Correlative Analysis Between Severity of Urolithiasis and Laboratory Parameters and Its Implication in Evaluation of the Probable Risk Profile

Rajeev T.P\textsuperscript{a}, Yashasvi Singh\textsuperscript{a, b}, Sasanka Kumar Barua\textsuperscript{a}, Debanga Sarma\textsuperscript{a}

Abstract

Background: Urolithiasis presents serious hazard which significantly elevates the cost of national health expenditure in almost every part of both the hemispheres. There is high risk of hospitalization with loss of valuable human resource and decreased productivity along with it. Risk factors still evade the exact etiology and search for optimal serum panel is still in its infancy. Urolithiasis incidence has gradually increased in last 3 decades which suggests that some constant metabolic and urinary parameters are implicated in the risk of occurrence of urinary stone. The present study is intended to identify a panel of serum parameters, urinary parameters, radiological characteristics and correlating it with the clinical severity of stone disease.

Methods: The present study was conducted at the Department of Urology at GMCH Guwahati. The authors retrospectively analyzed 151 patients undergoing stone surgery from a period of January 2016 to August 2017. Data comprised of all serum and urinary examinations done 1 week preoperatively and radiological scans within 1 month before surgery. Spearman test was used to determine correlation and analysis of variance (ANOVA) was applied for comparison between more than two categories.

Results: Stone multiplicity was positively correlated with upper tract stone sides ($r = 0.530$, $P < 0.01$), large stone volume ($r = 0.172$, $P < 0.02$), stone recurrence, urinary infection and urine protein. Upper tract stone sides number was positively correlated with upper tract obstruction sides ($r = 0.542$, $P < 0.03$), large stone volume ($r = -0.321$, $P < 0.01$). Upper tract obstruction sides number was positively correlated with large stone volume ($r = -0.848$, $P < 0.01$).

Conclusions: Results demonstrated that urinary tract obstruction and total stone volume significantly correlated with abnormal serum panel, urinary profile and were harbinger of complex stone pattern.

Keywords: Hypertension; CKD; Stone multiplicity; Upper tract obstruction

Introduction

Urolithiasis is an omnipresent pandemic disorder affecting large patient population worldwide and especially in South Asia. About 10% of people will experience nephrolithiasis in their lifetime and about 50-70% of those will have recurrences [1, 2]. Urolithiasis prevalence has been on an upswing in both sexes and in certain areas of the Indian subcontinent like NE India; the lifetime hazard appears to be even higher. A lot of capital has been used in the overall management of urinary stones worldwide but its forestalling has been a spurned field. A recent study based upon the National Health and Nutrition Examination Survey (NHANES) estimated that 19% of men and 9% of women will be diagnosed with a kidney stone by the age of 70 years [3]. Recurrence rates of renal stone are approximately 10% per year, 50% over a period of 5 - 10 years and 75% over 20 years period. The incidence rate of nephrolithiasis varies with geographical region of an individual country. Nearly 2 million people in India are affected with urolithiasis every year and many parts of the country have names denoted as a stone belt that is, Gujarat, Maharashtra, Punjab, Rajasthan, Delhi, Haryana and part of states on Northeast side[4]. There are a number of epidemiological arenas consisting of anatomic, metabolic, dietary and urinary factors that incline to the development of urolithiasis. The frequency with which these different risk factors occur in patients with recurrent stone disease and the role of genetic susceptibility are reviewed at the end of this topic. Upper urinary tract stones may lead to hydronephrosis and renal compromise. Nonetheless, hypertension, diabetes mellitus and chronic kidney disease (CKD) were proved to be correlated with nephrolithiasis. Many authors agree and it is our firm belief that stones in patients with CKD should be cleared [5]. However, the risk factor that may influence the calculus kidney damage is still unclear and needs further evaluation.

Nearly 75% of patients with nephrolithiasis form calcium stones mostly comprising of calcium oxalate or calcium phosphate to some extent, while uric acid stones make up less than 10 percent of all stones [6]. Renal stones consist of a variety of
crystalline and non-crystalline materials, knowledge of which influences clinical decision. Analysis of 24 h urine composition provides data on the possible contents of a stone but does not perfectly predict stone type. The frequent crystalline materials found in kidney stones are calcium oxalate, calcium phosphate, uric acid, and struvite though mutual coexistence for them in most cases has become an undeniable fact. Non-crystalline materials found in stones include blood and protein. The aim of this study is to establish a correlation between different calculus clinical patterns and laboratory parameters in patients with urolithiasis, in order to further explore the mechanism of these parameters changes after stone formation and also to find out some risk factors of kidney damage after urolithiasis occurs.

Methods

This retrospective study included 151 patients with urolithiasis who underwent surgery for renal, ureteric and vesical stones at the Department of Urology and Renal Transplantation between January 2016 to August 2017 and were in follow-up for at least 6 months in the Urology OPD. Diagnosis of stone diseases and their clinical implication were assessed by USG W/A, intravenous urogram (IVU), Non-contrast CT kidney-ureter-bladder scanning (NCCT KUB) and contrast-enhanced computed tomography (CECT (W/A + pelvis + urography)) when required accordingly.

“Stone multiplicity” was construed as multiple urinary tract stones present, disregarding their locations. “Upper urinary tract obstruction” was characteristically present when ureteric calculi or hydronephrosis caused by renal calculi were diagnosed. “Large stone bulk” was defined as stone’s size greater than 2 cm in diameter. Data on patient’s age at presentation, gender, co-morbidities, past medical and surgical history were recorded. Diagnosis of uncontrolled hypertension and diabetes mellitus was based on medical history and current use of medication. Height and weight were measured and body mass index was calculated.

All hematological and biochemical test results were obtained preoperatively, within 1 week before primary surgery. Authors evaluated the serum levels of urea, creatinine, sodium, potassium, magnesium, calcium, phosphorus, alkaline phosphate, uric acid and albumin. Patients were asked to collect morning urine samples which were examined for pH, specific gravity, protein and bacteria. Patients with borderline renal function and abnormal renal and liver function tests were excluded. Diagnosis of chronic obstructive pulmonary disease was based on medical history and current use of medication.

Table 1. Distribution Pattern of Categorical and Clinical Parameters With Overall Distribution

| Age (years) | Mean ± SD | 55.15 ± 14.26 |
|-------------|-----------|---------------|
| Gender      | Male/female | 87/64        |
| Recurrence  | Yes/no     | 50/101       |
| BMI         | Mean ± SD  | 25.21 ± 3.26 |
| HTN         | Present/absent | 48/103     |
| DM          | Present/absent | 29/122     |
| CKD         | Present/absent | 44/107      |
| DM + CKD    | Present/absent | 12/139     |
| Stone multiplicity | Present/absent | 85/66 |
| Upper tract stone site | 0/1/2 | 32/62/57 |
| Upper tract obstruction side | 0/1/2 | 16/65/67 |
| Large stone bulk | Present/absent | 41/110 |
| Ipsilateral kidney damage | Mild/moderate/severe | 48/80/23 |

Taking into account all the four respective groups but irrespective of gender group (P = 0.034). Median age was maximal for the ureteric stone group (Table 1). While examining the biochemical parameters it was seen that median value of serum urea was maximum in ureteric stone group but the overall distribution did not reach significant level (P = 0.950). Serum creatinine was analyzed in all the four groups as a constant variable both pre and postoperatively, and was maximal in the vesical calculus group when compared with other groups (P = 0.029) (Table 2). Protein metabolism that was specifically mapped upon in view of the dietary habits and serum albumin level was found to be significantly associated in all the four groups (P = 0.012). Though serum calcium concentration holds an important position in urolithiasis development, our analysis could not get a significant association except in obstruction (P = 0.001) group and large stone bulk group (P = 0.002) for the same. Serum sodium (P = 0.032) and magnesium (P = 0.045) both were significantly associated with stone formation among all the four groups. Among these parameters, male and the aged were significantly associated with bladder calculi. Renal calculi were associated with recurrence, UTI and higher urinary protein, whereas ureteral calculi were associated with higher serum sodium level. The combination of renal and ureteric calculi was associated with higher serum creatinine. One third of the patient in the entire cohort had recurrence in the follow-up period (6 months) with maximum number of cases in the kidney stone group with significant association (Chi square value = 4.706, P = 0.019) and correlation (r = 3.08, P = 0.042). Hypertension was as significantly associated (Chi square value = 8.528, P = 0.001) and correlated (r = 5.67, P = 0.036) as recurrence in the four stone categories and was present in 31.79% cases. CKD was present in 29.14% cases when compared with other groups (P = 0.042). Taking into account all the four groups but irrespective of gender group (P = 0.034). Median age was maximal for the ureteric stone group (Table 1). While examining the biochemical parameters it was seen that median value of serum urea was maximum in ureteric stone group but the overall distribution did not reach significant level (P = 0.950). Serum creatinine was analyzed in all the four groups as a constant variable both pre and postoperatively, and was maximal in the vesical calculus group when compared with other groups (P = 0.029) (Table 2). Protein metabolism that was specifically mapped upon in view of the dietary habits and serum albumin level was found to be significantly associated in all the four groups (P = 0.012). Though serum calcium concentration holds an important position in urolithiasis development, our analysis could not get a significant association except in obstruction (P = 0.001) group and large stone bulk group (P = 0.002) for the same.

Results

The median age for the entire cohort was 55.15 ± 14.26 years...
value = 8.348, P = 0.039) and correlated (r = -4.09, P = 0.027) with the stone groups. Multiple stones were managed in the same sitting if present on the same side and there was a gap of 3 to 6 months when present on the opposite side.

Upper tract stone sites incorporated 41.06% cases on a single site and 37.75% cases on two sites which was significantly associated (Chi square value = 14.743, P = 0.033) and correlated (r = -4.09, P = 0.027) with the same. Hydronephrosis of the upper tract resulting from obstruction occurred in 43.04% on one side and 44.37% on both the sides and it was significantly associated (Chi square value = 14.81, P = 0.049) and correlated (r = 2.69, P = 0.022) with the four stone groups. Large stone bulk was seen in 27.15% and was not significantly associated and correlated with the same compelling on the importance of delay in presentation to the OPD. The time lag in presentation augmented the ipsilateral kidney damage which towered at 52.98% for moderate damage and 15.23% for severe damage and was significantly associated (Chi square value = 14.27, P = 0.048) and correlated (r = 3.10, P = 0.038) with the groups in consideration.

The correlation determination for stone multiplicity with other clinical variables revealed that stone multiplicity was positively correlated with upper tract stone sides (r = 0.730, P < 0.01), large stone volume (r = 0.272, P < 0.02), stone recurrence (r = 0.632, P = 0.001), CKD (r = 0.062, P = 0.001), urine albumin (r = 2.63, P = 0.012) and urine infection (r = 1.80, P = 0.001). Upper tract stone sides number was positively correlated with age (r = 2.32, P = 0.002), upper tract obstruction sides (r = 0.742, P < 0.01), serum creatinine (r = 4.67, P = 0.002), serum sodium, and urinary infection, large stone volume (r = -0.321, P < 0.01), urine specific gravity (r = 1.87, P = 0.04) and urine infection (r = 8.06, P = 0.003). Upper tract stone obstruction sides number was positively correlated with age (r = 5.56, P = 0.033), BMI (r = 9.87, P = 0.044), serum potassium (r = 6.44, P = 0.021), serum creatinine (r = 1.56, P = 0.039), serum calcium (r = 5.06, P = 0.001), serum phosphate (r = 6.86, P = 0.042), urine albumin (r = 0.443, P = 0.002) and urinary infection (r = 0.443, P = 0.01), whereas negatively correlated with large stone volume (r = -0.748, P < 0.01) and serum magnesium (r = -4.67, P = 0.038). Large stone volume was positively correlated with age (r = 0.879, P = 0.033), urine albumin (r = 3.65, P = 0.011), serum phosphate (r = 4.01, P = 0.001), serum calcium (r = 0.801, P = 0.002) and CKD (r = 0.441, P = 0.023). Ipsilateral kidney damage was positively correlated with age, recurrence, CKD, hypertension, serum urea and serum creatinine, serum albumin (Table 4).

Discussion

In our patient population, bilateral upper tract calculi are correlated with higher serum sodium. Increased daily sodium intake tends to increase serum and urinary sodium in a significant manner which not only increases the net calcium excretion in the urine, but also increases urinary pH and decreases citrate excretion at the same time. Keeping the aforementioned aspect in consideration, the urinary concentration of calcium phosphate and monosodium urate also increased significantly. Thus, a high sodium intake was directly responsible for the crystalization of calcium salts in urine. Moreover, in our study, upper urinary tract obstruction side number was correlated with higher serum potassium, calcium and phosphate level, as well as lower magnesium level.

Calcium urolithiasis has hypercalcemia and hypercalciuria both as compelling and estimable factors requiring urgent and immediate action. Hypercalcemia exists when serum calcium level increases, together with measuring and calculation of physiologically active calcium when there is difference in the pH of blood or serum albumin [7]. In serum, calcium is either bound to albumin or is found in its free form (ionized).
It is the ionized calcium which is of concern because ionized calcium is the physiologically active form of calcium as calcium bound to albumin is inactive. Standard lab tests usually measure the total calcium. When albumin is low, patients have lower total calcium on lab tests. Hence, patients with lower serum albumin tended to have higher serum active calcium level. When the serum is alkaline, hydrogen ions bound to negatively charged albumin are released. These binding spots on albumin open for ionized calcium to bind, which reduces the amount of physiologically active calcium. Hypercalcemia and hypercalciuria increase the incidence of calcium stones, by increasing the urinary saturation of calcium salts and by binding negative-

| Variable             | Chi square coefficient | P value | Pearson r coefficient | P value |
|----------------------|------------------------|---------|-----------------------|---------|
| Gender               | 7.28                   | 0.063   | -0.015                | 0.859   |
| Recurrence           | 4.706                  | 0.019   | 3.08                  | 0.042   |
| HTN                  | 8.528                  | 0.001   | 5.67                  | 0.036   |
| DM II                | 5.635                  | 0.302   | 0.01                  | 0.907   |
| CKD                  | 8.66                   | 0.043   | 7.42                  | 0.027   |
| DM + CKD             | 2.92                   | 0.404   | -0.002                | 0.979   |
| Multiple stones      | 8.348                  | 0.039   | -4.09                 | 0.027   |
| Upper tract stone side| 14.743                 | 0.033   | -0.858                | 0.041   |
| Upper tract obstruction side| 14.81              | 0.049   | 0.269                 | 0.022   |
| Large stone bulk     | 2.072                  | 0.558   | 0.021                 | 0.798   |
| Ipsilateral kidney damage| 14.27                  | 0.048   | 3.1                   | 0.038   |

| Variable             | Multiplicity | Stone side | Ipsilateral kidney damage | Large stone bulk | Obstruction side |
|----------------------|--------------|------------|---------------------------|------------------|-----------------|
| Gender               | -0.87        | 0.081      | -0.7                      | 0.99             | 0.081           | 0.56            | 0.212           | 0.43            | -0.55           | 0.11            |
| Age                  | 0.65         | 0.239      | 2.32                      | 0.02             | 4.55            | 0.001           | 0.879           | 0.004           | 5.56            | 0.033           |
| Recurrence           | 0.632        | 0.001      | 8.06                      | 0.035            | 0.632           | 0.021           | 1.12            | 0.09            | 0.56            | 0.129           |
| BMI                  | 0.59         | 0.265      | 3.67                      | 0.86             | 0.89            | 0.1             | 0.974           | 0.134           | 9.87            | 0.044           |
| DM                   | 0.561        | 0.435      | 2.86                      | 0.77             | 0.661           | 0.101           | 0.45            | 0.172           | 0.36            | 0.067           |
| CKD                  | 0.062        | 0.001      | 3.44                      | 0.26             | 0.762           | 0.002           | 0.441           | 0.023           | 8.06            | 0.055           |
| HTN                  | 0.98         | 0.765      | 4.56                      | 0.14             | 0.98            | 0.001           | 1.02            | 0.56            | 0.67            | 0.113           |
| Serum Na             | 5.13         | 0.003      | 0.87                      | 0.87             | 0.113           | 0.81            | 0.167           | 0.87            | 0.86            | 0.088           |
| Serum K              | 5.92         | 0.96       | 4.36                      | 0.66             | 0.223           | 0.448           | 0.012           | 0.36            | 6.44            | 0.021           |
| Urea                 | 8.34         | 0.57       | 5.06                      | 0.24             | 0.458           | 0.01            | 0.183           | 0.26            | 0.97            | 0.954           |
| Creatinine           | 13.74        | 0.16       | 4.67                      | 0.002            | 0.886           | 0.001           | 0.393           | 0.47            | 1.56            | 0.039           |
| Serum ALP            | 14.81        | 0.24       | 1.86                      | 0.56             | 0.093           | 0.331           | 0.948           | 0.86            | 0.87            | 0.113           |
| Serum albumin        | 1.072        | 0.062      | 1.44                      | 0.97             | 0.088           | 0.001           | 0.11            | 0.44            | 9.36            | 0.121           |
| Serum Ca             | 14.27        | 0.85       | 3.97                      | 0.26             | 0.068           | 0.098           | 0.801           | 0.002           | 5.06            | 0.001           |
| Serum Mg             | 0.183        | 0.112      | 5.56                      | 0.09             | 0.29            | 0.1             | -0.631          | 0.072           | -4.67           | 0.038           |
| Serum PO4            | 0.293        | 0.18       | 0.56                      | 0.06             | 0.243           | 0.295           | 0.401           | 0.001           | 6.86            | 0.042           |
| Specific gravity     | 1.95         | 0.095      | 1.87                      | 0.04             | 0.416           | 0.233           | 0.198           | 0.98            | 0.44            | 0.098           |
| pH                   | 2.11         | 0.088      | 0.36                      | 0.081            | 0.665           | 0.376           | 0.1             | 0.11            | 0.775           | 0.1             |
| Urine infection      | 1.8          | 0.001      | 8.06                      | 0.003            | 0.885           | 0.431           | 0.442           | 0.689           | 0.443           | 0.01            |
| Urine albumin        | 2.63         | 0.012      | 0.445                     | 0.997            | 0.005           | 0.981           | 3.65            | 0.011           | 0.091           | 0.002           |
ly charged inhibitors of stone formation [8]. Increased level of
serum calcium should lead to analysis of both ionized calcium
and intact PTH with the aim to button in on patients who might
have undiagnosed hyperparathyroidism [9]. A study [10] stat-
ed that stone patients have a relative risk of hypercalcemia and
hypercalciuria nine to 18 times more than non-stone formers,
respectively. Nonetheless, by definition of hypercalcemia (se-
rum calcium > 2.75 mmol/L), only two patients (0.03%) in our
cohort were diagnosed as real hypercalcemia, which consistent
with the research of a prospective study [11]. In a large pro-
spective study, calcium intake was measured and high dietary
intake was inversely correlated with kidney stone risk (relative
risk: 0.56) [12]. In another large prospective cohort study of
men, the relative risk of stone formation for highest and low-
est quintiles of calcium intake was 0.69 [13]. Higher calcium
intake decreased the risk of stones in females by up to 28% in
a prospective cohort study [14]. Some studies indicated that
serum phosphate level was a significant risk factor of calcium
urolithiasis [15, 16]. The renal phosphate leak theory explains
calcium nephrolithiasis in recurrent stone cases by implicating
that the defect of renal tubules will prevent phosphate reab-
sorption [17]. The result is hyperphosphaturia, with low serum
phosphate level, causing an increase in the GIT absorption of
calcium, which results in an increased renal load of calcium re-
sulting in hypercalciuria. Nonetheless, the value of renal phos-
phate leak theory in the formation of calcium nephrolithiasis
is questionable. Few studies showed no consistent compelling
association of patient’s serum phosphate level with stone re-
currence [18].

When the urinary parameters of patients with idiopathic
hypercalciuria were equated to those with normocalciuria, pa-
tients with former were found to have increased urinary so-
dium and sodium intake [19]. In a randomized study compar-
ing low calcium and sodium diet and animal protein, the low
sodium and animal protein diet resulted in fewer stone recur-
rences [20]. Increased sodium diet was related to 61% increase
in nephrolithiasis risk in a large prospective study of women
[21]. In a randomized study of 210 patients with hypercalciuria
with calcium stones, a low sodium diet culminated in lower
urinary sodium, as well as lower urinary calcium and oxalate
excretion and resulted in normalization of urine calcium excre-
tion for one third of patients [22].

Negative correlation between upper urinary tract obstruc-
tion side number and serum magnesium was observed in our
series. Some authors indicated that magnesium can lower the
risk of stone formation by diverse mechanisms. Magnesium
chelates urinary oxalate, producing a soluble magnesium-ox-
alate complex than calcium oxalate and thus increases urinary
citrates level [23]. A low urinary magnesium level has been
seen in calcium stone formers with hypercalciuria. Clinically,
oral supplementation of magnesium citrate increases urinary
magnesium and citrate excretion. Magnesium supplement in
deficient patients proved to decrease the recurrence rate of uro-
lithiasis [24].

Serum potassium was observed to be positively correlated
with upper urinary tract obstruction side number. Upper uri-
nary tract obstruction invariably leads to hydrenephrosis and
renal insufficiency if ignored for long period of time. When
renal function deteriorates, the ability to effectively regulate
serum potassium via the Na+/K+-ATPase and multiple other
mechanisms exists, serum potassium will elevate continuously after it
reaches its limits.

Our analysis showed that older patients were correlated
with larger stone volume, higher risk of bilateral upper uri-
nary tract calculi and obstruction, and severe ipsilateral kid-
dney damage. Past studies have proved that older patients with
stone formation had more condition related to metabolic syn-
dromes than younger patients [25, 26]. The clubbing between
metabolic syndrome and nephrolithiasis has been established
by some past studies [27, 28]. One different scenario leading
to the aforementioned condition is the steady decline in renal
function that occurs with advanced age, as super-saturation
and nephrolithiasis have been attributed to renal tubular cell
damage [29]. Furthermore, older patients inclined to have
more peculiar presentations of urolithiasis, which cause delay
in the diagnosis and management [30]. This may explain why
bilateral upper urinary tract calculi and larger stone volume
were more frequent in older patients. In conclusion, older pa-
tients with urolithiasis usually have larger and more complex
stone disease. A random spot urine sample was used for urine
culture to establish organisms producing urease. The concomi-
tant finding of high urine pH (> 7.5) in few cases indicated that
the patient might have formed an infection stone (magnesium
ammonium phosphate + carbonate apatite). Low urine pH pre-
sents a risk for uric acid precipitation and subsequent stone
formation [31].

In our study, urinary tract infection (UTI) was positively
related with multiple stone, bilateral upper urinary tract
stone and obstruction, and larger stone volume. UTI has been
proved to be associated with the formation of stones, the most
common composition being struvite (magnesium ammonium
phosphate). Urease producing bacteria divides urea into am-
monia, resulting in an alkaline urinary pH with subsequent
struvite stone formation. Nevertheless, not all stones associated
with UTI are composed of struvite and not all are associated
with urea-splitting organisms [32, 33]. It is uncharted whether
non-struvite infected stones result from infection itself or be-
come secondary infected after formation. It has been observed
that pathogenic bacteria from stones could initiate renal in-
fammation leading to crystal aggregation and subsequent
nephrolithiasis. Moreover, the presence of bacteria could alter
the local microenvironment by metabolic activity which fur-
ther promotes lithogenesis [34, 35]. Rather than calculating the
total estimated GFR from serum creatinine, authors measured
the split GFR through Tc-99m DTPA renal scan which enabled
them to accurately evaluate the ipsilateral kidney damage level
caused by nephrolithiasis. Thus, authors were able to identify
the correlation between ipsilateral kidney damage, nephrolithi-
asis and various biochemical parameters.

In present study, older age, recurrence disease, hyperten-
sion (HTN) was positively correlated with the level of ipsi-
lateral kidney damage. There is a proven firm association be-
tween HTN and stone formation, as numerous studies have
pointed HTN as an independent predictor of nephrolithiasis
[36]. Few studies suggest that dysregulation in renal calcium
metabolism existed in patients with HTN, leading to increased
hypercalciuria [37]. Additionally vice versa, in patients with
nephrolithiasis, the incidence of HTN has been seen to be more than that of patients without nephrolithiasis [38]. It has been observed that uncontrolled hypertensive patients with nephrolithiasis have a greater risk for CKD as HTN is formidable associated with CKD. Recurrent stone formation history was proved to be significantly correlated with ipsilateral kidney damage. Amongst 171 patients with idiopathic calcium stones in a recent study [39], the 30 patients with reduced GFR (eGFR < 80 mL/min/1.73 m²) had significant incidence of recurrent disease with nearly one stone per patient per year.

In our study, negative correlation was observed between ipsilateral kidney damage and serum albumin level. Urine protein level was positively correlated with larger stone bulk and stone multiplicity. Although no correlation was observed between ipsilateral kidney damage and urine protein level, lower serum albumin level was frequently found in CKD patient mainly caused by proteinuria. It was observed in few studies that proteinuria was a surrogate outcome in CKD, with changes in proteinuria recommended as a surrogate for renal disease progression [40]. A study reported [1] atrophic kidney cortex (< 5 mm), proteinuria (> 300 mg/d), large stone bulk (> 1,500 mm²), pediatric age group and recurrent UTI, were predictors of poor renal outcome in upper urinary tract stone formation patients with CKD. This goes hand in hand with our observations. The authors identify several limitations of the current study. We did not categorize patients according to stone compositions because they were not regularly indentified in our cohort. The invaluable 24 h urine analysis for super-saturation profile, which includes urinary calcium, phosphate, citrate, oxalate, etc., was also not involved in our study.

Conclusions

Our analysis demonstrated upper urinary tract obstruction was more likely to correlate with abnormal electrolyte metabolism in terms of serum calcium, magnesium and phosphate. Older age and UTI were positively associated with larger stone volume and complex stone patterns. Ipsilateral kidney damage associated with upper tract obstruction is correlated with old age, recurrence, hypertension and low serum albumin.

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