Original Article

Effect of Beta vulgaris and Laurus Nobitis on Lipid Profile and Kidney in Hyperuricemia Rat

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ABSTRACT

Hyperuricemia is a major contributor to various chronic and metabolic diseases. It contributes to hyperlipidemia, elevated serum creatinine, hyperglycemia, and weight gain through multiple pathways and mechanisms. This study aimed to investigate the effects of *Beta vulgaris* and *Laurus nobilis* on reducing the risk of hyperuricemia-related metabolic disorders and kidney damage in a rat model. Twenty-four adult male albino rats (weighing approximately 200–220 g and aged 8–12 weeks) were housed in the animal facility. Hyperuricemia was induced in the model group by administering oxonic acid (250 mg/kg body weight). Treatment groups received either *Beta vulgaris* or *Laurus nobilis* following hyperuricemia induction. Kidney tissue samples were examined histopathologically, and biochemical tests were conducted on all rat groups. In rats treated with *Laurus nobilis* and *Beta vulgaris*, all biochemical parameters—excluding HDL—were significantly decreased compared to the hyperuricemia model rats ($P \leq 0.01$). Notably, cholesterol (49.00±6.48), triglycerides (47.25±2.22), LDL (34.50±3.11), uric acid (4.90±0.22), urea (46.00±0.82), creatinine (0.35±0.03), blood glucose (193.00±11.20), and weight gain (77.75±2.06) were lower. Histopathological analysis showed reduced nephron damage in rats treated with *Beta vulgaris* and *Laurus nobilis*. This study demonstrated that hyperuricemia induces kidney damage and metabolic disorders, including dyslipidemia, hyperglycemia, increased serum creatinine, urea, and weight gain in model rats. *Beta vulgaris* and *Laurus nobilis* significantly reduced these biochemical parameters and ameliorated histopathological signs of hyperuricemia, such as glomerular atrophy and hydropic changes in proximal tubular epithelial cells. *Laurus nobilis* exerted a more substantial effect on lipid profile, blood glucose, serum creatinine, weight, and urea levels than *Beta vulgaris*.

Keywords: Hyperuricemia, Metabolic disorder, *Beta vulgaris*, Laurus nobilis, Kidney damage

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1. Introduction

Hyperuricemia is recognized as a primary contributor to numerous chronic and metabolic diseases and is associated with a range of comorbidities. Studies show the prevalence of hyperuricemia in hospital patients is approximately 19.86% (1). Defined as an elevated serum uric acid level, hyperuricemia leads to uric acid accumulation in joints, resulting in gout. Additionally, high uric acid levels are implicated in cardiovascular disease, kidney damage, diabetes, and neurodegenerative diseases like Alzheimer's (2). This condition can also trigger metabolic disorders such as dyslipidemia and impair glucose metabolism. Furthermore, hyperuricemia initiates systemic inflammation through mechanisms including glomerular damage, nephropathy, and vascular inflammation (2, 3, 4). Hyperuricemia is typically defined as a serum uric acid level exceeding 6.8 mg/dL in males, though this threshold can vary with age (5). This condition results from either excessive uric acid production due to metabolic dysfunction or decreased uric acid excretion due to renal impairment. Uric acid in the bloodstream is primarily derived endogenously through the breakdown of nucleic acids, specifically adenine and guanine, during cell death, and exogenously from the purine catabolism end products in the liver, muscles, intestines, and vascular endothelium (6). Uric acid is freely filtered through the glomeruli, with approximately 90% reabsorbed into capillaries in the proximal tubules (6). Around 70% of uric acid is excreted by the kidneys, while the remaining 30% is eliminated through the intestines (7). Hyperuricemia contributes to hyperlipidemia, elevated serum creatinine, hyperglycemia, and weight gain through various pathways and mechanisms (8). It has been observed that triglycerides (TG), total cholesterol (TC), and low-density lipoprotein (LDL) increase with rising uric acid levels, while high-density lipoprotein (HDL) decreases inversely with uric acid levels (4, 9, 10). Although the physiological mechanisms linking uric acid to these metabolic disorders are complex and involve multiple pathways and interactions (11), some pathophysiological mechanisms have been identified. For example, increased TG synthesis promotes uric acid production by accelerating the conversion of ribose-5-phosphate to phosphoribosyl pyrophosphate (PRPP) via the NADP/NADPH metabolic pathway (11). Hyperuricemia can impair kidney function both directly, through uric acid accumulation, and indirectly, through mechanisms leading to nephropathy. Additionally, hyperuricemia exerts detrimental effects on the kidneys, heart, and brain tissues, largely due to the oxidative stress induced by free radicals or reactive oxygen species generated by elevated uric acid levels (2). *Beta vulgaris* possesses antioxidant and anti-inflammatory properties that have been leveraged in herbal medicine, including for reducing uric acid levels in rats (12, 13). *Beta vulgaris* L. can inhibit the xanthine oxidase enzyme, thereby preventing the conversion of xanthine to uric acid (14). Studies have also demonstrated its antihypercholesterolemic effects in rat models. Similarly, *Laurus nobilis* offers significant health benefits, notably in lipid profile regulation and antioxidant activity (15). Research has shown that *Laurus nobilis* can improve blood lipid profiles and potentially reduce the risk of cardiovascular disease (16). Additionally, its antioxidant effects may help mitigate kidney damage (17). This study aimed to evaluate the effects of *Beta vulgaris* L. and *Laurus nobilis* in reducing hyperuricemia-related risks, including dyslipidemia, hyperglycemia, weight gain, and kidney damage in a rat model.

2. Materials and Methods

2.1. Experimental (Laboratory) Animals

Twenty-four adult male albino rats (*Rattus norvegicus*) weighing approximately 200–220 g and aged 8–12 weeks were used in this study. The animals were housed in the animal facility under controlled conditions, with six rats per cage. The environment was maintained on a 12-hour light/dark cycle at a temperature of 22 ± 4°C, and the rats had ad libitum access to a standard pellet diet and tap water. The rats were divided into four groups, with six animals in each group. The hyperuricemia model group was administered oxonic acid to induce hyperuricemia. Following induction, two treatment groups were administered either *Beta vulgaris* or *Laurus nobilis* to assess therapeutic effects. The control group was neither exposed to hyperuricemia induction nor given treatment.

2.2. Preparation of *Beta Vulgaris*

Fruits were collected, shade-dried, and then ground into a fine powder using a mechanical grinder. The powder was stored in airtight containers. Beetroot extracts were prepared by soaking 1 gram of powder in 50 ml of 70% ethanol for 48–72 hours. The resulting solution was filtered and concentrated using a rotary evaporator. The concentrated extract was then lyophilized via freeze-drying to obtain a powder, which was stored in a dark container at 4°C until use.

2.3. Preparation of Laurus Nobilis

Five grams of dried bay leaf were boiled in 100 ml of distilled water. One ml of the bay leaf solution was administered to each rat daily by gavage, once per day, for two weeks.

2.4. Induction of Hyperuricemia

Hyperuricemia was experimentally induced in rats through intraperitoneal (IP) administration of oxonic acid at a dose of 250 mg/kg body weight, administered once daily for two weeks(18).

2.5. Biochemical examination and measurement

2.5.1. Collection of Blood Samples

At the end of the study, rats were fasted overnight and then anesthetized with an intraperitoneal injection of ketamine hydrochloride (50 mg/kg body weight) and xylazine (5 mg/kg body weight)(19). Blood samples were collected via cardiac puncture into gel tubes, then centrifuged at 3000 rpm for 15 minutes. The resulting serum was analyzed for biochemical parameters(20).

2.5.2. Estimation of Serum Biochemical

The following parameters were measured in serum using an automatic biochemistry analyzer (Cobas E 411, Roche, Germany)(21): serum uric acid (SUA), serum creatinine (SCr), urea, total cholesterol (TC), serum triglycerides (TG), high-density lipoprotein (HDL), low-density lipoprotein (LDL), and blood glucose (BG). The results are expressed in mg/dL.

2.5.3. Histopathological Examination

Histopathological tests were conducted to assess changes in renal tissue morphology and microscopic structure. Following collection, the renal cortex tissue was cleaned with 1% cold saline and preserved in 10% formalin. The samples were processed, embedded in paraffin blocks, and sectioned into 3 µm slices using a microtome. The fixed slices were stained with hematoxylin and eosin (H&E) and cover slips were affixed to the tissue slides using dibutyl phthalate polystyrene xylene (DPX). Examination was performed using a binocular microscope, and light micrographs of the fixed organs were captured.

2.5.4. Data Management

The biochemical data were analyzed using SPSS version 22. Mean and standard deviation were calculated for all biochemical tests. A one-way ANOVA test was employed to assess the statistical differences in mean biochemical parameters among the various rat groups. The results of the histopathological examinations are presented in figures and images.

3. Results

Table 1 illustrates the various biochemical parameters in the control, model, and treatment samples. Except for HDL, all biochemical parameters, including cholesterol, triglyceride, LDL, uric acid, urea, creatinine, blood sugar, and weight gain, were significantly decreased in the rat administered *Laurus nobilis* and *Beta Vulgaris* treatment compared to the hypouricemic model rat (P value ≤0.01). The mean in the hyperuricemia model rats were (49.00±6.48), (47.25±2.22), (34.50±3.11), (4.90±0.22), (46.00±0.82), (0.35±0.03), (193.00±11.20), and (77.75±2.06), respectively. Mean of cholesterol (35.25±2.99), triglyceride (27.50±3.00), HDL (10.25±0.68), and weight loss (-12.50±9.04) were even lower in the *Laurus nobilis*-treated rat compared to the control. While mean of LDL (21.13±2.10), uric acid (1.80±0.22), and creatinine (0.27±0.05) were almost similar to the control, and mean urea (39.00±3.56) and blood sugar (162.75±6.75) were higher than the control. Almost cholesterol, triglyceride, LDL, uric acid, and body weight in Laurus nobilis-treated rat were significantly decreased compared to the *Beta Vulgaris*-treated rat.

4. Discussion

The aim of this study was to assess the physiological effects of *Beta vulgaris* L. and *Laurus nobilis* on the regulation of uric acid, urea, creatinine, dyslipidemia, hyperglycemia, weight gain, and kidney damage in a hyperuricemic rat model. The study found that inducing hyperuricemia with intraperitoneal administration of oxonic acid (250 mg/kg body weight) resulted in a significant increase in uric acid levels, along with various metabolic disorders and kidney damage in the model rats. Elevated uric acid can induce dyslipidemia or hyperlipidemia, hyperglycemia, increased serum creatinine and urea levels, and weight gain through various complex mechanisms. Uric acid itself enhances the production of pro-inflammatory cytokines and gliosis in the hypothalamus, activating NF-κB, which contributes to dyslipidemia and glucose intolerance (22). Moreover, hyperuricemia may lead to kidney damage through multiple intricate pathways, including activation of the renin-angiotensin system (RAS), oxidative stress due to NADPH oxidase activation, mitochondrial dysfunction, epithelial-mesenchymal transition, endothelial dysfunction, and proliferation of vascular smooth muscle cells (23). The relationship between uric acid and kidney damage is complex, as elevated serum uric acid levels are associated with a pro-oxidative and pro-inflammatory state that can lead to kidney injury (2). Additionally, high uric acid levels significantly increased weight gain in the model rats. This finding is inconsistent with published literature that has demonstrated a positive correlation between body mass index (BMI) and uric acid, as adipose tissue in obese patients secretes more uric acid (24). Increased body weight may be related to alterations in lipid profile and insulin resistance in the rat model. Furthermore, high body weight could also result from water retention and kidney failure in hyperuricemic rats. *Beta vulgaris* contains several bioactive compounds, including betalains and polyphenols, which exert metabolic effects and scavenge free radicals (25). *Laurus nobilis* is rich in components such as phenolic acids, flavonoids, and alkaloids (26). A recent study indicated that daily administration of 1 g of *Beta vulgaris* L. and 5 g of *Laurus nobilis* could significantly reduce uric acid levels and ameliorate the physiological complications associated with hyperuricemia in rats. This finding aligns with existing literature (27, 12, 16). In another study, administering 1.56 g/kg body weight per day of beet powder resulted in a decrease of 6.35 mg/dL in uric acid levels and a reduction of 4.35 nmol/mL in malondialdehyde (MDA), while 1.8 g/kg body weight per day of beet powder demonstrated a greater effect than allopurinol (12). The beneficial effects of *Beta vulgaris* may be attributed to its ability to inhibit xanthine oxidase, thereby reducing uric acid production from xanthine (14). This action of *Beta vulgaris* on hyperuricemia may also positively influence other metabolic adverse effects associated with the condition, such as dyslipidemia, hyperglycemia, elevated serum creatinine, and weight gain. In the current study, *Laurus nobilis* and *Beta vulgaris* significantly reduced total cholesterol, triglycerides, LDL, uric acid, urea, creatinine, blood sugar, and weight gain induced by hyperuricemia, bringing these parameters closer to control levels. Both *Beta vulgaris* and *Laurus nobilis* were effective in decreasing total cholesterol (TC), triglycerides (TG), LDL, and blood glucose, likely due to their anti-inflammatory and antioxidant properties (28, 15, 27).

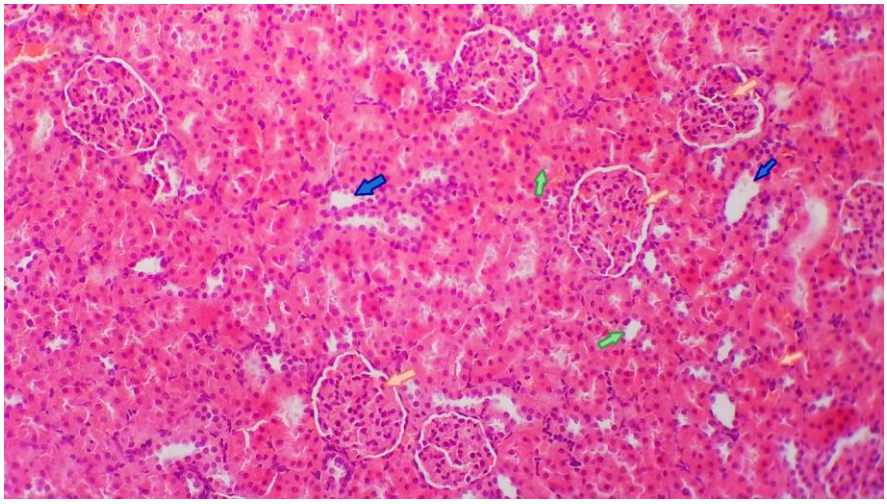

Figure 1. Shows the kidney of the control rat, where normal histological architecture of the kidney is seen, such as renal glomeruli (yellow arrow), proximal convoluted tubules (blue arrow), and distal convoluted tubules (green arrow), with H & E staining 10 X.

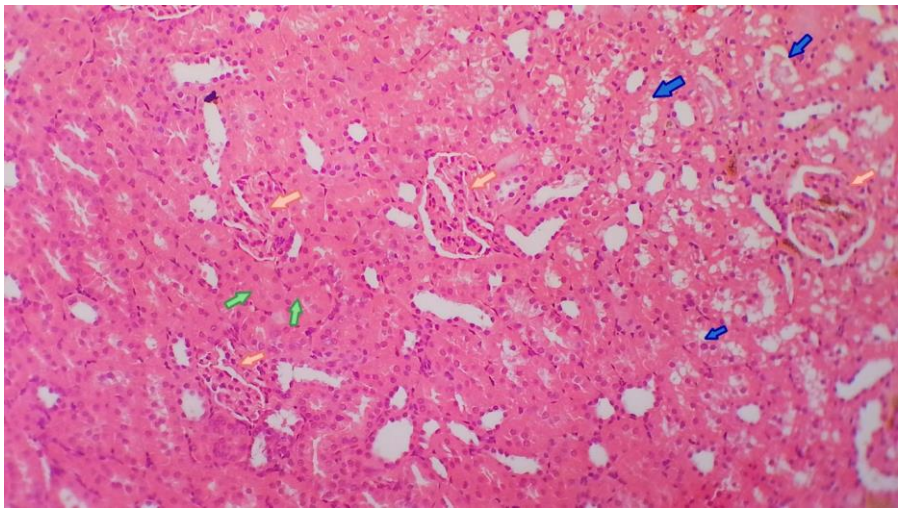

Figure 2. illustrates the kidney hyperuricemia model, with deterioration and atrophy of kidney glomeruli (yellow arrow). In proximal convoluted tubules, there are hydropic changes in the epithelial lining (blue arrow), whereas in distal convoluted tubules, there are no significant pathological changes (green arrow) H & E staining 10 X.

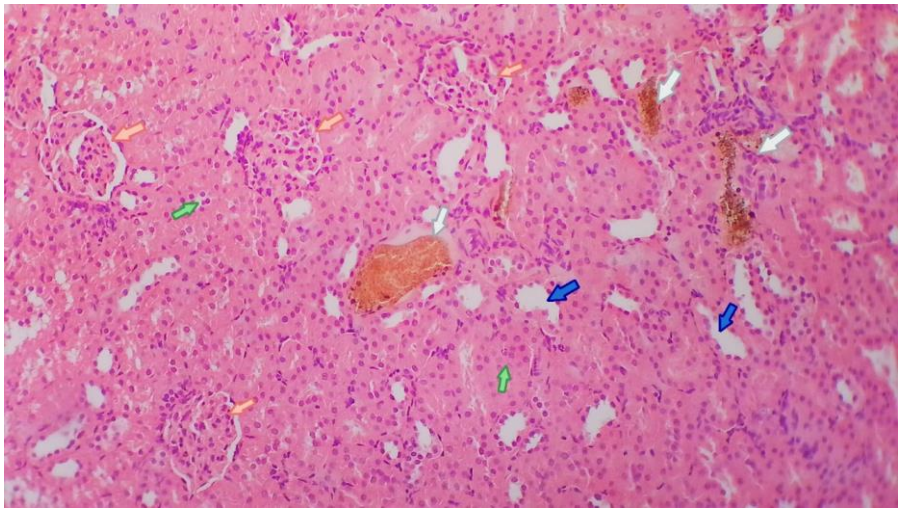

Figure 3. reveals that in the beetroot (*Beta Vulgaris*) model, there is degeneration of renal glomeruli indicated by narrowing or bowman space (yellow arrow), there are no significant pathological changes in proximal convoluted tubules (blue arrow) and distal convoluted tubules (green arrow), and throughout the section there is interstitial hemorrhage (white arrow). H & E staining 10 X.

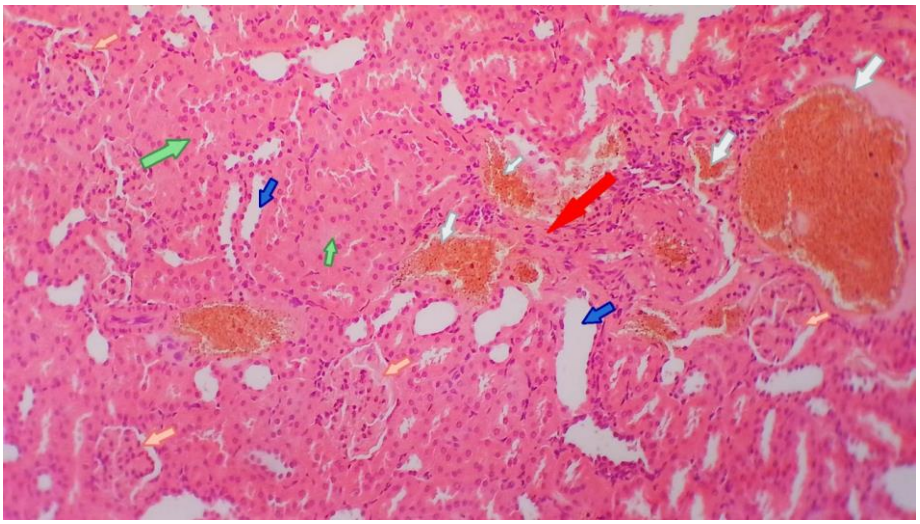

Figure 4. indicates kidney of Bay leaf model (Laurus nobilis), there is degeneration of renal glomeruli indicated by narrowing or bowman space (yellow arrow), there is no significant pathological changes in proximal convoluted tubules (blue arrow) and distal convoluted tubules (green arrow), throughout section there are interstitial hemorrhage (white arrow), and some area of fibrosis in renal tissue (red arrow) H & E staining 10 X.

Table 1. Mean and standard deviation of biochemical parameters in control, model and treatment rats

Groups	Control	Model	Laurus Nobilis	*Beta vulgaris*	Total	P. Value
	Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD	
Cholesterol (mg/dL)	37.00±4.32	49.00±6.48	35.25±2.99	41.00±5.60	40.56±7.08	0.01
Triglyceride (mg/dL)	35.50±5.57	47.25±2.22	27.50±3.00	34.50±2.38	36.19±7.99	0.000
HDL (mg/dL)	12.05±0.74	9.93±0.83	10.25±0.68	9.23±0.46	10.36±1.24	0.001
LDL (mg/dL)	21.75±2.06	34.50±3.11	21.13±2.10	27.00±1.41	26.09±5.89	0.000
Uric Acid (mg/dL)	1.10±0.08	4.90±0.22	1.80±0.22	2.25±0.29	2.51±1.50	0.000
Urea (mg/dL)	26.25±1.50	46.00±0.82	39.00±3.56	39.25±4.19	37.63±7.81	0.000
Creatinine (mg/dL)	0.24±0.03	0.35±0.03	0.27±0.05	0.28±0.04	0.28±0.05	0.015
Blood Sugar (mg/dL)	152.25±5.68	193.00±11.20	162.75±6.75	162.00±19.17	167.50±19.07	0.002
First Day Weight (gm)	210.00±8.16	205.00±5.77	235.00±19.15	210.00±8.16	215.00±15.92	0.013
Last Day Weith (gm)	259.50±15.86	282.75±4.57	222.50±23.70	256.50±15.29	255.31±26.58	0.002
Weight difference (gm)	49.50±10.25	77.75±2.06	-12.50±9.04	46.50±12.12	40.31±34.90	0.000

Beta vulgaris exhibits strong antioxidant activity, particularly in its ability to reduce malondialdehyde (MDA) levels in hypouricemic rats. *Laurus nobilis* has also demonstrated the capacity to reduce and scavenge free radicals such as DPPH, O2, and NO, as well as lipid peroxidation (12, 29, 26). Furthermore, *Beta vulgaris* possesses anti-inflammatory properties that can decrease levels of C-reactive protein (hs-CRP), intracellular adhesion molecule-1 (ICAM-1), vascular endothelial adhesion molecule-1 (VCAM-1), interleukin-6 (IL-6), E-selectin, and tumor necrosis factor-alpha (TNF-α) in rat models (27). However, a review study found no significant effect of *Beta vulgaris* on lipid profiles, including TC, TG, and LDL (30). *Laurus nobilis* exerts a greater physiological effect on lipid profile, blood glucose, serum creatinine, body weight, and urea levels compared to *Beta vulgaris*. In rats treated with *Laurus nobilis*, cholesterol, triglycerides, LDL, uric acid, urea, creatinine, and body weight were all significantly reduced compared to those treated with *Beta vulgaris*. The lower levels of urea and serum creatinine in the *Laurus nobilis* group indicate improved kidney function in this treatment cohort (31). This reduction in urea and serum creatinine levels may positively influence lipid and glucose metabolism while also mitigating kidney damage. The mean levels of cholesterol, triglycerides, HDL, and weight loss were lower in rats treated with *Laurus nobilis* compared to the control group, while the mean levels of LDL, uric acid, and creatinine were similar to those of the control group. In contrast, the mean levels of urea and blood sugar were elevated compared to the control. In the current study, the kidneys of hyperuricemic rats exhibited deterioration and atrophy of the glomeruli. The proximal convoluted tubules displayed hydropic changes in the epithelial lining, while the distal convoluted tubules did not show significant pathological changes. These findings align with existing literature indicating that hyperuricemia independently increases the risk of segmental glomerulosclerosis and tubular atrophy/interstitial fibrosis (32). Additionally, previous research has shown that kidney injury leads to increased interstitial fibrosis, macrophage infiltration, and autophagy due to various inflammatory processes, including the expression of NLRP3 and IL-1β, along with the activation of multiple cell-signaling pathways (3). Another study suggested that kidney damage in hyperuricemic rats is associated with uric acid-induced inflammatory activity, endothelial dysfunction, proliferation of vascular smooth muscle cells, and activation of the renin-angiotensin system (7). The filtration of uric acid in the glomeruli and its reabsorption in the proximal tubules may contribute to the observed atrophy and pathological changes in these nephron segments. In this study, both *Beta vulgaris* and *Laurus nobilis* were found to ameliorate kidney deterioration; however, some degeneration in the renal glomeruli was noted, indicated by narrowing of the Bowman space and occasional interstitial hemorrhages. No significant pathological changes were observed in the proximal or distal convoluted tubules compared to the hyperuricemia model. In the *Laurus nobilis* group, some areas of fibrosis in renal tissue were noted. The differences in histopathological appearance between the two treatment groups may relate to the biochemical activities of *Beta vulgaris* and *Laurus nobilis* and could be influenced by the doses used in this study. *Laurus nobilis* has also been shown to protect against degeneration of tubular epithelium and glomeruli in diabetic-induced rat models (33). In this study, both blood creatinine and urea levels—biomarkers of kidney function—demonstrated better kidney function in the *Laurus nobilis* group compared to the *Beta vulgaris* group, as indicated by lower levels of these substances (31). This study found that elevated uric acid levels induce kidney damage and various metabolic disorders, including dyslipidemia, hyperglycemia, increased serum creatinine and urea, and weight gain in model rats. Specific doses of *Laurus nobilis* and *Beta vulgaris* significantly reduced total cholesterol (TC), triglycerides (TG), LDL, uric acid, urea, creatinine, blood sugar, and weight gain induced by hyperuricemia, often returning these levels to those observed in normal control rats. *Laurus nobilis* had a greater physiological impact on lipid profiles, blood glucose, serum creatinine, weight, and urea compared to *Beta vulgaris*. Both *Laurus nobilis* and *Beta vulgaris* ameliorated the histopathological effects of hyperuricemia, including glomerular atrophy and hydropic changes in the epithelial lining of the proximal convoluted tubules. Author contribution.

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Authors' Contribution
Study concept of the study has been developed by: C.JK.
Acquisition and collecting of data was been arranged by: S.HM.
Analysis and interpretation of data was managed: J.KS.
Drafting of the manuscript has been prepared by: C.GR.
Critical revision of the manuscript for important intellectual content were managed by: D.HK and S.JM.
Statistical analysis was managed by: J.KS.
Administrative and technical support were done by: M.AS.

Ethics
We hereby declare all ethical standards have been respected in preparation of the submitted article. This study has been approved by the ethical committee of Sulaimani Polytechnic University.

Conflict of Interest
The authors declare that they have no conflict of interest.

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This study has been funded by the authors. It did not attain any financial support by any agencies.

Data Availability
This study has used the cleared raw data that have been collected from our laboratory (Biology Department at Sulaimani University). Data are available to use for further analysis.

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