A cute renal failure is considered by a sudden diminution in kidney function, fluctuating from a small decrease in glomerular filtration rate to a complete loss of kidney function with may results even to chronic loss of kidney function or end-stage renal disease. Chronic and acute kidney renal failure, regardless to its initiating cause, have inflammation, injury and immune system activation as an ordinary underlying mechanism.

Keywords: Tubular cell, Acute renal failure, End-stage renal disease, Chronic renal failure
jury comprise the release of reactive oxygen species and pro-inflammatory cytokines and chemokines which result to the activation of apoptotic pathways, intracellular Ca\(^{2+}\) accumulation and finally tubular cell damage. Firstly, in ischemic phase renal tubular epithelial and endothelial cells are principal producers of reactive oxygen species and are later accompanied by activated leucocytes, which is, oxidative burst connected to inflammation. These incidents disclose the position of reactive oxygen species in applying harmful consequences on inflammation, linking oxidative stress, cellular structure, and cell death. Reactive oxygen species (ROS) through interfaces with small metabolites such as proteins, lipids, and nucleic acids may permanently destroy or alter the function of these target molecules and related organelles and various cells. Additionally, kidney inflammation happens with the macrophage accumulation and infiltration of inflammatory cells (3-6). The influence of inflammatory mediators on tubular dysfunction and kidney hemodynamic depends on the pathological state, the type inflammatory mediators, and the site of inflammation. Recent studies shown that, mitochondrial ROS generation contributes in to the harmful cascade of events incited by ischemia/reperfusion directing to tubular cell death and acute renal damage (4-6). It should note that, renal structure is vulnerable to ischemia–reperfusion damage during a number of clinically important scenarios as mentioned above. Thus, the modalities should be toward firstly be first on preventive strategies and secondly administration of protective drugs such as antioxidant (5-8).

**Author’s contribution**

HN was the single author of the paper.

**Conflicts of interest**

The author declared no competing interests.

**Ethical considerations**

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**References**