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Original article

Gentamicin Induced Damage in Nephrotoxicity and Hepatotoxicity in Male Rabbits: The Protective Effect of Cinnamon

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ABSTRACT

One common aminoglycoside antibiotic that is well-known for its ability to treat gram-negative bacterial infections is gentamicin. Its nephrotoxic and hepatotoxic side effects, which are mostly brought about by the production of free radicals that harm kidney and liver cells, frequently restrict its therapeutic application. On the other hand, strong antioxidants found in cinnamon, a popular spice and therapeutic plant, have been demonstrated to have protective properties against oxidative stress. In order to assess the protective role of cinnamon, the current study examines how cinnamon affects gentamicin-induced changes in biochemical markers, liver enzymes in male rabbits. Treatment groups included a control group, a gentamicin-only (GIN) group, a cinnamon-only (CIN) group, and a combined cinnamon-gentamicin (CIN+GIN) group. The results showed that GIN group elevated liver enzymes, specifically aspartate transaminase (AST) and alanine transaminase (ALT), suggesting possible hepatotoxicity. The dose of cinnamon decreased these enzyme levels, while the combination therapy kept the AST levels close to control. Biochemical examination revealed that cinnamon reduced these alterations, particularly in the combination therapy group, while gentamicin increased total protein, urea, and creatinine levels, indicating renal and hepatic stress. The study concludes by highlighting the potential of cinnamon in reducing gentamicin-induced toxicity, specifically through its effects on lipid profiles, liver enzyme activities, body weight, blood profiles, and biochemical markers. This suggests that cinnamon could be used as an adjuvant therapy to reduce the negative effects of gentamicin".

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INTRODUCTION

A strong aminoglycoside antibiotic, gentamicin is used to treat a variety of bacterial illnesses, especially those brought on by Gram-negative bacteria. Gentamicin has been linked to severe adverse effects, including as hepatotoxicity and nephrotoxicity, despite its therapeutic effectiveness. About 10–20% of patients who get gentamicin develop nephrotoxicity, or kidney damage, mostly as a result of the drug's buildup in renal tubular cells, which causes inflammation and oxidative stress [1]. Furthermore, while less frequent, gentamicin-induced liver damage has been shown in animal research and is caused by comparable pathways, such as mitochondrial dysfunction and oxidative stress [2].

Kidney and inner ear issues can be brought on by gentamicin. Hearing loss and balance issues are examples of inner ear issues. These issues might not go away. It may be harmful to the growing fetus if taken while pregnant. Nonetheless, it

seems to be safe to use while nursing. Gentamicin is an aminoglycoside that functions by interfering with the bacteria's capacity to produce proteins, which usually results in the bacteria's death [3]. The bacterium Micromonospora purpurea naturally produces gentamicin, which was granted a patent in 1962 and authorized for use in medicine in 1964 [4]. By puncturing the bacterium's cell wall, the antibiotic is extracted from the Micromonospora culture. In an effort to boost expression and induce gentamicin secretion for a greater titer, current study is being conducted to comprehend the biosynthesis of this antibiotic. The World Health Organization's List of Essential Medicines includes gentamicin. Gentamicin is categorized as critically important for human medicine by the World Health Organization [5]. A generic version of the drug is accessible [6].

Gentamicin's nephrotoxic effects have been thoroughly investigated, and one of the main causes of kidney damage is oxidative stress. Normal kidney function is hampered by the overproduction of reactive oxygen species "ROS", which causes lipid peroxidation, protein oxidation, and "DNA" damage in kidney tissues. Increased blood urea nitrogen and serum creatinine levels, which are markers of kidney damage, are the clinical manifestation of this. Acute kidney injury "AKI", a potentially fatal syndrome that necessitates prompt medical attention, can result from severe gentamicin-induced nephrotoxicity [7]. The inner bark of various tree species of the genus Cinnamomum is used to make cinnamon, a spice. Throughout a wide range of cuisines, breakfast cereals, snack foods, bagels, teas, hot chocolate, and traditional foods, cinnamon is mostly employed as an aromatic condiment and flavoring addition. Cinnamaldehyde, the main ingredient and essential oil of cinnamon, along with a host of other components, such as eugenol, are responsible for the spice's flavor and aroma. Commercial spice products made from some tree species are referred to as cinnamon. They are all members of the Lauraceae family, namely the genus Cinnamomum. Commercial cinnamon production involves only a small number of species.

Cinnamomum verum, also known as "C". zeylanicum, is referred to as "Ceylon cinnamon" because it originated in Sri Lanka "formerly Ceylon". However, the majority of cinnamon sold internationally comes from four different species, which are more accurately called "cassia": C. burmanni, also known as Indonesian or Padang cinnamon; C. cassia, also known as Chinese or Chinese cinnamon; C. loureiroi, also known as Saigon cinnamon or Vietnamese cinnamon; and the less common C. citriodorum, also known

as Malabar cinnamon [8]. China produced 43% of the world's 226,753 tons of cinnamon in 2021 [9]. A well-known medicinal herb, cinnamon "Cinnamomum zeylanicum" has long been utilized for its antibacterial, anti-inflammatory, and antioxidant qualities. According to research, cinnamon includes potent antioxidants like eugenol and cinnamon aldehyde. Cinnamon may be used as a medicinal treatment to stop drug-induced organ damage because of these components' ability to scavenge free radicals and lower oxidative stress [10]. Cinnamon has demonstrated hepatoprotective and nephroprotective properties in a number of animal models, indicating that it may play a part in reducing gentamicin-induced toxicity [11]. Natural compounds like cinnamon have drawn more attention in recent years as potential preventative measures against drug-induced toxicity. Since cinnamon provides a safe, affordable, and easily accessible way to lessen these negative side effects, its potential to lessen gentamicin-induced liver and kidney damage is very encouraging.

Supplementing with cinnamon has been demonstrated to improve biochemical indicators of liver and kidney function, lower histopathological damage in animal models, and restore antioxidant enzyme levels [12]. To fully comprehend the precise mechanisms by which cinnamon carries out these protective benefits, more investigation is necessary. The primary objective of this research is to investigate the potential protective effects of cinnamon (Cinnamomum spp.) against nephrotoxicity and hepatotoxicity induced by gentamicin in male rabbits.

METHODS

Materials

This study employed cinnamon and gentamicin. In Al-Bayda City, cinnamon was bought from the public market for medical herbs. and in El-Bayda, Libya, gentamicin (Gentafar®, 10% Farvet, Holland) was bought at a pharmacy.

Experimental animals

We bought twenty mature male rabbits in good health from verified local farms. These rabbits were housed in a room that was appropriate for the trial duration and furnished in accordance with US-EPA 2004. The rabbits were kept in accordance with the US-EPA2004 for animal care and the Libyan Ministry of Agriculture's principles and guidelines. Each rabbit was kept in a suitable steel cage with a temperature between 22 and 26°C, a humidity level between 40 and 70%, and a clean environment with a 12-hour cycle of light.

For the course of the entire trial, a proper diet consisting of clean water and balanced feed has been supplied. The animals received the following treatment after being randomly assigned to four groups, each consisting of five rabbits: Group 1:

Gentamicin (50 mg/kg body weight) was administered orally to each rabbit on alternate days for 14 days. Group 2: Each rabbit received 200 mg/kg body weight of cinnamon orally every other day for 10 weeks. Group 3: Gentamicin (50 mg/kg body weight) and cinnamon (200 mg/kg body weight) were administered orally to each rabbit every day . For 14 days, Group 4 was administered 8 milliliters of distilled water orally as a control.

Experimental design

Throughout the 12-week trial period (two weeks of therapy plus ten weeks without treatment), all rabbit groups ("G1", "G2", "G3", and "G4") were observed. At the conclusion of the experiment, rabbits were killed to obtain a blood sample.

Blood biochemical parameters and enzyme activities

As soon as possible, the remaining fraction of the separated blood samples was put on ice. To create plasma, samples were centrifuged at 860 xg for 20 minutes. The plasma was then stored at -20°C until analysis was required. The Biuret technique, as described previously [13], was used to assess the total protein "TP" in stored plasma samples. Creatinine, urea, and plasma glucose concentrations were measured using [14] techniques. They discovered a way to measure total bilirubin in plasma [15]. To quantify the activity of plasma aspartate transaminase (AST; EC 2.6.1.1) and alanine transaminase ("ALT";" EC" 2.6.1.2), the [16] technique was employed. According to the approach outlined in previous study [17], the activity of alkaline phosphatase ("AlP"; EC 3.1.3.1) was measured in plasma.

Statistical analysis

Minitab software (version 17) or GraphPad Prism 8 were used for statistical analysis as needed. Following the identification of a normal distribution in the data, an "ANOVA" analysis using the Tukey multiple comparison test was performed to obtain a significance threshold of $P < 0.05$.

RESULTS

The effects of gentamicin, cinnamon, and their combination on the activities of "ALT" and "AST" in male rabbits table 1 show several interesting patterns, although no statistically significant changes are indicated by superscripts. Particularly with regard to the liver enzyme activity, the findings indicate varying reactions to each medication. "AST": The gentamicin-only "GIN" group had the highest "AST" levels (45.97±1.575 U/L), suggesting that gentamicin therapy elevated "AST" activity. Because "AST" is frequently released into the circulation when liver cells are injured, this rise in AST may indicate liver stress or possible hepatotoxic consequences from gentamicin consumption. The cinnamon-only "CIN" group had a somewhat lower "AST" level (35.78±0.954 U/L) than the control group "CON", which had an "AST" level of 38.96±1.662 U/L. This drop in the "CIN" group implies that cinnamon by itself could lower "AST" levels, which could indicate a stabilizing or protective impact on liver function. "AST" levels (38.83±1.469 U/L) in the combination therapy "CIN+GIN" were almost the same as the control, indicating that cinnamon may mitigate gentamicin-induced "AST" elevations and aid to keep enzyme activity closer to baseline levels. "ALT": The combination group "CIN+GIN" had the greatest "ALT" activity, measuring 45.68±1.409 U/L, surpassing even the "GIN" group's "ALT" level (41.79±1.318 U/L). This higher "ALT" in the "CIN+GIN" group may indicate that, although cinnamon by itself may lower enzyme activity, its protective impact against gentamicin's effect on "ALT" may be less potent than that against "AST". When administered without gentamicin, cinnamon may have a stabilizing impact on liver enzymes, as seen by the "CIN" group's lower "ALT" level (36.43±1.179 U/L), which was consistent with its effect on "AST". The "ALT" value of the control group (39.66±0.891 U/L) was comparable to that of the "GIN" group, indicating that gentamicin may have a mild influence on "ALT" without significantly raising it when compared to "AST. Additional studies should examine if these effects result from particular metabolic interactions between gentamicin and cinnamon chemicals in the liver.

Table 1. The activities of plasma enzymes of male rabbits treated withcinammion, gentamicinand their combination.

Parameter	*Experimental groups*			
Enzyme	**CON**	**CIN**	**GIN**	**CIN+GIN**
AST (U/L)	38.96 ± 1.662^{b}	35.78 ± 0.954^{b}	45.97 ± 1.575^{a}	38.83 ± 1.469^{b}
ALT (U/L)	39.66 ± 0.891^{b}	36.43 ± 1.179^{b}	41.79 ± 1.318^{a}	45.68 ± 1.409^{b}

The means ± SE for each treatment group is provided; n = 5. When mean values within a row did not share a common superscript letter (a, b, or c), significant differences ($p<0.05$) were observed.

Data on how cinnamon, gentamicin, and their combination affect male rabbits' table 2 biochemical parameters show both statistically significant and non-significant variations in the amounts of total protein, glucose, urea, and creatinine. An examination of these findings based on statistical significance and trends is provided below. G/dl of total protein: In comparison to the control "CON" and other treatment groups, the gentamicin-only "GIN" group had a substantially increased total protein level (6.90±0.296 g/dl) ($p<0.05$).

Given that gentamicin is known to impact liver function, elevated total protein in the "GIN" group might be a sign of increased protein synthesis or potential hepatic stress. In comparison to the control group (5.83±0.126 g/dl), the cinnamon-only "CIN" and combination therapy "CIN+GIN" groups showed decreased total protein levels (5.22±0.148 and 5.56±0.181 g/dl, respectively). This implies that, particularly when combined with gentamicin, cinnamon may have a moderating impact on protein synthesis or retention, assisting in bringing protein levels closer to normal. Glucose (mg/dl): The groups' levels of glucose varied significantly. High glucose levels were seen in both the "CON" and "CIN+GIN" groups (115.20±0.457 and 116.35±0.742 mg/dl, respectively), indicating that gentamicin and cinnamon may not have a substantial effect on glucose levels.

Significantly different from the other groups ($p<0.05$), the "CIN" group had the lowest blood glucose levels (91.17±1.751 mg/dl), indicating that cinnamon alone may help lower blood glucose because of its hypoglycemic and antioxidant qualities. At 100.45±2.065 mg/dl, the "GIN" group's glucose level was intermediate, lower than the control group's but higher than the "CIN" group's. This implies that gentamicin by itself could have a little hypoglycemia impact that is less noticeable when cinnamon is taken with it. Urea (mg/dl): Due to the nephrotoxic effects of gentamicin, the "GIN" group had the highest urea level (44.30±1.509 mg/dl), which was considerably higher than the control group's (38.00±0.437 mg/dl). This suggests that there may be renal stress or decreased urea clearance. Additionally, the "CIN+GIN" group had higher urea (42.58±0.905 mg/dl), indicating that although cinnamon may lessen the effects of gentamicin, some nephrotoxicity is still visible. In comparison to the control, the "CIN" group's urea level increased by 41.24±1.011 mg/dl, which was marginally higher but not statistically significant. This may suggest that whereas cinnamon by itself does not cause appreciable nephrotoxicity, it also does not totally negate the effects of gentamicin when taken in combination.

Due to the nephrotoxic effects of gentamicin, which are known to elevate creatinine levels, the "GIN" group had the highest creatinine level (1.08±0.070 g/dl), indicating substantial renal impairment. Creatinine levels in the "CIN" group were comparable to those in the control group (0.81±0.021 vs. 0.77±0.042 g/dl), indicating that cinnamon had no detrimental effects on renal function in terms of creatinine generation. Creatinine levels in the "CIN+GIN" group were (0.86±0.041 g/dl) closer to control values, suggesting that cinnamon may have a protective effect and lessen the nephrotoxicity of gentamicin. These results imply that cinnamon could offer male rabbits some metabolic defense against gentamicin-induced toxicity.

Table 2. Plasma biochemistry of male rabbits treated with cinammion, gentamicinand their combination.

Parameter	Experimental groups			
	CON	CIN	GIN	CIN+GIN
Total protein (g/dl)	5.83±0.126^{b}	5.22±0.148^{b}	6.90±0.296^{a}	5.56±0.181^{b}
Glucose (mg/dl)	115.20±0.457^{a}	91.17±1.751^{c}	100.45±2.065^{b}	116.35±0.742^{a}
Urea (mg/dl)	38.00±0.437^{b}	41.24±1.011^{ab}	44.30±1.509^{a}	42.58±0.905^{a}
Creatinine (g/dl)	0.77±0.042^{b}	0.81±0.021^{b}	1.08±0.070^{a}	0.86±0.041^{b}

The means ± SE for each treatment group is provided; n = 5. When mean values within a row did not share a common superscript letter (a, b, or c), significant differences ($p<0.05$) were observed.

DISCUSSION

The current study demonstrated that gentamicin altered the activities of biochemical parameters in plasma. Administration of gentamicin is known to cause hepatotoxicity, which results in increased levels of liver enzymes that show damage to the liver cells, such as "ALT" and "AST". Gentamicin causes hepatic cells to experience oxidative stress, which damages the cells and causes enzymes to flow into the blood. Because it neutralizes free radicals, cinnamon, which is high in bioactive antioxidants, has demonstrated promise in lowering oxidative damage in the liver. Gentamicin is well recognized for its hepatotoxic effects, which are mostly brought about by the production of reactive oxygen species "ROS", which harm and disrupt the function of liver cells. Increased levels of liver enzymes like "ALT" and "AST", which are indicators of liver damage, result from hepatocyte damage brought on by this oxidative stress. According to research by[18], gentamicin administration dramatically increased "ALT" and "AST" levels in animal

models, indicating cellular leakage and damage to liver tissue, mostly from oxidative stress. Cinnamon supplementation dramatically lowered "ALT" and" AST" levels in mice treated with gentamicin, indicating its hepatoprotective activity against oxidative damage, according researchby [19]. Because gentamicin is nephrotoxic, it frequently raises blood urea nitrogen "BUN" and creatinine levels, which are indicators of renal function. Kidney filtration capacity is hampered by glomerular and tubular damage brought on by oxidative stress in renal tissues caused by gentamicin. Antioxidants found in cinnamon, such cinnamon aldehyde, fight oxidative stress and may lessen kidney damage. Research by [20] demonstrated the nephroprotective function of cinnamon by showing that supplementing it significantly decreased "BUN" and creatinine levels in mice treated with gentamicin. Because gentamicin negatively affects insulin sensitivity and pancreatic cells, it may cause dysregulated blood glucose. Because gentamicin affects insulin sensitivity and pancreatic cells, it has been linked to hyperglycemia in animal studies. Oxidative stress brought on by gentamicin may hinder the action or secretion of insulin, raising blood glucose levels. Studies show that gentamicin-induced oxidative stress disrupts the body's normal glucose metabolism. [21] found that gentamicin-treated rabbits had elevated blood glucose levels, most likely due to pancreatic stress and irregularities in insulin regulation. Oxidative stress brought on by gentamicin may interfere with insulin signaling, raising blood glucose levels. Cinnamon has been demonstrated to increase insulin sensitivity and promote glucose absorption because of its insulin-mimetic qualities. According to [22], cinnamon supplementation decreased blood glucose in rats given gentamicin, suggesting that it might help alleviate the symptoms of hyperglycemia. Effect on Protein Levels Since most blood proteins are produced by the liver, gentamicin may result in decreased amounts of proteins, particularly albumin, due to liver dysfunction. Reduced protein synthesis due to gentamicin-induced liver injury can lead to hypoproteinemia and hypoalbuminemia. Cinnamon's preventive effect on liver cells helps to sustain protein synthesis by reducing hepatocyte damage. The preservation of albumin and total protein levels in mice administered gentamicin confirmed the hepatoprotective benefits of cinnamon, according a study by [23].

CONCLUSION

When given to rabbits, gentamicin causes severe oxidative stress, mostly affecting the liver and kidneys. Gentamicin produces free radicals that destroy vital enzymes and cellular membranes, resulting in cellular necrosis, t, and increased blood indicators like creatinine and "ALT" that show liver and kidney damage. In rabbits exposed to gentamicin, co-administration of cinnamon significantly lowers oxidative damage. Antioxidants found in cinnamon, such as cinnamon aldehyde, reduce the generation of free radicals and increase the activity of antioxidant enzymes, protecting the liver and kidneys' cells.

Conflict of interest. Nil

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الضرر الناجم عن الجنتاميسين في السمية الكلوية والسمية الكبدية في الأرانب الذكور: التأثير الوقائي للقرفة

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المستخلص

من المضادات الحيوية الشائعة من مجموعة الأمينوغليكوزيد والتي تشتهر بقدرتها على علاج الالتهابات البكتيرية سلبية الجرام الجنتاميسين. وكثيراً ما تحد الآثار الجانبية السامة للكلى والكبد، والتي تنجم في الغالب عن إنتاج الجذور الحرة التي تضر بخلايا الكلى والكبد، من استخدامها العلاجي. ومن ناحية أخرى، ثبت أن مضادات الأكسدة القوية الموجودة في القرفة، وهي من التوابل الشعبية والنباتات العلاجية، لها خصائص وقائية ضد الإجهاد التأكسدي. ومن أجل تقييم الدور الوقائي للقرفة، تدرس الدراسة الحالية كيف تؤثر القرفة على التغيرات التي يسببها الجنتاميسين في العلامات الكيميائية الحيوية وأنزيمات الكبد لدى الأرانب الذكور. وتضمنت مجموعات العلاج مجموعة تحكم ومجموعة جنتاميسين فقط ومجموعة قرفة فقط ومجموعة جنتاميسين وقرفة مجتمعة (CIN+GIN). وأظهرت النتائج أن مجموعة جين أظهرت ارتفاعًا في إنزيمات الكبد، وتحديدًا أسبارتات ترانس أمينيز (AST) وألانين ترانس أمينيز (ALT)، مما يشير إلى احتمال حدوث سمية كبدية. وقد أدت جرعة القرفة إلى خفض مستويات هذه الإنزيمات، في حين حافظ العلاج المركب على مستويات AST قريبة من السيطرة. وكشف الفحص الكيميائي الحيوي أن القرفة قللت من هذه التغيرات، وخاصة في مجموعة العلاج المركب، بينما زاد الجنتاميسين من مستويات البروتين الكلي واليوريا والكرياتينين، مما يشير إلى الإجهاد الكلوي والكبدي. وتختتم الدراسة بتسليط الضوء على إمكانات القرفة في تقليل السمية الناجمة عن الجنتاميسين، وتحديدًا من خلال تأثيراتها على ملفات الدهون وأنشطة إنزيمات الكبد ووزن الجسم وملفات الدم والعلامات الكيميائية الحيوية. وهذا يشير إلى أنه يمكن استخدام القرفة كعلاج مساعد لتقليل الآثار السلبية للجنتاميسين.

الكلمات المفتاحية :جنتاميسين؛ قرفة؛ أرانب؛ أنشطة إنزيمية.