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Authors
Lama, Daniel J
Safiullah, Shoaib
Yang, Albert
et al.

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THREE-DIMENSIONAL EVALUATION OF PERIRENAL FAT VOLUME IN PATIENTS WITH NEPHROLITHIASIS

Daniel J. Lama[1], **Shoaib Safiullah**[1], **Albert Yang**[2], **Tuyen Hoang**[1], **Zhamshid Okhunov**[1], **Jaime Landman**[1], and **Ralph V. Clayman, MD**[1] **[Professor of Urology]**

[1]Department of Urology, School of Medicine, University of California, Irvine.

[2]Department of Radiology, School of Medicine, University of California, Irvine

Abstract

INTRODUCTION: The concept of adipose tissue as an organ unto itself represents a new medical construct. Already differences in the volume of perirenal fat around a tumor-bearing-kidney have been described. We hypothesized that renal calculi may have similar impact on perirenal fat. Accordingly, we conducted a study utilizing 3D-imaging software to evaluate perirenal fat volume (PFV) in patients with nephrolithiasis.

METHODS: Among 40 patients with a history of unilateral nephrolithiasis who underwent percutaneous nephrolithotomy (PCNL) between 2005 and 2016, the following data were acquired: body mass index (BMI), past medical history, stone characteristics and composition (i.e. calcium oxalate (CO), calcium phosphate (CP), uric acid (UA), and struvite calculi). In addition, patients were stratified by dominant stone composition (≥ 50% fraction). Bilateral PFV measurements were obtained using the preoperative CT scan and specialized 3D-imaging software.

RESULTS: Themean PFV of stone-bearing kidneys was significantly greater than non-stone-bearing kidneys (397.3 and 323 cc, respectively; p=0.004), with the PFV difference in patients with CO-dominant stone-bearing kidneys reaching statistical significance (p=0.003). Subgroup analysis showed greater PFV surrounding the stone-bearing kidney regardless of gender (p=0.03), with male patients possessing significantly greater stone-bearing (p=0.01) and bilateral PFV (p=0.01) compared to females. No significant correlations were found between PFV and stone volume or stone density.

CONCLUSION: The PFV of calcium oxalate stone-bearing kidneys is significantly greater than non-stone-bearing kidneys.

Daniel J. Lama, 909-936-0516, djlama@uci.edu; Shoaib Safiullah, 847-293-8875, ssafiull@uci.edu; Albert Yang, 858-829-4087, alberty3@uci.edu; Tuyen Hoang , 949-824-3354, tuyrnh1@uci.edu; Zhamshid Okhunov, 714-456-7378, zokhunov@uci.edu; Jaime Landman, 714-456-3330, landmanj@uci.edu.

Data collection: Anastasia Cruz MD, Mitchell Daun MD, Adam Rudd MD.

Principal Investigator: Ralph V. Clayman MD, Professor of Urology, Department of Urology, Dean Emeritus, University of California, Irvine (UCI) School of Medicine, 333 City Blvd. West, Suite 2100, Orange, CA 92868, 714-456-6567, rclayman@uci.edu

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INTRODUCTION

Obesity has been studied extensively as a risk factor for nephrolithiasis.[1] In recent years, the prevalence of obesity and kidney stone disease in the United States has risen to 36.5% and 8.8%, respectively.[2,3] Although lifestyle and dietary abnormalities contribute to the formation of calculi, metabolic changes in urine composition in the setting of insulin resistance and the onset of diabetes mellitus have also been suggested as formidable lithogenic contributors.[4,5] Moreover, findings in the literature suggest that nephrolithiasis is a common finding among individuals with metabolic syndrome.[6,7]

The link between obesity and kidney stone disease was postulated in large observational studies that identified patients with nephrolithiasis as also often having a large body habitus. [8] Central adiposity provides a clinical index of adipose tissue surrounding the viscera and can predict the development of pathologic sequelae.[9,10]

The concept of visceral fat as an organ system unto itself has recently come into the medical consicouness. Indeed, variability in perirenal visceral adipose tissue was identified in kidneys affected by renal cell carcinoma was only first reported in 2012.[11] To our knowledge, to date, the volumetric differences in perirenal fat have not been evaluated in patients with nephrolithiasis.

Accordingly, we elected to study the possible relationship between perirenal fat volume (PFV) and the formation of renal calculi. Herein, we present findings from a retrospective evaluation of adipose tissue surrounding the kidney in patients with previously unoperated unilateral nephrolithiasis.

METHODS:

Retrospective Review

Retrospective data from 97 patients who underwent percutaneous nephrolithotomy (PCNL) by two experienced endourologists (RVC & JL) from 2010 to 2016 was accessed with Institutional Review Board approval. Patient information included gender, age, body mass index (BMI), and past medical history (e.g. type 1 or 2 diabetes mellitus (DM), history of hypertension (HTN), hyperlipidemia (HLD), and recurrent ipsilateral nephrolithiasis). Patients with a history of bilateral nephrolithiasis, renal cell carcinoma, or prior surgical therapy exclusive of ureteroscopy (i.e. extracorporeal shockwave lithotripsy, open or laparoscopic renal surgery, or percutaneous stone removal) were excluded. Stone data included stone diameter, stone volume (scalene ellipsoid formula[12]), location, Hounsfield units (HU), and chemical composition (calcium oxalate (CO), calcium phosphate (CP), uric acid (UA), and struvite calculi). Patients were also stratified according to the dominant stone composition (≥50% fraction) of the largest renal calculus following chemical analysis.

Measurement of Perirenal Fat Volume

PFV measurements of the stone-bearing and non-stone-bearing kidney were performed by radiologists under the supervision of senior faculty. The most recent computed tomography (CT) study prior to PCNL was used to measure PFV. Three-dimensional (3D) imaging

software (Vitrea® Advanced Visualization Software, Vital Images Inc., Minnetonka, MN) was used to measure the perirenal fat bilaterally according to the visible boundaries of Gerota's fascia on CT. The adrenal glands, kidneys, and renal hilar structures were subtracted from the surrounding perirenal fat to obtain the calculated PFV (cc), HU of the PFV, and a 3D rendering of the volumetric measurement (Figure 1).

Statistical Analysis

Continuous variables were summarized with descriptive statistics and categorical variables were summarized with frequency and percentage. Paired sample t-tests were performed for comparisons of unilateral and bilateral PFV. Pearsons correlation coefficient was computed for analysis of bilateral PFV and stone features. Statistical significance was defined as a p value of ≤ 0.05.

RESULTS

Forty patients met inclusion criteria. Twenty-four female patients and sixteen male patients had a BMI of 32 ± 9.4 kg/m^2 and 28.2 ± 6.7 kg/m^2, respectively. There was no significant difference in BMI with respect to gender (p=0.191). Sixteen patients (39%) had DM and 26 patients (65%) had a history of recurrent ipsilateral nephrolithiasis (Table 1). A total of 14 renal pelvic, 8 calyceal, and 18 staghorn calculi were treated with PCNL. No significant correlation was found between stone-bearing PFV and stone diameter, volume, or density.

In general, stone-bearing kidney PFV was significantly greater than non-stone-bearing kidney PFV (397.3 vs. 323 cc, respectively, p=0.004). Stone-bearing PFV was also greater for both male (p=0.04) and female (p=0.02) patients (Table 2). Compared to female patients, males had significantly more stone-bearing kidney PFV (545.4 vs. 271.4 cc, p=0.009), and total (i.e. bilateral) PFV (978.8 vs. 500.4 cc, p=0.01). A positive trend was observed between total PFV and age (r=0.29, p=0.146) (Figure 2), and total PFV and BMI (r=0.23, p=0.074) (Figure 3). Interestingly, the HU of stone-bearing kidney PFV was significantly greater than non-stone-bearing kidney PFV (−79.8 vs. −88.3 HU, respectively, p=0.023).

Differences in PFV with respect to patient co-morbidities are shown in Table 2. There was no difference detected between stone-bearing and non-stone-bearing PFV in diabetic patients, stone-bearing PFV in patients with DM compared to those without DM, among patients with diabetes vs. non-diabetic patients, or for BMI in diabetic vs. non-diabetic patients in the study. Of interest, a history of recurrent ipsilateral nephrolithiasis was associated with greater stone-bearing kidney PFV compared to patients with newly diagnosed renal calculi (445.2 vs. 298.7 cc, respectively, p=0.07).

After review of postoperative stone analyses, there were 19 patients with CO-dominant calculi (calcium oxalate monohydrate and dihydrate), 9 patients with CP-dominant calculi (carbonate apatite), 10 patients with UA-dominant calculi, and 2 patients with struvite calculi (Table 2). Among these groups, the difference between stone-bearing and non-stone-bearing kidney PFV for patients with CO-dominant calculi was significant (336.08 vs. 272.2 cc, p=0.003). In addition, the BMI of patients with CO-dominant calculi was significantly

less than patients with CP- or UA-dominant calculi (p=0.024). No other differences in BMI were found among stone composition groups.

DISCUSSION:

The implications of excess visceral adipose tissue have been studied in renal and systemic disease processes previously. Perirenal fat is identifiable as the visceral adipose tissue bound by Gerota's fascia in the retroperitoneum, yet the question as to the presence or absence of its specific involvement in renal disease is an area of limited study. Abdominal adiposity in general has been associated with the development of chronic kidney disease[13]; however studies have also shown an increased risk of nephropathy and hypertension with an accumulation of adipose tissue specifically in the renal sinus.[14,15] Importantly, the aforementioned findings also held true after adjusting for BMI and total visceral adipose tissue. Clinical observational studies and animal models have hypothesized that the sequelae of renal sinus lipomatosis are due to renal vein and lymphatic compression by excess adipose tissue, which can increase renal interstitial pressure, the reabsorption of sodium, and the risk of hypertension.[16,17] These findings suggest that perirenal fat accumulation may affect filtration and reabsorption processes in the kidney, possibly predisposing to lithogenesis. Considering that our PFV measurement calculated all adipose tissue bounded by Gerota's fascia, fat content in the renal sinus may have specific impact in our findings, generating inflammatory-based change in the kidney that may implicate the formation of renal calculi.

In addition to the mass effect hypothesis of adipose tissue surrounding the renal hilum, biochemical interactions between perirenal fat and the kidney are also thought to be possible contributors to renal disease. In a study by Ma et al., seven pigs fed with 16-weeks of a high fat/high carbohydrate diet demonstrated a significant increase in weight, systolic blood pressure, and PFV compared to controls.[18] *Ex vivo* samples of the renal arteries from experimental and control swine were then incubated with perirenal fat tissue from pigs fed with the aforementioned diet. Histologic examination of the renal vasculature demonstrated marked inflammatory cell infiltrate, an increase in reactive oxygen species, and elevated TNF-α levels, resulting in a damaged and dysfunctional renal endothelium. Hou et al. found that perirenal fat was associated with microalbuminuria in rats fed with a high-fat diet; indeed, further analysis revealed renal endothelial dysfunction and hormonal induction of inflammatory pathways which increased levels of glomerular reactive oxygen species.[19] Endothelial injury is common etiology of chronic kidney disease, and has also been suggested as a potential cause of urolithiasis.[20,21] The potential for similar pathologic hormonal signaling in human renal units has yet to be studied.

In patients with central obesity, excess adipose tissue is distributed around the visceral organs. Although BMI is often used as a clinical measure of morbidity, other metrics of visceral adipose tissue, such as waist-to-hip circumference, have been shown to be better indicators of all-cause mortality than BMI.[22] Similarly, we could find no statistically significant difference between BMI and bilateral PFV; indeed, greater PFV of the stone-bearing kidney occurred regardless of BMI (i.e. in both obese and non-obese patients). This

suggests that BMI does not predict PFV, nor is an appropriate measure of the risk of nephrolithiasis.

We noted differences in perirenal fat with respect to gender, with male patients bearing significantly more stone-bearing and total PFV compared to females. Similarly, Favre et al. measured PFV by manually outlining and summating abdominal CT slices of perirenal fat, which demonstrated significantly less adipose tissue bilaterally in women compared to men.[23] The discrepancy in PFV between males and females has been hypothesized to be a result of ectopic fat storage occurring to a greater extent in the subcutaneous tissue in women compared to storage in the viscera in men.[24] Furthermore, the increased storage of visceral adipose tissue in male kidneys may contribute to the possible gross and physiological effects of perirenal fat on the kidney and the greater risk of nephrolithiasis in men.[3]

An unexpected finding in our study was a significant difference in the HU of the perirenal fat itself between the stone-bearing and non-stone-bearing kidney PFV (−79.8 and −88.3 HU, respectively). Large depots of brown adipose tissue specifically, have been found surrounding the kidney[25] and also have demonstrated radiographic evidence of metabolic activity. A study by Bata et al. reported that activation of brown adipose tissue on positron emission tomography-computed tomography (PET-CT) also corresponded to a positive change in HU, suggesting that higher HU may characterize metabolically active brown adipose tissue.[26] Therefore, the positive difference in HU of the stone-bearing PFV may signify increased metabolic activity and suggests the possibility of a unique physiologic character of the perirenal fat in patients with unilateral nephrolithiasis. Whether this occurs prior to stone formation or after a calculus is already present (i.e. cause or effect) is a topic for further exploration.

In our study, patients specifically with CO-dominant calculi had significantly greater stone-bearing kidney PFV compared to the contralateral non-stone-bearing PFV. This is of interest in that CO-dominant calculi, as opposed to struvite or uric acid uroliths, form within the papillae and on Randall's plaques. In addition, calcium oxalate crystals are a proven source of inflammation within the tubules.[27] As such, one might hypothesize that the CO-dominant stone represents a pan-renal inflammatory condition that may impact the PFV.

There are few limitations in this study in addition to its retrospective nature. First, the number of patients without calcium oxalate stones was markedly small, limiting our ability to perform subcategory analysis and this warrants a further larger study of other stone types to assess for differences in PFV. Second, although our findings suggest that perirenal fat accumulation may play a role in the development of calcium-oxalate based nephrolithiasis, we did not evaluate if baseline differences in PFV exist between the right and left kidney in patients who have no history of stone disease or in diabetic patients who are presently without a history of renal calculi. Finally, the study includes a specific patient population of large stone formers undergoing a PCNL and these findings may not be generalized to all stone formers.

CONCLUSION:

Radiographic increases in PFV exist in patients with unilateral calcium oxalate nephrolithiasis. This difference was not observed in patients with calcium phosphate apatite, uric acid or struvite urolithiasis albeit with a small sample size. Future studies with larger number of patients are required to further elucidate the role of PFV in patients with nephrolithiasis.

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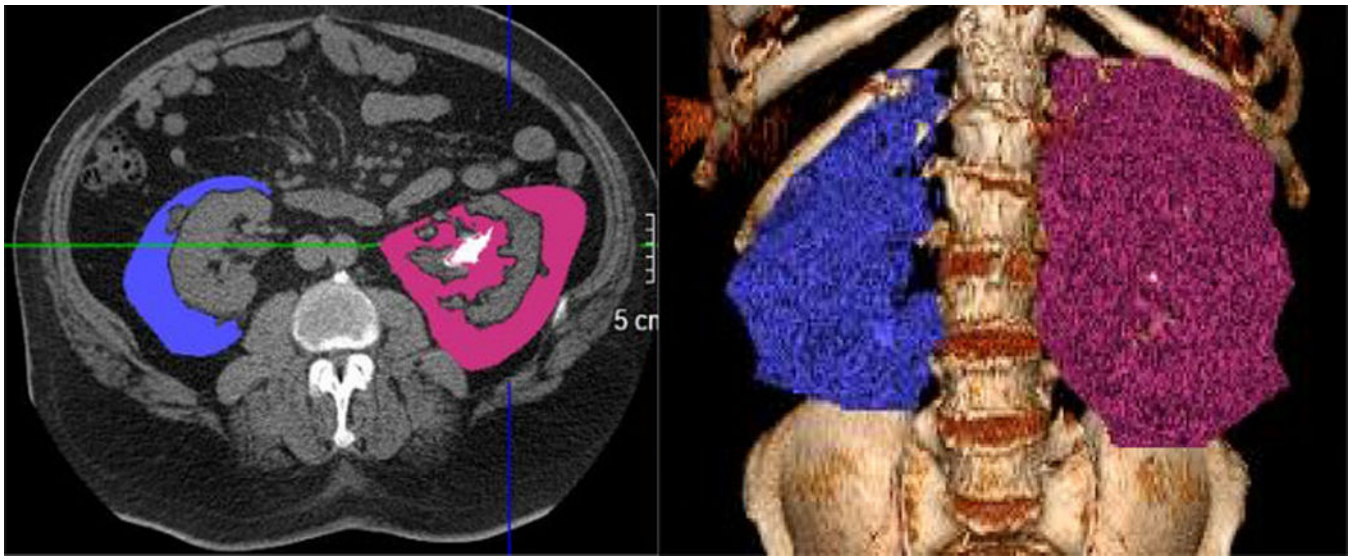

Figure 1:
Vitrea® software PFV measurements of the stone-bearing (pink) and non-stone-bearing (blue) kidney.

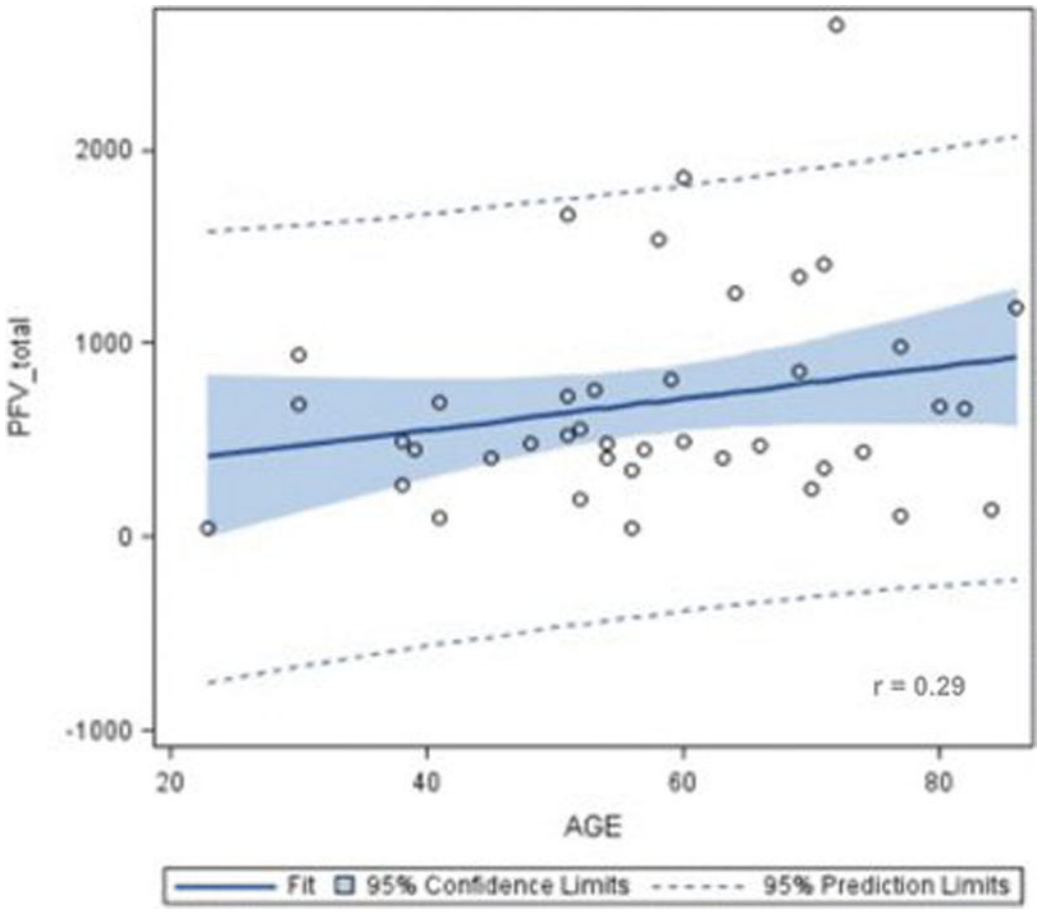

Figure 2:
Pearsons correlation between total PFV and patient age.

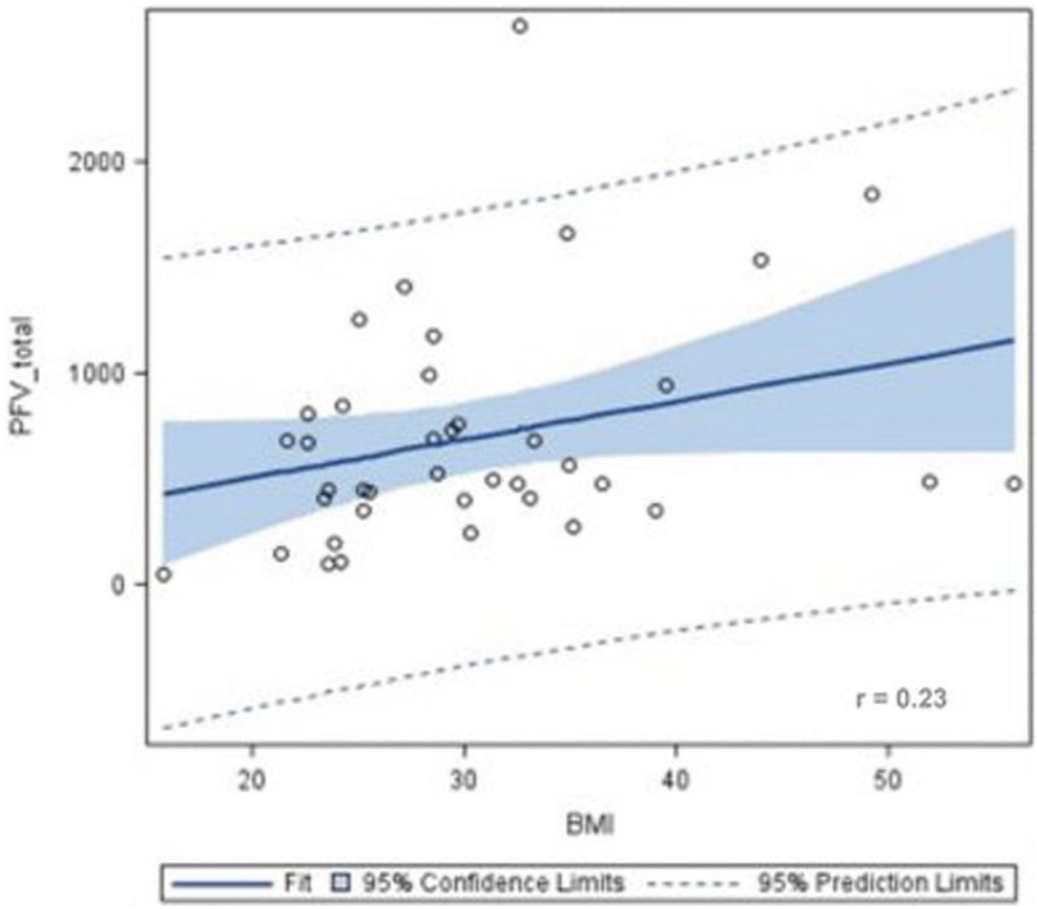

Figure 3:
Pearsons correlation between total PFV and patient body mass index.

Table 1:

Patient features per dominant stone composition group assignment.

Variable	Calcium oxalate (*n*=19)	Calcium phosphate (*n*=9)	Uric acid (*n*=10)	50/50 NH, CP (*n*=2)	Total (*n*=40)
Age (mean ± SD)	61.3 ± 14.1	54 ± 17.1	58.5 ± 15.4	56.5 ± 0.7	57.9 ± 15.5
Female (%)	11 (58)	6 (67)	5 (50)	2 (100)	24 (58)
BMI, kg/m^2 (mean ± SD)	27.8 ± 6.3	34.1 ± 10.2	33.0 ± 9.9	25.2 ± 0.1	30.4 ± 8.5
Underweight[a] (%)	1 (5.3)	–	–	–	1 (2.5)
Normal[b] (%)	6 (31.5)	2 (22.2)	2 (20)	–	10 (25)
Overweight[c] (%)	6 (31.5)	1 (11.1)	3 (30)	2 (100)	12 (30)
Obese[d] (%)	5 (26.3)	4 (44.4)	4 (40)	–	13 (32.5)
Morbidly obese[e] (%)	1 (5.3)	2 (22.2)	1 (10)	–	4 (10)
Diabetes mellitus (%)	7 (36.8)	3 (33)	6 (60)	–	16 (39)
Hyperlipidemia (%)	6 (31.6)	1 (11)	3 (30)	–	10 (24)
Hypertension (%)	11 (58)	8 (89)	4 (40)	–	23 (56)
Axial diameter, cm (mean ± SD)	1.6 ± 0.7	3.5 ± 1.1	2.9 ±0.8	2.5 ± 0.8	2.4 ± 1.1
Coronal diameter, cm (mean ± SD)	1.9 ± 0.8	4.7 ± 1.7	3.3 ± 1.9	4.7 ± 4.5	2.9 ± 1.8
Sagittal diameter, cm (mean ± SD)	1.2 ± 0.3	3.2 ± 1.6	2.3 ± 0.8	2.3 ± 1.4	2.0 ± 1.2
Stone volume, cm^3 (mean ± SD)	2.8 ± 3.8	23.6 ± 20.9	3.1 ± 0.31	25.9 ± 33.9	10.5 ± 14.8
Stone density, HU (mean ± SD)	1000.6 ± 276.7	828.1 ± 187.1	475.6 ± 107.3	770.5 ± 101.8	819 ± 303.1
Stone−bearing kidney PFV density, HU (mean ± SD)	−80.7 ± 16	−80.4 ± 22.3	−85.6 ± 12	−43.6 ± 6	−75.0 ± 29.8
Non−stone−bearing kidney PFV density, HU (mean ± SD)	−84.8 ± 15.8	−92.8 ± 5.3	−92.9 ± 6	−89.6 ± 1.2	−84.3 ± 27.1

SD standard deviation

[a] BMI <18.5

[b] BMI 18.5 – 24.9

[c] BMI 25 – 29.9

[d] BMI 30 – 39.9

[e] BMI >40

Table 2:

Comparisons of mean PFV of the stone-bearing and non-stone-bearing kidneys.

Variable	Mean PFV of stone-bearing kidney (cc)	Mean PFV of non-stone-bearing kidney (cc)	Mean difference (cc)	95% Confidence Interval	*p-value*
Male (*n*=16)	545.45	433.29	112.1	6.2–218	0.039
Female (*n*=24)	271.4	229	42.4	6.6–78.1	0.022
Obese[a] (*n*=17)	461.81	361.38	100.4	−7.8–208.6	0.066
Non-obese[b] (*n*=23)	343.8	291.49	52.3	15.7–88.8	0.007
Diabetes mellitus (*n*=16)	502	406.1	96	−21.7–213.4	0.103
Hypertension (*n*=12)	302.53	277.81	24.7	−17.58−67.0	0.224
Calcium oxalate (*n*=19)	336.08	272.17	63.9	24.4–103.4	0.003
Calcium phosphate (*n*=9)	410.36	261.96	148.4	−63.4–360.2	0.145
Uric acid (*n*=10)	518.25	501.46	16.8	−40.2–86.5	0.557
Struvite (*n*=2)	248.52	152.11	96.4	−633.2–826	0.342

[a] BMI ≥ 30

[b] BMI ≤ 29.9