Cervical Cancer: What Should We Tell Women About Screening?

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Cervical cancer is the second most common cancer among women worldwide, with significantly higher rates in developing areas, especially in Africa, the Caribbean, and Latin America (Parkin, Bray, Ferlay, & Pisani, 2005). In contrast, incidence and mortality rates of cervical cancer in the United States have declined significantly among women of all ethnic and racial groups; it is not among the top 10 leading causes of new cancer cases in women (American Cancer Society [ACS], 2006b; Edwards et al., 2005).

Although the statistics are encouraging, women continue to be diagnosed with a disease that can be prevented through risk reduction and appropriate screening with the Papanicolaou (Pap) test (ACS, 2006a). Cervical cancer is a fairly unique cancer in that the natural history of the disease is well known. The most significant risk factor is the human papillomavirus (HPV), a sexually transmitted disease, which is present in approximately 99% of cervical cancers (Walboomers et al., 1999). The conventional Pap test is inexpensive and widely available, with a reported sensitivity rate as high as 77% in detecting precursor lesions (Andy, Turner, & Neher, 2004). However, 9,700 women will be diagnosed with cervical cancer this year and 3,700 women will die from it (ACS, 2006a). Current estimates suggest that 50% of women diagnosed with cervical cancer have never had a Pap test and another 10% have not had a Pap test in the past five years (Nuovo, Melnikow, & Howell, 2001).

Etiology and Risk Factors

Several known risk factors are associated with the development of cervical cancer. The most significant is the acquisition of HPV. In the United States, HPV is the most common sexually transmitted disease, infecting an estimated 15% of the population. The lifetime risk of getting HPV is estimated to be as high as 75% (Koutsky, 1997). More than 100 types of HPV exist. Fortunately, most are benign and resolve without treatment. Low-risk HPV types may cause visible, benign lesions or warts known as condylomata acuminate. High-risk HPV types tend to persist and are associated with the development of precancerous lesions and cervical cancer. Approximately 15 high-risk HPV types are associated with the development of cervical cancer, especially HPV types 16, 18, 33, and 45. HPV 16 and 18 cause more than half of cervical cancers, and the two types are associated with more than a 200-fold increased risk of developing invasive cervical cancer (Munoz et al., 2003).

Transmission of HPV occurs by genital contact with an infected partner. The virus enters through a break in the squamous epithelium, where infection stimulates the replication of the epithelium. Proteins expressed by HPV bind to the p53 tumor suppressor protein, interfering with normal cell proliferation and blocking apoptosis from occurring, which allows the damaged cells and the HPV infection to thrive. Time from exposure to infection may be one to eight months (Mehring, 2005). The risk of acquiring HPV increases with the number of lifetime sexual partners and is not 100% preventable with condom use because HPV infection can be transmitted on body surfaces that are not covered by a condom (Centers for Disease Control and Prevention [CDC], 2003).

Long-term use of oral contraceptives has been associated with an increased risk of developing cervical cancer. Results from a large literature review found that women, including HPV-positive women,

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