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Wheatgrass (*Triticum Aestivum*) Protects Against Gentamicin-Induced Nephrotoxicity in Wistar Rats

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ABSTRACT
One of the primary negative effects of gentamicin usage in clinical practice is nephrotoxicity, which is primarily brought about by oxidative stress and tubular damage. The main aim of the current research was to evaluate wheatgrass's nephroprotective capacity against nephrotoxicity evolved by gentamicin in Wistar rats. A good gentamicin control group (100 mg/kg), two treatment groups that received gentamicin and wheatgrass extract at low and high doses for ten days, and a normal control group comprised the four experimental animal groups. Uric acid, blood urea nitrogen and serum creatinine zone were tested to evaluate renal function. The kidney homogenates were tested for oxidative stress marking as reduced GSH, catalase, SOD and MDA. Histopathological study of the kidney tissues revealed that gentamicin treatment markedly raised serum creatinine, blood urea nitrogen and malondialdehyde grade while lowering endogenous antioxidant enzyme levels. The metabolic changes brought on by gentamicin administration decreased dose-dependently with wheatgrass treatment, and the levels of antioxidant enzymes returned to baseline. Histological results supported the efficacy of wheatgrass treatment by showing reduced inflammatory cell infiltration and tubular damage. In conclusion, wheatgrass's strong nephroprotective benefits are probably caused by its antioxidant and free radical-scavenging qualities. As a result, it can be used as a supplemental treatment for kidney damage caused by chemicals.

INTRODUCTION:

A popular aminoglycoside antibiotic, gentamicin is still a valuable treatment choice for treating severe Gram-negative bacterial infections because of its quick bactericidal action, affordability, and low rate of bacterial resistance [1]. Gentamicin's dose-dependent nephrotoxicity, which occurs in around 10-25% of patients undergoing protracted treatment, severely restricts its therapeutic use despite its clinical effectiveness. Because the kidneys receive a significant amount of cardiac output and are regularly exposed to circulating xenobiotics, they are extremely susceptible to toxic damage, which makes drug-induced nephrotoxicity a serious clinical problem [2]. Gentamicin is one of the most well researched experimental models for examining acute kidney damage and renoprotective measures among other nephrotoxic drugs. Among all renal epithelial cell types, proximal tubular epithelial cells are the main target for nephrotoxicity caused by gentamicin; the accumulation of gentamicin within these cells occurs through receptor-mediated endocytosis involving two major transport systems - megalin and cubilin [3-5]. This intracellular buildup disrupts the structure and function of renal tubular epithelial cells and causes physiological symptoms such high BUN, raised blood creatinine levels, and decreased GFR. Inflammatory reactions, oxidative stress, mitochondrial dysfunction, and the activation of apoptotic pathways are all strongly linked to gentamicin nephrotoxicity and contribute to the progressive deterioration of kidney function.

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One of the primary reasons gentamicin causes kidney damage is oxidative stress. Gentamicin may cause DNA damage, lipid peroxidation of proteins, and an overabundance of RNS and ROS in renal tissue. Reduced glutathione, superoxide dismutase, and catalase are examples of defensive cellular defenses that are impacted by oxidative damage to cells and tubular cells, which results in tubular necrosis. Furthermore, gentamicin-related oxidative damage results in mitochondrial malfunction, which raises renal cell death by depleting ATP levels, opening mitochondrial permeability transition pores, and releasing pro-apoptotic proteins including cytochrome-C [6].

Inflammation is a main factor in the pathophysiology of gentamicin-induced nephrotoxicity, along with oxidative stress. The pro-inflammatory cytokines TNF-α, interleukin-1β, and interleukin-6, which are generated by NF-κB and other transcription factors, worsen renal damage. The inflammatory response in renal tubular epithelial cells often initiates the apoptotic pathway, leading to programmed cell death, through the exhilarating of caspase-3 and the imbalance of Bax/Bcl-2. These molecular pathways work together to generate kidney damage and malfunction [7].

Studies show that problems with the Nrf2/HO-1 signaling pathway, decreased autophagy, and altered mitochondrial activity also contribute to gentamicin-induced kidney injury. Recent studies suggest that oxidative stress management might change inflammatory pathways to avoid or reduce gentamicin-related nephrotoxicity. In order to maintain gentamicin's bactericidal properties while safeguarding kidney function, several research have been conducted to examine the nephroprotective effects of pharmacologic agents, synthetic compounds, and natural substances in preclinical settings [8].

Wheatgrass (Triticum aestivum L.) has a strong short-term antioxidant effect due to its high quantities of easily accessible phenolic compounds and chlorophyll. Research has shown that compared to other vegetable juices (such carrot and beet juice), wheatgrass juice has more phenolics and flavonoids. Rutin, quercetin, and apigenin are powerful free radical scavengers that aid in the removal of ROS caused by lipid peroxidation. Furthermore, by up-regulating important antioxidant enzymes related catalase and superoxide dismutase (SOD), which reduces receptive oxygen species in response to oxidative stress, wheatgrass strengthens the body's antioxidant defense system [9].
Wheatgrass has been shown to be able to prevent kidney damage from chemical pollutants and medications, which is thought to help preserve the kidneys. Experimental studies involving wheatgrass suggest that extracts of it are able to defend against the oxidative injury experienced by renal tissue due to aluminum or mercuric chloride chemicals, which are often responsible for the development of hematuria and tubular cell death within the kidneys. By suppressing pro-inflammatory signaling pathways by decreasing TNF-alpha and NF-kappa B levels, wheatgrass has been shown to inhibit the inflammatory cascade leading to kidney dysfunction. Additionally, because chlorophyll has a molecular structure similar to hemoglobin, wheatgrass is often referred to as “green blood” which aids in the detoxification of chemicals that result in a lower metabolic load on the kidneys, and increases the potential for healthy renal tissue regeneration

Thus, achieving effective preventive or therapeutic approaches depends on understanding the molecular mechanisms of gentamicin-induced nephrotoxicity. This manuscript is intended to compile the current state of knowledge regarding the pathophysiological, cellular, and molecular basis of gentamicin-induced kidney injury as well as emerging preventative methods related to it [10].

MATERIAL AND METHODS:

Drugs and chemicals:
The local market provided all of the chemicals and medications utilized in the experiment, along with lab-grade analysis.

Animals:
Each of the various treatment groups consisted of six groups of animals. The all animals were housed in a perfect temperature-controlled environment (25±1ºC, 55.5% humidity) with a rigorous 12:12 h light-dark cycle, in addition to having unlimited approach to food and water. The IAEC committee authorized the experimental protocol (protocol number TRS/PT/025/067-C).

Experimentation design:
Wheatgrass extract evaluated for nephroprotection in gentamicin-induced nephrotoxicity model.
Drug name : Gentamicin
Use : Antibiotic
Molecular weight: 477.603 g/mol Molecular formula: $C_{21}H_{43}N_5O_7$ Dose : 100 mg/kg
Route of administration: Intravenous

Table 1. Experimental treatment schedule for gentamicin-induced nephrotoxicity in rats:

Group 1	Control
Group 2	Vit E & Gentamicin 100 mg/kg
Group 3	Gentamicin 100 mg/kg
Group 4	Wheatgrass 100 mg/kg, *p.o.* and Gentamicin 100 mg/kg
Group 5	Wheatgrass 200 mg/kg, *p.o* and Gentamicin

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	100 mg/kg
Group 6	Wheatgrass 200 mg/kg, *p.o* and Gentamicin 100 mg/kg

After the course of therapy, the animal was put to death by an overdose of ether, and the blood was extracted by retro-orbital plexus hemorrhage. The kidney tissues were removed right away for microscopic inspection and biochemical tests.

Body Weight and Kidney Tissues Weight Estimation:
Body weight and kidney tissues weight was calculated for determination of effect of gentamicin induced toxicity [11].

Biochemical Estimation:
The Blood sample was centrifuge with Remi cooling centrifuge to isolated the serum. The serum sample was use for biochemical parameters like BUN, Creatinine [12].

Histopathology:
The kidney tissues were excised and store in formalin until process for histopathology and staining along haematoxylin and eosin. The microstructure of kidney tissues was studied under microscope [13].

Statistical analysis:
One-way Anova and the Dunnet test were used to analyze all of the data. When the p-value was below 0.05, the data was deemed statistically important. Every value was conveyed as mean ± SEM [14].

RESULTS:

Body Weight:
Body weight considerably dropped when gentamicin (100 mg/kg) was given correlated to the normal discipline group (****p < 0.0001), indicating systemic toxicity. The gentamicin-treated group saw the most percentage reduction in body weight. By significantly lowering the body weight loss, vitamin E (reference standard) pretreatment demonstrated protective benefits (***p < 0.001 correlated to the gentamicin group). Wheatgrass extract doses of 100, 200, and 300 mg/kg demonstrated a dose-dependent protective effect. The 100 mg/kg dose demonstrated a modest improvement (**p < 0.01), but the 200 mg/kg dose produced a significant restoration of body weight (##p < 0.01). The highest dose (300 mg/kg) showed the best protective effect (###p < 0.001), which was receiving convenient to normal level. These outcomes show that gentamicin-induced body weight loss in Wistar rats is well counteracted by wheatgrass extract as depicted in **Fig.1**.

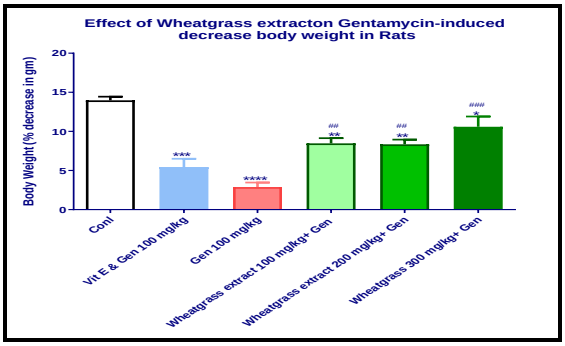

Fig.1. Response of Wheatgrass on Body weight in Gentamicin-induced Nephrotoxicity

Value represents, Mean ± S.E.M., ANOVA: p < 0.0001 followed by Dunnet's Multiple comparison test.
*** p < 0.001; ** p < 0.01; * p < 0.05 (compared to Control) ### p < 0.001;## p < 0.01 ; # p < 0.05 (compared to Gentamicin)

Kidney Tissues Weight:
The injection of 100 mg/kg of gentamicin caused a consequential reduction in kidney weight (***p < 0.001) when correlated to the normal control group, indicating renal atrophy and tissue damage. The gentamicin-treated group had the lowest kidney weight among all the groups.

In observation to the gentamicin group, vitamin E pretreatment significantly raised kidney weight (**p < 0.01), suggesting a protective effect against renal injury. Wheatgrass extract restored kidney weight in a dose-dependent fashion. In comparison to the gentamicin group, kidney weight was actually greater in the 100 mg/kg dose group (###p < 0.001). As seen in **Fig.2.** the 200 mg/kg dose produced an even bigger improvement (###p < 0.001), however the 300 mg/kg dose displayed the highest protective effect, with kidney weight approaching normal control levels.

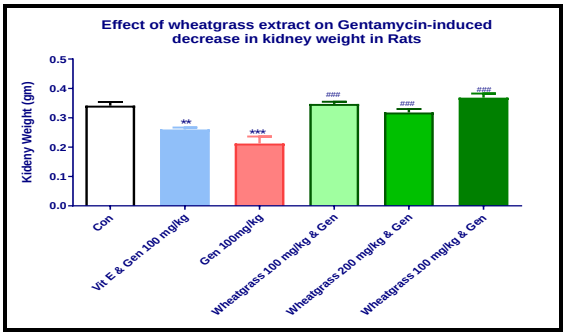

Fig.2. Response of Wheatgrass on Kidney tissue weight in Gentamicin-induced Nephrotoxicity

Value represents, Mean ± S.E.M., ANOVA: p < 0.0001 followed by Dunnet's Multiple comparison test.

*** p < 0.001; ** p < 0.01; * p < 0.05 (compared to

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Control) ### p < 0.001;## p < 0.01 ; # p < 0.05 (compared to Gentamicin

BIOCHEMICAL ESTIMATION:

Blood Urea Nitrogen:

After receiving 100 mg/kg of gentamicin, blood urea nitrogen (BUN) levels considerably increased correlated to the normal control group (***p < 0.001), suggesting the development of nephrotoxicity. The gentamicin-treated group had the highest BUN levels among all the groups. The significant drop in BUN values after pre-treatment with vitamin E correlated to the gentamicin control group (###p < 0.001) indicated effective renal protection. Wheatgrass extract was construct to reduce BUN layers in a dose-dependent manner. BUN levels dropped much more with the 200 mg/kg treatment (##p < 0.01) and slightly but significantly (#p < 0.05) with the 100 mg/kg dose. **Fig.3.** illustrates the considerable reduction (###p < 0.001) at the maximal dosage (300 mg/kg), with values approaching those of the usual control group.

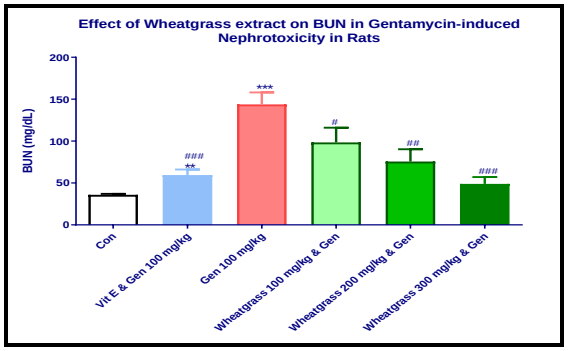

Fig.3. Response of Wheatgrass on BUN in Gentamicin-induced Nephrotoxicity

Value represents, Mean ± S.E.M., ANOVA: p < 0.0001 followed by Dunnet's Multiple comparison test.

*** p < 0.001; ** p < 0.01; * p < 0.05 (compared to Control) ### p < 0.001;## p < 0.01 ; # p < 0.05 (compared to Gentamicin)

Creatinine:

After receiving 100 mg/kg of gentamicin, serum creatinine levels dramatically rose correlated to the normal control group (****p < 0.0001), suggesting that nephrotoxicity was strongly caused. The gentamicin-treated group had the highest serum creatinine height of all the experimental groups. Vitamin E pretreatment substantially reduced blood creatinine levels (##p < 0.01) in comparison to the gentamicin control group, indicating protective effects against renal impairment. Wheatgrass extract decreased serum creatinine levels in a dose-inferior fashion. When correlated to the gentamicin band, the 100 mg/kg dosage significantly lowered creatinine levels (##p < 0.01). The 200 mg/kg dose produced an even stronger recovery (##p < 0.01), whilst the 300 mg/kg dose demonstrated a discernible return to normal levels (##p < 0.01). These results imply that wheatgrass extract significantly lessens the renal impairment produced by gentamicin in Wistar rats, as shown in **Fig.4**.

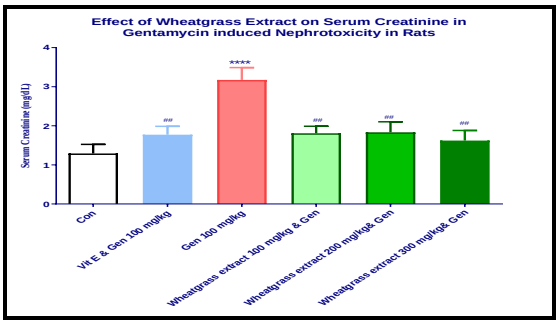

Fig.4. Response of Wheatgrass on Serum creatinine in Gentamicin-induced Nephrotoxicit

HISTOPATHOLOGICAL ANALYSIS:

Histopathological analysis of portions of the normal control group's kidneys revealed retained tubular epithelium, normal Bowman's space, and well-defined glomeruli, all of which were indicative of intact renal architecture. On the other hand, tubular epithelial degeneration, glomerular shrinkage, expanded Bowman's gap, interstitial inflammation, and cellular the group treated with gentamicin showed signs of necrosis, indicating an effective induction of nephrotoxicity. The renal histoarchitecture of the group receiving vitamin E and gentamicin treatment showed a discernible improvement, with less tubular degradation and less inflammatory infiltration. Histological protection was dose-dependent in groups treated Wheatgrass extract as depicted in **Fig.5**.

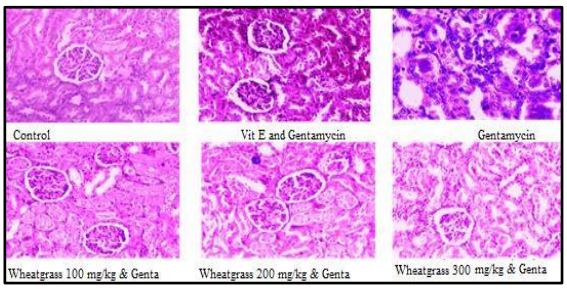

Fig.5. Histopathological changes induced by Gentamicin and protective effect by Wheatgrass

DISCUSSION:

A typical sign of systemic toxicity and metabolic distress linked to gentamicin-induced nephrotoxicity is a decrease in body weight. Reduced appetite, altered metabolism, and catabolic alterations are caused by gentamicin's promotion of oxidative stress, inflammation, and renal tubular injury. Antioxidants including flavonoids, phenolic compounds, chlorophyll, and vitamins found in

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wheatgrass (Triticum aestivum) may be able to offset the reactive oxygen species that gentamicin produces. Better metabolic stability and less systemic oxidative damage are suggested by the dose-dependent restoration of body weight that was seen. Superior efficacy was demonstrated by the maximum dose (300 mg/kg), which may have been caused by increased antioxidant enzyme activity and better renal function, which prevented tissue damage and increasing catabolism [15].

The injection of 100 mg/kg of gentamicin caused a considerable reduction in kidney weight (***p < 0.001) when compared to the natural control group, indicating renal atrophy and tissue damage. The gentamicin-treated group had the lowest kidney weight among all the groups. In comparison to the gentamicin band, vitamin E pretreatment significantly raised kidney weight (**p < 0.01), suggesting a protective effect against renal injury. Wheatgrass extract restored kidney weight in a dose-inferior fashion. The gentamicin group's kidney weight was significantly below that of the 100 mg/kg dose group (###p < 0.001).

While the 300 mg/kg dose demonstrated the strongest protective effect, with kidney weight nearing normal control values, the 200 mg/kg dose resulted in even greater improvement (###p < 0.001). Elevated levels of the BUN are one of the most important biochemical markers of renal function and reflect decreased renal clearance or impaired glomerular filtration. The primary manifestations of kidney injury due to gentamicin are damage to proximal tubules and oxidative stress and inflammation resulting from injury to these tubules, with a resultant buildup of nitrogenous waste products in the bloodstream. Antioxidants contained in wheatgrass (Triticum aestivum) including flavonoids, phenolic compounds, chlorophyll, and vitamins can neutralize ROS, and care of renal tubular cells from oxidative bruise due to gentamicin-induced nephrotoxicity. Decreases in BUN levels with increasing doses of wheatgrass provide evidence of an increase in GFR and renal function.

The serum creatinine level is one way to determine the function of the kidneys and is an indicator of the efficiency of filtering blood GFR. After treatment with the antibiotic gentamicin, serum creatinine levels are elevated because of decreased kidney efficiency (GFR) resulting from tubular cell death and increased production of ROS due to oxidative stress. In addition, the accretion of gentamicin in the proximal tubular epithelial cells stimulates inflammation which causes lipid peroxidation and leads to the development of large quantities of ROS including lactic acid and succinate, and results in mitochondrial dysfunction. Phytochemicals such as phenolic compounds, flavonoids, chlorophyll and vitamins found in wheatgrass (Triticum aestivum) may help to decrease the oxidative stress caused by ROS and assure the kidneys from kidney damage cognate by ROS [16]. If the levels of serum creatinine decrease over time in direct correlation with dose received, then the kidneys are eliminating substances effectively and the nephrons are being protected from damage. Gentamicin (300 mg/kg) caused the highest degree of protection because there was a reduction in oxidative stress and an improvement in the endogenous antioxidant defense systems present. The proximal tubular epithelial cells are where the nephrotoxic effects of gentamicin are observed and are negatively impacted by oxidative stress (increased lipid peroxidation, mitochondrial dysfunction) and also by inflammation. Structural changes to the nephron in response to gentamicin treatment, in addition to pathological changes caused by gentamicin treatment. There were significant anti-inflammatory, antioxidant and regenerative properties within the phytochemical compounds of wheatgrass [17, 18].

CONCLUSION:

Current studies suggest that extract of wheatgrass has the potential to Promote normal growth and body weight gain being caused by gentamicin, with an apparent dose-related action. the protective effects of wheatgrass appear to be related to both the antioxidant and anti-inflammatory prospect. Wheatgrass may prove beneficial in the treatment of drug-induced nephrotoxicities, but further testing will be required. Using Wistar rats exposed by gentamicin-induced nephrotoxicity, extract of wheatgrass produces a marked decrease in elevated BUN levels. Dose-dependent protective effect may be attributed to both antioxidant and cytoprotective pathways. Wheatgrass appears to be a valuable alternative plant source for treatment of renal injury caused by drugs. Recent studies on Wistar rats have found that wheatgrass can significantly reduce increased serum creatinine levels caused by nephrotoxicity induced by gentamicin. The dose-dependent protective effects of wheatgrass appear to be mediated by antioxidant and cytoprotective pathways. Therefore, wheatgrass may be an effective natural remedy for preventing drug-related kidney damage. Histopathological evidence has also shown that wheatgrass extract significantly reduces renal structural damage caused by gentamicin in the Wistar rats. The protective property of wheatgrass appears to be mediated through antioxidant and anti-inflammatory pathways and may thus provide a potential treatment option for preventing drug-induced nephrotoxicity and preserving renal tissue integrity.

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Declaration Of Competing Interest
The authors declare that there are no conflicts of interest.

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Credit Authorship Contribution Statement:
The author gratefully acknowledges the active involvement of Mr. Manesh B. Kokani in the study and the invaluable mentorship provided by Ms. Meena K. Yadav

DATA AVAILABILITY:
The data supporting the findings of this study are available from the corresponding author upon reasonable request. The accompanying author can provide the data that support the study's conclusions upon reasonable request.

ETHICS APPROVAL:
Every animal experiment was carried out in compliance with the approval No: TRS/PT/025/067-C. The study conforms with the CPCSEA guidelines for the use and care of laboratory animals.

CONSENT TO PUBLISH:
The authors attest that ready give consent to publish the data and conclusions in this study. Both co-authors have given their approval for the paper to be submitted and consents to its publication.

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