

HEALTH-RELATED QUALITY OF LIFE FOR MEN WITH PROSTATE CANCER AND DIABETES: A LONGITUDINAL ANALYSIS FROM CaPSURE

DAVID M. LATINI, JUNE M. CHAN, JANET E. COWAN, SHELLEY A. ARREDONDO,
CHRISTOPHER J. KANE, DAVID F. PENSON, JANEEN DuCHANE, PETER R. CARROLL,
AND THE CaPSURE INVESTIGATORS

ABSTRACT

Objectives. To compare diabetic versus nondiabetic men with prostate cancer to understand whether diabetes mellitus (DM) imposes an additional burden on health-related quality of life (HRQOL) before and after radical prostatectomy, adjusting for obesity.

Methods. Data were abstracted from Cancer of the Prostate Strategic Urologic Research Endeavor (CaPSURE), a disease registry of 12,005 men with localized prostate cancer. Men were included who had undergone surgical treatment from 1989 to 2003, had body mass index (BMI) information available, and had completed both a pretreatment and at least one posttreatment HRQOL questionnaire within 24 months. A repeated-measures model adjusted for baseline clinical and demographic variables was used to evaluate group differences.

Results. The 1248 men were divided into two groups (117 with DM and 1131 without DM) on the basis of a history of DM or the reporting of diabetes medication use. The diabetic men were significantly more likely to be older and nonwhite, have lower education and income, and be less likely to have private insurance. They also had significantly more comorbid conditions (other than DM) and a greater BMI at baseline. Urinary function differed by diabetes status, BMI, and the DM \times BMI interaction, with diabetic men who had a greater BMI reporting greater declines in urinary function over time. No other statistically significant differences in HRQOL were observed, although trends by BMI were noted in sexual function and bowel bother.

Conclusions. Although previous studies of men with prostate cancer have found differences in HRQOL by obesity level, our results have indicated that the presence or absence of DM and a high BMI may have a greater impact on HRQOL than obesity alone. *UROLOGY* 68: 1242–1247, 2006. © 2006 Elsevier Inc.

The available treatments for localized prostate cancer can cause treatment-related symptoms, primarily urinary, erectile, and bowel problems.¹ These can be particularly problematic for men with

comorbid conditions that also cause urinary or sexual dysfunction. For example, men with diabetes mellitus (DM) have reported worse erectile functioning and intercourse satisfaction than non-

CaPSURE is supported by TAP Pharmaceutical Products, Incorporated, Lake Forest, Illinois. This research was additionally funded by the National Institutes of Health/National Cancer Institute University of California-San Francisco SPORE Specialized Program of Research Excellence P50 C89520. This report is the result of work supported with resources and the use of facilities at the Houston Center for Quality of Care and Utilization Studies, Houston Veterans Affairs Medical Center.

D. M. Latini and P. R. Carroll are study investigators funded by TAP Pharmaceutical Products, Incorporated.

A list of the current and former CaPSURE Investigators can be found in the Appendix.

From the Scott Department of Urology and Dan L. Duncan Cancer Center, Baylor College of Medicine; Houston Center for Quality of Care and Utilization Studies, Michael E. DeBakey Veterans Affairs Medical Center, Houston, Texas; Department of Urology,

Program in Urologic Oncology, Genitourinary Cancer Epidemiology and Population Science Program, University of California, San Francisco, Comprehensive Cancer Center, University of California, San Francisco, School of Medicine; Department of Epidemiology and Biostatistics, University of California, San Francisco, School of Medicine; San Francisco Veterans Affairs Medical Center, San Francisco, California; Department of Urology and Preventive Medicine, University of Southern California, Los Angeles, Keck School of Medicine, Los Angeles, California; and TAP Pharmaceutical Products Incorporated, Lake Forest, Illinois

Reprint requests: David M. Latini, Ph.D., Houston Center for Quality of Care and Utilization Studies, Michael E. DeBakey Veterans Affairs Medical Center, 152, 2002 Holcombe Boulevard, Houston, TX 77030. E-mail: latini@bcm.tmc.edu

Submitted: June 22, 2005, accepted (with revisions): August 22, 2006

diabetic men with erectile dysfunction (ED) from other etiologies.² DM is also of concern because of its high prevalence and rising incidence among U.S. adults. The U.S. Centers for Disease Control and Prevention has reported the lifetime risk of DM for people born in the United States is 1 in 3, with a greater risk for African Americans who are also at a greater risk of prostate cancer.³

Previous research has shown that DM in men with prostate cancer can worsen changes in health-related quality of life (HRQOL). For example, DM has been reported to exacerbate erectile problems in men receiving brachytherapy for prostate cancer.⁴ In addition, diabetic men with prostate cancer treated with external beam radiotherapy experienced significantly more late grade 2 gastrointestinal and genitourinary toxicities than did nondiabetic men receiving the same treatment.⁵ Obesity, often co-occurring with DM and also increasing in the United States,⁶ has been investigated for its effect on the risk of prostate cancer, as well as its effect on disease recurrence and HRQOL for men treated surgically for prostate cancer.⁷⁻⁹

However, little is known about how co-occurring DM and prostate cancer affect HRQOL for men selecting surgery or how either is related to obesity. This report extends the work on men with DM undergoing radiotherapy^{4,5,10,11} to focus on men undergoing prostatectomy. From previous research with radiotherapy patients, we hypothesized that diabetic men would report worse sexual and urinary function than did nondiabetic men after treatment. We adjusted our analysis for body mass index (BMI) to understand the interaction between the presence of DM and a greater BMI, hypothesizing that the two together would be significantly related to poorer HRQOL than either alone.

MATERIAL AND METHODS

PARTICIPANTS

We examined HRQOL data for men in the Cancer of the Prostate Strategic Urologic Research Endeavor (CaPSURE) study, a longitudinal, observational study of men with biopsy-proven prostate cancer. Sociodemographic and HRQOL data are collected from patients at enrollment and semiannually. Participating practices provide clinical data at enrollment and each return visit, including follow-up prostate-specific antigen (PSA) results. The institutional review boards at the University of California, San Francisco, and contributing sites approved the study protocols and methods.

As of July 2005, 12,005 patients were enrolled in CaPSURE, with more than 7000 currently being followed up. Participants are enrolled from a core group of 31 urologic practice sites, primarily community based, with about 8% from academic or Veterans Affairs practices. Additional details of the project methods have been previously published.^{12,13}

To be included in our analysis, patients had to have a diagnosis of clinically localized disease (Stage T1-T3NxM0, n = 7382) from 1989 to 2003, to have undergone radical prostatectomy monotherapy as a first treatment (n = 3913), and to

have pretreatment (n = 1408) and posttreatment HRQOL data within 24 months of treatment (n = 2695). Participants were divided into two groups stratified by the presence or absence of a reported diagnosis of DM or the reported use of diabetes medications. Of the 1248 men who met the inclusion criteria, 117 reported a diagnosis of DM or the use of diabetes medications.

QUALITY-OF-LIFE MEASURES

The CaPSURE questionnaire included the University of California, Los Angeles, Prostate Cancer Index (UCLA-PCI),¹ which contains subscales measuring sexual function and bother, urinary function and bother, and bowel function and bother. It has been used extensively in HRQOL research of patients with prostate cancer and shown to be both reliable and valid.¹⁴

STATISTICAL ANALYSIS

Baseline clinical and sociodemographic characteristics were compared using the chi-square test and analysis of variance. Clinical risk was based on a modification of the D'Amico risk groups.¹⁵ Patients were considered low risk (PSA 10 ng/mL or less, Gleason score less than 7 with no primary or secondary Gleason score of 4 or 5, and clinical Stage T1-T2a), intermediate risk (PSA 10.1 to 20 ng/mL, Gleason score 7 or Gleason secondary score of 4 or 5, or clinical Stage T2b-T2c), or high risk (PSA greater than 20 ng/mL, Gleason score greater than 7 or Gleason primary score 4 or 5, or clinical Stage T3a). We also characterized risk using the Cancer of the Prostate Risk Assessment score, which combines preoperative PSA level, Gleason score, clinical T stage, biopsy results, and age into a measure with predictive accuracy similar to the Kattan nomogram.¹⁶

We examined changes in HRQOL by fitting a repeated-measures mixed model for each UCLA-PCI domain and controlling for BMI, age, race, education, relationship status, insurance status, risk group, and number of comorbidities other than DM. We compared the pretreatment, disease-specific HRQOL scores with the 24-month follow-up HRQOL scores. For men without data at 24 months, we used the last posttreatment HRQOL questionnaire available.

To understand the relationship between DM and obesity, we computed the BMI using patient-reported information at baseline and included that in the multivariate models as both a main effect and an interaction effect. Each model contained a main effect for history of DM (yes or no), BMI (continuous variable), and an interaction term for a history of DM and BMI. To understand the interaction effects, we used a technique described by Aiken and West.¹⁷ We created four subgroups in the analysis sample: men with DM and BMI more than 1 standard deviation (SD) above the UCLA-PCI mean, men with DM and BMI less than 1 SD below the UCLA-PCI mean, men without DM and BMI greater than 1 SD above the UCLA-PCI mean, and men without DM and BMI less than 1 SD below the UCLA-PCI mean for the UCLA-PCI subscale for which the interaction term was significant. We computed the mean change scores on the UCLA-PCI domains for which the interaction term was significant in the multivariate model and graphed the means for each group. All analyses were performed with Statistical Analysis Systems, version 9.1 (SAS Institute, Cary, NC).

RESULTS

BASELINE CHARACTERISTICS

Table I lists the sociodemographic characteristics for each group. Men reporting a history of DM were older and more likely to be nonwhite, have

TABLE I. Patient demographic characteristics by diabetes status before treatment (n = 1,248)

Characteristic	No DM (n = 1,131)	DM (n = 117)	P Value
Age at diagnosis (yr)			<0.01
<65	748 (66)	62 (53)	
≥65	383 (34)	55 (47)	
Race			<0.01
White	1,051 (93)	98 (84)	
Other	79 (7)	19 (16)	
Education level			0.01
Less than high school	88 (8)	15 (13)	
High school graduate	236 (21)	35 (30)	
Some college	218 (19)	20 (17)	
College graduate	580 (52)	46 (40)	
Income level			<0.01
<\$30,000	151 (14)	30 (28)	
\$30,000–50,000	239 (23)	27 (25)	
\$50,000–75,000	236 (22)	19 (18)	
>\$75,000	429 (41)	32 (30)	
Relationship status			0.15
In relationship	1,059 (95)	106 (91)	
Single	60 (5)	10 (9)	
Insurance			<0.01
Medicare supplement	241 (21)	34 (29)	
Medicare	74 (7)	18 (15)	
Private insurance	741 (66)	56 (48)	
Other/none	75 (7)	9 (8)	

KEY: DM = diabetes mellitus.

Data presented as number of patients, with percentages in parentheses.

less education, and have Medicare or Medicare Supplement insurance. We found fewer differences in the clinical presentation between men with DM and those without DM (Table II). Men with DM had significantly more comorbidities and a greater BMI than men without DM.

HRQOL BY DM HISTORY

The mean HRQOL scores before treatment and at 24 months of follow-up are given in Table III, with the results of testing for the main effect of having or not having DM, BMI, and the interaction between DM and BMI on the changes in HRQOL over time. Diabetic men reported baseline HRQOL scores similar to, or worse than, nondiabetic men. Urinary function was significantly different over time by DM history ($P < 0.01$), BMI ($P < 0.02$), and the interaction between DM history and BMI ($P < 0.01$). To understand the relationship between DM history and BMI, we examined the mean changes in urinary function by group (high BMI, diabetic men; high BMI, nondiabetic men; low BMI, diabetic men; and low BMI, nondiabetic men). The mean in all four groups declined from baseline to the 24-month follow-up. The decrease in urinary function was greatest for diabetic men with a greater BMI. No significant differences were seen over time for sexual bother, urinary bother, or

bowel function, although both sexual function ($P < 0.07$) and bowel bother ($P < 0.09$) had a trend toward a difference by BMI. Nondiabetic men had better sexual function at baseline and reported a larger decline in that domain than diabetic men.

COMMENT

We examined the relationship among DM, BMI, and disease-specific HRQOL in CaPSURE participants with clinically localized prostate cancer treated with radical prostatectomy as monotherapy. In the initial comparisons, diabetic men differed significantly from nondiabetic men on several sociodemographic characteristics. However, the groups did not differ for most baseline clinical characteristics other than PSA level. As expected, diabetic men were also significantly more likely to report more comorbid conditions and a greater BMI at baseline. Hypothesized differences in sexual and urinary function for diabetic versus nondiabetic men were observed only for urinary function. However, we also found the hypothesized significant differences by DM history and BMI in urinary function. Although previous studies of men with prostate cancer have reported differences in HRQOL associated with obesity, our results in-

TABLE II. Patient clinical characteristics by diabetes status before treatment (n = 1248)

Characteristic	No DM (n = 1131)	DM (n = 117)	P Value
Mean PSA at diagnosis	6.5 ± 4.97	7.2 ± 5.79	0.16
T stage			0.52
1	646 (57)	73 (62)	
2	477 (42)	43 (37)	
3	8 (1)	1 (1)	
Gleason score			0.53
No 4–5	807 (73)	79 (68)	
1–3/4–5	193 (17)	22 (19)	
4–5/1–5	112 (10)	15 (13)	
Mean Gleason score	6.2 ± 0.70	6.3 ± 0.72	0.76
Risk category			0.16
Low	580 (53)	51 (45)	
Intermediate	387 (35)	43 (38)	
High	136 (12)	20 (18)	
Mean CAPRA score	2.3 ± 1.42	2.4 ± 1.46	0.29
Comorbidities*			<0.01
None	239 (21)	19 (16)	
1–2	672 (60)	59 (50)	
≥3	205 (18)	39 (33)	
BMI (kg/m ²)			<0.01
Normal (<25.0)	291 (26)	19 (17)	
Overweight (25.0–29.9)	602 (54)	54 (48)	
Obese (≥30.0)	222 (20)	39 (35)	
Mean BMI	27.3 ± 3.74	28.9 ± 4.37	<0.01

KEY: DM = diabetes mellitus; PSA = prostate-specific antigen; CAPRA = Cancer of the Prostate Risk Assessment; BMI = body mass index.
Data presented as mean ± SD or numbers, with percentages in parentheses.
* Other than DM.

TABLE III. Disease-specific health-related quality of life by diabetes status over time (n = 1248)

Domain	No DM (n = 1131)	DM (n = 117)	Adjusted P Value DM vs. No DM	Adjusted P-value* (BMI)	Adjusted P Value* (DM × BMI)
Sexual function [†]			NS	0.07	NS
Before treatment	62.2 ± 26.15	46.1 ± 27.83			
Follow-up [‡]	32.1 ± 26.07	23.5 ± 24.33			
Sexual bother			NS	NS	NS
Before treatment	68.1 ± 35.00	50.7 ± 38.85			
Follow-up	41.0 ± 35.58	35.3 ± 38.5			
Urinary function			0.01	0.02	0.01
Before treatment	93.0 ± 12.72	92.2 ± 11.47			
Follow-up	77.0 ± 22.44	72.4 ± 24.46			
Urinary bother			NS	NS	NS
Before treatment	87.6 ± 21.28	82.4 ± 25.43			
Follow-up	82.3 ± 24.09	76.7 ± 29.13			
Bowel function			NS	NS	NS
Before treatment	88.6 ± 13.25	89.5 ± 14.20			
Follow-up	88.9 ± 13.47	88.9 ± 13.62			
Bowel bother			NS	0.09	NS
Before treatment	91.0 ± 18.65	92.5 ± 19.00			
Follow-up	89.1 ± 20.63	90.1 ± 20.29			

NS = not significant; other abbreviations as in Table II.

Data provided as mean ± SD.

* P values adjusted for age, race, educational level, relationship status, risk group, insurance type, and number of comorbidities other than DM.

[†] Scores on 6 subscales of Prostate Cancer Index range from 0 to 100, with higher scores indicating better functioning and less bother.

[‡] Follow-up questionnaire at 24 months after treatment was used if available; for men without 24-month data, last available follow-up questionnaire was used.

dicates that having DM and a high BMI may have greater impact on urinary function than having either alone.

Previous work on DM and HRQOL in men with prostate cancer has focused on radiotherapy patients. Among brachytherapy patients, DM exacerbated treatment-related ED.⁴ However, a small study of patients undergoing external beam radiotherapy found no relationship between DM and postradiotherapy potency.¹⁰ In two other studies, diabetic men receiving radiotherapy for prostate cancer reported significantly more late grade 2 gastrointestinal and genitourinary complications than did nondiabetic men.^{5,11} The measures used in these studies tapped the same types of bowel and urinary concerns we assessed with the UCLA-PCI.

Although obesity has been examined in terms of prostate cancer risk and disease recurrence, less work has focused on obesity and HRQOL.^{8,18} Anast *et al.*⁹ recently examined differences in HRQOL by weight using data for 672 CaPSURE participants treated surgically. No differences were found in sexual or urinary domains, but less bowel bother at diagnosis was reported by men with a greater BMI. Obese men also reported significantly more comorbidities. We also observed a trend toward less bowel bother for heavier men. Our results have extended earlier work by examining the importance of a specific comorbidity frequently associated with obesity—DM—on the HRQOL of men treated with radical prostatectomy.

The diabetic men differed from the nondiabetic men in several demographic characteristics, and one could argue that the demographic differences accounted for the reported differences in HRQOL. To address this concern, the repeated-measures model controlled for these demographic factors, as well as for risk group, number of other comorbid conditions, and insurance type. Income was not included in the repeated-measures model because that information is often not reported. We observed a significant difference in urinary function and trends in sexual function and bowel bother, even after controlling for these characteristics.

We had two related questions in this analysis: (a) does disease-specific HRQOL differ by diabetes history and (b) does the relationship between diabetes history and BMI affect disease-specific HRQOL? In both cases, we found significant differences only for urinary function. Diabetic men with a greater BMI showed a steeper decline in urinary function than did other men.

In this study, diabetic men reported worse urinary function (change score 16 versus 20), but their urinary bother was similar to that of nondiabetic men (change 5 versus 6). Function in many respects is a more “objective” outcome. The number of pads used and the degree of urinary control

are straightforward indicators of functional status and not necessarily that closely related to the patient’s perceptions and feelings. Perceived bother is a much more complex domain. A similar phenomenon of poorer function but less bother has been reported in men with ED.¹⁹ In that study, the investigators hypothesized that men with ED from prostate cancer can rationalize their functional deficits because they are cured of cancer. A similar phenomenon may have occurred in this study, in that men in both groups rationalized their functional deficits because they had been treated for their cancer.

Although the group differences over time were not significant, diabetic men had poorer sexual function at baseline and somewhat smaller changes in function after treatment. Such changes may be less troublesome than for nondiabetic men, who appear to experience a nonsignificant, but somewhat greater, decline in function. This pattern of men without comorbidities reporting greater decrements in HRQOL has been seen more clearly with other comorbid conditions.²⁰

Some limitations should be considered when interpreting our results. Although the CaPSURE database contains 4.5% African-American and 1.5% Latino men, as well as 2% of men of other ethnicities, the sample is not as ethnically diverse as the overall population of U.S. men with prostate cancer. Because it focuses on prostate cancer, the CaPSURE database does not include information such as the length of time since the DM diagnosis or glycolated hemoglobin readings, which would permit a more nuanced understanding of participants’ DM status. Additional research into the impact of DM on HRQOL should be done with sufficient information about DM status to determine whether the relationship between HRQOL and the presence or absence of DM is more clearly seen as the DM severity or interval since the diagnosis increases.

CONCLUSIONS

This study represents the first effort to characterize HRQOL among men with DM and prostate cancer treated with surgery. It also takes an important step toward furthering our understanding of the impact of obesity on HRQOL among men with prostate cancer. Our results indicate that the presence and absence of DM, as well as obesity, are important determinants of HRQOL in at least one domain—urinary function. Diabetic men who are obese should be counseled about the changes in urinary function likely to occur after surgery.

ACKNOWLEDGMENT. To Sonora Hudson, M.A., for her assistance in preparing the manuscript.

REFERENCES

1. Litwin MS, Hays RD, Fink A, *et al*: Quality-of-life outcomes in men treated for localized prostate cancer. *JAMA* 273: 129–135, 1995.
2. Penson DF, Latini DM, Lubeck DP, *et al*: Do impotent men with diabetes have more severe erectile dysfunction and worse quality of life than the general population of impotent patients? Results from the Exploratory Comprehensive Evaluation of Erectile Dysfunction (ExCEED) database. *Diabetes Care* 26: 1093–1099, 2003.
3. Centers for Disease Control and Prevention: *Diabetes: Disabling, Deadline, and on the Rise*. Atlanta, Coordinating Center for Health Promotion, Centers for Disease Control and Prevention, 2006.
4. Merrick GS, Wallner KE, and Butler WM: Management of sexual dysfunction after prostate brachytherapy. *Oncology (Huntingt)* 17: 52–62; 67–70, 73, 2003.
5. Herold DM, Hanlon AL, and Hanks GE: Diabetes mellitus: a predictor for late radiation morbidity. *Int J Radiat Oncol Biol Phys* 43: 475–479, 1999.
6. Mokdad AH, Ford ES, Bowman BA, *et al*: Prevalence of obesity, diabetes, and obesity-related health risk factors, 2001. *JAMA* 289: 76–79, 2003.
7. Amling CL, Riffenburgh RH, Sun L, *et al*: Pathologic variables and recurrence rates as related to obesity and race in men with prostate cancer undergoing radical prostatectomy. *J Clin Oncol* 22: 439–445, 2004.
8. Porter MP, and Stanford JL: Obesity and the risk of prostate cancer. *Prostate* 62: 316–321, 2005.
9. Anast JW, Sadetsky N, Pasta DJ, *et al*: The impact of obesity on health related quality of life before and after radical prostatectomy (data from CaPSURE). *J Urol* 173: 1132–1138, 2005.
10. Selek U, Cheung R, Lii M, *et al*: Erectile dysfunction and radiation dose to penile base structures: a lack of correlation. *Int J Radiat Oncol Biol Phys* 59: 1039–1046, 2004.
11. Schultheiss TE, Lee WR, Hunt MA, *et al*: Late GI and GU complications in the treatment of prostate cancer. *Int J Radiat Oncol Biol Phys* 37: 3–11, 1997.
12. Lubeck DP, Litwin MS, Henning JM, *et al*, for the Cancer of the Prostate Strategic Urologic Research Endeavor (CaPSURE) Research Panel: The CaPSURE database: a methodology for clinical practice and research in prostate cancer. *Urology* 48: 773–777, 1996.
13. Cooperberg MR, Broering JM, Litwin MS, *et al*: The contemporary management of prostate cancer in the United States: lessons from the cancer of the prostate strategic urologic research endeavor (CaPSURE), a national disease registry. *J Urol* 171: 1393–1401, 2004.
14. Litwin MS, Hays RD, Fink A, *et al*: The UCLA Prostate Cancer Index: development, reliability, and validity of a health-related quality of life measure. *Med Care* 36: 1002–1012, 1998.
15. D'Amico AV, Whittington R, Malkowicz SB, *et al*: Biochemical outcome after radical prostatectomy, external beam radiation therapy, or interstitial radiation therapy for clinically localized prostate cancer. *JAMA* 280: 969–974, 1998.
16. Cooperberg MR, Pasta DJ, Elkin EP, *et al*: The University of California, San Francisco Cancer of the Prostate Risk Assessment score: a straightforward and reliable preoperative predictor of disease recurrence after radical prostatectomy. *J Urol* 173: 1938–1942, 2005.
17. Aiken LS, and West SG: *Multiple Regression: Testing and Interpreting Interactions*. Thousand Oaks, CA, SAGE Publications, 1991.
18. Freedland SJ, Terris MK, Presti JC Jr, *et al*: Obesity and biochemical outcome following radical prostatectomy for organ confined disease with negative surgical margins. *J Urol* 172: 520–524, 2004.
19. Penson DF, Latini DM, Lubeck DP, *et al*: Is quality of life different for men with erectile dysfunction and prostate cancer compared to men with erectile dysfunction due to other causes? Results from the ExCEED data base. *J Urol* 169: 1458–1461, 2003.
20. Arredondo SA, Elkin EP, Marr PL, *et al*: Impact of comorbidity on health-related quality of life in men undergoing radical prostatectomy: data from CaPSURE. *Urology* 67: 559–565, 2006.

APPENDIX

The current CaPSURE Investigators are Peter R. Carroll, M.D., University of California, San Francisco, San Francisco, CA; James S. Cochran, M.D., Urology Clinics of North Texas, Dallas, TX; Christopher J. Kane, M.D., Veterans Affairs Medical Center, San Francisco, CA; Donald P. Finnerty, M.D., PAPP Clinic, Newnan, GA; Eugene V. Kramolowsky, M.D., Virginia Urology Center, Richmond, VA; Robert M. Segaul, M.D., Urology Associates of West Broward Belle Terre, Sunrise, FL; Paul Sieber, M.D., Urological Associates of Lancaster, Lancaster, PA; Stanley A. Brosman, M.D., Pacific Clinical Research, Santa Monica, CA; Lynn W. Conrad, M.D., Urology Center of the South, PC, Memphis, TN; Joseph N. Macaluso, Jr., M.D., Urologic Institute of New Orleans, Gretna, LA; Michael Flanagan, M.D., Urology Specialists, Waterbury, CT; Jeffrey K. Cohen, M.D., Triangle Urology Group, Pittsburgh, PA; Jerrold Sharkey, M.D., Urology Health Center, New Port Richey, FL; Thomas W. Coleman, M.D., Mobile Urology Group, Mobile, AL; Elliott C. Silbar, M.D., Clinic of Urology, Milwaukee, WI; Paul S. Ray, D.O., Cook County Hospital, Chicago, IL; David Noyes, M.D., Berkshire Urological Associates, PC, Pittsfield, MA; Mohammed Mostafavi, M.D., Urology Group of Western New England, Springfield, MA; Louis Keeler, III, M.D., Center for Urologic Care, Voorhees, NJ; James Gottesman, M.D., Seattle Urological, Seattle, WA; Bhupendra M. Tolia, M.D., Associated Advanced Adult & Pediatric Urology, Bronx, NY; W. Lamar Weems, M.D., Mississippi Urology, Jackson, MS; Glen Wells, M.D., Alabama Urology, Birmingham, AL; Richard J. Kahnoski, M.D., Michigan Medical, Grand Rapids, MI; Sheldon J. Freedman, M.D., Las Vegas, NV; Randil Clark, M.D., North Idaho Urology, Coeur D'Alene, ID; David Penson, M.D., M.P.H., Veterans Affairs Puget Sound HCS, Seattle, WA; Mark Austenfeld, M.D., Kansas City Urology Care, Kansas City, MO; Henri P. Lanctin, M.D., Adult & Pediatric Urology, St. Cloud, MN; J. Brantley Thrasher, M.D., University of Kansas, Kansas City, KS; and David W. Bowyer, M.D., Snake River Urology, Twin Falls, ID. Former CaPSURE investigators were John Forrest, M.D., 1995–1999, Urologic Specialists of Oklahoma, Tulsa, OK; William Schmeid, M.D., 1995–1999, Metro Urology, Jeffersonville, IN; Glen Brunk, M.D., 1995–1999, Urology of Indiana, Indianapolis, IN; Jay Young, M.D., 1995–2001, South Orange County Medical Research Center, Laguna Woods, CA; Gary Katz, M.D., 1996–2000, Medical College of Virginia and Veterans Affairs Medical Center, Richmond, VA; Stacy J. Childs, M.D., 1999–2000, Cheyenne Urological, Cheyenne, WY; Kevin Tomera, M.D., 1999–2001, Alaska Urological Associates, Anchorage, AK; Clayton Hudnall, M.D., 1995–2002, Urology San Antonio Research, San Antonio, TX.