NEPHROPROTECTIVE ACTIVITY OF *MERREMIA EMARGINATA* BURM. AGAINST CISPLATIN-INDUCED NEPHROTOXICITY IN RATS

Sudhavani.V*,Chinnikrishnaiah.V,RaghuMoorthy.V,Raghavendra.H.G,RangaNayakulu.D

Abstract

Cisplatin is an effective anticancer drug, but its clinical use is limited by dose-dependent nephrotoxicity. Oxidative stress, inflammation, mitochondrial dysfunction, and tubular epithelial injury are among the major mechanisms involved in cisplatin-induced renal damage. *Merremia emarginata* Burm. is a medicinal plant traditionally used for various therapeutic purposes and contains bioactive phytochemicals with antioxidant and anti-inflammatory potential. The aim of this study is to evaluate the nephroprotective potential of *M. emarginata* against cisplatin-induced nephrotoxicity in rats. A literature-based experimental framework was considered with emphasis on serum creatinine, blood urea nitrogen, renal antioxidant status, lipid peroxidation, and histopathological changes. The available pharmacological evidence suggests that plant-derived antioxidants may reduce cisplatin-associated renal injury by limiting reactive oxygen species formation and preserving endogenous antioxidant defenses. Further experimental studies are required to establish the specific efficacy, optimal dose, and active constituents of *M. emarginata* in this model.

Keywords: *Merremia emarginata*, cisplatin, nephrotoxicity, nephroprotection, oxidative stress, antioxidants, renal injury.

Introduction

Cisplatin is widely used in the treatment of several solid tumors, including ovarian, testicular, bladder, lung, and head-and-neck cancers. Despite its therapeutic effectiveness, nephrotoxicity remains one of its most important adverse effects.

The kidney is particularly vulnerable because cisplatin accumulates in renal tubular cells, especially in the proximal tubules. This can lead to oxidative stress, mitochondrial dysfunction, DNA damage, inflammation, and apoptosis [1].

The resulting renal injury is commonly associated with increased serum creatinine and blood urea nitrogen, reduced glomerular filtration, electrolyte disturbances, and structural tubular damage.

Oxidative stress plays a central role in this process. Cisplatin increases the formation of reactive oxygen species and can reduce endogenous antioxidants such as glutathione, superoxide dismutase, and catalase [2].

Medicinal plants with antioxidant and anti-inflammatory properties have therefore attracted interest as possible nephroprotective agents.

Merremia emarginata Burm., belonging to the family Convolvulaceae, has been used traditionally in herbal medicine and is reported to contain flavonoids, phenolic compounds, and other secondary metabolites that may contribute to biological activity [3].

The aim of this article is to examine the possible nephroprotective activity of *M. emarginata* against cisplatin-induced renal damage.

Materials and Methods

This article follows a literature-supported experimental design commonly used in nephroprotective studies.

Experimental animals may be divided into normal control, cisplatin-treated toxic control, standard nephroprotective treatment, and one or more groups receiving *M. emarginata* extract.

The plant extract may be prepared from authenticated material using ethanol or another suitable solvent, followed by filtration and concentration.

The principal renal biochemical parameters include serum creatinine, blood urea nitrogen, uric acid, and electrolyte levels.

Oxidative-stress markers may include malondialdehyde, reduced glutathione, superoxide dismutase, catalase, and glutathione peroxidase.

Kidney tissue should also be examined histopathologically to assess tubular degeneration, necrosis, inflammation, and structural preservation.

Results

In cisplatin nephrotoxicity, serum creatinine and blood urea nitrogen typically increase as renal filtration and tubular function decline.

A nephroprotective treatment would therefore be expected to reduce these elevations.

Cisplatin also promotes lipid peroxidation, often reflected by increased malondialdehyde levels.

At the same time, endogenous antioxidant defenses may decrease.

A beneficial plant extract would be expected to reduce lipid peroxidation while preserving or restoring glutathione, superoxide dismutase, and catalase activity.

The phytochemical profile of *M. emarginata* suggests that antioxidant constituents may contribute to such effects.

Flavonoids and phenolic compounds can scavenge free radicals and may also support endogenous antioxidant pathways.

Histological improvement would provide additional evidence of nephroprotection.

Cisplatin commonly causes tubular epithelial degeneration, cellular swelling, necrosis, and inflammatory infiltration.

A protective effect would be reflected by better preservation of renal architecture.

Discussion

The nephrotoxicity of cisplatin is strongly linked to oxidative and inflammatory mechanisms.

Reactive oxygen species damage membrane lipids, proteins, and mitochondrial structures, leading to tubular cell dysfunction.

Antioxidant therapy may therefore reduce some aspects of this injury.

The potential nephroprotective effect of *M. emarginata* is scientifically plausible because medicinal plants rich in flavonoids and phenolic compounds often show free-radical-scavenging and membrane-protective effects.

However, direct experimental evidence specifically involving *M. emarginata* and cisplatin should be evaluated carefully.

A positive effect on serum creatinine alone would not be sufficient.

A strong nephroprotective conclusion should be supported by multiple biochemical and histological parameters.

Another important issue is extract standardization.

The concentration of active compounds may vary according to plant source, extraction solvent, harvesting conditions, and storage.

For reproducibility, phytochemical characterization and marker-compound analysis should be included in future studies.

Conclusion

Merremia emarginata Burm. may possess nephroprotective potential against cisplatin-induced renal toxicity.

Its possible mechanisms include reduction of oxidative stress, preservation of endogenous antioxidant enzymes, inhibition of lipid peroxidation, and protection of renal tubular structure.

However, the current evidence should be regarded as preliminary unless supported by direct controlled studies.

Future research should evaluate dose-response relationships, renal biomarkers, antioxidant parameters, inflammatory markers, and detailed histopathology.

Isolation and characterization of the active constituents are also necessary before the plant can be considered a reliable nephroprotective agent.

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