of inflammatory cells like macrophages and subsequent disease progression (4, 5). This drug also stimulates nephropathy in rat models and employed as a model for human nephrotic syndrome. Therefore, the nephropathy related to this drug is frequently detected by proteinuria, edema, ascites, hypoproteinemia, dyslipidemia, and hypercoagulability (6,7). Some investigators use the nephropathy of adriamycin as a rodent model of chronic renal failure following the development of chronic proteinuric kidney failure by this agent (3). The toxicity by this drug is conducted by mitochondrial dysfunction, oxidative markers, pro-inflammatory interleukins, and other inflammatory mediators (8). Furthermore, in

the renal toxicity of this drug, a programmed cell death by pyroptosis was recently suggested which leads to renal dysfunction (9). The renal tissue is a high-energy requirement organ, and is abundant of mitochondria and the renal tubular cells are particularly rich of this organelle. Several studies showed that mitochondrial disturbance, redox imbalance and renal damage are interrelated (9). Moreover, other studies detected that inflammatory response and the damage of oxidative stress are behind the toxicity of adriamycin for the kidney tubular damage. *Likewise, reactive oxygen species* controls endoplasmic reticulum stress and apoptosis, which attribute to the renal tubular cell injury and acute renal failure (10). Finally, renal tubular epithelial cell damage have a pivotal role in the development of chronic kidney disease too. Chronic renal failure also found as a worldwide public health issue; hence, agents which subsides the generation of *reactive oxygen species* could play a vital role in reducing adriamycin-induced renal tubular damage (11,12). Thereby further investigations of this aspect of adriamycin-adverse effects suggests.

Authors' contribution

MM is a single author of this manuscript.

Conflicts of interest

The author declares that she has no competing interests.

Ethical issues

Ethical issues (including plagiarism, data fabrication and double publication) have been completely observed by the author.

*Corresponding author: Mahnaz Momenzadeh, Email: Mahnazmomenzadehf@gmail.com, Mahnaz.momenzadeh@pharm.mui.ac.ir

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