Antiurolithiatic and antioxidant efficacy of *Musa paradisiaca* pseudostem on ethylene glycol-induced nephrolithiasis in rat

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**Abstract:** *Musa paradisiaca* has been used in the treatment of urolithiasis by the rural people in South India. Therefore, we plan to evaluate its efficacy and possible mechanism of antiurolithiatic effect to rationalize its medicinal use.

**Material and Methods:** Urolithiasis was induced in hyperoxaluric rat model by giving 0.75% ethylene glycol (EG) for 28 days along with 1% ammonium chloride (AC) for the first 14 days. Antiurolithiatic effect of aqueous-ethanol extract of *M. paradisiaca* pseudostem (MUSA) was evaluated based on urine and serum biochemistry, microscopy of urine, oxidative/nitrosative indices, kidney calcium content, and histopathology.

**Results:** Administration of EG and AC resulted in increased crystalluria and oxaluria, hypercalciuria, polyuria, crystal deposition in urine, raised serum urea, and creatinine as well as nitric oxide concentration and erythrocytic lipid peroxidation in lithiatic group. However, MUSA treatment significantly restored the impairment in above kidney function tests as that of standard treatment, cystone in a dose-dependent manner.

**Conclusions:** The present findings demonstrate the efficacy of MUSA in EG-induced urolithiasis, which might be mediated through inhibiting various pathways involved in renal calcium oxalate formation, antioxidative effect, and potential to inhibit biochemical markers of renal impairment.

**Key words:** Histopathology, *Musa paradisiaca*, oxidative/nitrosative stress, rat, urolithiasis

Urolithiasis or formation of urinary stone causes a major impact on public health and economy globally since last two decades.[¹²] It is also considered as the third most common problem of the urinary tract with an estimated lifetime risk of around 2%–5% in Asia, 8%–15% in America and Europe, and 20% in the Middle East countries.[¹³] In spite of substantial progress in the study of physiological manifestation of urolithiasis, its exact mechanism is still not clearly understood. The recent proposed mechanism of stone formation involves urinary supersaturation, nucleation, precipitation, growth, aggregation of crystals, and their retention in renal tubular epithelial cells with crystal matrix.[¹¹,¹²] These processes are modulated by a variety of urinary macromolecules which become incorporated in the growing crystals and eventually constitute organic component or matrix.

Modern diagnostic and therapeutic aids such as extracorporeal shock wave lithotripsy, ureteroscopy, and percutaneous nephrolithotomy had revolutionized the urological practices but cannot altered the recurrence of stone formation and also have adverse side effects. Pharmaceutical agents such as thiazide and citrate diuretics, alkali therapy, allopurinol, etc., have also limited efficacy in addition to their less tolerability.[¹⁰] Indeed, urolithiasis is a multifactorial disease and involves in alteration of several biochemical pathways. Therefore, the present situation demands a newer approach of therapy. Several recent studies have highlighted the effectiveness of medicinal plants and natural compounds for treatment and management of urolithiasis.[³,⁵‑¹⁰] Moreover, herbal remedies are known to contain multiple constituents, acting through multiple pathways such as antioxidant, analgesic, diuretic, pH neutralizing, etc., *Musa paradisiaca* is a commonly available plant in India. The remote people of South India had been using the juice of *M. paradisiaca* pseudostem for...
kidney stone disease as indigenous folk remedies. Although some preliminary published reports were available,[11] its antiurolithiatic efficacy was not explored scientifically till now. Hence, the study was designed to evaluate the possible mechanism of antiurolithiatic effect of aqueous ethanol extract of *M. paradisiaca* pseudostem (MUSA) in ethylene glycol (EG) induced lithiatic model in rats.

### Materials and Methods

#### Animals
The animal care and handling was in accordance with the internationally accepted standard guidelines for use of animals,[10] and the protocol was approved by the Institutional Animal Ethical Committee (approval no F.26-1/2015-16/JDR). Sixty male albino Wistar rats (weighing 150–200 g) were procured from the Laboratory Animal Research Division of Indian Veterinary Research Institute and housed in clean polypropylene cages under controlled temperature 25°C ± 2°C, humidity 45%–55%, and 12 h light-dark cycle throughout the experimental period. Animals were given standard diet and had free access to food and water *ad libitum* throughout the study. However, food was withdrawn while collecting 24 h urine samples inside metabolic cage.

#### Plant Material and Extraction
The plant *M. paradisiaca* was collected from horticulture section of the Institute and was identified and authenticated from Botanical Survey of India, Central National Herbarium, Howrah, India. The pseudostem of *M. paradisiaca* (MUSA) was separated from whole plant and cut into pieces, washed with distilled water, dried, and uniformly powdered using an electric grinder. Hydro-ethanol extract of the powdered plant material was extracted in room temperature (37°C) using solvent system (aqueous: Ethanol 1:1) and filtered using filter paper (Whatman no. 40). The solvent was evaporated using rotary evaporator under pressure. The extract was dried *in vacuo* and stored refrigerated until use.[12] The recovery percentage was 16% (w/w).

#### Acute Toxicity Study
The oral acute toxicity study was carried out as per OECD guideline 423 using male Wistar rats before evaluation of the antiurolithiatic activity.[6,7] Increasing dosage of MUSA (500, 1000, 2000, and 3000 mg/kg) were orally administered to groups of three animals for each and the general behavior of rats were observed at hourly interval up to 12 h, and daily for next 2 weeks. The median lethal dose (LD$_{50}$) cutoff dose was found to be 3000 mg/kg for MUSA. Hence, the therapeutic dose was taken as 100 mg/kg to 300 mg/kg body weight for antiurolithiatic study.

#### Study on Animal Model of Urolithiasis
Antiurolithiatic activity of the test plant material was determined by hyperoxaluric rat model of calcium oxalate (CaOx) urolithiasis as described previously.[3,10] Forty-eight male Wistar rats were divided with matched body weights into eight groups of six animals each, which were then randomly selected to receive various treatments. Rats of Group I served as untreated healthy control without receiving any treatment. Group II rats act as lithiatic control received 0.75% (w/v) EG along with 1% (w/v) ammonium chloride in drinking water for 14 days for induction of crystal followed by EG alone for next 14 days. Group III, Group IV, Group V, Group VI, and Group VII rats received stone-inducing treatment for 14 days followed by 100 mg/kg, 150 mg/kg, 200 mg/kg, 250 mg/kg, and 300 mg/kg body weight of MUSA orally through gastric gavage for next 14 days. Group VIII rats received stone-inducing treatment for 14 days followed by standard drug-cystone at 100 mg/kg orally through gastric gavages for next 14 days.

#### Sampling
Twenty-four hours urine samples were collected on day 0, day 14, and day 28, housing rats individually in the metabolic cages. Following volume and pH determination, part of each urine sample was acidified to pH 2 with 5M HCl. Both acidified and nonacidified urine sample were centrifuged at 1500 g for 10 min to remove debris and supernatants and were stored at −20°C until analyzed. Blood was collected through cardiac puncture from individual animals under ether anesthesia at day 0, day 14, and day 28 for kidney function test and oxidative/nitrosative stress indices. Animals were sacrificed after the end of study and both the kidneys were excised for histopathology and estimation of tissue calcium content.

#### Biochemical Analysis of Urine and Serum
In acidified urine sample, calcium and phosphorus were determined using commercial kits. Oxalate content of acidified urine was estimated using quantitative ELISA kit (Quibiotech, China). Microscopic examination of urine was performed in nonacidified samples. Blood urea nitrogen (BUN), creatinine, total protein (TP), and albumin were estimated in serum using commercial kits.

#### Estimation of Oxidative/Nitrosative Stress Indices
Lipid peroxidases (LPO) level in 10% red blood cell (RBC) hemolysate was estimated spectrophotometrically following the method of Placer *et al.*[13] Nitric oxide (NO) level of blood plasma was measured by nitrate reduction on copper-cadmium alloy (Cu–Cd alloy), followed by color development with Griess reagent (0.1% naphthalene diamine dihydrochloride in 3 N hydrochloric acid and 1% sulfanilamide 1:1).[14] Superoxide dismutase (SOD) was measured in the supernatant of 10% RBC hemolysate following the method of Minami and Yoshikawa.[15] Each unit of SOD activity is defined as the quantity of enzyme that inhibits auto-oxidation of pyrogallol by 50% under suitable experimental conditions. Catalase (CAT) activity in 10% RBC hemolysate was estimated spectrophotometrically at wavelength of 240 nm by standard procedure,[16] and the values were expressed in units per milligram of hemoglobin. Glutathione (GSH) was estimated in packed RBC following di-thiobis-2-nitro benzoic acid method.[17]

#### Urine Microscopy and Crystal Deposition Study
First 3 h morning urine sample was taken for microscopic examination for detection and counting of crystals. Crystal deposition score in urine was estimated using the semi-quantitative scoring methods described previously.[9] Briefly, ten randomly selected microscopic fields were examined under ×10 magnification, and crystal deposits were graded according to the scale: 0 = <1 crystal, 1 = ≤1–10, 2 = ≤11–30, 3 = ≤31–50, 4 = ≤51–75, and 5 = >75 crystals.[9]
Histopathology and Kidney Calcium Content
The kidneys were rinsed in ice-cold physiological saline and weighed. The left kidney was fixed in 10% neutral buffered formalin. Sectioned at 5 μm thickness and stained with hematoxylin and eosin for histopathological examination. The right kidney was taken for estimation of tissue calcium content by atomic absorption spectrophotometer.

Statistical Analysis
The data were expressed as mean ± standard error of mean and median effective concentration (EC₅₀ value) with 95% confidence interval. All statistical comparisons between the groups were made by t-test (comparison between two groups) or ANOVA with post hoc Dunnett's test (SPSS software, version 24, IBM corporation, USA). P < 0.05 is considered statistically significant.

Results
Serum and Urine Biochemistry
All the day 0 parameters of urine and serum biochemistry recorded just before the start of experiment (baseline parameters) were similar among all the groups. Tables 1 and 2 summarise the change in serum and urine biochemistry among different groups at different interval of study, respectively. EG treatment cause significant impairment of renal functions as evident from significantly (P < 0.01) raised BUN, creatinine and TP concentration in all grouped rats except healthy control on day 14, and lithiatic group on day 28. On contrary, treatment with MUSA significantly (P < 0.01) reduced the concentration of BUN, creatinine and TP in almost dose-dependent manner. There was almost 50% reduction of above parameters in rats treated with test extract at the dose rate of 250 mg/kg body weight as compared to untreated lithiatic group [Table 1]. The volume of urine, urinary oxalate and calcium concentration increased significantly (P < 0.01) on day 14 in all groups as compared to healthy control rats [Table 2]. The values were further increased on day 28 in lithiatic group (Group B) but treatment with MUSA significantly reduced the polyuria, hypercalciuria and oxaluria in dose-dependent manner. On contrary, the pH of urine decreased significantly (P < 0.01) after stone-inducing treatment, but increased significantly after treatment with MUSA and the value was statistically similar to standard treatment.

Table 1: Effect of aqueous ethanol extract of M. paradisiaca pseudostem and standard drug on serum biochemical parameters

| Parameters       | Day | Untreated control (Group I) | Lithiatic control (Group II) | 100 mg/kg (Group III) | 150 mg/kg (Group IV) | 200 mg/kg (Group V) | 250 mg/kg (Group VI) | 300 mg/kg (Group VII) | Cystine 100 mg/kg (Group VIII) |
|------------------|-----|------------------------------|-----------------------------|-----------------------|----------------------|---------------------|------------------------|------------------------|-------------------------------|
| BUN (mg/dl)      | 14  | 13.85±0.50                   | 25.37±0.89**                | 24.32±0.74**          | 24.85±0.78**         | 25.68±0.70**        | 24.07±0.54**           | 23.96±0.63**            | 28.72±0.52**                  |
|                  | 28  | 14.60±0.58                   | 41.22±0.95**                | 30.19±0.88**          | 28.39±0.53**         | 24.18±0.38**        | 19.42±0.49**           | 19.99±0.50**            | 18.97±0.59**                  |
| Creatinine (mg/dl) | 14  | 0.59±0.03                    | 1.21±0.07**                 | 1.15±0.08**           | 1.18±0.07**          | 1.02±0.05**         | 1.26±0.07**            | 1.16±0.08**             | 0.99±0.08**                   |
|                  | 28  | 0.61±0.02                    | 1.92±0.07**                 | 1.64±0.06**           | 1.62±0.06**          | 1.35±0.04**         | 0.91±0.03**            | 0.93±0.04**             | 0.82±0.04**                   |
| TP (mg/dl)       | 14  | 5.33±0.20                    | 7.15±0.34**                 | 6.92±0.07**           | 6.90±0.23**          | 6.95±0.48**         | 7.12±0.19**            | 7.08±0.28**             | 6.98±0.21**                   |
|                  | 28  | 4.91±0.13                    | 8.27±0.16**                 | 7.20±0.20**           | 6.76±0.16**          | 5.57±0.02**         | 4.90±0.24**            | 5.04±0.28**             | 5.31±0.19**                   |
| Albumin (mg/dl)  | 14  | 3.65±0.13                    | 4.08±0.21                   | 4.01±0.09             | 3.51±0.14            | 4.18±0.15           | 4.02±0.16              | 3.96±0.28               | 3.79±0.20                     |
|                  | 28  | 3.45±0.08                    | 4.21±0.37                   | 4.02±0.06             | 3.92±0.15            | 3.86±0.09           | 3.51±0.10              | 3.54±0.14               | 3.41±0.13                     |

Values mean±SE significantly differs *P<0.05 and **P<0.01 versus untreated healthy control (Group I). *P<0.05 and **P<0.01 versus lithiatic group (Group II).

Alteration of Oxidative/Nitrosative Indices
The mean LPO and NO activity increased significantly (P < 0.01) on day 14 in all the rats received EG treatment and further increased (P < 0.01) on day 28 in lithiatic control rats as compared to untreated rats [Table 3]. However, MUSA treatment significantly reduced the above oxidative/nitrosative stress in dose-dependant manner, and the mean values were statistically similar to rats treated with standard drug, cystone. On contrary, the antioxidant enzymes GSH, SOD, and CAT were decreased on day 14 but increased significantly after treatment with MUSA, and the values were similar to standard treatment and untreated control [Table 3].

Urine Microscopy and Crystal Deposition Score
The microscopic examination of urine revealed absence of any crystal in healthy rats [Figure 1a]. Significantly increased amount of crystals were present in the urine sample of untreated lithiatic rats on day 28 [Figure 1b], however, treatment with MUSA reduced the crystalluria in dose-dependent manner [Figure 1]. The crystal deposition score of urine revealed a significant increased score of crystal deposit in lithiatic group [Figure 2]. Treatment with test extract at dosage 250 mg/kg significantly reduced the crystal count as well as crystal size and it was almost 50% of the mean crystal deposit score of lithiatic group.

Histopathology and Kidney Calcium Content
The microscopic section of kidney revealed the presence of crystals most commonly in renal tubules in cortex region and less commonly in medulla, mononuclear cell infiltration, nerosis, tubular degeneration, casts, and fibrin deposits in lithiatic group [Figure 3b]. Rats received MUSA at dosage 100 mg/kg [Figure 3c] and 150 mg/kg [Figure 3d] also possesses crystal in the kidney section along with mono nuclear cell infiltration and hemorrhage but severity was lesser extent to urolithic group. There was complete absence of crystal in the kidney section in rats received MUSA at dosage 200 mg/kg [Figure 3e], 250 mg/kg [Figure 3f], 300 mg/kg [Figure 3g], and cystone 100 mg/kg body weight [Figure 3h] but hemorrhages and infiltration of cells was found in some cases, however, the architecture of kidney is almost similar to healthy control rats [Figure 3a]. The tissue Ca²⁺ concentration was significantly increased in urolithic rats (P < 0.01) as compared to healthy control [Figure 4]. However, treatment with MUSA significantly reduces the tissue Ca²⁺ content.
Table 2: Effect of aqueous-ethanol extract of *M. paradisiaca* pseudostem and standard drug on 24 h urinary biochemical parameters

| Parameters   | Day | Untreated control (Group I) | Lithiatic control (Group II) | 100 mg/kg (Group III) | 150 mg/kg (Group IV) | 200 mg/kg (Group V) | 250 mg/kg (Group VI) | 300 mg/kg (Group VII) | Cystone (100 mg/kg) (Group VIII) |
|--------------|-----|-----------------------------|-------------------------------|-----------------------|----------------------|---------------------|---------------------|------------------------|----------------------------------|
| Volume (ml)  | 14  | 7.20±0.28                   | 10.52±0.38**                 | 11.02±0.72**          | 10.72±0.71**        | 11.50±0.64**        | 10.60±1.04**         | 11.30±0.46**            | 13.70±0.45**                    |
|              | 28  | 7.35±0.48                   | 13.50±0.79**                 | 11.00±0.54**          | 10.37±0.48**        | 9.50±0.46**         | 8.50±0.46**          | 8.51±0.34**             | 8.35±0.41**                    |
| pH           | 14  | 7.27±0.18                   | 6.10±0.13**                  | 6.30±0.42**           | 6.21±0.27**         | 5.92±0.22**         | 6.12±0.10**          | 6.01±0.29**             | 5.73±0.07**                    |
|              | 28  | 6.79±0.26                   | 5.72±0.35*                   | 5.87±0.18*            | 5.95±0.13*          | 6.12±0.12*          | 6.29±0.19*           | 6.22±0.23*              | 6.28±0.05*                     |
| Ox (mg)      | 14  | 0.69±0.12                   | 2.37±0.20**                  | 2.58±0.17**           | 1.97±0.42**         | 2.13±0.26**         | 2.56±0.15**          | 2.31±0.19**             | 1.80±0.13**                    |
|              | 28  | 0.78±0.10                   | 2.83±0.22**                  | 2.05±0.18**           | 1.76±0.21**         | 1.55±0.37**         | 1.28±0.11**          | 1.29±0.16**             | 1.15±0.05**                    |
| Ca (mg)      | 14  | 5.19±0.15                   | 7.28±0.16**                  | 7.55±0.19**           | 6.87±0.20**         | 7.85±0.18**         | 8.19±0.13**          | 7.74±0.16**             | 7.43±0.12**                    |
|              | 28  | 4.53±0.11                   | 9.54±0.26**                  | 7.75±0.23**           | 6.57±0.18**         | 6.13±0.18**         | 5.14±0.12**          | 5.17±0.18**             | 5.02±0.09**                    |
| P (mg)       | 14  | 4.26±0.17                   | 4.54±0.21                    | 4.59±0.26             | 4.61±0.19           | 4.49±0.23           | 5.11±0.26            | 4.81±0.19               | 4.37±0.19                       |
|              | 28  | 3.01±0.23                   | 3.31±0.20                    | 3.04±0.11             | 3.21±0.20           | 3.29±0.17           | 2.81±0.14            | 2.87±0.16               | 2.78±0.18                       |

Values mean±SE significantly differs *P<0.05 and **P<0.01 versus untreated healthy control (Group I), *P<0.05 and **P<0.01 versus lithiatic group (Group II). SE=Standard error, Ca=Calcium, Ox=Oxalate, P=Phosphorus, MUSA=Aqueous-ethanol extract of *M. paradisiaca* pseudostem

Figure 1: Effect of *Musa paradisiaca* pseudostem and standard treatment on microscopic picture of urine in rats (*×100*). (a) Untreated healthy control, (b) lithiatic control, (c) rat treated with *Musa paradisiaca* pseudostem at 100 mg/kg, (d) rat treated with *Musa paradisiaca* pseudostem at 150 mg/kg, (e) rat treated with *Musa paradisiaca* pseudostem at 200 mg/kg, (f) rat treated with *Musa paradisiaca* pseudostem at 250 mg/kg, (g) rat treated with *Musa paradisiaca* pseudostem at 300 mg/kg, (h) rat treated with cystine at 100 mg/kg body weight

Discussion

*M. paradisiaca* is a commonly available plant in India. The pseudostem contributes approximately 12.67% of plant biomass and traditionally used for the treatment of diabetes, diarrhea, peptic ulcer, inflammation, rheumatism, hypertension, burn, and wound. It contains rich source of bioactive compounds, and the aqueous juice of pseudostem had been used traditionally by the rural people of South India against nephritis, uremia, and urolithiasis. Although substantial researches had been conducted in MUSA, its antiurolithiatic property is yet to be scientifically validated. Hence, the study was designed to evaluate the antiurolithiatic and renoprotective efficacy of MUSA in EG-induced rat model.

CaOx stone formation is a multifactorial process involving various etiological factors. In the present study, male rats were selected because the presence of testosterone in male promotes stone formation by suppressing osteopontin expression in the kidney and increasing urinary oxalate excretion.

Estrogen appears to inhibit stone formation by increasing osteopontin expression in the kidney and decreasing urinary oxalate excretion. Hyperoxaluric rat model is the most potent experimental model for preclinical evaluation of antiurolithiatic efficacy of medicinal herbs because the physiological process mimics the etiology of kidney stone formation in human and animal. The hepatic enzymes metabolize EG to oxalic acid by glyoxalate mechanism, which combine with calcium ion in the renal tubular epithelium to form CaOx crystals and hyperoxaluria. The impairment of renal function was also clearly indicated by elevation of serum creatinine, BUN, TP in lithiatic group as compared to normal, which has been restored by MUSA treatment, and the values were statistically similar to standard cystine treatment. Maximum reduction of the renal impairment was recorded in the rats administered with MUSA at dosages 250 mg/kg body weight. Similar finding was also reported by taking various medicinal herbs such as *Ipomoea eriocarpa*, *Asparagus racemosus*, *Hordeum vulgare*, *Alcea rosea*, and Gokshuradi polyherbal Ayurvedic formulation.
### Table 3: Effect of aqueous-ethanol extract of M. paradisiaca pseudostem and standard drug on oxidative/nitrosative stress indices

| Parameters | Day | Untreated control (Group I) | Lithiatic control (Group II) | 100 mg/kg (Group III) | 150 mg/kg (Group IV) | 200 mg/kg (Group V) | 250 mg/kg (Group VI) | 300 mg/kg (Group VII) | Cystone (Group VIII) |
|------------|-----|-----------------------------|-----------------------------|----------------------|---------------------|---------------------|---------------------|---------------------|---------------------|
| LPO (nmol) | 14  | 1.778±0.291                 | 3.822±0.336**               | 4.236±0.236**        | 4.236±0.236**       | 3.742±0.109**       | 3.984±0.464**       | 3.742±0.109**       | 4.302±0.069**       |
| MDA/mg Hb  | 28  | 2.546±0.299                 | 10.467±0.692**              | 7.938±0.216**        | 5.365±0.274**       | 4.643±0.176**       | 2.840±0.234**       | 2.228±0.179**       | 2.227±0.160**       |
| SOD (U/mg Hb) | 14  | 0.819±0.034                 | 0.321±0.012**               | 0.608±0.043**        | 0.525±0.052**       | 0.507±0.071**       | 0.608±0.030**       | 0.660±0.042**       | 0.641±0.051**       |
| CAT (U/mg Hb) | 28  | 0.101±0.049                 | 0.397±0.020**               | 0.557±0.024**        | 0.571±0.044**       | 0.678±0.037**       | 0.840±0.052**       | 0.899±0.042**       | 0.944±0.079**       |
| GSH (mmol/mg Hb) | 14  | 0.252±0.023                 | 0.311±0.036*                | 0.335±0.012*         | 0.408±0.021*        | 0.421±0.061*        | 0.339±0.028*        | 0.327±0.030*        | 0.309±0.016*        |
| NO (µmol/ml) | 14  | 0.161±0.038                 | 0.247±0.013**               | 0.298±0.021**        | 0.440±0.035*        | 0.495±0.048*        | 0.522±0.027*        | 0.541±0.043*        | 0.534±0.024*        |
| 28         | 0.327±0.022                 | 0.243±0.009**               | 0.264±0.009*           | 0.314±0.009**       | 0.308±0.017*        | 0.376±0.032*        | 0.305±0.014*        | 0.301±0.007*        | 0.329±0.012*        |
| 28         | 0.397±0.017                 | 0.308±0.022                 | 0.243±0.013**          | 0.214±0.013**       | 0.317±0.013**       | 0.327±0.014*        | 0.334±0.009*        | 0.359±0.024*        | 0.323±0.012*        |
| NO (µmol/ml) | 14  | 0.320±0.040                 | 5.445±1.684**               | 5.775±0.188**        | 6.105±0.123**       | 6.278±0.248**       | 5.911±0.224**       | 5.701±0.185**       | 5.837±0.360**       |
| 28         | 2.743±0.054                 | 8.285±0.201**               | 6.808±0.101**          | 5.716±0.178**       | 4.103±0.216**       | 3.372±0.134**       | 3.619±0.119**       | 2.998±0.078**       |

Values mean±SE significantly differ *P<0.05 and **P<0.01 versus untreated healthy control (Group I), *P<0.05 and **P<0.01 versus lithiatic group (Group II). LPO=Lipid peroxidases, MDA=Malondialdehyde, SOD=Superoxide dismutase, CAT=Catalase, GSH=Glutathione, NO=Nitric oxide, MUSA=Aqueous-ethanol extract of M. paradisiaca pseudostem.
Panigrahi, et al.: Antiurolithiatic efficacy of Musa paradisiaca pseudostem

Figure 3: Histology of representative kidney section stained with H and E stain (×100). (a) Untreated healthy control, (b) lithiatic control, (c) rat treated with Musa paradisiaca pseudostem at 100 mg/kg, (d) rat treated with Musa paradisiaca pseudostem at 150 mg/kg, (e) rat treated with Musa paradisiaca pseudostem at 200 mg/kg, (f) rat treated with Musa paradisiaca pseudostem at 250 mg/kg, (g) rat treated with Musa paradisiaca pseudostem at 300 mg/kg, (h) cystone at 100 mg/kg body weight

Figure 4: Renal tissue calcium content. Data are expressed as Mean±SEM. *P<0.05, **P<0.01 versus untreated healthy control (Group A). (A) Untreated healthy control, (B) lithiatic control, (C) rat treated with Musa paradisiaca pseudostem at 100 mg/kg, (D) rat treated with Musa paradisiaca pseudostem at 150 mg/kg, (E) rat treated with Musa paradisiaca pseudostem at 200 mg/kg, (F) rat treated with Musa paradisiaca pseudostem at 250 mg/kg, (G) rat treated with Musa paradisiaca pseudostem at 300 mg/kg, (H) rat treated with cystone at 100 mg/kg body weight

The microscopic examination of renal tissue showed intratubular and interstitial CaOx crystal deposits, mononuclear cell infiltration, dilated renal tubule, etc., in untreated rats, consistent with finding of others. The presence of polycrystalline, rosette-like arranged CaOx crystals is evidence of adhesion and retention of particles within the renal tubules. In contrast, treatment with MUSA significantly reduced the CaOx crystal deposit evident by histology of kidney and microscopy of urine.

Conclusions

The present findings demonstrate the efficacy of MUSA in EG-induced urolithiasis, which might be mediated through inhibiting various pathways involved in renal CaOx formation, antioxidant effect, and potential to inhibit biochemical parameters involving in impairment of renal function. Therefore, future studies may be established to quantify the active components in the extracts and also to evaluate its efficacy and safety for clinical use.

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Conflicts of Interest

There are no conflicts of interest.

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