Causes of Death in Elderly Prostate Cancer Patients and in a Comparison Nonprostate Cancer Cohort

Craig J. Newschaffer, Koichiro Otani, M. Kathleen McDonald, Lynne T. Penberthy

Background: Prostate cancer tends to affect older men and to progress relatively slowly. Since the prevalence of comorbidity increases with advancing age, competing causes of death are important contributors to death rates among prostate cancer patients. Accurate determination of the underlying causes of death in older men dying with prostate cancer may thus also be more difficult. Methods: We compared the distribution of underlying causes of death in decedents from a population-based cohort of elderly prostate cancer patients to that from a population-based comparison cohort of elderly men without prostate cancer. Among decedents from the prostate cancer patient cohort, we examined associations of patient demographics, disease stage, and initial treatment, with assignment of a prostate cancer underlying cause of death (versus any other cause) by use of multivariable logistic regression. In the subgroup of prostate cancer patient decedents having underlying causes of death other than prostate cancer, the underlying cause distribution was compared with that in nonprostate cancer cohort decedents. Results: Prostate cancer was the underlying cause for 39% (95% confidence interval [CI] = 36.3–41.9) of the decedents in the prostate cancer cohort. Causes of death among prostate cancer patients not dying of prostate cancer were similar to those among the nonprostate cancer cohort decedents. However, in those who were aggressively treated, the adjusted odds of other cancer causes of death were 51% higher (odds ratio [OR] = 1.51; 95% CI = 1.08–2.10) than that in nonprostate cancer patient decedents, while in those treated with watchful waiting the adjusted odds were 34% lower (OR = 0.66; 95% CI = 0.47–0.93). Conclusions: Initial treatment may influence the underlying cause of death reported in vital statistics for prostate cancer patients. [J Natl Cancer Inst 2000;92:613–21]

Prostate cancer is the second most common cause of cancer death among U.S. men, with 39,000 deaths attributed to the disease in 1998 (1). The vast majority of men diagnosed with and dying of this disease are elderly. As the incidence of prostate cancer increases with age, so too does the prevalence of multiple chronic morbidity. By 80 years of age, more than half of U.S. men report at least two prevalent chronic diseases (2). While prostate cancer is an important contributor to mortality in elderly men, high prevalence of comorbidity coupled with the tumor’s relatively slow progression (3) means that competing causes of death are substantive contributors to mortality in prostate cancer patients. For example, in one cohort of subjects with local-stage prostate cancer who were followed closely for 15 years, nearly 90% of the observed deaths were believed to be from causes other than prostate cancer (4).

Attribution of the underlying cause of death in older individuals with multiple chronic disease can be a difficult process (5), and only limited work has been done describing clinical and demographic factors associated with the ascribed cause of death among prostate cancer patients. Associations between age, race, stage, initial treatment, and comorbid cardiovascular disease with prostate cancer underlying cause of death have been recently reported among decedents in a prostate cancer patient cohort (6). However, equally important to understanding influences on cause-of-death reporting in prostate cancer patients is an examination of nonprostate cancer causes of death. Furthermore, examination of the distribution of causes of death among prostate cancer patients will be more informative when compared with the distribution of causes of death among men without the disease. Here, we explore the underlying causes of death listed for decedents in a large, population-based cohort of prostate cancer case subjects and compare their patterns of nonprostate cancer deaths to those among decedents from a cohort of men without prostate cancer.

Subjects and Methods

Study Population

The population under study consisted of two groups of decedents. The first was 1207 decedents from a cohort of 1996 prostate cancer patients residing in Virginia who were diagnosed from 1987 through 1989. Decedents from this cohort were ascertained through record linkage (described below under “Vital Status Follow-up”) completed in 1997, ascertaining deaths through the end of 1995. All patients were aged 67 years or more at diagnosis. Data on case subjects were drawn from Medicare and Virginia Cancer Registry (VCR) databases. As discussed previously (7), neither data source has perfect case ascertainment sensitivity and specificity, and the two sources likely include complementary data. Therefore, subjects with cancer were followed only if there was successful linkage between Medicare and VCR identifying information and if both sources showed an incident prostate cancer diagnosis within 6 months. Linkage was accomplished with the use of a three-step algorithm matching Social Security number (SSN), first name, last name, sex, and date of birth. The definition of a first-incident prostate cancer based on Medicare inpatient claims data is shown in Appendix I. The age threshold of 67 years was adopted to ensure that all men had at least 2 years of Medicare eligibility prior to diagnosis to allow for review of comorbidity.

The second group of 2906 decedents was drawn from a nonprostate cancer
cohort (n = 6586) comprising male Medicare beneficiaries aged 67 years or more living in Virginia who were hospitalized for benign prostatic hyperplasia (BPH) from 1987 through 1989 with no history of prostate cancer. Medicare data were used to identify these men (criteria described in Appendix I), and lack of prostate cancer history was determined via review of 2 years’ previous Medicare inpatient claims and a crosscheck with linked VCR records. Since Medicare hospital inpatient diagnostic codes were the only available source of comorbidity data, we needed to select a nonprostate cancer group with at least one hospitalization so that there was equivalent opportunity to report comorbidity. A hospitalization for BPH was selected because the disease has minimal direct effect on survival but typically confers morality that, during the study period, commonly led to inpatient interventions (8,9). Pathologic analysis of tissue excised during partial resection for suspected BPH could lead to a diagnosis of prostate cancer; however, these patients would have prostate cancer coded in the first diagnostic position on the Medicare claim and, consequently, would be included in the prostate cancer, not the BPH, group. Record linkage to identify decedents in this cohort followed the same procedure as that for the prostate cancer cohort. The protocol for linkage and analysis of study data was approved by human subjects committees at Saint Louis University (St. Louis, MO) and the Centers for Disease Control and Prevention (Atlanta, GA).

Vital Status Follow-up

Vital status was ascertained in hierarchic fashion by use of Virginia Department of Health (VDH) Vital Statistics, National Death Index Plus (NDI-Plus), and, when necessary, death certificates supplied from states other than Virginia. First, VDH vital statistics files were matched to identifiers of subjects in the cancer and nonprostate cancer cohorts. When SSNs were available, matches had to agree exactly on the SSN and, allowing for minor misspellings and suffix differences, the name. In cases where no SSN was available, matches were based on exact name and date-of-birth agreement. VDH vital statistics files contain information on the date and cause of death. The underlying cause of death is included as coded by state nosologists completing death certificate review for submission to the National Center for Vital Statistics. Fields are also available for up to four additional causes of death.

To identify members of each cohort dying outside the state of Virginia, identifier information for all subjects not matching VDH vital statistics files was submitted for NDI-Plus database matching. The criteria for accepting matched deaths from NDI-Plus are described in Appendix Table II. The NDI-Plus database contains information on date and, for decedents in participating states, cause of death. An additional 54 decedents were identified. For those identified from states not participating in the cause-of-death reporting component of NDI-Plus (19 of the 54 subjects), we obtained death certificates directly from state health departments. By special arrangement, the code combinations from these death certificates were reviewed by state of Virginia nosologists who applied their standard algorithm for vital statistics reporting to assign an underlying cause of death (10).

Underlying causes of death were categorized as prostate cancer, heart disease, other cancer, cerebrovascular disease, chronic obstructive pulmonary disease (COPD), pneumonia, diabetes, nephritis, septicemia, Alzheimer’s disease, hypertension, and other. Throughout this article, the category “other cancer” refers to cancer at any site other than the prostate. The specific conditions used, other than prostate cancer, are the 10 most common underlying causes of death in white and African-American men over age 65 years in 1995 (11).

Other Study Variables

The Medicare files were the source of information on patient age at diagnosis and race. In these files, race was coded only as white, black, and other. For the cancer cohort, the VCR provided information on summary tumor stage (local, regional, or distant) at diagnosis. Unfortunately, during the study period, only 17% of the men diagnosed with prostate cancer who were reported to the VCR had more detailed American Joint Committee on Cancer staging data available (12).

Both the VCR and Medicare claims include data on the initial course of treatment of cancer patients. The VCR considers the first four postdiagnostic months as the initial treatment period. Therefore, we combined VCR initial treatment data with inpatient diagnostic and procedure codes from the index prostate cancer Medicare claim and any from the subsequent 4 months. We categorized initial treatment in several different ways. First, we used a five-level classification: no treatment, surgery, radiation therapy, combination therapy (either surgery or radiation therapy in combination with another treatment modal-
to have less comorbidity. As expected, overall cumulative mortality in the prostate cancer cohort, at 60.5%, was greater by roughly 40% than that in the nonprostate cancer cohort.

Initial analyses estimated proportional mortality rates in both cohorts. Because prostate cancer was the underlying cause of death for 39% (95% confidence interval [CI] = 36.3–41.9) of the decedents in the prostate cancer cohort (see Fig. 1), proportionate mortality rates were uniformly lower for other leading causes in the prostate cancer cohort. Note that, for any cause accounting for at least 3% of deaths (heart disease, cerebrovascular disease, COPD, pneumonia, diabetes, and all the other causes), the difference was statistically significant at \( P < .05 \). To examine associations between the demographic, prognostic, and treatment factors and the underlying cause of death among decedents in the prostate cancer cohort, we estimated a multivariable logistic regression model predicting a prostate cancer underlying cause of death (as compared with all other causes of death). Older age at diagnosis was associated with reduced odds of prostate cancer cause of death, with decedents aged 85 years or older having 44% lower odds (OR = 0.56; 95% CI = 0.36–0.87) of prostate cancer listed as their underlying cause than decedents diagnosed between the ages of 67 and 74 years. A positive CCI score was also associated with reduced odds of a prostate cancer underlying cause of death (OR = 0.61;
This result is counter to that reported by Satariano et al. (6), and is consistent with the hypothesis that prostate cancer patients who have died of causes other than prostate cancer have a cause-of-death distribution similar to that in the nonprostate cancer cohort. All other causes had proportionate mortality similar across all groups. These stratified comparisons were followed with logistic regression models that estimated adjusted relative ORs for other cancer underlying cause of death comparing prostate cancer cohort decedents dying of noncancer causes, stratified by treatment with nonprostate cancer cohort decedents. Table 4 shows these estimates. Model 1 stratifies the prostate cancer decedents into three groups: no initial treatment, aggressive treatment, and other treatment. Model 2 separates the aggressive-treatment groups into surgery, radiation therapy, and combined-modality treatment. Model 3 eliminates the combined-modality group, recoding according to whether radiation therapy or surgery was present as one of the modalities (10 case subjects with indications of both surgery and radiation therapy are excluded from the model 3 analysis). The reference group is always the nonprostate cancer cohort. All ORs are adjusted for age, race, community-level education, comorbidity, and time to death. Untreated decedents in the prostate cancer cohort with other underlying causes of death still had a one-third lower odds (OR = 0.66; 95% CI = 0.47–0.93) of an other cancer underlying cause of death than decedents in the nonprostate cancer cohort. The aggressively treated group had higher adjusted odds (OR = 1.51; 95% CI = 1.08–2.10) of an other cancer underlying cause of death than the nonprostate cancer cohort decedents. Separating into specific initial treatments led to broader CIs, but OR estimates were still suggestive of positive associations of aggressive treatments with the listing of some other cancer as underlying cause of death.

We next examined the distribution of specific other cancer underlying causes of death in the treatment subgroups to determine whether this pattern was being driven by cancer attributed
to a particular site. Table 5 shows these distributions along with the distribution from the nonprostate cancer cohort. In general, the distribution of other cancers is similar across the groups—no major type of cancer appeared to be underrepresented among the untreated prostate cancer decedents nor did one type appear overrepresented among the aggressively treated decedents. Table 5 also shows the proportion of other cancer decedents with other cancer comorbidity, i.e., those who had indications of other cancers at, or prior to, diagnosis. A smaller proportion of other cancer decedents in the aggressively treated group had other cancers at, or prior to, diagnosis. A smaller proportion of other cancer comorbidity, i.e., those who had indications of other cancers at, or prior to, diagnosis. A smaller proportion of other cancer comorbidity, i.e., those who had indications of other cancers at, or prior to, diagnosis. A smaller proportion of other cancer comorbidity, i.e., those who had indications of other cancers at, or prior to, diagnosis. A smaller proportion of other cancer comorbidity, i.e., those who had indications of other cancers at, or prior to, diagnosis. A smaller proportion of other cancer comorbidity, i.e., those who had indications of other cancers at, or prior to, diagnosis.

**DISCUSSION**

In this investigation of underlying causes of death in elderly prostate cancer patients, we found that, although it was the single most common underlying cause of death, prostate cancer was the underlying cause in fewer than half of the decedents. A prostate cancer cause of death was associated positively with factors suggestive of more severe disease (i.e., late stage and palliative treatment) and negatively with factors suggestive of the presence of more or more severe competing risks (i.e., advanced age and positive CCI scores).

In a previous study of prostate cancer proportionate mortality, Satariano et al. (6) found cardiovascular comorbidity, but not other comorbidities, to be associated with decreased odds of a prostate cancer underlying cause of death. On the basis of this finding, these authors speculated that cardiovascular comorbidity may have special prognostic significance for prostate cancer patients. We, too, found cardiovascular comorbidity to reduce the odds of a prostate cancer cause of death, yet we believe that the negative association between cardiovascular comorbidity and prostate cancer proportionate mortality could occur even if there was no special prognostic significance of cardiovascular disease in prostate cancer patients. Like any competing risk, deaths due to the independent mortality risk posed by cardiovascular disease would dilute the contribution of prostate cancer deaths to the total decedent pool and thereby lower prostate cancer proportionate mortality. Because cardiovascular disease is the leading competing risk, it is not surprising that the association between cardiovascular disease and prostate cancer proportionate mortality is large and statistically significant; however, this association does not mean cardiovascular disease is any more of a threat in prostate cancer patients than in other men.

Satariano et al. (6) also observed no difference in prostate cancer proportionate mortality in patients with noncardiovascular comorbidity and in patients without comorbidity. However, we saw a similar magnitude-negative association with prostate cancer underlying cause for other comorbid diseases as for cardiovascular disease. Differences in comorbidity data sources or coding algorithms could contribute to this heterogeneity across studies.

We also performed a series of analyses focusing on the group of prostate cancer cohort decedents who did not have prostate cancer listed as their underlying cause of death. The rationale for this analysis is that, in the absence of major difficulties in the attribution of underlying cause of death, these decedents should have the same distribution of underlying causes of death as a comparison cohort of elderly men without prostate cancer. For the prostate cancer cohort overall, we found this to be the case.

This result is generally consistent with other studies that have examined the quality of vital statistics reporting of prostate cancer cause of death. Researchers from the National Cancer Institute (NCI), Bethesda, MD, have periodically examined underlying cancer cause of death data in large cohorts of cancer patients (23–25). Two of the NCI studies (23,24) focused on the proportion of cancer patient decedents with an underlying cancer cause who had agreement between underlying cause and primary incident cancer sites (deemed the "detection rate"). For incident prostate cancer case subjects, this rate was 93.5%, one of the higher detection rates reported among the major cancer sites. If we estimate the detection rate from our data, we find a much lower rate of 77%; however, our elderly cohort excludes the 20% of prostate cancer patients diagnosed under the age of 65 years (26,27). This excluded group has a higher likelihood of dying of their disease; consequently, excluding them drives down this detection rate.

The other NCI investigation (25) compared population cancer-specific mortality rates derived from vital statistics (deaths with a cancer underlying cause over total population at risk, not
just cancer patients) with life table-derived, cancer-specific mortality rates based on Surveillance, Epidemiology, and End Results (SEER)-based incidence and relative survival rate estimates. Relative survival rates capture cancer-related mortality but do not rely on attribution of specific causes of death. For prostate cancer, vital statistics-based and life table-derived population mortality was similar at 23.1 and 24.7 per 100,000, respectively. Note that, while these rates were estimated for the general population, if restricted to those with prostate cancer, the percentage difference between the rates would not change. However, these studies did not examine particular subgroups of cancer patients. It is conceivable that there were problems in cause of death reporting within subgroups of prostate cancer patients and that these may have offset each other when all of the patients were examined together. In fact, this is what was suggested from our data with respect to prostate cancer subgroups defined by initial treatment. Decedents treated initially with watchful waiting were less likely to have a different cancer as the underlying cause of death than comparison cohort decedents, while decedents aggressively treated were more likely to have another cancer as an underlying cause. This intriguing pattern persisted after adjustment for known covariates. We were, however, unable to create prostate cancer subgroups defined simultaneously by stage and initial treatment because of the small size of a number of the resulting strata. Separate analyses that compared local-, regional-, and distant-stage prostate cancer groups.

Fig. 2. Distribution of underlying causes of death among decedents in the prostate cancer cohort with underlying causes other than prostate cancer along with the distribution for the nonprostate cancer cohort. Causes shown include the top 10 leading causes of deaths for U.S. white and black males aged 65 years and older as reported in (11). Cancer is stratified into prostate cancer and other cancer. PCa = prostate cancer; COPD = chronic obstructive pulmonary disease. *P values shown at the top of each pair of bars are for two-sided tests of differences between proportions.

<table>
<thead>
<tr>
<th>Underlying cause of death</th>
<th>Prostate cancer cohort: nonprostate cancer decedents (n = 2877), proportionate mortality rate (%)</th>
<th>Nonprostate cancer cohort decedents (n = 343)</th>
<th>Aggressive initial treatment‡ (n = 201)</th>
<th>Other initial treatment§ (n = 191)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart disease</td>
<td>37.7</td>
<td>40.8</td>
<td>32.3</td>
<td>42.9</td>
</tr>
<tr>
<td>Other cancer</td>
<td>18.9</td>
<td>13.7</td>
<td>29.9</td>
<td>17.8</td>
</tr>
<tr>
<td>Cerebrovascular disease</td>
<td>8.7</td>
<td>10.2</td>
<td>7.5</td>
<td>9.4</td>
</tr>
<tr>
<td>COPD‡</td>
<td>5.8</td>
<td>5.8</td>
<td>4.5</td>
<td>4.7</td>
</tr>
<tr>
<td>Pneumonia</td>
<td>4.9</td>
<td>5.2</td>
<td>5.5</td>
<td>3.7</td>
</tr>
<tr>
<td>Diabetes</td>
<td>1.6</td>
<td>2.3</td>
<td>2.5</td>
<td>0.5</td>
</tr>
<tr>
<td>Nephritis</td>
<td>1.3</td>
<td>1.5</td>
<td>1.5</td>
<td>2.6</td>
</tr>
<tr>
<td>Septicemia</td>
<td>1.2</td>
<td>1.7</td>
<td>1.0</td>
<td>1.0</td>
</tr>
<tr>
<td>Alzheimers</td>
<td>0.9</td>
<td>1.2</td>
<td>1.0</td>
<td>1.0</td>
</tr>
<tr>
<td>Hypertension</td>
<td>0.3</td>
<td>0.9</td>
<td>1.0</td>
<td>0.5</td>
</tr>
<tr>
<td>Other</td>
<td>18.5</td>
<td>16.6</td>
<td>13.4</td>
<td>15.7</td>
</tr>
</tbody>
</table>

*Causes shown include the top 10 leading causes of deaths for U.S. white and black males aged 65 years and older as reported in (11). Cancer is stratified into prostate cancer and other cancer.
†Nonprostate cancer cohort comprised patients hospitalized with index diagnosis of benign prostatic hyperplasia and no indications of prostate cancer.
‡Aggressive treatment combines the surgery, radiation-therapy, and combination-therapy categories shown in Tables 1 and 2.
§Hormones or orchiectomy (no prostate surgery or radiation therapy).
*Two-sided test for difference of two independent binomial proportions.
¶COPD = chronic obstructive pulmonary disease.
their race was other or missing or their ecologic education was missing.

and coded in Table 2.

assigned to another type of cancer. Conversely, among patients neoplasia, if not assigned to prostate cancer, would likely be

because patients with cachexia and other signs of progressive
tice might result in a higher frequency of other cancer codes

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confounded by stage of disease at diagnosis.

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other cancer proportionate mortality. This result indicates that

with the nonprostate cancer cohort showed no differences in

other cancer proportionate mortality. This result indicates that

the findings with respect to initial treatment were not likely

confounded by stage of disease at diagnosis.

One possible explanation for the differing distribution of

other cancer deaths in initial treatment groups is information bias; i.e., assignment of the underlying cause is dependent on

knowledge of treatment approach. Health-care providers com-

pleting death certificates for prostate cancer patients known to

have received aggressive treatments may have been less likely to

attribute deaths to the prostate tumor because of their beliefs

regarding the effectiveness of aggressive treatment. Such a prac-
tice might result in a higher frequency of other cancer codes

because patients with cachexia and other signs of progressive

neoplasia, if not assigned to prostate cancer, would likely be

assigned to another type of cancer. Conversely, among patients

known to have been followed with observation only, there may

have been an increased tendency on the part of health-care pro-

viders to code deaths consistent with sequelae of late-stage can-
cer to the prostate tumor because of the lack of definitive initial

therapy. There was no particular site disproportionately repre-

sented among the other cancer decedents in either treatment group.

Because our definition of aggressive treatment included ra-
diation therapy and Medicare claim codes for radiation therapy

are not site specific, we considered the possibility that case

subjects designated as aggressively treated might include a

greater proportion of individuals with coprimary cancers. If this

t were the case, the higher proportion of decedents with other
cancer underlying causes seen in the aggressively treated group
could be due solely to this enrichment with individuals having
coprimary cancer. However, this does not appear likely because

the prostate cancer cohort decedents with other cancer underly-
ing cause of death who were aggressively treated actually had a lower proportion with indications of coprimary cancer than those with no initial treatment or other initial treatment. In addition, the proportion of other cancer deaths was actually higher among aggressively treated prostate cancer case subjects who had prostate surgery than among those who had radiation therapy.

The few available direct comparisons of underlying cause of death reported on national vital statistics to a “gold standard” cause of death determination in prostate cancer case series do not rule out information bias. Unfortunately, studies that have used autopsy data as the gold standard (28–30) have generally been comprehensive investigations of all major causes of death and consequently have not stratified by cancer site or attempted to identify prevalent cancer case subjects among decedents dying of other causes. Recently, however, there have been studies focusing on prostate cancer patients that used medical records reviews as a gold standard [16; Funkhouser E: manuscript in preparation]. Percent agreement between the death certificate and the record review ranged from approximately 80% to 90%. The possibility that underlying cause of death might be misreported in as many as one fifth of all prostate cancer decedents does not appear to be unreasonable. Our findings suggest that a focus of future direct validation studies might be on other cancer cause of death and that special attention should be paid to stratifying by initial treatment.

There are, of course, a number of limitations to the data and methods that we used here. Although great care was taken in establishing linkages, there may still be errors in matching cancer registry, Medicare, and vital statistics data. In addition, while the Medicare and cancer registry data sources do not impose severe restrictions on the study population, the VCR was not yet fully population based (it included hospitals comprising 85% of the beds in the state at that time), and the Medicare data excluded a small fraction of patients with incomplete Medicare enrollment or a Health Maintenance Organization affiliation during the study period. Also, bear in mind that available information on comorbidity was imperfect, coming from administrative data, and disease severity and initial treatment data were restricted to what was available from the cancer registry. In interpreting our findings, we were careful to remember that factors associated with increased proportionate mortality are not necessarily associated with increased mortality risk (11); we urge readers to likewise keep this in mind.

Underlying cause of death data as reported in vital statistics is relied on heavily in population-based surveillance and observational epidemiologic studies. Most cancer registries, including SEER, employ vital records linkages as their principal means of incorporating cause of death information into their databases. Several recent prostate cancer studies, including several focusing on treatment, have used prostate cancer-specific mortality as ascertained from vital statistics, either directly or via a cancer registry, as a primary end point. This study, however, points out the possibility of an important limitation in vital statistics data, i.e., nondifferential misclassification of cause of death according to initial treatment status. We realize that our data analysis approach involved a large number of proportionate mortality rate comparisons, with only one major difference emerging. However, we agree with the perspective on multiple comparisons advanced by a number of authors (31–33) where, in addition to bearing in mind the increased likelihood of such a finding being attributable to chance, the reader is also urged to consider carefully the plausibility of the hypothesis. The hypothesis of information bias that we propose seems sufficiently reasonable to warrant further consideration. Cause of death across initial treatment groups should be investigated thoroughly in direct comparisons of vital statistics-based cause of death information to medical records review and autopsy.

Appendix I. Inpatient Medicare Claims ICD-9-CM Diagnostic Code Criteria for incident prostate cancer (PCa) and incident benign prostatic hyperplasia (BPH)

Prostate Cancer
Either: 1) first position malignant neoplasm of the prostate code (185) or 2) second through fifth position 185 with either: a. a prostatectomy procedure code (60.3, 60.4, 60.5, or 60.6); b. a transurethral prostatectomy (TURP) procedure code (60.2); or c. a biopsy procedure code (60.11 or 60.12) in any procedure code field.

The additional presence of a diagnostic code indicating personal history of prostate cancer (V10.46) would result in exclusion.

The presence of a diagnostic code of 185 or V10.46 on any inpatient claim 2 years prior to a claim meeting the above criteria would also result in an exclusion.

Benign Prostatic Hyperplasia
Either: 1) first position benign prostatic hyperplasia (600) or 2) second through fifth position 600 with either: a. a bladder neck obstruction diagnostic code (596.0); b. a retention of urine diagnostic code (788.2); c. a urinary tract infection, site not specified, diagnostic code (599.0) in the first diagnostic code field.

The presence of a diagnostic code of 600 on any inpatient claim 2 years prior to a claim meeting the above criteria would also result in an exclusion.

Note: Any men meeting the above criteria for either prostate cancer or BPH who were not continuously enrolled in Medicare or who belonged to a Medicare Health Maintenance Organization in the year of diagnosis or any of the prior 2 years are excluded, since all of their utilization data are not captured in the Medicare files.

Appendix II. Criteria for accepting matched deaths from the National Death Index (NDI)*

<table>
<thead>
<tr>
<th>NDI match class</th>
<th>NDI definition</th>
<th>Study decision rule</th>
</tr>
</thead>
<tbody>
<tr>
<td>2</td>
<td>Exact match on SSN; no match on one or more of last name, first name, middle name, sex, state of birth, birth month, and birth year</td>
<td>Accept with three or more matches on: last name, first name, birth month, birth year (+/− 01), birth day</td>
</tr>
<tr>
<td>4</td>
<td>SSN unknown with &lt;8 matches on last name, first name, middle initial, birth year, birth month, birth day, sex, race, marital status, and state of birth</td>
<td>Accept with last name match plus matches on three of the following: first name, birth month, birth year (+/− 01), birth day</td>
</tr>
<tr>
<td>5</td>
<td>SSN known and does not match</td>
<td>Accept with last name match or SSN off on just one digit plus matches on first name, birth month, birth year (+/− 01), and birth day</td>
</tr>
</tbody>
</table>

*SSN = Social Security number.
REFERENCES


(22) Rothman KJ. No adjustments are needed for multiple comparisons. Epidemiology 1994;5:48-56.


Notes

1. Editor’s note: SEER is a set of geographically defined, population-based, central cancer registries in the United States, operated by local nonprofit organizations under contract to the National Cancer Institute (NCI). Registry data are submitted electronically without personal identifiers to the NCI on a biannual basis, and the NCI makes the data available to the public for scientific research.

Supported by the Centers for Disease Control and Prevention under the Prevention Research Centers Cooperative Agreement No. U48/CCU710806-03.

We thank Ron Hyman and the nosologist of the Virginia Department of Health for their review of causes of death listed on non-Virginia death certificates.

Manuscript received May 28, 1999; revised January 10, 2000; accepted January 28, 2000.