Published in final edited form as:

Urology. 2009 June ; 73(6): 1352–1355. doi:10.1016/j.urology.2008.12.073.

Effect of Obesity on Urethroplasty Outcome

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Abstract

OBJECTIVES—To determine whether obesity increases the risk of urethroplasty failure.

METHODS—A total of 381 patients underwent urethroplasty and had complete body mass index (BMI) data. Stricture recurrence-free survival was defined as subjective and objective improvement in urinary flow, an absence of radiographic evidence of stricture, and no further need for urethral instrumentation. Cox proportional hazards regression analysis was used to identify both univariate and multivariate predictors of urethroplasty outcome.

RESULTS—The median patient age was 40 years (range 18–90). The median follow-up was 5.9 years (range 1 month to 10 years). Of the 381 patients, 60 developed recurrent stricture (15.7%). Overweight (BMI 25–30 kg/m²) and obese (BMI 30–35 kg/m²) patients were more likely to have urethroplasty failure, reaching significance and near significance on univariate and multivariate analysis, respectively ($P = .03$ and $P = .07$, respectively). Patients who were severely (BMI 35–40 kg/m²) or morbidly obese (BMI >40 kg/m²) did not have an increased risk of urethroplasty failure.

CONCLUSIONS—Although obesity's relationship with urethroplasty failure is not linear, it does appear to affect outcomes after urethroplasty.

Obesity is a global epidemic, and the prevalence has continued to increase in industrialized nations and nations undergoing economic advancement.¹ The World Health Organization has estimated that 250 million people are obese worldwide, and that by 2025, that number will have increased by 50 million.¹ In the United States, >50% of the population is overweight and approximately 20% is extremely overweight or obese.¹ Within urology, obesity has been linked to stress urinary incontinence, erectile dysfunction, infertility, stone formation, and renal cell carcinoma.²

Urethral stricture disease can lead to lower urinary tract symptoms, bladder dysfunction, renal damage, urethral cancer, and Fournier's gangrene. In the United States, urethral stricture results in >5000 inpatient hospitalizations yearly.³ Urethroplasty to treat stricture disease is successful, durable, and cost effective.⁴

Perineal surgery for obese patients with urethral stricture disease is perceived as more difficult; therefore, we sought to determine whether obesity was predictive of a worse outcome after urethroplasty. We hypothesize that the risk of stricture treatment failure would be greater for obese individuals.

MATERIAL AND METHODS

Patient Population

After institutional review board approval, the University of California, San Francisco, urethroplasty database was reviewed. Approximately 495 patients had been treated with urethroplasty for urethral stricture disease by a single surgeon (J.W.M.) from 1995 to 2004 at 3 hospitals—the University of California, San Francisco, Medical Center, San Francisco General Hospital, and the San Francisco Veterans Affairs Medical Center. Of the 495 patients, 381 had complete body mass index (BMI) data and were analyzed. The patients were included in the study if they had undergone urethroplasty and had complete BMI data. The patients were excluded if they did not have complete BMI data.

Follow-up

Clinical history and uroflowmetry were performed at 3-month intervals for the first year and yearly thereafter. Voiding cystography was performed at catheter removal, and retrograde urethrography was performed during follow-up at 3 months and 1 year. Additional fluoroscopic images were obtained if subjective obstructive symptoms were present. In addition, additional fluoroscopic imaging was obtained when the peak urinary flow rate was <15 mL/s or a change in the voiding pattern was seen on uroflowmetry during other follow-up visits.⁵ When needed, information for referral patients was gathered by written or telephone correspondence. Stricture recurrence-free survival was defined as subjective and objective improvement in urinary flow, the absence of radiographic evidence of stricture, and no further need for urethral instrumentation.

Statistical Analysis

Stricture recurrence-free survival curves were estimated using the Kaplan-Meier method. Patients were censored either at the last evaluation or death. The 9 most common medical comorbidities in the database were selected: smoking, chronic obstructive pulmonary disease, diabetes mellitus, coronary artery disease, peripheral vascular disease, connective tissue disorders, liver disease, advanced age, and obesity. Additional stricture-related variables were also evaluated, including trauma, hypospadias, previous dilation, direct vision internal urethrotomy, urethroplasty, stricture length, and type of repair. For the BMI, the definitions of normal (18 – 25 kg/m²), overweight (25 – 30 kg/m²), obese (30 – 35 kg/m²), severely obese (35 – 40 kg/m²), and morbidly obese (>40 kg/m²) were used.

Cox proportional hazards regression analysis was used to identify both univariate and multivariate predictors of urethroplasty outcome. Backward step-wise modeling with the likelihood ratio statistic was used for variable entry and removal, which was set at 0.05 and 0.1, respectively. All potential risks were included in the multivariate model to increase the

accuracy and decrease bias. Hazard ratios were calculated to provide an estimate of the relative risk of the analyzed variables.

The α value was set at 0.05 for all analyses, and 95% confidence intervals were calculated. The analysis was performed using commercially available statistical software (SPSS, Chicago, IL).

RESULTS

We identified 381 patients who met the inclusion and exclusion criteria. Their median age was 40 years (range 18-90). The median follow-up was 5.9 years (range 1 months to 10 years). Of the 381 patients, 60 (15.7%) developed stricture recurrence. The patient population was generally healthy, with comorbidities such as chronic obstructive pulmonary disease, smoking, coronary artery disease, chronic obstructive pulmonary disease, diabetes mellitus, peripheral vascular disease, connective tissue disease, and liver disease present in only 4%, 17%, 12%, 4%, 4%, 3%, 2%, and 1% of patients, respectively. Only 10% of the patients were >65 years of age. Obesity was the most prevalent comorbidity and was present overall in 70% of patients.

The stricture etiology, location, length, and treatment are listed in Table 1. The stricture length was similar among the groups except for those with a BMI <25 kg/m², who had shorter strictures. Stricture location was similar across the weight divisions. The stricture etiology was most commonly associated with trauma, which included previous catheterization, except in the morbidly obese population. The incidence of lichen sclerosis did not correlate directly with weight. Only 111 patients (29%) had not undergone previous treatment and 189 (50%), 172 (45%), and 106 (28%) had undergone previous direct vision internal urethrotomy, dilation, and urethroplasty, respectively. End-to-end urethroplasty was used in most cases (n = 209, 55%). Other procedures included buccal mucosa graft urethroplasty (n = 78, 21%), fasciocutaneous flap (n = 61, 16%), and other procedures (n = 33, 8%) or a combination of procedures. The use of the different surgical approaches was similar across the weight groups.

Figure 1 shows the Kaplan-Meier curve stratified by the BMI classification and follow-up data. This analysis suggested that patients with a BMI of 25-30 kg/m² and 30-35 kg/m² (overweight and obese) were at an increased risk of stricture recurrence compared with patients with a BMI of 0-25 kg/m² (normal) and >35 kg/m² (severe obesity and morbid obesity). Within each surgical approach (eg, end to end, buccal graft), obesity had a similar effect on outcomes, as it did on the surgical approaches aggregated. The same held true when considering the influence of obesity on outcomes after anterior or posterior repair (data not shown).

Table 2 lists the univariate Cox regression analysis results (significance, $P < .05$). Diabetes, BMI of 25-35 kg/m², previous direct vision internal urethrotomy, and previous urethroplasty each proved to be a significant ($P < .05$) negative influence on urethroplasty outcome. Although the simple end-to-end procedure was protective ($P < .01$), longer strictures and fasciocutaneous flaps were more prone to failure ($P < .01$).

Table 3 lists the multivariate Cox regression analysis results (significance, $P < .05$). After taking all variables into consideration, smoking and previous urethroplasty were more predictive of failure ($P < .05$ and $P < .03$, respectively), and obesity (BMI 25-35 kg/m²) neared significance ($P < .07$).

COMMENT

Given their comorbidities, obese patients theoretically have an increased risk of poor outcomes after surgical intervention.^{6,7} Surgeons frequently cite an increased risk of complications when considering therapy for obese patients.⁷⁻⁹ Obesity has been linked to stress urinary incontinence, erectile dysfunction, infertility, nephrolithiasis, and renal cell carcinoma.² The degree of risk associated with obesity and its affect on surgical outcomes needs to be better defined. A definitive understanding of the risk and complication rates will allow surgeons to offer the best therapeutic modality to obese patients. We attempted to determine whether obesity is a risk factor for urethroplasty failure using a retrospective analysis of our urethroplasty database.

On univariate analysis, diabetes mellitus ($P < .05$), BMI 25-35 kg/m² ($P < .03$), previous direct vision internal urethrotomy ($P < .05$), previous urethroplasty ($P < .01$), stricture >4 cm ($P < .01$), and a history of fasciocutaneous flap placement ($P < .01$) were predictive of urethroplasty failure. As expected, use of end-to-end urethroplasty was protective ($P < .01$). On multivariate analysis, end-to-end urethroplasty was again protective ($P < .01$). A BMI of 25-35 kg/m² showed a strong trend toward significance ($P < .07$), and smoking history ($P < .05$) and previous urethroplasty ($P < .03$) were significant predictors of urethroplasty failure.

Although our data have revealed that patients who are overweight (BMI 25-30 kg/m²) and obese (BMI 30-35 kg/m²) develop urethroplasty failure more frequently, the severely and morbidly obese did not. It is unclear why a continued dose-response relationship was not seen with an increasing BMI. Potentially, morbidly obese patients are more sedentary and avoid activities that could put their urethroplasty at risk of failure, thus leading to the protective role of a BMI >35 kg/m². Additionally, morbidly obese people are less likely to engage in activity that results in increased perineal pressure, such as bicycling. Furthermore, obese individuals possess a significant cushion of perineal fat that might protect against straddle-type injury.

A potential exists that severe and morbidly obese people are receiving more medical attention.¹⁰ As such, unrecognized comorbidities in overweight and mildly obese patients, such as diabetes and coronary artery disease, might have confounded the multivariate results. It is possible that an ascertainment bias on the part of both patients and providers exists. As such, heavier patients are less likely to notice or be bothered by a change in urination and stricture recurrence. In addition, the provider might be more likely to tolerate minor or even moderate symptoms before recommending additional intervention.

We found results similar to those of Elliott et al.,¹¹ who studied the incidence of urethral stricture after primary treatment for prostate cancer among approximately 6000 men from the CaPSURE database. Obesity and age were significant predictors of stricture treatment on

multivariate analysis. Debate exists whether increased stricture rates arise from technical difficulties encountered in the obese patient or whether obesity influences wound healing.¹¹

Our data are somewhat in contradiction to an earlier report by Brandes.⁹ Compared with nonmorbidly obese patients, patients with a BMI >40 kg/m² had increased urethroplasty failure (50% vs 24%), longer operative times (284 vs 256 minutes), and a longer length of stay (2.2 vs 1.2 days). However, of the 111 patients in the series, only 14 were morbidly obese (BMI >40 kg/m²). Although the study by Brandes benefited from the large portion of morbidly obese patients, it would be interesting to know the additional categorization of the BMI categories to better compare that study's results with ours.

Anema et al.¹² studied a series of 185 patients who had undergone urethroplasty in the high lithotomy position. They found that obesity was not a risk factor for the development of neurapraxia, rhabdomyolysis, or compartment syndrome. In their study, the length of stricture, duration of surgery, and use of the lithotomy position were risk factors for the development of complications. We did not analyze complication rate or type.

The present study was of the experience of a single surgeon (J.W.M.) with a high-volume urethroplasty practice. Taking data from a single surgeon eliminates the confounding influence of variable techniques on outcomes. Another strength of our analysis was the long follow-up time and the large number of patients included.

Our study was limited because the BMI is only a surrogate for body fat composition and is a simple indicator of obesity.⁸ The BMI tends to overestimate the body fat in athletes and individuals with a muscular build.⁸ It can also underestimate the body fat in older persons and others who have lost muscle mass. Nevertheless, the BMI is a universally used benchmark for obesity. In addition, a large portion of the patients did not have complete BMI data available for analysis. Additional limitations of our study included those inherent to its retrospective design. Our findings in the severely and morbidly obese patient were based on a small sample size, because these patients accounted for only 11% (n = 42) of the entire population. The analysis was also limited in that the outcome measure was solely related to urethroplasty failure or success.

CONCLUSIONS

Although the relationship of obesity with urethroplasty failure is not linear, it does appear to affect the outcomes after urethroplasty.

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Figure 1.
Kaplan-Meier estimate of stricture recurrence-free survival according to World Health Organization body mass index category.

Table 1

Stricture length, location, etiology, and treatment stratified by BMI

Variable	BMI (kg/m ²)				
	Normal <25 (n = 116)	Overweight 25-30 (n = 143)	Obese 30-35 (n = 80)	Severe Obesity 35-40 (n = 19)	Morbid Obesity >40 (n = 23)
Stricture length (cm)					
Mean	2.93	3.8	4.1	3.9	4.0
Range	0.6-10	1-15	0.5-19	0.7-10	1-20
Location (n)					
Anterior	81 (70)	106 (74)	65 (81)	16 (84)	17 (74)
Posterior	28 (24)	31 (22)	14 (18)	3 (16)	5 (22)
Both	7 (6)	6 (4)	1 (1)	0 (0)	1 (4)
Etiology (n)					
Trauma	74 (64)	67 (47)	35 (44)	10 (53)	5 (22)
Idiopathic	31 (27)	54 (38)	33 (41)	8 (42)	16 (69)
Hypospadias	9 (8)	11 (7)	8 (10)	1 (5)	0 (0)
Lichen sclerosis	2 (1)	11 (7)	4 (5)	0 (0)	2 (9)
Treatment (n)					
End to end	80 (69)	68 (48)	38 (47)	7 (37)	16 (70)
Buccal onlay	17 (15)	34 (24)	19 (24)	6 (32)	2 (9)
Fasciotomaneous	10 (9)	31 (21)	15 (19)	4 (21)	2 (10)
Combined	9 (7)	10 (7)	8 (10)	2 (10)	4 (17)

BMI, body mass index.

Table 2

Univariate analysis of potential prognostic factors for stricture-free survival

Variable	HR (95% CI)	P Value
Smoking	1.5 (0.8-2.7)	.19
Comorbidity		
Chronic obstructive pulmonary disease	1.4 (0.5-4.6)	.54
Diabetes mellitus	2.4 (1.0-5.9)	.05*
Coronary artery disease	1.2 (0.6-2.5)	.64
Peripheral vascular disease	0.6 (0.1-4.2)	.60
Connective tissue disease	2.7 (0.9-8.7)	.09
Liver disease	2.5 (0.9-6.5)	.11
Age >65 y	1.0 (0.4-2.4)	.96
BMI 25-35 kg/m ²	1.9 (1.1-3.3)	.03*
Stricture etiology		
Trauma	0.8 (0.5-1.3)	.38
Hypospadias	2.0 (1.0-4.3)	.06
Previous treatment		
Dilation	1.1 (0.6-1.8)	.83
Direct vision internal urethrotomy	1.7 (1.0-2.8)	.05*
Urethroplasty	2.1 (1.3-3.5)	< .01*
Stricture length quartiles (cm)		< .01
0-1.7	—	—
1.7-2.5	1.0 (0.4-2.6)	.99
2.5-4.0	1.3 (0.5-3.2)	.60
>4.0	2.8 (1.3-5.9)	< .01*
Repair type		
End to end	0.4 (0.2-0.7)	< .01*
Buccal mucosa graft	1.1 (0.6-2.0)	.76
Fasciocutaneous flap	2.4 (1.4-4.3)	< .01*
Combined	1.8 (0.9-3.8)	.09

HR, hazard ratio; CI, confidence interval; BMI, body mass index.

* Statistically significant.

Table 3

Multivariate analysis of potential prognostic factors for stricture-free survival

Variable	HR (95% CI)	P Value
Smoking	1.8 (1.0-3.2)	.05*
Comorbidity		
Chronic obstructive pulmonary disease	1.3 (0.4-4.6)	.65
Diabetes mellitus	1.9 (0.8-4.8)	.16
Coronary artery disease	1.0 (0.4-2.3)	.94
Peripheral vascular disease	0.6 (0.1-5.5)	.56
Connective tissue disease	1.4 (0.4-4.7)	.64
Liver disease	1.0 (0.1-9)	.98
Age >65 y	0.8 (0.3-2.1)	.71
BMI 25-35 kg/m ²	1.7 (1.0-3.2)	.07*
Stricture etiology		
Trauma	1.1 (0.6-2.1)	.67
Hypospadias	1.6 (0.6-3.9)	.31
Previous treatment		
Dilation	0.8 (0.4-1.3)	.34
Direct vision internal urethrotomy	1.7 (1.0-2.9)	.06*
Urethroplasty	1.8 (1.1-3.1)	.03*
Stricture length quartiles (cm)		.46
0-1.7	—	—
1.7-2.5	1.2 (0.4-3.0)	.76
2.5-4.0	0.9 (0.4-2.6)	.97
>4.0	1.7 (0.7-4.3)	.23
Repair type		
End to end	0.5 (0.3-0.8)	< .01*
Buccal mucosa graft	0.8 (0.4-1.5)	.44
Fasciocutaneous flap	1.0 (0.4-2.4)	.97
Combined	0.9 (0.1-9.1)	.98

Abbreviations as in Table 2.

* Statistically significant.