Ovarian cancer is the most lethal gynecologic cancer. Early-stage diagnosis is difficult, and chemotherapy treatments often are not durable. Despite challenges, progress has been made since the 1990s; healthcare professionals now have an increased understanding of the disease biology and can identify hereditary ovarian cancer and provide screening recommendations. The recognized importance of complete staging, cytoreductive surgery, and new effective treatments has made an improvement in five-year survival.

Ovarian cancer incidence is about half that of endometrial cancer; however, the mortality rate of ovarian cancer exceeds the combined rates of cervical and endometrial cancer. In 2007, estimates indicate that 22,430 new cases will be diagnosed and 15,280 deaths will be attributed to ovarian cancer (Jemal, Siegel, Ward, Murray, Xu, & Thun, 2007). The median age for ovarian cancer diagnosis is 63, and the incidence rate increases steadily in women until age 80, when it declines (National Cancer Institute, 2006).

Ovarian tumors can be benign, of low malignant potential, or invasive cancers. The major pathologic ovarian cancers are epithelial, germ cell, or sex-cord stromal. Epithelial is the most prevalent, accounting for more than 90% of ovarian tumors (Ozols, Rubin, Thomas, & Robboy, 1997), and arises from cells on the surface of the germinal epithelium or mesothelium of an ovary. The tumors are classified further as borderline or invasive and according to cell type (see Figure 1). Germ cells are precursors of the ova, and the most common malignant result is dysgerminoma. Sex-cord stromal cells secrete hormones that connect different components of the ovary together, often causing a granulose-cell tumor. This article will focus on the most commonly occurring ovarian cancer, epithelial.

Pathophysiology

The primary method of ovarian cancer metastasis is by the exfoliation of cells that implant along the surfaces of the peritoneal cavity; lymphatic and hematogenous dissemination also occurs but is less common. The exact molecular events that trigger the development of ovarian cancer have not been determined; however, epithelial ovarian cancer develops because of the malignant transformation of the epithelium on the surface of the ovary. The origin of ovarian cancer centers on reproduction and ovulation. Two general hypotheses of ovarian cancer pathogenesis are the incessant ovulation theory and the excess gonadotropin secretion theory. Incessant ovulation theory posulates that the risk of ovarian cancer is directly related to the number of ovulatory cycles and the repetitive trauma and repair ovulation causes (Fathalla, 1971). The excess gonadotropin secretion theory proposes that excess secretion of gonadotropin promotes high estrogen concentration, which may lead to epithelial proliferation and possible malignant transformation (Cramer & Welch, 1983).

Risk Factors

Genetics, hormones, reproduction, and lifestyle have been implicated as risk factors for ovarian cancer. Most ovarian cancer is not attributed to a specific cause. The most significant risk factor, hereditary ovarian cancer syndrome, increases the possibility of developing ovarian cancer by 25%–50% (Daly & Obrams, 1998). Women have a 4%–5% increased risk if a single family member was diagnosed with ovarian cancer, whereas those with two or more family members have a 7% increased risk (Daly & Obrams). Other hereditary syndromes are site-specific ovarian cancer, breast-ovarian cancer, and the family

At a Glance

- Ovarian cancer is the most lethal gynecologic cancer.
- Most patients with ovarian cancer are diagnosed at advanced stages of disease.
- Improvements in staging, cytoreductive surgery, and adjuvant therapies have improved five-year survival.

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