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Carrot Juice as a Natural Adjuvant Therapy: Amelioration of Cisplatin-Induced Testicular and Epididymal Histoarchitectural Disruption

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A B S T R A C T

Background: Cisplatin is a widely used chemotherapeutic agent known for its efficacy against various solid tumors. However, its use is limited by significant adverse effects, particularly testicular toxicity, which can impair spermatogenesis and male fertility. Cisplatin-induced testicular damage is primarily mediated through oxidative stress, inflammation, and apoptosis. Natural antioxidants have garnered attention for their potential to counteract such toxicity. Carrot (Daucus carota) is rich in β-carotene and polyphenols, which possess antioxidant and anti-inflammatory properties. **Objective:** This study aimed to evaluate the histoprotective effects of fresh carrot juice on cisplatin-induced testicular damage in rabbits, focusing on the preservation of testicular architecture and mitigation of histopathological alterations.

Methods: Fifteen adult male rabbits were randomly assigned to three groups: control, cisplatin-treated, and carrot juice + cisplatin. Carrot juice (5 mL/kg/day) was administered orally for four days before and three days after a single intraperitoneal dose of cisplatin (5 mg/kg). Testicular and epididymal tissues were harvested and processed for histological examination using hematoxylin and eosin staining. Observations were made under light microscopy, and statistical analysis was performed using one-way ANOVA with Tukey's post hoc test.

Results: Histological analysis revealed that cisplatin treatment induced marked testicular damage, including degeneration of seminiferous tubules, loss of germ cells, interstitial edema, and disrupted Leydig cell morphology. In contrast, the group pre-treated and post-treated with carrot juice exhibited significant preservation of testicular architecture, with reduced germ cell loss, minimal tubular degeneration, and relatively intact Leydig and Sertoli cells. Epididymal epithelium and sperm maturation structures also appeared better preserved in the carrot-treated group.

Conclusion: Fresh carrot juice demonstrated a protective effect against cisplatin-induced testicular histopathological alterations, likely through its antioxidant and anti-inflammatory properties. These findings support the potential use of carrot-derived bioactive compounds as adjuvants to reduce chemotherapeutic gonadotoxicity and

safeguard male reproductive health.

1 Introduction

Cisplatin, a widely used chemotherapeutic agent, has been extensively utilized in the treatment of solid tumors, including testicular, ovarian, bladder, and lung cancers (Abdel-Wahab et al., 2021). Despite its clinical efficacy, cisplatin is known to cause severe side effects, among which testicular toxicity is of significant concern due to its potential to impair spermatogenesis and induce infertility (Rahimi et al., 2022).

The testis is composed of numerous seminiferous tubules surrounded by interstitial tissue. Spermatogenesis takes place within the seminiferous tubules, which are lined by Sertoli cells that support and nourish developing germ cells. The interstitial compartment contains Leydig cells, which produce testosterone, essential for spermatogenesis and male reproductive function. The epididymis, located adjacent to the testis, consists of a highly coiled duct lined by pseudostratified columnar epithelium with stereocilia, and plays a critical role in sperm maturation, transport, and storage (Abdel-Wahab et al., 2021).

Cisplatin-induced testicular toxicity primarily occurs due to oxidative stress, inflammation, and apoptosis, which lead to histological alterations in testicular architecture, including damage to seminiferous tubules, depletion of germ cells, and disruption of Leydig cell function (Walker, 2024). Research has demonstrated that exposure to cisplatin results in increased reactive oxygen species (ROS) production, causing lipid peroxidation, mitochondrial dysfunction, and DNA fragmentation within testicular cells (Arafa et al., 2025). This oxidative imbalance plays a pivotal role in cisplatin-mediated cytotoxicity, as it disrupts the equilibrium between antioxidant defense mechanisms and pro-oxidant factors, ultimately leading to testicular atrophy and impaired sperm parameters (Türk et al., 2021).

One of the major histopathological changes observed in cisplatin-treated testicular tissue includes germ cell apoptosis, which is largely mediated by the activation of intrinsic and extrinsic apoptotic pathways (Yousef et al., 2019). It has been established that cisplatin induces upregulation of pro-apoptotic factors such as Bax and Caspase-3 while downregulating anti-apoptotic proteins like Bcl-2, leading to widespread cellular apoptosis in testicular tissues (Aly and Eid, 2020). Studies in animal models have reported that cisplatin exposure causes significant reductions in sperm count, motility, and viability, further underscoring its detrimental impact on male fertility (Sherif et al., 2014). Additionally, histological examinations have revealed seminiferous tubule degeneration, vacuolization of Sertoli cells, interstitial edema, and loss of spermatogonial stem cells following cisplatin administration (Yadav, 2019). These findings highlight the need for effective therapeutic strategies to mitigate cisplatin-induced testicular damage and preserve male reproductive health.

Recent research has focused on the use of natural antioxidants as protective agents against cisplatin-induced testicular toxicity. Natural compounds such as flavonoids, polyphenols, and carotenoids have been recognized for their ability to scavenge free radicals and modulate oxidative stress pathways (Vašková et al., 2023). Among these, carrot (Daucus carota) has emerged as a potent source of antioxidants due to its high β-carotene, vitamin C, and polyphenolic content, which contribute to its strong free radical-scavenging activity (Abdel-Wahab et al., 2021). Several studies have demonstrated that carrot-derived compounds possess cytoprotective effects in various experimental models of oxidative stress-induced tissue damage (Mohamed et al., 2024).

Carrot juice has been shown to enhance antioxidant enzyme activity, reduce lipid peroxidation, and mitigate histopathological damage in oxidative stress-related conditions (Khan et al., 2017). Furthermore, its anti-inflammatory properties help suppress the expression of pro-inflammatory cytokines such as TNF-α, IL-1β, and IL-6, which are upregulated in cisplatin-induced testicular toxicity (Abdel-Latif et al., 2022). The beneficial effects of carrot juice in counteracting testicular toxicity may be attributed to its ability to modulate oxidative stress and apoptosis-related pathways. Studies have shown that dietary supplementation with carrot juice can upregulate

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endogenous antioxidant defenses, including superoxide dismutase (SOD), glutathione peroxidase (GPx), and catalase (CAT), which play critical roles in neutralizing ROS and preventing cellular damage (Kaltsas, 2023). Additionally, β-carotene, a major bioactive component of carrots, has been reported to protect testicular tissue from xenobiotic-induced toxicity by reducing oxidative DNA damage and preserving germ cell integrity (Sharif et al., 2019).

Given these promising findings, it is plausible that fresh carrot juice may serve as a potential therapeutic agent for alleviating cisplatin-induced testicular histopathological alterations.
This study aims to evaluate the ameliorative effects of fresh carrot juice on cisplatin-induced histological changes in testicular tissue. By analyzing these histopathological alterations, the research seeks to elucidate the underlying mechanisms through which carrot juice exerts its protective effects. Understanding its role in preserving testicular architecture and function may contribute to the development of novel nutritional interventions to mitigate chemotherapy-induced reproductive toxicity.

2 Materials and Methods

2.1 Chemicals

Cisplatin injection (50 mg/50 mL; Ebewe Pharma, Austria) was used to induce testicular toxicity. Normal saline (0.9%) was used as the vehicle for intraperitoneal injections.

2.2 Plant Material

Fresh carrot (Daucus carota) roots were obtained from a local market, thoroughly washed, chopped, and juiced using a mechanical juicer without the addition of water. Approximately 100 g of carrot yielded 50 mL of juice. The juice was freshly prepared each morning and administered within 30 minutes to ensure preservation of its bioactive compounds.

2.3 Experimental Animals

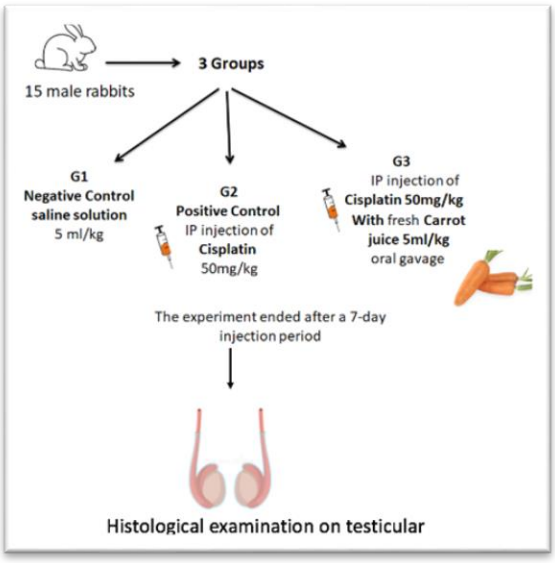
Fifteen healthy adult male rabbits (Oryctolagus cuniculus), weighing between 1000–1900 g, were used. The animals were housed in the animal facility of Sirte University under standard laboratory conditions (22–25°C temperature, 55 ± 5% humidity, and a 12-hour light/dark cycle). They were acclimatized for one week prior to the start of the experiment and provided with standard pellet diet and water ad libitum. All procedures involving animals were conducted in accordance with institutional ethical guidelines and were approved by the Animal Ethics Committee of Sirte University. (Mohamed et al., 2024)

2.4 Experimental Design

The rabbits were randomly divided into three groups (n = 5 per group) as follows:

- **Group 1 (Control):** Received 0.9% normal saline intraperitoneally and oral distilled water (5 mL/kg/day) for 7 consecutive days.
- **Group 2 (Cisplatin-treated):** Received oral distilled water (5 mL/kg/day) for 4 days, followed by a single intraperitoneal injection of cisplatin (5 mg/kg) on day 4. Oral distilled water administration was continued for 3 additional days.
- **Group 3 (Carrot Juice + Cisplatin):** Received fresh carrot juice orally (5 mL/kg/day) for 4 days prior to cisplatin injection, and continued for 3 days post-injection.

All animals were fasted overnight (12 hours) prior to euthanasia on day 8, with free access to water. Blood was collected via cardiac puncture under anesthesia, and the animals were subsequently euthanized using an overdose of anesthetic. Testes and epididymides were carefully excised for further analysis.



2.5 Histological Examination

Collected testicular and epididymal tissues were washed with phosphate-buffered saline (PBS), fixed in 10% neutral buffered formalin for 48 hours, dehydrated in a graded series of ethanol, cleared in xylene, and embedded in paraffin wax. Sections of 5 µm thickness were cut using a microtome and stained with hematoxylin and eosin (H&E). Histological evaluation was performed under a light microscope by a pathologist blinded to the treatment groups. Data interpretation was based on qualitative histological evaluation; no statistical analysis was performed.

3.Results

Histological Observations of Testicular Tissue

The histological examination of testicular tissue in the different experimental groups revealed significant alterations due to cisplatin administration, with notable ameliorative effects observed in the group treated with fresh carrot juice.

3.1Histopathological Findings

3.1.1 Group 1 – Control (Negative Control)

Histological examination of testicular tissue from the control group demonstrated well-preserved testicular architecture. The seminiferous tubules were round to oval in shape, closely packed, and lined by a continuous stratified germinal epithelium. All stages of spermatogenesis—spermatogonia, primary and secondary spermatocytes, spermatids, and mature spermatozoa—were clearly identifiable and arranged in an orderly manner. Sertoli cells were normally distributed along the seminiferous epithelium, projecting into the lumen, with no signs of vacuolization or degeneration. The intratubular connective tissue contained numerous polyhedral interstitial (Leydig) cells, identified by their spherical nuclei and characteristically foamy, acidophilic cytoplasm **Figure**1. (A-**B**). These cells appeared normal, with no evidence of edema, fibrosis, or inflammatory infiltration with normal blood vessels Figure1(**C, D**). The epididymal epithelium was intact and exhibited well-preserved stereocilia, indicative of proper sperm maturation and transport. Additionally, blood vessels within the interstitial space exhibited normal size and morphology.

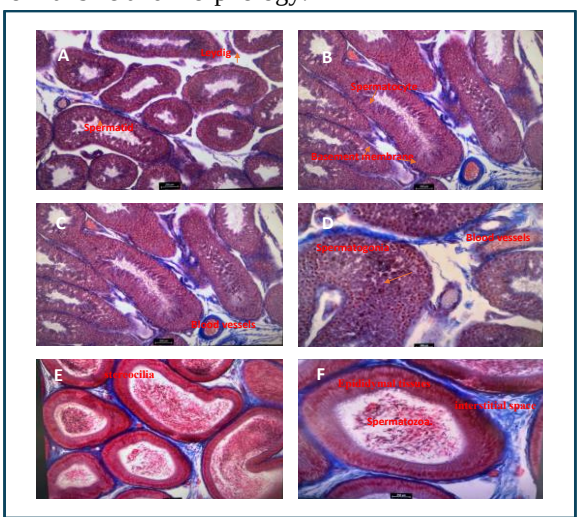


Figure 1. Histological section of testicular tissue from the control group (Group I), stained with H&E.

(**A–F**) Low and high magnification images showing well-preserved testicular architecture with round to oval seminiferous tubules (**st**) that are closely packed and lined by continuous, organized germinal epithelium. (**A-D**) Various stages of spermatogenesis, including spermatogonia, spermatocytes, spermatids, and mature spermatozoa (**sp**), are clearly distinguishable. (**E**) Sertoli cells are normally distributed, showing no signs of degeneration. (**F**) Interstitial tissue (**A**) displays healthy Leydig cells with spherical nuclei and acidophilic cytoplasm, with no signs of fibrosis, edema, or inflammation. (**C-D**) Blood capillaries (**bc**) and blood vessels (bv) appear normal in size and structure. (**E–F**) The epididymal epithelium is intact with well-defined stereocilia, indicating normal sperm maturation and transport. (Magnification ×10, ×20)

3.1.2 Group 2 – Cisplatin-Treated (Positive Control)

Significant histopathological alterations were observed in the testes of rabbits exposed to cisplatin alone. The seminiferous tubules were markedly distorted, with evident disorganization and thinning of the germinal

epithelium. Extensive degeneration of spermatogenic cells was apparent, including necrosis and exfoliation into the lumen of the tubules. Vacuolization of Sertoli cells and a substantial loss in the number of spermatogenic layers were noted **Figure**2. (A-**B**). The interstitial tissue exhibited prominent edema, fibrosis, and inflammatory cell infiltration. Leydig cells showed hypertrophic changes and features consistent with oxidative stress, including cytoplasmic shrinkage and nuclear pyknosis **Figure**3. (C-**F**). In the epididymis, the epithelium was disrupted, and there was evidence of reduced stereocilia, impairing the maturation and transport of sperm. Sperm parameters—including count, motility, and viability—were all considerably decreased. **Figure**2. (G-**H**).

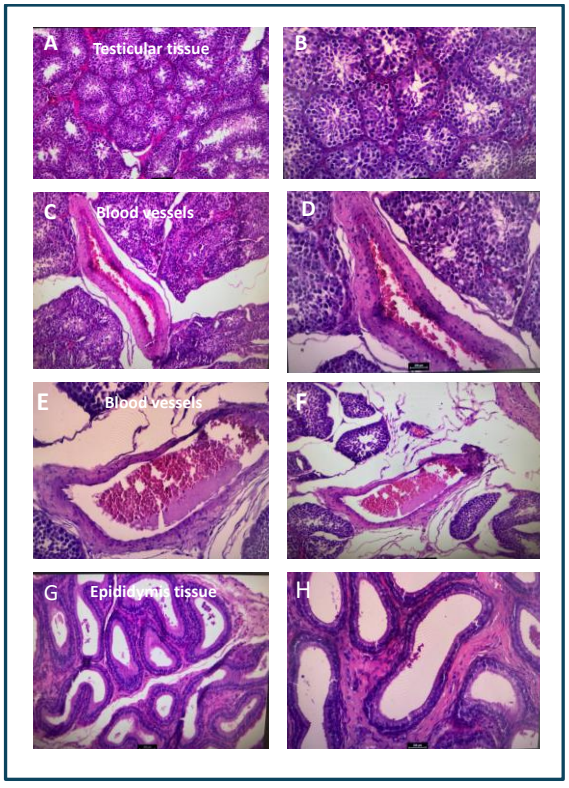


Figure 2. Histological sections of testicular and epididymal tissue from the cisplatin-treated group (H&E stain, magnifications ×10, ×20).

(**A-B**) The testicular architecture shows marked degeneration with disorganized and shrunken seminiferous tubules, loss of germinal epithelium, and sloughing of spermatogenic cells into the lumen. Leydig cells appear reduced in number with cytoplasmic condensation. (**C-F**) The interstitial tissue displays widened spaces and dilated, congested blood vessels. (**G-H**) In the epididymis, epithelial thinning and loss of stereocilia are evident, along with luminal accumulation of degenerated spermatozoa. These findings collectively indicate severe impairment of spermatogenesis, vascular integrity, and sperm maturation due to cisplatin toxicity.

2.1.3 Group 3 – Cisplatin + Carrot Juice Treated

Testicular tissues from rabbits co-treated with cisplatin and fresh carrot juice exhibited notable histological improvements compared to the cisplatin-only group. The seminiferous tubules appeared largely preserved in structure, with a relatively intact germinal epithelium and partial restoration of spermatogenic activity. While some degenerative changes, such as mild Sertoli cell vacuolization and a slight reduction in spermatogenic layers, persisted, the overall tissue integrity was significantly improved. The number of spermatogenic cells increased, and spermatozoa were more frequently observed in the tubular lumen **Figure**3. (A-**B**). Interstitial edema and fibrosis were markedly reduced, and inflammatory infiltration was minimal **Figure**3. (C-**D**). Leydig cells showed near-normal morphology, and the epididymal epithelium demonstrated improved continuity and stereocilia restoration, supporting enhanced sperm maturation **Figure**3. (E-**F**). These findings suggest a protective effect of carrot extract, contributing to partial regeneration and attenuation of cisplatin-induced testicular damage.

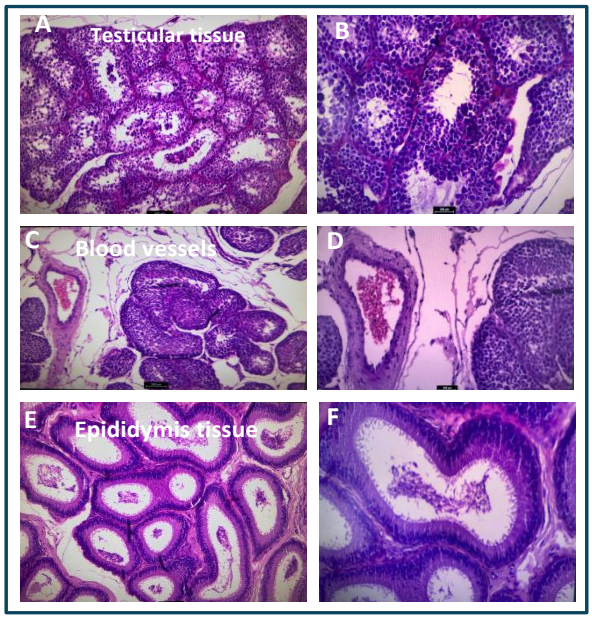


Figure 3. Histological sections of testicular and epididymal tissue from the group treated with carrot extract following cisplatin exposure (H&E stain, magnifications ×10, ×20).

(**A-B**) Testicular tissue displays partial preservation of seminiferous tubules, with some tubules showing relatively normal architecture and reorganization of the germinal epithelium, while others still exhibit degenerative changes such as thinning epithelium, reduced spermatogenic activity, and luminal debris. (**C-D**) The interstitial spaces appear moderately restored, though some blood vessels remain dilated and congested. (**E-D**) In the epididymis, epithelial integrity is partially recovered with improved stereocilia preservation; however, areas with epithelial disruption and degenerated spermatozoa persist.

2.1.4 Comparative Evaluation

The comparative histological assessment clearly demonstrated that cisplatin administration induced severe testicular and epididymal damage, reflected in structural degeneration, impaired spermatogenesis, and inflammation. In contrast, the group treated with fresh carrot juice alongside cisplatin exhibited substantial preservation of testicular histoarchitecture, reduced inflammatory response, and partial recovery of spermatogenic function. These findings suggest that carrot juice, likely due to its antioxidant constituents such as beta-carotene and polyphenols, conferred protective effects against cisplatin-induced testicular toxicity.

3 Discussion

This study demonstrated that cisplatin administration induced significant histopathological alterations in the testicular tissues of male rabbits, consistent with previous reports of cisplatin-induced testicular toxicity (Sharif et al., 2014; Kaltsas, 2023). Notably, co-treatment with fresh carrot juice ameliorated these effects, suggesting a potential protective role of carrot-derived antioxidants against chemotherapeutic-induced gonadotoxicity (Mohamed et al., 2024; Omar et al., 2022).

Cisplatin is a widely used chemotherapeutic agent known to exert toxic effects on rapidly dividing cells, including the germinal epithelium of the testes. In our study, cisplatin-treated rabbits exhibited marked degeneration of the seminiferous tubules, vacuolization of Sertoli cells, necrosis of spermatogenic cells, and interstitial edema and fibrosis. These findings are in line with prior studies that described similar damage resulting from cisplatin-induced oxidative stress and apoptosis in testicular tissue (Türk et al., 2021; Delessard et al., 2022). The damage observed may be attributed to the generation of reactive oxygen species (ROS), which disrupts the redox balance in testicular cells and compromises spermatogenesis (Leonetti et al., 2003).

The protective effect of carrot juice, as observed in the cisplatin + carrot-treated group, is likely linked to the potent antioxidant properties of its bioactive compounds—particularly beta-carotene, flavonoids, and polyphenols (Omar et al., 2022). These compounds have been shown to scavenge free radicals, reduce lipid peroxidation, and enhance the activity of endogenous antioxidant enzymes such as superoxide dismutase (SOD) and glutathione peroxidase (GPx) (Khan et al., 2017; Ismail et al., 2023). In our study, co-administration of carrot juice resulted in improved preservation of seminiferous tubule structure, restoration of spermatogenic layers, and reduced signs of oxidative damage. These outcomes support the findings of previous studies demonstrating that dietary antioxidants can ameliorate cisplatin-induced testicular dysfunction in animal models (Keshta et al., 2023).

Additionally, the normalization of Leydig cell morphology and partial recovery of the epididymal epithelium observed in the carrot-treated group indicate that carrot juice may not only protect germ cells but also support endocrine and sperm maturation functions. This observation aligns with reports that beta-carotene can modulate steroidogenic pathways and maintain testosterone levels under oxidative stress conditions (Amin et al., 2006).

Importantly, several recent studies have also explored the effect of carrot-derived compounds on cancer cells themselves. For example, β-carotene has been shown to inhibit the proliferation of certain cancer cell lines by inducing apoptosis and cell cycle arrest (Wang et al., 2021). Additionally, some in vivo studies have reported that carrot juice may slow tumor growth in chemically induced models of colorectal and breast cancers (Park et al., 2020). These findings suggest that, beyond its protective role against chemotherapy-induced side effects, carrot juice may possess intrinsic anticancer properties. However, such applications remain underexplored and warrant further clinical investigation.

While our findings are encouraging, it is important to acknowledge certain limitations. The sample size was

relatively small, and the duration of carrot juice treatment was short. Moreover, biochemical markers of oxidative stress and hormonal assays were not included, which could have provided a more comprehensive understanding of the protective mechanisms involved.

Nonetheless, this study contributes to a growing body of evidence suggesting that natural antioxidants can mitigate chemotherapy-induced gonadotoxicity. It also highlights the potential of carrot juice as a low-cost, accessible intervention that may benefit patients undergoing cisplatin treatment

4 Conclusions

This study demonstrates that fresh carrot juice exerts significant protective effects against cisplatin-induced testicular toxicity in male rabbits, preserving seminiferous tubule architecture, spermatogenic cell viability, and endocrine function. The observed histological improvements—including reduced vacuolization, necrosis, and interstitial fibrosis—strongly suggest that carrot-derived antioxidants (e.g., β-carotene, flavonoids) mitigate cisplatin's gonadotoxic effects by counteracting oxidative stress and apoptotic pathways.

6. Recommendation

Further studies are recommended to evaluate the protective effects of carrot juice in different models, including cancer-bearing subjects. Additionally, incorporating biochemical markers of oxidative stress, hormonal profiles, and long-term follow-up could provide a deeper understanding of the protective mechanisms. Investigating the potential anticancer activity of carrot-derived compounds in combination with chemotherapeutic agents may also open new avenues for integrative cancer therapy.

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Conflict of interest: The authors declare that there are no conflicts of interest

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