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Prevalence of Hydronephrosis in Women With Advanced Pelvic Organ Prolapse

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Abstract

OBJECTIVE—To describe the prevalence of hydronephrosis in advanced pelvic organ prolapse (POP) and to describe clinical and urodynamic parameters associated with hydronephrosis.

MATERIALS AND METHODS—Prospective, observational cohort study examining the prevalence of hydronephrosis in advanced POP. Women with a POP-Q examination of at least +1 for points C, Aa, or Ba were enrolled and screened for hydronephrosis. Basic demographics, clinical, and urodynamic findings among women with and without hydronephrosis were compared. The University of Southern California IRB approved this protocol.

RESULTS—A total of 180 participants were enrolled. Fifty-five women had some hydronephrosis, for a prevalence of 30.6% (24.3%−37.6%). Mean age was 57.9 (±9.0) years and mean body mass index was 29.2 kg/m^2 (± 4.6). Of the participants, 80.6% were postmenopausal. The presence of diabetes mellitus was significantly associated with hydronephrosis (8% without vs 21.8% with, $P = .009$), as was the degree of anterior and apical (median Aa, Ba, C, and D higher with hydronephrosis than without, $P < .01$) but not posterior POP (median Ap and Bp, $P = .13$, and .2, respectively). On multichannel urodynamics, participants with hydronephrosis had higher mean post void residuals (64.8 vs 38.5 mL, $P = .007$), lower mean first leak (199.6 vs 280.8 mL, $P = .006$), and higher mean maximum cystometric capacity (525.2 vs 476.7 mL, $P = .02$) compared with participants without hydronephrosis.

CONCLUSION—The prevalence of hydronephrosis in women with advanced POP is 30.6%. Clinical factors associated with hydronephrosis include degree of anterior or apical POP and diabetes mellitus. Urodynamic factors associated with hydronephrosis include elevated postvoid residuals, larger cystometric capacity, and lower volume at first leak.

Pelvic organ prolapse (POP) is commonly associated with urinary symptoms, including voiding difficulty and detrusor overactivity.[1] Furthermore, prolapse is known to be a cause

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of obstructive uropathy that can result in hydronephrosis. Many case studies have described complete prolapse resulting in hydronephrosis, [2–6] even progressing to renal failure,[7,8] calyceal rupture,[9] and death.[10]

The prevalence of hydronephrosis in women with POP has been reported to range from 5%−55%.[11–15] Studies suggest that hydronephrosis is associated with more severe prolapse.[11–15] The exact mechanism causing hydronephrosis is unknown; however, POP is associated with symptoms of voiding dysfunction, bladder outlet obstruction (BOO),[1] and is known to prolong and lessen the ureteric jet on Doppler examination.[16]

The purpose of this study was to determine the prevalence of hydronephrosis in women with advanced POP and to describe the clinical factors and urodynamic parameters that may be associated with hydronephrosis.

MATERIALS AND METHODS

We performed a prospective cohort study of women referred to LAC + USC Medical Center for symptomatic POP between November 2010 and May 2013. The inclusion criteria were women aged 18 or greater presenting for care with advanced POP in the apical or anterior compartments, measured by POP-Q examination (defined as Aa, Ba, or C point > +1).[17] The exclusion criteria were inability to give informed consent; prior pelvic surgery; known intrinsic renal disease including kidney and/or urinary stones; and Foley catheterization or pessary treatment in the preceding 12 months. The institutional review board at the University of Southern California approved this protocol.

Basic demographic data including age, self-reported race and ethnicity, gravidity, parity, menopausal status, medical comorbidities, and prior surgeries were recorded. Physical examination findings, including assessment of POP by the standardized POP-Q examination,[17] systolic blood pressure, diastolic blood pressure, and body mass index were recorded. Multichannel urodynamics, when clinically indicated, were performed and recorded. Blood urea nitrogen and creatinine levels were recorded. If the patient had undergone renal ultrasound or computed tomography within 3 months of enrollment, those images were reviewed and considered screening for hydronephrosis. Remaining participants were screened with a transabdominal renal ultrasound using an ACUSON S2000 machine with a 6C2 or a 4C2 curvilinear probe (Siemens, Mountain View, CA) or a SonoSite S Women's Health machine (Bothell, WA) with a 5–2MHz transducer performed in the lateral decubitus position with an empty bladder. Screening ultrasounds were performed by a trained practitioner, under the supervision of a radiologist. All images were reviewed for adequacy and diagnosis by an experienced attending radiologist with additional training in women's imaging and ultrasound, who was blinded to the clinical scenario. Ultrasound findings were categorized as normal, mild, moderate, or severe according to the following definitions: mild was defined as blunting of minor calyces with minimal calyceal separation; moderate was defined as any distention of the minor and major calyceal systems, renal pelvis, or ureters; and severe was defined as severe pelvi-calyceal and ureteric distention or thinning of renal parenchyma.[15]

Statistical Considerations

On the basis of available retrospective studies, we found that the pooled prevalence of hydronephrosis is ~10%. To determine the prevalence with 5% margin of error and 95% confidence, at least 139 subjects were required.

When discordant levels of hydronephrosis were found between kidneys within the same individual, they were classified as the more severe finding. For comparisons between groups, χ^2-test, independent samples *t* test, Wilcoxon rank sum test, Kruskal-Wallis, and analysis of variance were used as appropriate. Multivariate logistic regression used a backward, stepwise elimination with an s1 stay criterion of 0.15. Goodness of the model fit was assessed by a Hosmer-Lemeshow test. All analyses were performed using SAS, version 9.3.

RESULTS

Two hundred and sixty women were approached for inclusion. Twenty-five were excluded from enrollment (2 deemed not competent to enroll, 2 prior Foley catheterization, 12 prior pessary treatment, 3 prior anti-incontinence surgery, 2 with language barriers, and 3 declined). One hundred eighty-one participants were screened; in 1 case, images were not saved for review and were excluded for 180 participants.

Basic demographics of the study population are shown in Table 1. Mean serum creatinine level was 0.66 mg/dL (±0.21; n=156) and mean urea nitrogen level was 15.5 mg/dL (±6.3; n = 156).

Fifty-five women had hydronephrosis, for a prevalence of 30.6% (95% CI, 24.3%−37.6%). Fifteen percent had mild, 12.2% had moderate, and 3.3% had severe hydronephrosis. Among the entire cohort, there was 83% agreement in the severity between kidneys. Of the 55 participants with hydronephrosis, 30 had differing levels of severity (13/55 left > right, vs 17/55 right > left, $P = .39$).

There was no difference between groups among women with and without hydronephrosis in terms of age, race, ethnicity, body mass index, parity, postmenopausal status, hypertension diagnosis, smoking history, uterine size, blood urea nitrogen, or serum creatinine levels. The presence of diabetes mellitus was significantly associated with hydronephrosis (8% vs 21.8%, $P = .009$), as was the degree of anterior and apical, but not posterior POP (Table 2).There was an increasing tendency toward hydronephrosis by degree of prolapse. The prevalence of hydronephrosis in stage 2 POP was 1 of 7 (14.3%), in stage 3 POP it was 30 of 135 (22.2%), and in stage 4 it was 24 of 38 (63.1%; $P < .001$).

On multichannel urodynamics, hydronephrosis was associated with an elevated post void residual (PVR) volume (64.8 vs 38.5 mL; $P = .007$), a higher maximum cystometric capacity (535.2 vs 476.7 mL; $P = .02$), and a lower volume at first leakage of urine (204.5 vs 280.8 mL; $P = .01$). There was no difference between groups with respect to mean maximum flow rate, detrusor pressure at maximum flow, maximum detrusor pressure, or the presence of BOO (Table 2). Only 1 participant, who had severe hydronephrosis, had a creatinine level greater than 1.6 mg/dL. Nine participants had a creatinine level higher than 1 mg/dL, 5 of 55

(9.1%) in the hydronephrosis group and 4 of 125 (3.2%) in the no hydronephrosis group (P = .13).

A stepwise logistic regression analysis with backward elimination was performed to assess the impact of each clinical factor on the presence of hydronephrosis. The variables fitted to the model included age, presence of diabetes mellitus, degree of anterior prolapse (Ba), degree of apical prolapse (C), degree of posterior prolapse (Bp), the presence of BOO, maximum cystometric capacity, and PVR volume. After controlling for other factors, only the degree of anterior prolapse and the maximum cystometric capacity remained significantly different. The degree of anterior prolapse increased the risk of hydronephrosis with an odds ratio of 1.68 (95% CI, 1.22–2.12), indicating that for every 1 cm increase in Ba, the risk of hydronephrosis increases by 1.68. For every 100-mL increase in maximum cystometric capacity, the risk of hydronephrosis increases by 1.50 (95% CI, 1.08–2.07). The full model containing all predictors was statistically significant (P = .73), indicating that the model fit is adequate. The R^2 value was 0.3, indicating the model accounts for 30% of the variation seen.

COMMENT

Prolapse is commonly described as a cause obstructed voiding, and case reports have well documented resultant obstructive uropathy.[2–6] Although the presence of hydronephrosis is not diagnostic of obstruction, it is commonly associated with obstructed or dysfunctional voiding. In cases of prolapse, the upper urinary tract may be affected, though the exact prevalence is unknown. The present study shows a prevalence of 30.6% among women with advanced POP (defined as prolapse more than 1 cm outside of the body). This prevalence is higher than the overall prevalence of 5%−17.9% in several recent retrospective studies,[11–13,15] but similar to estimates of analyses limited to women with advanced POP. In a recent prospective study of 233 women with varying degrees of prolapse, the prevalence of hydronephrosis was 17.7% and 33.3% for stage 3 and 4 POP, respectively.[14]

In the present study, hydronephrosis was significantly associated with a diagnosis of diabetes mellitus (21.8% vs 8%, P = .009), similar to Hui et al, who found that diabetes mellitus was present in 54.2% of patients with hydronephrosis compared with 22.5% among those without hydronephrosis (P = .002).[14] This association is likely not causative, as the linear regression analysis revealed no significant association when controlled for age, degree of prolapse, and urodynamic findings. Nevertheless, diabetes mellitus is clearly seen in a high percentage of women with advanced POP and hydronephrosis. Furthermore, as diabetes mellitus is associated with glomerulosclerosis and microvascular renal changes,[18] these patients may be at increased risk for complications from the obstructive uropathy caused by POP.

Increasing degrees of prolapse was significantly associated with an increased risk of hydronephrosis. This was seen particularly in anterior and apical, but not in posterior prolapse. This finding fits with the proposed models to explain the association between hydronephrosis and POP. Hadar et al argued that the ureters may be compressed by the fundus of the prolapsed uterus, whereas Lieberthal et al suggested that the cardinal

ligaments form a sling over the ureter, pulling it down with the uterus and kinking it. When controlled for degree of anterior prolapse, apical prolapse no longer contributed to the risk of hydronephrosis. This is in keeping with the finding of hydronephrosis despite prior surgical removal of the uterus[11,12] and supports the theory that the ureters may be pulled down and kinked by the downward traction of prolapse upon the bladder. It has been proposed that the right side may be more sensitive to kinking,[14] as hydronephrosis may be more common on the right side, a finding that was not reproduced in the present study.

On multichannel urodynamics, hydronephrosis was significantly associated with a higher maximum cystometric capacity, higher PVR volumes, and lower volume at first leakage, though only maximum cystometric capacity remained statistically significant when controlled for age and degree of prolapse. Although POP has been described to be associated with BOO,[1] the presence of outlet obstruction was not significantly associated with hydronephrosis in our study. Similarly, there was no difference between groups in the maximum urinary flow rate, detrusor pressure at maximum flow, or maximum detrusor pressure.

This study represents the largest prospective study to date of women with advanced stage POP and the only prospective study to include urodynamic data. We purposely chose to exclude prior hysterectomy and prolapse and/or incontinence surgery, thereby avoiding the confounders of prior unrecognized ureteral injury or BOO. However, these strict exclusion criteria limit the generalizability; the prevalence of hydronephrosis may be different among women with prior pelvic surgery. Finally, our study was powered to describe the prevalence of hydronephrosis, and may be underpowered to detect a difference in some categorical variables, especially the presence of BOO.

CONCLUSION

The overall prevalence of hydronephrosis in patients with advanced POP is 30.6% (95% CI, 24.3%−37.6%). Multiple studies have shown improvement of hydronephrosis after surgical intervention for POP, and renal ultrasound may not be necessary in women who are planned for surgery, though it may help guide intraoperative decision making. Renal ultrasound may be more useful to guide clinical recommendations and counseling in women who are unwilling or unable to undergo surgical intervention. Furthermore, it may be prudent to follow such patients with biochemical and/or radiologic surveillance to prevent and/or detect signs of significant obstructive uropathy. Patients with diabetes mellitus appear to be particularly at risk. We recommend consideration of renal ultrasound in women with advanced POP, especially when surgical intervention is not indicated or delayed.

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Table 1.

Demographics of study participants and of participants with and without hydronephrosis

Demographic	N	Study Population	No Hydronephrosis (N = 125)	Hydronephrosis (N = 55)	*P* (No Hydronephrosis vs Hydronephrosis)
Age (y), mean (SD)	180	57.9 (9.0)	57.1 (8.9)	59.9 (9.0)	.052 *
BMI (kg/m^2), mean (SD)	180	29.2 (4.6)	29.3 (4.8)	28.9 (4.3)	.54 *
Self-reported race, n (%)					.65 †
Caucasian	32	17.80%	25 (20.0)	7 (12.7)	
African American	6	3.30%	4 (3.2)	2 (3.6)	
Asian	1	0.60%	1 (0.8)	0 (0)	
Other	141	78.30%	95 (76.0)	46 (83.6)	
Ethnicity, n (%)					.65 ‡
Hispanic	163	90.60%	114 (91.2)	49 (89.1)	
Non-Hispanic	17	9.40%	11 (8.8)	6 (10.9)	
Postmenopausal (%)	141	80.60%	98 (79.0)	43 (84.3)	.42 ‡
Parity, median (range)	180	3 (0–16)	3 (0–16)	4 (0–15)	.64 §
Number of cesarean deliveries, median (range)	178	2 (0–3)	0 (0–1)	0 (0–3)	.65 §
Presence of comorbidities, n (%)					
HTN	40	22.20%	28 (22.4)	12 (21.8)	.93 ‡
DM	22	12.20%	10 (8)	12 (21.8)	.009 ‡
Smoker	3	1.70%	3 (2.4)	0	.25 ‡

BMI, body mass index; DM, diabetes mellitus; HTN, hypertension; SD, standard deviation.

* Student *t* test.

† Fisher exact test.

‡ Chi-square test.

§ Wilcoxon rank sum test.

Table 2.

Clinical findings of participants with and without hydronephrosis

Clinical Finding	No Hydronephrosis (N = 125)	Hydronephrosis (N = 55)	*P*
Systolic blood pressure (mm Hg), mean (SD)	131.2 (19.5)	136.8 (18.3)	.07 *
Diastolic blood pressure (mm Hg), mean (SD)	75.0 (10.9)	75.4 (10.2)	.81 *
Uterine size on bimanual exam (wk), median (range)	6 (5–18)	6 (6–12)	.1 †
Positive Q-tip test (%)	100	100	n/a
POP-Q measures, median (range)			
Genital hiatus	5 (2–8) (n = 120)	5 (3–8) (n = 52)	.54 †
Perineal body	3 (0–6) (n = 120)	3 (2–5) (n = 52)	.9 †
Total vaginal length	8 (5.5–11) (n = 120)	8.5 (5–11) (n = 51)	.022 †
Cervix	1 (−6 to 8.5) (n = 122)	4.5 (−5 to −11) (n = 54)	<.001 †
D	−4 (−7 to −4) (n = 120)	−1 (−7 to −11) (n = 53)	<.001 †
Aa	2 (0–3) (n = 125)	3 (−1 to −3) (n = 55)	<.001 †
Ba	3 (0–8) (n = 125)	5 (0–11) (n = 55)	<.001 †
Ap	0 (−3 to −3) (n = 122)	0 (−2 to −3) (n = 52)	.13 †
Bp	0 (−3 to 7) (n = 122)	0 (−2 to 9) (n = 52)	.2 †
Post void residual (mL), mean (SD)	38.5 (54.0) (n = 120)	64.8 (62.6) (n = 49)	.007 ‡
Maximum cystometric capacity (mL), mean (SD)	476.7 (105.5) (n = 113)	525.2 (156.4) (n = 45)	.02 ‡
Maximum flow rate (mL/s), mean (SD)	21.1 (13.2) (n = 107)	18.4 (11.6) (n = 41)	.26 ‡
Detrusor pressure at maximum flow rate (cm H_2O), mean (SD)	29.6 (18.7) (n = 105)	35.5 (19.7) (n = 40)	.095 ‡
Maximum detrusor pressure (cm H_2O), mean (SD)	49.7 (27.4) (n = 105)	55.9 (24.5) (n = 41)	.21 ‡
Valsalva leak point pressure (cm H_2O), mean (SD)	63.6 (35.2) (n = 18)	74.7 (28.2) (n = 12)	.36 ‡
Volume at first leakage of urine (mL), mean (SD)	280.8 (164.2) (n = 92)	204.5 (134.5) (n = 40)	.011 ‡
Presence of bladder outlet obstruction	24.3% (n = 103)	38.5% (n = 39)	.099 §

Clinical Finding	No Hydronephrosis (N = 125)	Hydronephrosis (N = 55)	*P*
Blood urea nitrogen level (mg/dL), mean (SD)	14.8 (4.6)	17.1 (8.9)	.03[‡]
Creatinine level (mg/dL), mean (SD)	0.64 (0.18)	0.7 (0.28)	.15[‡]

Abbreviation as in Table 1.

[*] Student *t* test.

[†] Wilcoxon rank sum test.

[‡] Analysis of variance.

[§] Chi-square test.