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Prostate Specific Antigen Testing Among the Elderly—When To Stop?

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Abstract

Purpose—Prostate specific antigen testing is common in the elderly despite evidence that older men without aggressive prostate cancer are unlikely to benefit from diagnosis and treatment. We evaluated the relationship between prostate specific antigen and the risk of aggressive prostate cancer developing in men of various ages.

Materials and Methods—This longitudinal cohort study consisted of 849 men (122 with and 727 without prostate cancer) with serial prostate specific antigen measurements participating in the Baltimore Longitudinal Study of Aging. The primary outcome measure was the proportion of men by prostate specific antigen and age who died of prostate cancer or in whom aggressive prostate cancer developed (death from prostate cancer, a prostate specific antigen 20 ng/ml or greater, or Gleason score 8 or greater).

Results—No participants between 75 and 80 years old with a prostate specific antigen less than 3.0 ng/ml died of prostate cancer. In contrast, men of all ages with a prostate specific antigen of 3.0 ng/ml or greater had a continually increasing probability of death from prostate cancer (Fisher's exact test $p < 0.001$). The time to death or diagnosis of aggressive prostate cancer after age 75 years was not significantly different between the prostate specific antigen categories of 3 to 3.9 and 4 to 9.9 ng/ml ($p = 0.634$), whereas the time to death or diagnosis of high risk prostate cancer was significantly longer for the prostate specific antigen category of less than 3 vs 3 ng/ml or greater ($p = 0.019$).

Conclusions—Men 75 to 80 years old with a prostate specific antigen less than 3 ng/ml are unlikely to die of or experience aggressive prostate cancer during their remaining life, suggesting that prostate specific antigen testing might be safely discontinued for these men.

Keywords

prostate-specific antigen; early detection of cancer; prostatic neoplasms; aged

The widespread use of prostate specific antigen has resulted in the detection of prostate cancer at an earlier stage and has most likely contributed to a 30% decrease in prostate cancer mortality through 2001.¹ However, due to the lead time associated with PSA based screening it has been

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suggested that up to 30% of prostate cancers detected in the contemporary era may have otherwise remained clinically silent during life.² Of men diagnosed with prostate cancer more than 85% undergo active treatment and, thus, it is possible that there was unnecessary treatment of cancers that would have remained undetected in the absence of PSA testing.³

Older men with low risk disease may be at risk for unnecessary treatment due to an increased possibility of competing causes of death.⁴ Nevertheless, PSA screening rates in older men remain high.^{5,6} One survey demonstrated that the prevalence of PSA screening within the last year was higher in men older than 80 years than in those 50 to 59 years old (56% vs 48%).⁵ Another study revealed high rates of screening even in men 85 years or older with limited chances of surviving 10 years.⁶ This finding suggests that for some men who undergo screening, treatment of pCA is unlikely to alter mortality.

Although routine prostate cancer screening in men older than 75 years is not endorsed by some professional organizations,⁷ high rates of screening in the elderly may relate to concern for potentially missing lethal cancer by not screening.⁸ Thus, evidence showing a lack of adverse outcomes in the absence of screening could reduce unnecessary screening. As baseline PSA levels are predictive of the development of cancer for up to 2 to 3 decades after the test, we hypothesized that PSA in elderly men might also be associated with the probability of lethal cancer developing later in life, thereby informing a decision about the need for further PSA testing.⁹

METHODS

Study subjects were participants in the Baltimore Longitudinal Study of Aging, a prospective institutional review board approved cohort study sponsored by the National Institute on Aging (Bethesda, Maryland).¹⁰ Since 1958 this open enrollment study has included 1,806 men from the Baltimore, Maryland and Washington, D. C. regions of the United States.

Since 1991 PSA measurements have been performed at each evaluation. Participants with a PSA greater than 4.0 ng/ml and/or an abnormality on digital rectal examination underwent a transrectal ultrasound directed prostate biopsy. PSA before 1991 was measured using frozen sera samples stored at -70°C. All PSA measurements were performed using a standard monoclonal immunoradiometric assay (TandemTM-R, Hybritech Inc., San Diego, California).

Study Cohort

Figure 1 shows selection criteria for the study. Of the 1,806 men in the BLSA 1,201 had PSA measurements available. The study cohort consisted of men 40 years or older, excluding those with a diagnosis of prostate cancer with no PSA data before diagnosis (38), those who underwent simple prostatectomy for prostate enlargement with no PSA data before prostate surgery (80), those taking finasteride (Proscar®) at any time (47), those with an unknown cause of death (54), those with incomplete diagnostic information (2), those with a single PSA that was suspected to be a laboratory error (1) and those with no PSA data after age 40 years (130). After exclusions the study population consisted of 849 men of whom 122 had a diagnosis of prostate cancer. Of these men 79% were white, 17% were black and 4% were Asian or other ethnic groups.

The outcome of interest in this study was the development of high risk pCA based on PSA and age. We first defined this outcome as death from prostate cancer. Of 849 participants 18 (2%) had confirmed death due to prostate cancer at a median age of 83 years. Median year of diagnosis was 1990 and median year of death was 1993.9. This proportion of cancer deaths is similar to the 2% probability of death from prostate cancer by age 85 years for white men in the United States in the Surveillance, Epidemiology and End Results Program

(<http://www.seer.cancer.gov>). Cause of death was determined by intermittent telephone followup of inactive participants, correspondence from relatives and searches of the National Death Index.

As advanced prostate cancer can be associated with significant morbidity from sequelae such as bony metastases and hormonal therapy, we also examined a more expanded high risk definition that included subjects with a PSA of 20 ng/ml or greater (12), Gleason score 8 or greater (5), or death from prostate cancer (18). Prior studies have shown that a PSA greater than 20 ng/ml and Gleason score 8–10 are not only associated with a high risk of death from prostate cancer¹¹ but also with a greater likelihood of systemic progression with its associated morbidity.¹² Of the 122 men with a diagnosis of prostate cancer 35 were considered to have high risk disease using these criteria. Of these men 6 (17%) were alive and 29 (83%) had died of any cause as of August 31, 2006. The stage and grade of cancer were not consistently available for all subjects with a diagnosis of prostate cancer and, therefore, these subjects were not included in analyses.

Of 727 men without a diagnosis of prostate cancer 542 (75%) were alive and 185 (25%) were deceased. Of these 185 men 24 (3.3%) were diagnosed with prostate cancer at autopsy. Autopsy detected prostate cancers were included in the group without prostate cancer because autopsies were not performed routinely in all men in this cohort, and those with autopsy detected prostate cancer were unaware of a diagnosis of prostate cancer during their lifetime.

Statistical Analyses

Study characteristics were compared using pooled t tests in the case of equal variances and Cochran t tests in the case of unequal variances. All statistical tests were 2-sided with $p < 0.05$ considered statistically significant. Subjects at high risk for death from prostate cancer were considered events in all analyses.

PSA measures were censored at diagnosis of prostate cancer, at simple prostatectomy for benign disease, at death for those subjects without a diagnosis of prostate cancer or as of August 31, 2006 for those without prostate cancer who were alive. We stratified data by age decade and PSA to compare followup times between groups. Followup time was the time to death or diagnosis of high risk disease for those alive, and at administrative censorship in August 2006 for the remainder of the population. We compared PSA distributions by age decade between subjects in the BLSA and published data from the NHANES using the Kolmogorov-Smirnov 2-sample test.¹³

Beginning at age 60 to 65 years and for each 5-year age period to age 80 years or older we determined the probability of high risk prostate cancer (event) subsequently developing in a given age range by PSA cutoffs of less than 1, less than 1.5, less than 2, less than 2.5, less than 3 and 3.5 ng/ml or greater. We chose these cutoffs because urologists are increasingly concerned about the presence of prostate cancers in men with PSA values less than 4.0 ng/ml and because there is no PSA value below which a diagnosis of prostate cancer can be excluded.¹⁴ The probability of an event was calculated as the ratio of the number of subjects diagnosed with high risk prostate cancer within a given age interval-to-the total number of subjects in the same interval.

The distribution of time to event stratified by PSA was estimated using the Kaplan-Meier method. In this analysis time 0 was the first visit after age 75 years. We focused on age 75 years because average life expectancy is approximately 10 years.¹⁵ The natural history of nonhigh risk pCA is to behave in a relatively indolent fashion for a decade or more, making it unlikely that the diagnosis and treatment of such a cancer would be beneficial.⁴ Poisson regression was used to test for equality of survivor functions.

RESULTS

Comparisons between cohorts with and without the diagnosis of prostate cancer were not statistically significantly different except age at last PSA and age at death or censoring were greater for those subjects with prostate cancer compared to those without prostate cancer (table 1). Subjects without prostate cancer had shorter times from first to last PSA and from last PSA to censoring than men with a diagnosis of prostate cancer. The number of repeat PSA measurements and PSAs at first and last visits were significantly greater for men with prostate cancer compared to those without the disease. Followup time by PSA strata within age groups was similar except for subjects 60 to 69 years old with a PSA less than 1 vs 1 to 1.9 ng/ml ($p = 0.03$), and with a PSA less than 1 vs 3 ng/ml or greater ($p = 0.004$). PSA distributions by age decade were also similar when comparing data from the BLSA to population based data from the NHANES, suggesting that the PSA data in the BLSA are representative of the United States population (fig. 2).

In the BLSA men across all age groups and PSA values were diagnosed with prostate cancer (fig. 3, gray triangles, and table 2). However, of the 154 participants older than 75 years with a PSA less than 3 ng/ml none died of prostate cancer and high risk prostate cancer developed in only 1. This participant had a PSA of 2.9 ng/ml at age 75 years and did not die of prostate cancer. There were an additional 5 men with prostate cancer who had a PSA of 3 ng/ml or less after age 75 years (table 2). None of these 5 men had high risk disease. Four men died of causes other than prostate cancer and 1 is alive at age 85, 8 years after prostate cancer diagnosis.

Of the 108 subjects older than 75 years with a PSA of 3 ng/ml or greater 10 died of prostate cancer and 18 had high risk disease (table 2). In this group 90 men did not have a diagnosis of high risk prostate cancer, including 75 who were never diagnosed with cancer (median time to censoring 12.5 years) and 15 who were diagnosed with nonhigh risk cancer (median time to censoring 17 years).

In the men with a PSA of 3 ng/ml or greater at age 75 to 80 years, the probability of later death from prostate cancer continued to increase whereas no subject 75 to 80 years old with a PSA less than 3.0 ng/ml died of prostate cancer (Fisher's exact test $p < 0.001$) (fig. 4, A). Similarly in subjects with high risk prostate cancer there remained a significant difference in the probability of high risk disease for those with a PSA of 3 ng/ml or greater vs less than 3.0 ng/ml (Fisher's exact test $p < 0.001$) (fig. 4, B). A probability graft of various PSA cut points also demonstrates the transition to an increased probability of death or high risk prostate cancer above the PSA cut point of 3.0 ng/ml (fig. 5).

The distribution of time from the first visit after age 75 years (time 0) to death from prostate cancer or the development of high risk disease is shown by the PSA categories of less than 3.0, 3 to 3.9 and 4 to 9.9 ng/ml (fig. 6). The time to diagnosis or death was not significantly different between the PSA categories of 3 ng/ml or more ($p = 0.634$) but was different, in contrast, for the PSA categories of less than 3 vs 3 ng/ml or greater ($p = 0.019$).

DISCUSSION

Professional organizations have published varying recommendations on prostate cancer screening. The American Cancer Society and American Urological Association recommend annual screening beginning in the 40s for high risk and in the 50s for average risk men.^{16–18} In contrast, the U.S. Preventive Services Task Force states that the balance of benefits and harms with screening cannot be determined for men younger than 75 years.⁷ They also state that it is likely that the harms outweigh the benefits for men older than 75 years.

This issue of the age at which screening should be discontinued remains controversial and there are few published data to guide this question. According to the American Cancer Society screening is appropriate for individuals with at least a 10-year life expectancy. However, an estimation of life expectancy can be challenging in the individual patient and, thus, it would be useful if objective variables were available to inform decisions.

In this regard we found that men with a PSA less than 3 ng/ml at age 75 to 80 years are unlikely to experience life threatening prostate cancer during their remaining years of life compared to those with a PSA of 3 ng/ml or greater. In our cohort the PSA distributions by age and the lifetime risk of death from prostate cancer were similar to those of the general United States population. Thus, if confirmed, these findings may be useful in determining more concrete guidelines for when PSA based screening might be safely discontinued.

It is noteworthy that many studies have reproducibly shown the strong relationship between baseline PSA and subsequent prostate cancer diagnosis. Thus, our finding that a PSA later in life is predictive of subsequent lethal disease is not surprising.

Gann et al were the first to our knowledge to show that a single PSA measurement was associated with the risk of a prostate cancer diagnosis in the next 10 years.¹⁹ In their analysis prostate cancer risk increased incrementally with PSA. Lilja et al subsequently demonstrated that a baseline PSA test at age 44 to 50 years was predictive of a prostate cancer diagnosis up to 25 years later.⁹ The authors suggested that these data could be used for risk stratification in prostate cancer screening programs.

Finally we previously reported that a low PSA in older men could be used to reduce the intensity of screening without increasing the rate of undetected prostate cancer.²⁰ Our current findings go beyond this observation to suggest that the risk of a diagnosis of lethal prostate cancer is lower than the lifetime risk of death from prostate cancer in the United States if PSA is less than 3 ng/ml at age 75 to 80 years. Thus, discontinuation of routine PSA screening in these men may not increase the rates of undetected lethal disease. However, we cannot conclude that further PSA testing would not be beneficial for symptomatic men regardless of PSA and age.

Strengths of our study include the cohort design, involving men with clinical characteristics similar to those of the general United States population and the extended longitudinal followup. Limitations include the use of frozen sera for PSA assay and the inability to assess prostate cancer stage and grade in all diagnosed cases. Also, although a majority of deaths occurred at the beginning of the PSA era (early 1990s), the potential impact of treatment on prostate cancer mortality is unknown. In addition, our results may not be applicable to a population with a different racial distribution.

CONCLUSIONS

The optimal approach to prostate cancer screening remains controversial. To date there is limited evidence with which to inform the decision of when to discontinue prostate cancer screening. Our findings suggest that men 75 to 80 years old who have a PSA less than 3 ng/ml are unlikely to be diagnosed with high risk prostate cancer during life. Therefore, these men may represent an ideal target group for discontinuation of PSA testing, which could dramatically reduce the costs associated with screening, as well as the potential morbidity of additional evaluations and/or treatment in a population unlikely to experience benefit.

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Abbreviations and Acronyms

BLSA

Baltimore Longitudinal Study of Aging

NHANES

National Health and Nutrition Examination Survey

pCA

prostate cancer

PSA

prostate specific antigen

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Figure 1.

Selection criteria for study population. High risk pCA definition 1—men who died of prostate cancer. High risk pCA definition 2—men with PSA 20 ng/ml or greater (12), Gleason score 8 or greater (5), or prostate cancer as underlying cause of death (18). *BPH*, benign prostatic hyperplasia.

Figure 2.

Kernel density plot of PSA distribution for NHANES data (broken line) and BLSA (solid line) data for ages 50 to 59 (A), 60 to 69 (B), 70 to 79 (C) and 80 years or older (D). Two-sample Kolmogorov-Smirnov test was used for comparison.

Figure 3.

Relationship between PSA and age for subjects without prostate cancer (dots), subjects with high risk prostate cancer who died of disease (closed circles), those with PSA greater than 20 ng/ml or Gleason score 8 or more (open circles), and those with prostate cancer not considered high risk (triangles). Each mark represents PSA at subject visit so that subject could be represented more than once. For simplicity figure does not show data on subjects without high risk prostate cancer who did not have PSA information before age 75 years. Lower right panel represents study group (PSA less than 3.0 ng/ml and older than 75 years).

Figure 4.

Probability of death from prostate cancer (A). For example, probability of death from prostate cancer in subject with PSA 3 ng/ml or greater after age 70 to 75 years was approximately 7%. Probability of high risk prostate cancer (B) (death from prostate cancer, or PSA 20 ng/ml or more, or Gleason score 8 or more).

Figure 5.

Probability plot of death (A) or development of high risk prostate cancer (B) at age 75 years with PSA cut points as indicated along x-axis. For example, probability of life threatening prostate cancer developing in subject with PSA less than 3 ng/ml was approximately 3.4%. For each of PSA cut points 95% CIs were determined using bootstrap approach. Note confidence intervals for probability of high risk disease with PSA greater than 3.5 ng/ml do not overlap with confidence intervals for lower PSAs.

Figure 6.

Kaplan-Meier estimate of time to death from prostate cancer (A) or high risk prostate cancer (B) from first subject visit after age 75 years. Time to diagnosis of high risk prostate cancer was not significantly different between PSA categories of 3 ng/ml or more ($p = 0.634$). Time to diagnosis of high risk prostate cancer differed significantly for PSA less than 3 vs 3 ng/ml or greater ($p = 0.019$). PSA less than 3 (solid line), 3 to 3.9 (broken line) and 4 to 10 (dot dash line) ng/ml were measured at visit after age 75 years.

Table 1

Description of diagnostic groups*

	Total	Ca	No Ca	p Value Comparisons Ca vs No Ca
No. subjects	849	122	727	
Age at first PSA test:				
Median (range)	53 (40–92)	55.6 (40.1–86)	52.4 (40–92)	
Mean (SD)	55.2 (11.8)	56.8 (11.5)	54.9 (11.9)	0.120
Age at last PSA:				
Median (range)	66.8 (40–95.3)	69.7 (47.2–88.2)	66.4 (40–95.3)	
Mean (SD)	66.1 (12.7)	69.4 (9.4)	65.5 (13.1)	<0.001
Yrs followup from first to last PSA test:				
Median (range)	10.5 (0–38.8)	17 (0–30.6)	9.9 (0–38.8)	
Mean (SD)	11.3 (8.9)	15.8 (8.9)	10.6 (8.7)	<0.001
Time from last PSA or Ca diagnosis to death or censoring:*				
Median (range)	6.1 (0–39.1)	8.6 (0–24.6)	5.9 (0–39.1)	
Mean (SD)	7.4 (5.3)	9 (4.7)	7.1 (5.4)	<0.001
No. PSA values repeated:				
Median (range)	4 (1–15)	5 (1–15)	4 (1–14)	
Mean (SD)	4.7 (3)	5.4 (3.3)	4.6 (2.9)	0.005
Yrs between PSA tests:				
Median (range)	2.1 (0.1–21.5)	2 (0.1–12.7)	2.1 (0.1–21.5)	
Mean (SD)	2.9 (2.1)	2.9 (2.2)	2.9 (2)	0.456
PSA (ng/ml) at first visit:				
Median (range)	0.76 (0.05–18.97)	1.25 (0.1–17.1)	0.7 (0.05–18.97)	
Mean (SD)	1.41 (1.95)	2.38 (2.95)	1.25 (1.67)	0.001
PSA (ng/ml) at last visit:				
Median (range)	1.5 (0.05–351.9)	5.1 (0.3–351.9)	1.23 (0.05–18.97)	
Mean (SD)	3.6 (14.4)	13 (36.3)	2.02 (2.29)	0.001
Age at diagnosis:				
Median (range)	Not applicable	72.7 (47.1–90)	Not applicable	
Mean (SD)		72.5 (9.2)		
Age at death/censoring:†				
Median (range)	75.2 (44.5–102.2)	83.1 (57.5–102.2)	73.1 (44.5–100)	<0.001
Mean (SD)	73.9 (12.9)	81.5 (9.4)	72.6 (12.9)	

* Time from last PSA test to censoring for those without pCA, or time from pCA diagnosis to death or censoring for those with prostate cancer.

† Age at death for deceased subjects, age at censoring for subjects still living.

Table 2
Men with PSA histories stratified by age and PSA

	No Ca	Nonhigh Risk Ca	Death From pCA	High Risk pCA
No. younger than 75 yrs:				
PSA less than 3.0 ng/ml	625	65	13	26
PSA 3.0 ng/ml or greater	127	58	10	20
No. older than 75 yrs:				
PSA less than 3.0 ng/ml	148	5	0	1
PSA 3.0 ng/ml or greater	75	15	10	18

A subject in 1 row could be represented in additional rows as/if age or PSA increased. Subjects without high risk pCA who did not have PSA data before age 75 years were excluded from table for simplicity.