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A POPULATION-BASED CASE-CONTROL STUDY OF PSA AND DRE SCREENING ON PROSTATE CANCER MORTALITY

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Abstract

Objectives—The efficacy of screening for prostate cancer (PC) with digital rectal exam (DRE) and prostate specific antigen (PSA) has not been proven in randomized clinical trials. In an earlier case-control study we found that DRE may reduce PC mortality. This case-control study assesses the association between both PSA and DRE testing and prostate cancer mortality.

Methods—Case subjects included 74 Olmsted County residents who died from 1992-2005 with PC as the underlying cause of death. From one to three community control subjects (alive at time of case's death) were matched to each case. Medical records were reviewed to identify DRE's and PSA determinations performed from 0 to 5 years before the date the case was diagnosed (index date). Tests performed in the absence of symptoms were considered to be "screening." Conditional logistic regression was used to estimate odds ratios (OR) and 95% confidence intervals (CI) for the association of screening (defined in multiple ways) and PC mortality.

Results—From 1-5 years before the index date, control subjects were more likely than case subjects to have had a prior screening PSA or DRE (81.3% vs. 60.8%, p = 0.0005). The unadjusted OR (95% CI) associated with a prior screening PSA or DRE was 0.34 (0.18, 0.63), while the OR adjusted for potential confounders was 0.35 (0.17, 0.71). PSA testing was frequently done in conjunction with DRE, making evaluation of individual effects difficult.

Conclusion—This case-control study suggests a potential benefit of screening by PSA and/or DRE on prostate cancer mortality.

Keywords

screening; prostate-specific antigen; digital rectal examination; prostate cancer; mortality

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INTRODUCTION

Prostate cancer (PC) remains the most common non-dermatologic cancer among US males, with over 234,000 new cases estimated in 2006.[1] It is also the third leading cause of cancer death among men, with over 27,000 deaths in 2006. The early detection of aggressive PC, when it is theoretically more amenable to curative treatment, is generally considered desirable. Further, if screening is effective, it should lead to a decrease in population-based PC mortality rates.

Screening for PC is controversial as most men with PC do not die from it, and no randomized trials have proved its efficacy in reducing mortality. The recommended screening methods are digital rectal examination (DRE) and measurement of serum prostate specific antigen (PSA) levels. While PC mortality rates have declined since 1990,[1,2] factors other than increased use of PSA may be responsible. These include increased use of prostatectomy and hormonal therapy, both of which have been shown to be effective in randomized trials.[3,4] Two large randomized screening trials, the Prostate, Lung, Colorectal and Ovarian (PLCO) Cancer Screening Trial and the European Randomized Study of Screening for Prostate Cancer (ERSPC) are underway, but the results may not be known for several years.[5,6] In view of this uncertainty, varying guidelines exist regarding when or if to screen for PC.[7,8]

In the absence of data from randomized trials, case-control studies in which PC screening history of men who died of PC (case subjects) is compared to that of surviving men (control subjects), can be used to estimate the effectiveness of PC screening.[9] Among case-control studies that have evaluated the use of DRE, one suggested a benefit [10] while two did not.[11, 12] Three of four case-control studies performed during the PSA era found screening to be beneficial[13-15] and one did not.[16] However, few of these studies were population-based and some relied partly on recall of screening. The goal of this project was to extend our prior work[10] by conducting a population-based case-control study using medical records to estimate the effectiveness of screening by PSA testing and/or DRE in reducing PC mortality.

MATERIAL AND METHODS

STUDY SETTING

This is a population-based, case-control study of PC screening in Olmsted County, Minnesota. Epidemiologic studies in Olmsted County are possible because its population is served by a largely unified medical care system which has accumulated comprehensive clinical records over a long period of time. Through the Rochester Epidemiology Project (REP), a medical records linkage system, all community medical records for county residents from the few providers of care in the county are easily retrieved for research purposes.[17] The 2000 county decennial census population was 124,277 with 90% being white. With the exception of a higher proportion employed in health care, county demographics are similar to those of United States whites.

IDENTIFICATION OF CASE SUBJECTS

Resources of the REP[17] were used to identify all Olmsted County residents who died from 1/1/1992 through 3/31/2005 with PC listed on their death certificate as the direct or underlying cause of death (Part I). Further, to assess the impact of PSA testing (began in 1987 in Olmsted County), only cases with a PC diagnosis in 1987 or later were included. Once identified, the complete community (inpatient and outpatient) medical records were reviewed to verify the diagnosis of PC, establish a date of diagnosis, and confirm residence in Olmsted County at the time of death and at the date of diagnosis of PC. Criteria for diagnosis included: 1) autopsy-proven gross or histologic evidence of PC, and 2) biopsy-proven histologic evidence of PC.

The date of diagnosis (index date) was assigned to each case as the date of first confirmatory evidence of PC.

SELECTION OF CONTROL SUBJECTS

Once the diagnosis of PC was confirmed and residency verified for each case, potential controls (men alive at the time of the case's death) were identified from an enumeration of the Olmsted County population through the records linkage system of the REP.[17] Controls were required to have been seen at the Mayo Clinic within ±1 year of the case subject's diagnosis, be male and to be alive at the time of the case's death. For each case, a computerized matching algorithm was used to select a maximum of five potential controls who had a birth year within ±1 year of the case's, and year of first ever clinic registration within 5 years of that of the matched case.[18] Once selected, the medical records of potential controls were reviewed to verify Olmsted County residence at time of the case's index date and to confirm the absence of PC on or before the index date. While controls could not have had PC prior to their matched case's date of diagnosis, they could have been later diagnosed with PC. This screening process resulted in 192 controls; 7 cases had 1 control, 16 had 2, and 51 had 3 matched controls. PC subsequent to the index date occurred in 15 controls, none of whom later died of PC.

SCREENING (EXPOSURE) ASSESSMENT

The community medical records of case and control subjects were reviewed by a single trained nurse abstractor (blinded to case/control status) to record dates of each DRE and serum PSA determination. In addition, pertinent DRE findings such as estimated prostate size, consistency (nodular, hard, soft), symmetry, and possible indications for the DRE including notation of obstructive urinary symptoms, hematuria, bone pain, pelvic pain, and penis pain were recorded. At each visit a test was defined as screening if performed in the absence of documented concurrent obstructive urinary symptoms or PC-related symptoms. Further, a DRE performed in an asymptomatic man in response to an elevated PSA, or a PSA test performed in response to abnormal DRE findings, was not considered a screening test. The retrospective coding of tests as screening or not is subject to misclassification if the test was done for symptoms and/or clinical findings that were not noted in the medical record. The potential for multiple tests leading to the diagnosis of PC in the cases resulted in an *a-priori* decision to focus on tests done 1-5 years prior to the index date. Others have approached this problem by considering screening tests as those performed prior to the "symptom reference date", the date on which prostate cancer was first suspected in the case.[14,16] The rationale for this is that once a patient exhibits symptoms or PC is suspected, he is no longer a candidate for screening. Thus, we determined the symptom reference date for each case as the earliest date of hematuria, bone pain or perineal pain within 5-years prior to the index date. Hence, we also defined screening as a serum PSA determination or DRE (in the absence of symptoms) prior to the case's symptom reference date. To yield similar potential screening intervals, the case's symptom reference date was also used for their matched control. We previously demonstrated high intra-abstractor reliability regarding the presence of abnormal DRE findings (100% based on a re-abstraction of data for 41 cases after a three-month interval) for the nurse abstractor in this study.[10]

POTENTIAL CONFOUNDERS

Age and duration of medical record, two major potential confounders of the association between screening and PC deaths, were controlled for through the matching process. The marital status at the index date and educational attainment of each man were recorded. Comorbidity was assessed by calculating the Charlson score using information from the diagnostic medical index of the REP,[17] excluding PC.

STATISTICAL METHODS

Consistent with the study design, the association between screening and PC mortality was assessed using conditional logistic regression models and reported as odds ratios (OR) and 95% confidence intervals (CI), with adjustment for marital status, Charlson comorbidity score, and education. OR's <1 suggest a benefit to screening, and if the upper limit of the 95% CI is <1, the benefit is significant at the 0.05 alpha level.

RESULTS

GROUP COMPARISONS

Due to matching criteria, the 74 cases and 192 controls were similar regarding mean birth year (1918.2 and 1919.6, respectively), duration of medical record (42.0 and 42.4 yrs.), index year (1993.2 and 1993.3). Mean (SD) age at index date was 74.4 (10.4) for cases and 73.2 (10.0) for controls, while age at death for cases was 79.4 (10.1) years. Age at last follow-up for controls was longer at 82.1 (9.2) years, as controls were required to be alive at time of their matched case's death. Controls were slightly, but not significantly, more likely to be married/have a partner (80% vs. 73%, p = 0.24) and to have completed college (36% vs. 32%, p = 0.59), and more frequently had Charlson comorbidity scores of 2 or higher (37% vs. 21%, p = 0.01). For the 74 PC cases, clinical stage at diagnosis was T1, T2, T3, and metastatic (T4 or N+) for 6, 30, 8, and 29, respectively, with one not available. The median PSA level at time of diagnosis was 22 ng/ml, with 37% having a PSA >50 ng/ml. Initial PC therapy within 90 days of diagnosis was hormonal alone for 38, hormonal with radiation or surgery for 10, surgery alone for 7, radiation alone for 5, and none for 14.

PSA/DRE TESTING 1-5 YEARS PRIOR

The frequency of DRE and serum PSA testing is summarized in Table I. From 1-5 years before the index date, control subjects were generally more likely than case subjects to have had a screening DRE (78.7% vs. 56.8%, p=0.0003), screening PSA (39.6% vs. 24.3%, p=0.02), or either (81.3% vs. 60.8%, p=0.0005). Nearly all serum PSA determinations were done in conjunction with a DRE, making interpretation of separate effects difficult. The unadjusted OR (95% CI) associated with a screening PSA or DRE from 1-5 years before the index date was 0.34 (0.18, 0.63), and remained significant and essentially the same (0.35 [0.17, 0.71]) with adjustment for marital status, education, and Charlson score. Counting all tests (screened + symptomatic) within the 1-5 year period prior to the index data, 85% of controls and 72% of cases had a DRE or PSA, OR=0.41(95%CI 0.21,0.80).

PSA/DRE TESTING IN OTHER INTERVALS

With a narrowing of the screening window to 2-5 years prior to the case's index date, a highly significant, protective adjusted odds ratio of 0.50 (0.26, 0.94) was observed. However, with expansion of the screening window to include screening PSA or DRE tests from within 7 days to 5 years prior to the index date, the point estimate for the association was still protective (adjusted OR = 0.58), similar beneficial odds ratios were obtained, but the confidence interval included 1 (Table I). By contrast, further expansion of the screening window to include tests from 0 days to 5 years prior to the index date resulted in a non-significant association (1.05 [0.45, 2.44]). Eight cases and four controls had their only screening test performed in the week prior to PC diagnosis. As noted by Hosek et al., inclusion of the final test may bias the OR towards the null unless symptom-initiated tests are excluded.[17] Thus, although we attempted to identify all symptom-initiated tests, expanding the screening window to include tests performed up to 0 days before diagnosis may include symptom initiated tests. Inclusion of screening tests on or before the symptom reference date resulted in a protective, but non-significant, adjusted OR of 0.78 (95% CI 0.36, 1.69).

COMMENT

FINDINGS

In the current study we assessed the potential effectiveness of screening for PC in a population-based case-control study. With the use of various screening windows and definitions, our estimated OR's varied considerably, though nearly all suggested a benefit. With screening defined as having a test 1-5 years prior to diagnosis, we found that screening with either DRE or PSA was generally associated with over a 50% reduction in PC mortality. With only tests done from the symptom reference date to 5 years prior, we found a 22% reduction in mortality. Our earlier population-based study suggested a beneficial association between screening with DRE and death due to PC.[10] Nearly all of the cases in that study were diagnosed prior to the availability of serum PSA testing. Following the introduction of PSA testing in Olmsted County in 1987, PC mortality rates began to decline starting in the early 1990's.[2] The decline in mortality was about 23% and was consistent with national data.[1] This population-based case-control study suggests that the addition of PSA to DRE screening may partially explain the observed mortality decline.

OTHER STUDIES (TABLE II)

Three studies have evaluated screening with DRE prior to the PSA era (Table II).[10-12] All reported protective odds ratios of 0.9,[11] 0.7[12] and 0.5,[10] though only the latter was statistically significant. Four other groups have recently published case-control studies of PC screening in the PSA era, with three suggesting a benefit. Kopec et al. evaluated the association between serum PSA testing and development of metastatic PC in Toronto and 5 surrounding counties.[15] Medical records were used to identify serum PSA testing in asymptomatic men and they found a protective OR of 0.65 (95% CI 0.45, 0.93) for PSA screening. Weinmann et al. conducted a case-control study of the association between DRE or serum PSA screening and PC mortality using Kaiser Permanente Northwest enrollees and found a protective OR of 0.7 (0.5, 1.1).[13] In a much larger study from four Kaiser Permanente sites,[14] 69% of controls and 61% of cases among White participants had a prior screening DRE or serum PSA determination based on chart review with a beneficial OR of 0.73 (0.55, 0.97). Concato et al. evaluated the association between serum PSA or DRE screening with all cause mortality and PC mortality within the Veteran's Affairs system and found no significant benefit (OR = 1.1 for both endpoints).[16] Rates of PSA screening in controls were low (13%) as compared to the present study (40%) and the Kopec study (27%). In total, seven of eight case-control studies have suggested a protective OR, although not all were statistically significant. The probability of finding seven of eight OR's being <1 is .031 (one-sided), which either suggests a benefit or some very consistent biases.

While the literature suggests a beneficial effect of screening on PC mortality, the risks of screening should also be considered. These risks include false positive screening tests resulting in unnecessary worry and PC biopsy with its related complications and costs,[20] and false negative screening tests resulting in false reassurance. Further, true positive tests have led to additional PC diagnoses and treatments for both lethal and non-lethal cases, with unnecessary treatment complications, costs and worry among the latter.

LIMITATIONS

Our study has several potential limitations inherent to retrospective designs. It is difficult to retrospectively ascribe a test as done for "screening" purposes. Further, the appropriate window within which to define screening is debatable.[9] We designated tests done in the absence of documented urinary systems as likely due to screening. Any such designation is subject to the quality and quantity of symptom documentation in the medical record. Similar results were found when including all (screening + symptomatic) tests. We used several fixed screening

windows and found essentially consistent results. We also looked at screening prior to the "symptom reference date" as was suggested by Weinmann[14] and found an OR consistent with theirs, though determination of such a date is rather subjective. Study power was limited (55% to detect an OR of 0.5 or less; assuming 81% screening for controls) due to the time period of interest and the samples size of the cases. As did others[15] we found it difficult to separate the effects of PSA and DRE, as PSA was usually done in conjunction with DRE. While published findings on the association of body mass index (BMI) with PC outcomes had been mixed, a recent review noted a consistent link between increased BMI and death due to PC.[21] Abstraction of BMI data was not performed as part of the original protocol; therefore, it's not clear how adjustment for BMI would have impacted the results. Finally, nearly all of our study subjects were Caucasian, reflecting the local demographics.

One might ask if the length of our study was adequate to find an effect due to screening. The time interval required to see a potential benefit to PC screening is not known, as it is a function of the pre-clinical duration of PC, treatment effectiveness, and survival following advanced stage PC. The perception is that it may be quite long, possibly due to the excellent survival of men with PC, most of who have localized disease at detection. However, a successful screening program should reduce the number of men with advanced stage disease at detection, for whom long-term survival is poor (as low as 23% by 5 years).[22] In our study, the median (25^{th},75^{th} percentile) time from diagnosis to death for our cases (many with advanced stage PC) was only 3.8 (2.2,6.3) years. If the conversion to advanced stage disease occurred during the prior 5 years (our screening window), then it is plausible many of these cancers could have been detected at the localized stage by screening, with a mortality effect seen in a relatively short period.

STRENGTHS

Despite these potential limitations, our study strengths include the population-based nature of the design, which means the results are less subject to referral, provider, and specialty-practice biases. Additionally, our screening documentation is based on high quality documented medical data, as opposed to surveys or interviews, limiting the likelihood of recall bias.

CONCLUSIONS

This case-control study suggests a potential benefit of PSA and DRE screening on PC mortality. The results are consistent with a growing number of similar case-control studies. Nonetheless screening recommendations await the conclusion of ongoing randomized trials and formal risk-benefit analyses.

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TABLE I

Screening PSA and DRE testing in case and control subjects

Prior DRE's and PSA's	Case, % (n=74)	Control, % (n=192)	Crude OR (95% CI)	Adjusted OR* (95% CI)
Screening tests done 1-5 years prior to PC diagnosis				
PSA	24.3	39.6	0.38 (0.18, 0.79)	0.36 (0.16, 0.81)
DRE	56.8	78.7	0.34 (0.18, 0.62)	0.37 (0.19, 0.71)
PSA or DRE	60.8	81.3	0.34 (0.18, 0.63)	0.35 (0.17, 0.71)
Screening PSA or DRE done over varying intervals				
2-5 years prior	56.8	73.4	0.45 (0.25,0.81)	0.50 (0.26,0.94)
30 days-5 years prior	64.9	84.9	0.32 (0.17, 0.60)	0.34 (0.17, 0.69)
15 days-5 years prior	66.2	84.9	0.33 (0.17, 0.64)	0.36 (0.18, 0.73)
7 days-5 years prior	74.3	84.9	0.51 (0.26, 0.99)	0.58 (0.28, 1.20)
0 days - 5 years prior	85.1	87.0	0.90 (0.41, 1.97)	1.05 (0.45, 2.44)
Screening PSA or DRE done on or before symptom reference date†	67.6	72.9	0.67 (0.32, 1.39)	0.78 (0.36, 1.69)

*Adjusted for matching factors plus marital status, education, and Charlson score.

†Earliest date of hematuria, bone pain, or perineal pain within the 5 years prior to the PC diagnosis. Median (25^{th}, 75^{th} percentile) is 0.06 (0, 2.7) years prior to PC diagnosis. The same date was used for controls.

Note: Screening refers to tests done in the absence of documented symptoms.

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TABLE II

Other case-control studies of prostate cancer screening

Author (Year Published)	Setting	Case Definition	Number Cases/ Controls	Screening Definition (Controls Screened, %)	Adjusted OR (95% CI)
Friedman (1991)[11]	Kaiser, SF Bay area	Metastatic PC, 1979-85	139/139	DRE 3mos to 10 yrs prior (71%)	0.9 (0.5,1.7)
Richert-Boe (1998)[12]	Large HMO	PC death	150/299	DRE 10 years prior (n/a)	0.8 (0.5,1.5)
Jacobsen (1998)[10]	Population-based, Olmsted County	PC death, 1976-91	173/346	DRE 1-10 years prior (81%)	0.3 (0.2,0.5)
Weinmann (2004)[13]	Kaiser Northwest	PC death, 1992-99	171/342	Prior DRE or PSA (75%)	0.7 (0.5,1.1)
Weinmann (2005)[14]	Kaiser, 4 sites	PC death, 1997-01	608/608	DRE or PSA 10 years prior (69%)	0.7 (0.5,1.0)†
Kopec (2005)[15]	Population-based, Toronto	Metastatic PC, 1999-02	236/462	Prior PSA (27%)	0.7 (0.5,0.9)
Concato (2005)[16]	VA medical centers	PC death after metastatic PC, 1991-99*	136/136	DRE or PSA in prior 5 years (13% PSA, 41% either)	1.1 (0.6,2.1)
Current (2006)	Population-based, Olmsted County	PC death, 1992-05	74/192	DRE or PSA: 1-5yrs prior (40% PSA, 81% either)	0.4 (0.2,0.7)
				Symptom date-5yrs prior (73%)	0.8 (0.4,1.7)

*Deaths from any cause (n=501) after metastatic PC were also analyzed with a PSA screening OR (95% CI) = 1.1 (0.7, 1.6).

†Tabled OR is for whites. The OR for blacks was 1.0 (0.6, 1.4).