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Alpha Pinene Mitigates Cisplatin Induced Acute Kidney Failure

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Abstract

Background: Cisplatin is a very effective chemotherapy drug that is used to treat a wide range of malignancies. Its therapeutic efficacy is limited by tumor resistance and significant adverse effects that are connected to the dose, including hearing and renal impairment, as well as decreased bone marrow function.

Aim of Study: Alpha-pinene's nephroprotective efficacy against cisplatin-induced nephrotoxicity rats was the focus of this investigation.

Material and Methods: 24 Sprague-Dawley male rats were separated into three groups (n=8), (1) Normal group, (2) Model group (cisplatin 7.5mg/kg, single dose, intraperitoneal), (3) α-Pinene group (50mg/kg, p.o. for 18 days). Cisplatin was administrated to all groups on day 15, excluding the normal group. Kidney function markers, antioxidant status, and inflammatory marker were analyzed.

Results: Cisplatin elevated kidney function markers and inflammatory markers. Further, the antioxidant level was significantly diminished in model group.The results showed that alpha-pinene pretreatment boosted antioxidant status while dramatically lowering renal function markers and inflammatory markers.

Conclusion: Alpha-pinene has strong antioxidant and anti-inflammatory features, which protect against CP-induced nephrotoxicity.

Key Words: *Cisplatin – Acute Kidney Injury – α-Pinene.*

Introduction

NEPHROTOXICITY refers to kidney damage caused by various substances, including drugs and chemicals **[1]**. Common nephrotoxic agents include antibiotics, NSAIDs, anticancer drugs, and antifungals **[2]**. Risk factors for drug-induced nephrotoxicity include patient age, gender, pre-existing renal insufficiency, and drug dosage **[3]**.

Cisplatin, a widely used chemotherapeutic agent, is associated with significant nephrotoxicity, limiting its clinical efficacy **[4]**. The drug accumulates in renal tubular cells through transport-mediated processes, causing damage to various cellular structures **[5]**.

While initially thought to be related to its platinum moiety, cisplatin nephrotoxicity is now understood to involve complex mechanisms, including the formation of toxic metabolites **[6]**. The drug's nephrotoxic effects are mediated through multiple pathways, such as oxidative stress, inflammation, and apoptosis induction **[7]**. Understanding these mechanisms may lead to novel therapeutic interventions tolimit nephrotoxicity while maintaining cisplatin antineoplastic efficacy.

A direct correlation between antioxidant effects and nephroprotection indicates that oxidative stress is a key factor in cisplatin-induced kidney injury **[8]**. Reactive oxygen species (ROS) generation, mitochondrial malfunction, and lysosomal membrane leakiness are also the mechanisms that cause inflammation and apoptosis during cisplatin nephrotoxicity **[9]**.

Recent studies have demonstrated the critical role of inflammation in kidney damage brought on by cisplatin. By activating pro-inflammatory signaling pathways, such as NF-κB, cisplatin increases the production of inflammatory mediators and the influx of neutrophils **[4]**.

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Abbreviations:

AKI : Acute kidney injury.
IL-6 : Interleukin 6.
TNF-α : TNF-α: Tumor necrosis factor alpha.
NFκB : Nuclear factor kappa B.

Nevertheless, these preventative measures frequently only work in part, which emphasizes the necessity of combinatorial approaches that maintain cisplatin's anticancer effectiveness [10].

Alpha pinene, a monoterpene found in many plants, has diverse biological activities. It demonstrates anti-inflammatory effects by suppressing MAPKs and NF-κB pathways, reducing the production of inflammatory mediators like IL-6, TNF-α, and NO in mouse macrophages [11]. It exhibits antibacterial and insecticidal properties and is used in the fragrance and flavor industries [12]. It prevents oxidative stress, DNA damage, and apoptosis caused by UVA by regulating DNA repair proteins and inflammatory mediators [13].

Consequently, the previously discussed endeavors establish a basis for the exploration of the prospective nephroprotective properties of alpha pinene in cisplatin induced AKI.

Material and Methods

The research was carried out in the Animal House, Faculty of Medicine, Alexandria University, Academic Building, Mouwasat Hospital, May 2021.

Drugs and chemicals:

Cisplatin (cisplatin 10mg/10ml vial) was obtained from Mylan S.A.S., Saint-Priest, France. Alpha-pinene was procured from Sigma Aldrich, St. Louis, Missouri, USA. Carboxy methyl cellulose (CMC) was acquired from the El-Gomhouria Company for Trading Chemicals and Medical Appliances, Alexandria, Egypt.

Animals:

24 male Sprague-Dawley rats (180-230g) were acquired from the animal house of the Medical Research Institute (Alexandria, Egypt). Rats were housed in cages (3 rats/cage) in well-ventilated and pathogen-free rooms under standard circumstances (12-hr light/dark cycle, temperature range of 25°C ± 2°C, and relative humidity of 55%±5%, with water and food available ad libitum). Acclimatization was allowed for one week before starting the experiment. All animal experiments followed the ARRIVE guidelines and adhered to the U.K. Animals (Scientific Procedures) Act, 1986, and the National Research Council's Guide for the Care and Use of Laboratory Animals, which has been approved by the Research Ethics Committee at Damanhour University (Ref. No. 521PO25).

Experimental design:

After adaptation, the rats were randomly separated into 3 groups (n=8) and treated in the following manner. In the normal group, the rats were orally administered a vehicle of 0.5% CMC aqueous solution every day for 18 days. In the model group, rats were intraperitoneally (i.p.) administered 7.5mg/kg cisplatin on day 15. In the α-pinene group, the rats were orally administered 50mg/kg/day alpha pinene suspended in 0.5% CMC aqueous solution for 18 days and 7.5mg/kg, i.p., single dose cisplatin on day 15. The doses of cisplatin [14], and alpha pinene [15,16] were selected based on previous literature.

On day 19, all animals were euthanized using thiopental (70mg/kg, i.p.), and blood samples were obtained via left ventricular puncture [17]. Serum was acquired and analyzed for urea and creatinine levels.

The kidneys were isolated and weighed instantly. The right kidneys were preserved in 10% buffered formalin for histopathological and immuno-histopathological studies. The left kidneys were homogenized 1:10 (weight/volume) with phosphate-buffered saline (pH=7.4) and centrifuged at 12,000 rpm for 10 minutes. The supernatants were separated, kept at −80°C, and used for assessment of renal oxidative stress, apoptosis, inflammation, and other molecular markers.

Biochemical examination:

Kidney function assessment:

Serum creatinine (SCR) levels (n=8) were assessed using a CREATININE (COLORIMETRIC) kit (Vitro Scient, Egypt). Serum urea levels (n=8) were assessed by an enzymatic colorimetric method (urease)-modified Berthelot reaction using a VITRO SCIENT Kit (VitroScient, Egypt). All these parameters were assayed according to the manufacturer's protocol.

Oxidative stress marker evaluation:

The homogenate was used for evaluation of total antioxidant capacity by using Total Antioxidant Capacity Assay Kit (No. ab65329) according to the manufacturer's instructions.

Inflammatory assessment:

Interleukin-6 (IL-6) was measured in renal homogenate using Rat IL-6 ELISA Kit (No. NBP1-92697) according to the manufacturer's instructions.

Histopathological examination:

Following fixation for 24 hours in 10% buffered formalin, routine processing in alcohol and xylene and embedding of tissues from the right kidney (n=5) in melted paraffin were performed. The tissues were sectioned into five-micron-thick sections with a microtome, processed, and stained with hematoxylin and eosin (H&E) using a conventional protocol [18]. The histopathological architecture of the kidneys in the control and experimental groups was examined using a light microscope at 400X magnification in a blinded manner.

Statistical analysis:

All results are represented as mean ± SD using one-way analysis of variance (ANOVA), followed by post-hoc Tukey to spot the differences between the experimental groups. GraphPad Prism version 8.0 (GraphPad Prism Software Inc.) was utilized for the analysis. *p*-values <0.05 were regarded as statistically significant.

Results

Effects of α-Pinene on kidney function parameters in cisplatin-induced AKI in rats:

In Fig. (1) Model group showed a significantly higher serum level of (A) Creatinine and (B) Ureacompared to normal group by (one and half-folds increase, $p<0.0001$) and (three and half-folds increase, $p<0.0001$), respectively. However, α-Pinene group showed a significant decrease in serum creatinineand urea levels by (25% decrease, $p<0.03$) and (39% decrease, $p<0.0001$), respectively, compared to model group.

Effects of α-Pinene on oxidative stress parameters in cisplatin-induced AKI in rats:

Regarding the model group, total antioxidant capacity level in renal homogenate was decreasedsignificantly by (50% decrease, $p<0.0001$) compared to normal group. On the other hand, α-Pinene group demonstrated a significant increase in total antioxidant capacity level by (59%, $p<0.008$) compared to model group as demonstrated in Fig. (2).

Effects of α-Pinene on inflammatory markers in cisplatin-induced AKI in rats:

As illustrated in Fig. (3), the level of IL-6 was elevated significantly in model group by (145% increase, $p<0.0001$) when compared with normal group. On the contrary, IL-6 level in renal homogenate was reduced significantly in α-Pinene groupby (27% decrease, $p<0.03$) in comparison with model group.

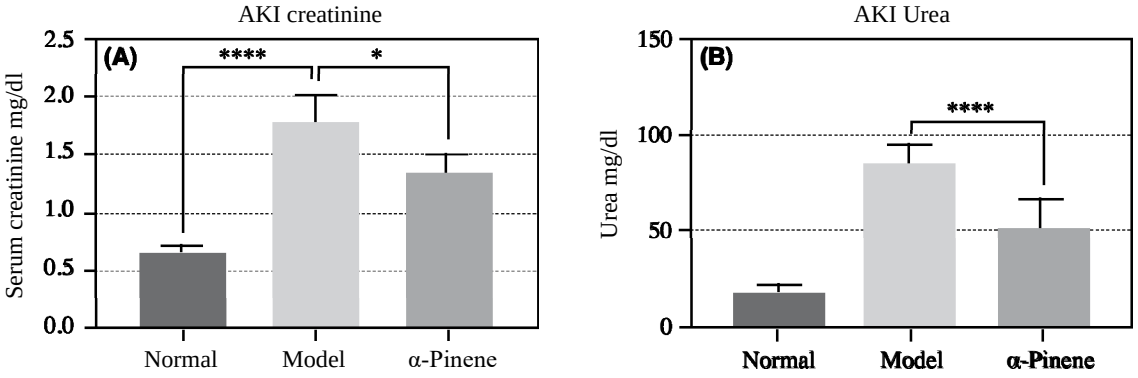


Fig. (1): The effect of α pinene (50mg/kg/P.O./18 days) on kidney function in cisplatin induced AKI in rats. (A) SCR level and (B) Urea level. Data are expressed as mean ± SD. Significant difference are presented at $p<0.05$. *Indicates significant difference of groups between brackets.

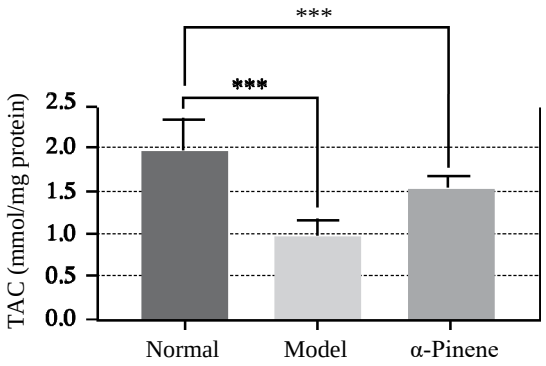


Fig. (2): The effect of α pinene (50mg/kg/P.O./18 days) on total oxidant capacity in cisplatin induced AKI in rats. Data are expressed as mean ± SD. Significant difference are presented at $p<0.05$. *Indicates significant difference of groups between brackets.

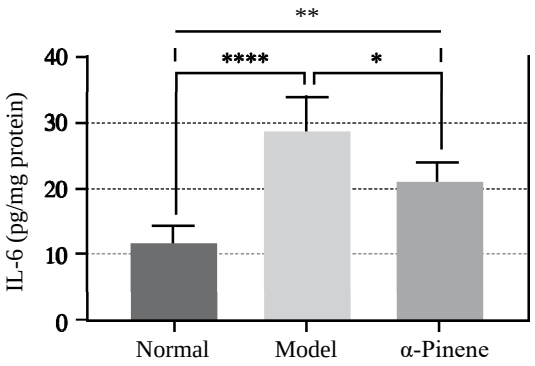
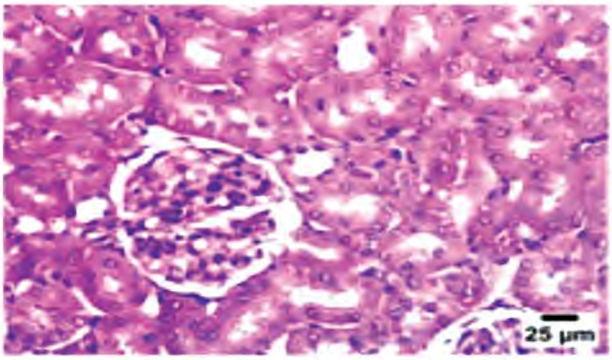


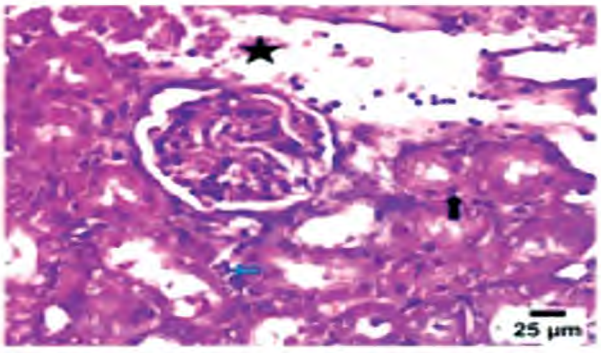
Fig. (3): The effect of α pinene (50mg/kg/P.O./18 days) on IL-6 in cisplatin induced AKI in ratsData are expressed as mean±SD. Significant difference are presented at $p<0.05$. *Indicates significant difference of groups between brackets.

Effects of α-Pinene on renal histological changes in cisplatin-induced AKI in rats:

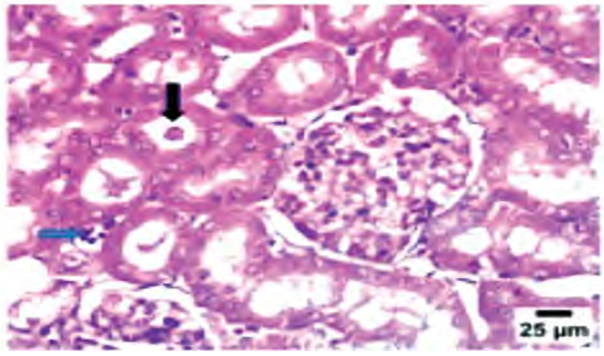
The histological photomicrograph from rats in the normal group showed normal histological structure of renal tubules as shown in Fig. (4A). The model group displayed remarkable deleterious histological alterations such as preglomerular congestion (star), presence of pyknotic nuclei in some renal tubules (black arrow) indicating apoptosis, and few number of mononuclear inflammatory cells between renal tubules (blue arrow) as shown in Fig. (4B). The α-Pinene group revealed renal cast formation (black arrow) and presence of aggregates of few numbers of mononuclear inflammatory cells between renal tubules (blue arrow) Fig. (4C).



(A)



(B)



(P)

Fig. (4): The effect of α pinene (50mg/kg/P.O./18 days) on renal histological changes in cisplatin-induced AKI in rats. (A) Normal group, (B) Model group, and (C) α-pinene group.

Discussion

Rats in the current investigation received a single intraperitoneal (i.p.) injection of cisplatin (7.5mg/kg), which caused histological alterations in renal tubular cells and decreased kidney function as indicated by a substantial rise in serum creatinine and urea levels indicating cisplatin-induced nephrotoxicity. These results are consistent with earlier research *[19]*.

Nephrotoxicity was noted in the earliest clinical trials of cisplatin therapy. Nowadays, it is understood that around one-third of individuals undergoing cisplatin treatment experience cisplatin nephrotoxicity *[20]*.

It is thought that the generation of reactive oxygen species (ROS), the accumulation of lipid peroxidation products in the kidneys, and the inhibition of the antioxidant system are the primary causes of cisplatin-induced AKI *[21]*.

Cisplatin is transformed into a highly reactive form inside the cell, where it quickly reacts with antioxidant molecules that contain thiols, such glutathione. As a result, glutathione depletion causes the cell to experience more oxidative stress *[22]*.

Alpha pinene has a strong antioxidant effect and is mostly effective at preventing lipid peroxidation *[23]*. Also, it not only stops free radicals from forming but also boosts antioxidant enzymes like SOD, CAT, GPx, and GSH that are necessary for the defense against ROS *[24]*. In line with prior investigations, our results showed that pretreatment with alpha pinene restored the redox status by increasing total antioxidant capacity.

It is increasingly recognized that, in addition to direct cellular toxicity, inflammation plays a part in the pathogenesis of cisplatin nephrotoxicity. In the past decade, numerous mediators of inflammatory renal injury have been identified *[25]*.

Oxidativestress, mediated by cisplatin-induced injury, is an activator of the NFκB transcription factor, which, in turn, promotes the production of proinflammatory cytokines, including TNF-α *[26]*.

Consequently, the expression of several inflammatory cytokines and chemokines is increased in the kidney after cisplatin injury such as the expression of IL-1β, IL-18, and IL-6 *[27]*.

The cytokine IL-6 is multifaceted. Endothelial cells generate large levels of it in reaction to proinflammatory triggers like TNF-α *[28]*. Renal damage increases IL-6 both locally and systemically, which encourages neutrophil infiltration and makes the injury worsen *[29]* which confirmed by our model.

In pathological situations, alpha-pinene acts as a potent inhibitor of inflammation. In addition to

suppressing microglial cells, it can prevent the synthesis of inflammatory cytokines like TNF-α, NF-κB, and IL-1β **[30]**. Consistent with these studies, this research demonstrated that alphapinene inhibits inflammation and cytokines production, such as IL-6; consequently, kidney injury was reduced, and the confirmation was done by improving histological changes.

Conclusion:

Theresults obtained from this study showed that alpha pinene exhibits a nephroprotective effect against cisplatin induced nephrotoxicity by enhancing kidney function, improving histological changes in kidney, decreasing oxidative stress, and reducing inflammation indicating that a-pinene acts as a promising molecule for the attenuation cisplatin nephrotoxicity.

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ألفا بينين يخفف من تلف الكلى الحاد الناجم عن السيسبلاتين

السيسبلاتين دواءٌ كيميائى فعّالٌ جدًا، يُستخدم لعلاج طيفٍ واسعٍ من الأورام الخبيثة. وتُحدّد فعاليته العلاجية بمقاومته للأورام، بالإضافة إلى آثاره الجانبية الكبيرة المرتبطة بالجرعة، بما فى ذلك ضعف السمع وتلف الكلى، بالإضافة إلى انخفاض وظائف نخاع العظم. ركّزت هذه الدراسة على فاعلية ألفا–بينين فى حماية الكلى من التلف الناتج عن السيسبلاتين حيث قُسِّم ٢٤جرذًا ذكرًا من سلاله سبراغ–داولى إلى ثلاث مجموعات تتالف كل مجموعه من ثمانيه جرذان :(١) المجموعة الاولى الطبيعية، (٢) المجموعة الثانيه تم حقنها بعقار سيسبلاتين ٥. ٧مللى جرام/كيلو جرام ، جرعة واحدة، داخل الصفاق)، (٣) مجموعة الثالثه تم حقنها بماده ألفا–بينين (٥٠ مللى جرام/كيلو جرام، عن طريق الفم لمدة ١٨ يومًا). تم حقن السيسبلاتين لجميع المجموعات فى اليوم الخامس عشر، باستثناء المجموعة الطبيعية. وتم تحليل مؤشرات وظائف الكلى، وحالة مضادات الأكسدة، ومؤشرات الالتهاب. حيث وجد ان السيسبلاتين رفع مؤشرات وظائف الكلى والالتهابات. علاوة على ذلك، انخفض مستوى مضادات الأكسدة بشكل ملحوظ فى المجموعة الثانيه. أظهرت النتائج أن المعالجة المسبقة بألفا بينين عززت حالة مضادات الأكسدة، بينما خفضت بشكل كبير مؤشرات وظائف الكلى والالتهابات. يمكن الاستنتاج ان ماده ألفا بينين يتميز بخصائص قوية مضادة للأكسدة والالتهابات، مما يحمى من السمية الكلوية الناتجة عن عقار السيسبلاتين.