

Educating Women Regarding the Early Detection of Endometrial Cancer— What Is the Evidence?

Jennifer M. Tiffen, RN, MS, APN, and Suzanne M. Mahon, RN, DNSc, AOCN®, APNG

Endometrial cancer, cancer of the lining of the uterus, is the most common gynecologic malignancy in the United States, with an estimated 40,880 new cases diagnosed in 2005 (American Cancer Society [ACS], 2005a). Fortunately, mortality rates for endometrial cancer are relatively low, with 96% of women living five years or longer after being diagnosed with localized disease. Most women (72%) are diagnosed with localized disease (Ries et al., 2002) because early-stage, localized disease most often presents with abnormal uterine bleeding, leading many women to seek prompt medical attention, ultimately resulting in early detection (ACS, 2005a).

Endometrial cancer usually is diagnosed in women aged 50–65 years, with a peak incidence occurring in postmenopausal women. Two major types of endometrial cancer exist: type I and type II. Type I carcinomas, the more common, are associated with unopposed estrogenic stimulation, usually occur in younger postmenopausal women, and often are diagnosed at a low-grade stage, when prognosis is good. Type II carcinomas are thought to be unrelated to estrogenic stimulation, usually are diagnosed in older women, and have a poorer prognosis (Goodfellow, 2005; Ronnett, Seidman, Zaino, Ellenson, & Kurman, 2005). Most identified risk factors are associated with type I carcinomas, whereas older age is the only identified risk factor for type II (Purdie, 2003). Known risk factors for type I carcinomas include obesity, unopposed estrogen use, early menarche, late menopause, nulliparity, polycystic ovarian syndrome, tamoxifen therapy, diabetes, hypertension, and a history of atypical hyperplasia (ACS, 2005a; Barakat, 1998; Cooper & Stegmann, 2005; Purdie).

The administration of unopposed estrogen-replacement therapy has been linked to a relative risk of endometrial cancer of 2.3, with an increased risk as years of use increase (Grady, Gebretsadik, Kerlikowske, Ernster, & Petitti, 1995). Current practice usually includes the administration of progesterone in combination with estrogen, which does not appear to increase risk (Barakat, 1998).

Tamoxifen therapy has been associated with increased risk of endometrial cancer. The reported relative risk is 7.5 (Fisher et al., 1994). Although other agents for hormonal manipulation in the treatment of breast cancer have been developed, tamoxifen continues to be an effective treatment for breast cancer despite the associated risk for endometrial cancer. Fortunately, endometrial

cancers diagnosed in users of unopposed estrogen and tamoxifen tend to be similar to those diagnosed in the general population, with a good prognosis for most women (Barakat et al., 2000).

The diagnosis of known or suspected hereditary nonpolyposis colorectal cancer syndrome (HNPCC) increases a woman's risk of developing endometrial, colon, and ovarian cancers and, more rarely, cancers of the renal pelvis, bladder, brain, and stomach. HNPCC is an inherited cancer susceptibility syndrome accounting for approximately 5% of all endometrial cancers. Mutations in the *MLH1* gene and *MSH2* gene can increase the risk of developing endometrial cancer by as much as 60% (Bandera, 2005; Vasen et al., 2001). Genetic testing for the *MLH1* and *MSH2* genes is readily available on a commercial basis and is recommended

Jennifer M. Tiffen, RN, MS, APN, is a clinical instructor in the Department of Medical-Surgical Nursing at the University of Illinois in Chicago, and Suzanne M. Mahon, RN, DNSc, AOCN®, APNG, is a clinical professor in the Division of Hematology/Oncology in the Department of Internal Medicine at Saint Louis University in Missouri.

Digital Object Identifier: 10.1188/06.CJON.102-104