Reducing Prostate Cancer Morbidity and Mortality in African American Men: Issues and Challenges

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Prostate cancer is the most commonly diagnosed cancer in men in the United States. It disproportionately affects African American men when compared to other ethnic groups. African American men are two to three times more likely to die of prostate cancer than white men. The reasons for the disparity remain unclear, but several factors may be involved, such as age, race, nationality, nutrition, exercise, and family history of cancer. Detection of prostate cancer in high-risk African Americans is important but continues to be controversial. This article reviews the current issues and challenges regarding prostate cancer in African American men. Nurses play a vital role in the health care and education of patients; therefore, they must be aware of the issues.

Prostate cancer is the most prevalent form of cancer in American men (Ries et al., 2007). Approximately 95% of all prostate cancers develop in the glandular cells of the prostate ducts and are classified as adenocarcinomas. However, about 4% of prostate cancers are believed to arise from the lining of the prostatic urethra, tumors that arise from neuroendocrine stem cells, and tumors that are believed to be the result of aberrations in cell transformation (Theodorescu & Krupski, 2005).

Facts and Figures

The American Cancer Society (ACS, 2007a) estimated that 218,890 new cases of prostate cancer would be diagnosed in men in the United States in 2007 and that one in six men will be diagnosed with prostate cancer during their lifetimes. Prostate cancer, accounting for approximately 9% of cancer deaths, is exceeded only by lung cancer as the leading cause of cancer deaths in men. ACS (2007a) estimated that 27,050 men would die from prostate cancer in 2007 in the United States. However, improvements in prostate cancer screening, diagnosis, and treatment in the past decade have led to significant reductions in prostate cancer mortality. Data from the National Cancer Institute (NCI) Surveillance Epidemiology and End Results (SEER) program indicated that the prostate cancer mortality rate among men of all races and ages was 27.9 per 100,000 from 2000–2004 (Ries et al., 2007), compared with 32.9 per 100,000 from 1996–2000 (Ries et al., 2003). Similar trends have been observed in survival. The five-year relative survival rate for men of all races and ages was 99% from 1996–2003 (Ries et al., 2007).

At a Glance

- Prostate cancer disproportionately affects African American men when compared to other ethnic groups.
- Despite decades of healthcare advances, prostate cancer treatment continues to have complications that may affect patients physically and psychosocially.
- Awareness of current issues regarding prostate cancer in African Americans will inform nurses who care for individuals at high risk.

Prostate cancer affects all population groups. However, when incidence, mortality, and survival rates are compared by race and ethnicity, African American men are shown to bear a disproportionate burden. Prostate cancer is the most common form of cancer diagnosed in African American men and the second most common cause of cancer-related death. The ACS...
(2007b) estimated that 30,870 cases of prostate cancer would be diagnosed in African American men and that an estimated 4,240 African American men would die from the disease in 2007.

NCI and Centers for Disease Control and Prevention reported the overall rate of newly diagnosed prostate cancer and the overall prostate cancer mortality rate to be higher in African American men than in men of other racial and ethnic population groups (Ries et al., 2007). In addition, although the five-year relative survival rate of African American men newly diagnosed with prostate cancer has increased in recent decades, it lags far behind that of other racial and ethnic groups.

The mortality rate for prostate cancer from 2000–2004 was 62.3 per 100,000 among African Americans; 25.6 per 100,000 among whites; 21.2 per 100,000 among Hispanics; 18.0 per 100,000 among American Indians and Alaska natives; and 11.3 per 100,000 among Asians and Pacific Islanders (Ries et al., 2007). In addition, the five-year overall survival rate of African American men diagnosed with prostate cancer was 94.9% compared to 98.9% in white men for the years 1996–2003 (Ries et al., 2003).

Genetics

Although the causes of prostate cancer remain unclear, several factors have been found to increase the risk of developing the disease: age, race, nationality, family history of cancer, and diet and nutrition (Cohen, Kristal, & Stanford, 2000; Friedenreich, McGregor, Courneya, Angyalfi, & Elliott, 2004; Gronberg, 2003). Older African American men who have a first-degree relative (i.e., father or brother) with prostate cancer are at increased risk of developing the disease (Steinberg, Carter, Beatty, Childs, & Walsh, 1990). Some factors (i.e., diet and nutrition) can be modified, whereas others (i.e., age, race, nationality, and family history) cannot.

Age

The risk of developing prostate cancer increases rapidly after age 50 (Zoorob, Anderson, Cefalu, & Sidani, 2001). Approximately two-thirds of all prostate cancers are diagnosed in people older than 65 (ACS, 2007c). The probability of being diagnosed with prostate cancer is 1 in 19,299 for men younger than 40 years, 1 in 45 for men aged 40–59 years, and 1 in 7 for men aged 60–79, with an overall lifetime risk of developing prostate cancer of 1 in 6 (NCI, 2007). A 50-year-old man has an estimated 42% lifetime risk of developing histologic evidence of prostate cancer and a 2.9% risk of death from prostate cancer (Scher, Isaacs, & Zellefsky, 2000).

Race

According to the SEER program, African American men (255.5 per 100,000) in the United States have a higher rate of prostate cancer than white men (161.4 per 100,000), Hispanics (140.9 per 100,000), and Asians and Pacific Islanders (96.5 per 100,000) (Ries et al., 2007). Compared with men of other races, African American men also are more likely to be diagnosed at advanced stages and have a higher mortality rate. A clinical trial that examined the outcomes of 288 African American and 975 white men with prostate cancer found that African Americans had poorer prognoses than whites and that prostate cancer was diagnosed at more advanced stages in African Americans (Thompson et al., 2001). Although the five-year survival rates for African Americans have improved since the 1980s, African Americans continue to be 2.4 times more likely to die from prostate cancer than whites (ACS, 2007b). The reasons for racial differences in prostate cancer are not clear.

Nationality

The incidence of prostate cancer varies worldwide. The United States, Canada, and Scandinavia have the highest prostate cancer rates (ACS, 2007c). The disease is less common in Asia, Africa, Central America, and South America (ACS, 2007c), whereas China and other parts of Asia have the lowest rates (Gronberg, 2003). Singh and Siahpush (2001) found that immigrants to the United States (i.e., foreign-born individuals) have lower mortality rates than individuals who were born in the United States. The reason is not well understood.

Family History

Men with relatives who have been diagnosed with prostate cancer are considered to be at high risk. Whitemore, Wu, et al. (1995) conducted a study of African Americans, whites, and Asians in the United States and reported a twofold to threefold increase in risk of developing prostate cancer when a first-degree family member (i.e., father, brother, or son) has been diagnosed with the cancer. Another study (Bock, Peyser, Montie, & Cooney, 2005) showed that the age of diagnosis of prostate cancer has been decreasing over successive generations. The authors suggested that a man who has a father or uncle of a particular age who was diagnosed with prostate cancer is 1.30 times more likely to be diagnosed with prostate cancer at the same age that the father or uncle was diagnosed. Bratt (2002) reported that men with a family history of prostate cancer are diagnosed, on average, six to seven years earlier than those without a positive family history. This is most likely caused by familial genetic mutations, which may be associated with a higher risk of prostate cancer (Boyle, Gianluca, & Giles, 2003). Bratt noted that more than 40% of men diagnosed before age 55 may have a hereditary basis. In addition, risk is higher for men who have a brother with prostate cancer than for those who have a father with prostate cancer.

Thus, prostate cancer seems to have an inherited or genetic component (ACS, 2007c; Ahaghotu et al., 2004; Cooney, 1998; NCI, 2007; Nieder, Taneja, Zeegers, & Ostrer, 2003). One study (Carter et al., 1993) of 44,788 pairs of twins in the Scandinavian region examined the risk of cancers among monozygotic and dizygotic twins and found that the largest susceptibility to cancer accounted for by a genetic component was prostate cancer. The authors calculated that 42% of prostate cancers were associated with genetic factors.

ACS (2007d) suggested that several hormones and genes may be responsible for the development of prostate cancer, including the gene hereditary prostate cancer 1 (HPC-1) and BRCA-1 and BRCA-2 gene mutations. NCI (2005) noted that endogenous hormones, such as androgens and estrogens, also may be associated with the development of prostate cancer. Shuch et al. (2004)
found a strong association between epidermal growth factor receptor (EGFR) (which plays a critical role in prostate cancer signal transduction) overexpression and African Americans, thus suggesting that race contributes significantly to EGFR expression in prostate cancer. However, the research on the gene mutations and growth factors remains preliminary.

Diet and Nutrition

Dietary factors, such as high consumption of red meat and high-fat dairy products and low consumption of fruits and vegetables, are less understood; which specific factors contribute to risk of developing prostate cancer are not clear (ACS, 2007c; Kolonel, Nomura, & Cooney, 1999). In an often-cited multisite study, an estimated 10%–15% of the difference in prostate cancer incidence among whites, African Americans, and Asian Americans was associated with differences in saturated-fat intake (Whittemore, Kolonel, et al., 1995). Rodriguez et al. (2006) found that high consumption of cooked, processed meats high in animal fat may contribute to increased risk for prostate cancer among African American men. Several studies (Fairfield & Fletcher, 2002; Lu et al., 2001; Willett, 2001) have suggested that addition or reduction of certain foods and supplements in the diet may decrease the risk of prostate cancer. Kolonel et al. (2000) suggested a small positive association between a diet high in saturated fat and prostate cancer incidence. Prostate cancer is more common in countries where people consume more dairy and meat products than in countries where people have a basic diet of soybean products, vegetables, and rice.

A diet high in antioxidants (e.g., lycopene, found in fruits and vegetables such as tomatoes, pink grapefruit, and watermelon) may reduce risk (Miller et al., 2002). Several studies have suggested that lycopene inhibits prostate cancer (Giovannucci, Rimm, Liu, Stampfer, & Willett, 2002; Tang, Jin, Zeng, & Wang, 2005); however, one study (Kolonel et al., 2000) reported no association between lycopene and inhibition of prostate cancer. Ongoing studies are focused on the association. Kolonel et al. (2000) examined the protective effects of vegetables, fruits, and legumes against prostate cancer and suggested that legumes may have protective properties primarily because of their high-fiber content and possibly because of their phytoestrogen characteristics. Because estrogen may lower the risk of prostate cancer, a rationale exists. Vitamin E (Chan & Giovannucci, 2001), vitamin D, and the mineral selenium (Platz & Helzlsouer, 2001) also may decrease the risk of developing prostate cancer (ACS, 2007c).

Screening

Prostate cancer screening is highly controversial; its effects have not been well established in randomized clinical trials. Several organizations have created recommendations for prostate cancer screening (see Table 1). In one study (Clarke-Tasker & Dutta, 2005), 86.9% of a sample of 67 African American men agreed that their physicians would think they should have a digital rectal examination (DRE) and a prostate-specific antigen (PSA) test performed. Currently, two large, ongoing, randomized screening trials in Europe (the European Randomized Study of Screening for Prostate Cancer) and the United States (Prostate, Lung, Colorectal and Ovary Cancer study) are examining whether screening men for signs of prostate cancer saves lives (International Prostate Screening Trial Evaluation Group, 1999).

Several studies have noted that certain races respond differently to prostate cancer screenings. Cotter, Gern, Ho, Chang, and Burk (2002) and Steele, Miller, Maylahn, Uhler, and Baker (2000) found lower rates of prostate cancer screening in African American men and Hispanic men compared to white men. McFall, Hamm, and Volk (2006) found that African Americans had a stronger interest in forming health-promotion activities to help the larger community understand prostate cancer and early detection, whereas whites were more concerned with individual understanding about prostate cancer screening. However, in the study by McFall et al., Hispanics were less aware of the important issues regarding prostate cancer screenings compared with African Americans and whites.

Detection

DRE usually is the initial assessment of the prostate. It may find nodules, asymmetry in the prostate gland, and texture differences. Physical examination alone is not reliable to differentiate a cyst from cancer; thus, biopsy may be necessary. Findings from DRE may be the beginnings of cancer staging. DRE has been reported to have a sensitivity range of 49%–69.2% and a specificity range of 18%–99.5% (Mistry & Cable, 2002), a wide range. Therefore, the U.S. Food and Drug Administration has approved PSA testing along with DRE to help detect prostate cancer in men (NCI, 2004).

The PSA blood test improves the chances of prostate cancer detection. PSA level helps to establish an age-related diagnosis that could lead to suspicion of prostatitis, benign prostatic hyperplasia, or prostate cancer. A large, multicenter trial (Catalona et al., 1994) suggested the use of serum total PSA greater than 4 ng/ml as the threshold to start testing for prostate cancer by way of biopsy. Gann, Hennekens, and Stampfer (1995), who evaluated the validity of PSA testing to detect prostate cancer, reported an overall specificity of 91% and sensitivity of 46%. Schroder et al. (2000) noted that 20%–50% of clinically significant prostate cancers may occur in men with serum total PSA less than 4 ng/ml. One study (Thompson et al., 2004) showed that the rate of prostate cancer was 10.1% among men with PSA levels of 0.6–1.0 ng/ml and rose to 26.9% among men with PSA levels of 3.1–4.0 ng/ml.

PSA levels may differ among races. Cheng et al. (2005), who examined serum PSA levels among Singapore Chinese (1.43 ng/ml), African American (1.46 ng/ml), white (1.28 ng/ml), Latino (1.18 ng/ml), and Japanese American (1.22 ng/ml) men, reported that PSA mean levels for African Americans were higher than for the other ethnic groups. Thompson et al. (2001) reported that African American men were more likely than white men to have higher PSA levels, as well as higher Gleason scores. In addition, Fowler, Sanders, et al. (2000) reported a significant racial difference in total PSA levels when comparing African Americans to whites. When percent-free PSA was tested, African American men’s was 35.2% compared to 29.2% for white men.
Diagnosis

Biopsy usually is warranted to make a diagnosis and to determine the Gleason score. A biopsy is taken after a suspicious PSA level suggests the presence of prostate cancer, and the sample is assigned a grade. Although African American men often have been reported to have a higher incidence of prostate cancer, a few studies (Brett et al., 2004; Fowler, Bigler, Miles, & Yalkut, 2000) have found no racial differences in repeat-biopsy cancer detection. The Gleason system is used to describe the degree of differentiation of the cancer cells. Scores range from grade 2–10. The Gleason system assigns a grade to each of the two largest areas of cancer in the prostate tissue sample; those grades range from 1 (least aggressive) to 5 (most aggressive). The two grades are added together to produce the Gleason score. Higher Gleason scores represent poorly differentiated (more aggressive) cancer cells that are more likely to metastasize.

Several studies (Du et al., 2006; Latini et al., 2006; Sohayda, Kupelian, Altsman, & Klein, 1999) have found that African Americans had biopsy Gleason scores significantly higher when compared with whites. The reasons are unclear but could be related to delayed diagnosis of prostate cancer among African Americans or the biology of tumors among African Americans.

Pathophysiology

Higher incidence and mortality of prostate cancer among African American men compared to white men may be attributable to differences in androgen levels (e.g., dihydrotestosterone [DHT]). Several studies have noted that testosterone is higher in African American men than in whites. The CYP3A4 gene is associated with mediating the testosterone metabolism pathway and has a potential to mutate, causing a greater bioavailability of testosterone to be metabolized and converted into DHT, a potent androgen that has been noted to play a role in the formation of prostate cancer (Lamharzi et al., 2003; Zeigler-Johnson, 2001). Testosterone is the major circulating androgen in the body, and DHT is a tenth the concentration of circulating testosterone (Mohler et al., 2004). Higher amounts of DHT in the prostate may cause an increase of androgen stimulation, which could cause prostate cancer. Mohler et al. examined androgen in prostate tissue of African American and white men who had received radical prostatectomy for prostate cancer; they found no differences in tissue levels of testosterone and DHT by race. However, high-grade prostatic intraepithelial neoplasia, a precursor of prostate cancer, was found to be a significant predictor of prostate cancer (Carver et al., 2004) and is more common in African American men than in white men (Fowler, Bigler, Lynch, Wilson, & Farabaugh, 2001).

Table 1. Summary of Prostate Cancer Screening Recommendations

<table>
<thead>
<tr>
<th>ORGANIZATION</th>
<th>RECOMMENDATION</th>
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<tbody>
<tr>
<td>American Academy of Family Physicians (2007)</td>
<td>Insufficient evidence exists on which to make a recommendation for or against routine screening for prostate cancer using prostate-specific antigen (PSA) testing or digital rectal examination (DRE).</td>
</tr>
<tr>
<td>American Cancer Society (2006)</td>
<td>Annual examination begins at age 50 for patients with at least a 10-year life expectancy. Men at higher risk (African Americans and those having first-degree relatives with prostate cancer) should begin testing at age 45. Information should be provided to all men about what is known and what is uncertain about the benefits and limitations of early detection and treatment of prostate cancer so that they can make informed decisions about testing.</td>
</tr>
<tr>
<td>American College of Physicians (1997)</td>
<td>Physicians should describe potential benefits and known harms of prostate cancer screening, diagnosis, and treatment; listen to the patient’s concerns; and individualize the decision to screen.</td>
</tr>
<tr>
<td>American College of Preventive Medicine (Ferrini &amp; Woolf, 1998)</td>
<td>Population screening with DRE and PSA is not recommended. Men aged 50 years or older with a life expectancy of more than 10 years should be given information about the potential benefits and harms of screening to allow them to make their own choices about screening.</td>
</tr>
<tr>
<td>American College of Radiology (Ferrini &amp; Woolf, 1998)</td>
<td>Begin annual DRE and PSA screening at age 50 and annual PSA screening beginning at age 40 for African American men and other men with a positive family history of prostate cancer.</td>
</tr>
<tr>
<td>American Medical Association (2001)</td>
<td>Both PSA and DRE should be offered annually, beginning at age 50, to men who have at least a 10-year life expectancy and to younger men who are at high risk.</td>
</tr>
<tr>
<td>American Society of Internal Medicine (U.S. Preventive Services Task Force, 2002)</td>
<td>Most appropriate candidates for prostate cancer screening include men older than 50 years of age and younger men with an increased risk of developing prostate cancer with at least 10 years of expected life or more.</td>
</tr>
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</table>
Several options are available for prostate cancer treatment (i.e., surgery, radiation therapy, chemotherapy, hormonal therapy, and watchful waiting). Surgical techniques, such as a laparoscopic radical prostatectomy (LRP), decrease the chance of impotence. LRP using a robotic interface (the da Vinci system) allows for a nerve-sparing approach, which increases the chance of potency after the operation. A telescopic instrument, the laparoscope with a small camera, is inserted into the abdomen through a small incision at the umbilicus. The surgeon is able to view inside the abdomen and perform the surgery without making a large incision. Usually, four or more small incisions are made in the abdomen for the surgical instruments. Because the technique is less invasive, patients spend much less time in the hospital (about two nights) as compared to traditional prostatectomy (approximately five nights).

Controversy has surrounded the use of radical prostatectomy, radiation therapy, and conservative management for prostate cancer. Several studies (Hoffman et al., 2003; Underwood et al., 2004; Zeliadt, Potosky, Etzioni, Ramsey, & Penson, 2004) have reported that African American men were 26% less likely to use aggressive therapy, such as radical prostatectomy, than conservative therapies (i.e., radiation therapy, androgen deprivation therapy, and expectant management [watchful waiting]) when compared to whites.

Several factors may influence the type of treatment an individual receives: age and expected life span, stage and grade of cancer, and potential treatment side effects. Several studies have reported racial differences in the use of certain treatment modalities for localized prostate cancer (Denberg, Beaty, Kim, & Steiner, 2005; Spencer, Fung, Wang, Rubenstein, & Litwin, 2004; Underwood et al., 2004). However, Peters and Armstrong (2005) completed a systematic review of prostate cancer treatment outcomes as they relate to race; from the 29 studies they included in their analysis, no differences were found between races after controlling for tumor and patient characteristics. Denberg et al. found that African American men were less likely to receive curative therapy, such as radical prostatectomy.

Survivorship

African American men have a lower survival rate once diagnosed with prostate cancer than other ethnic groups (Ries et al., 2007). Other than genetics, diet, treatment type, and screening, as mentioned earlier, comorbidities may have a significant role in low survival rates in this group. Freeman et al. (2004) found that African American men had greater mortality from prostate cancer and other comorbidities than their white counterparts. In addition to comorbidities, a few studies (Datta et al., 2005; Grossfeld et al., 2002) have shown that African Americans have a higher risk of recurrence of prostate cancer when compared to whites.

The complex sociodemographic characteristics of African Americans in relation to health care also may have a critical role in the survival of African Americans diagnosed with and treated for prostate cancer, regardless of type of treatment. Oakley-Girvan et al. (2003) found that socioeconomic status is associated with later-stage diagnosis of prostate cancer and poor survival and that African Americans are more likely than whites to be diagnosed with advanced disease. Although the reasons for the low survival rate within this population are unclear, numerous variables contribute to poorer survival outcomes for African Americans compared to whites.

Conclusion

Despite advances in the healthcare system, health disparities remain wide, particularly regarding prostate cancer. Studies (Agho & Lewis, 2001; Wilkinson, List, Sinner, Lanting, & Chodak, 2003) have reported numerous factors that may increase the risk of prostate cancer in African American men. Although much controversy surrounds prostate cancer screening, early detection and early diagnosis appear to play a significant role in treatment. Men at high risk for prostate cancer, particularly African Americans, seem to lack knowledge, awareness, and resources, which ultimately affects their health. Research is needed to examine the effects of prostate cancer screening on mortality. Because of minorities’ hesitation to participate in research studies and the dearth of randomized clinical trials that focus on prostate cancer and contain significant numbers of minorities (NCI, 2000), specifically African Americans, more randomized clinical trials are needed that examine the risks of prostate cancer, prostate cancer treatment outcomes, and the potential benefits of prostate cancer screenings. In addition, more studies are needed to help identify interventions that will narrow the prostate cancer gap between African Americans and whites.

Implications for Nursing

Nurses should be more aware of the psychosocial, behavioral, and physical factors that affect individuals, particularly African Americans, who are at high risk for or have been diagnosed with prostate cancer. Nurses have an important role in informing high-risk individuals about prevalence, screening options, treatment options, and other pertinent information about prostate cancer so that men can make informed decisions about prostate cancer screening and treatment. Nurses should make every patient contact an opportunity to educate. Continuous awareness of the physical and psychosocial effects of prostate cancer, especially as they relate to minorities, has the potential to help decrease morbidity and mortality among the population.

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References


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