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Opportunities for Cancer Risk Reduction in Gynecology Surgery

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INTRODUCTION

In the United States, ovarian cancer is the sixth greatest cause of cancer-related death in women (US). 95% of all malignant ovarian neoplasms are epithelial ovarian cancers (EOC). High grade serous EOC is the most fatal subtype, accounting for 70-75% of all instances of ovarian cancer. This subtype typically manifests late in life, with a 5-year overall survival rate of 35%. Prior efforts to improve the prognosis of EOC focused on early detection. The clinical findings have been disappointing-early-stage malignancies were low grade and slow-growing, while high-grade lethal cancers went undiagnosed until late stage. False-positive results necessitated additional testing and unneeded procedures, both of which were costly and risky. These investigations emphasised the variations in aetiology and clinical behaviour among EOC morphologic subgroups. Understanding the unique genetic and immunohistochemical properties of histologic subtypes and grades, as well as the numerous exposures linked with each morphology, garnered scientific interest and resources.

DESCRIPTION

As a result of this research, the "ovarian" cancer paradigm has shifted to one in which cancers of the fallopian tube, peritoneum, and ovary is classified based on aetiology, molecular and immunohistochemical characteristics, grade, and clinical behaviour rather than location. The fallopian tube is central in the carcinogenesis of serous, endometrioid, clear cell, and undifferentiated epithelial carcinomas, according to this model. Extirpative and occlusive tubal operations are gaining popularity as ovarian cancer prevention strategies. The terms ovarian carcinoma and ovarian cancer will be used interchangeably in this review article to refer to epithelial cancer of the ovaries, fallopian tubes, and peritoneum [1-3].

The clinical behaviour of each ovarian cancer subtype and grade reflects its genetic make-up and immunohistochemistry characteristics. Low-grade serous, endometrioid, and mucinous carcinomas grow slowly and manifest early. High-grade serous, high-grade endometrioid, clear cell, and undifferentiated carcinomas have aggressive behaviour and present at an advanced stage. Endometrioid and clear cell histologies are thought to be the result of benign endometriosis lesions that progress to atypical endometriosis and subsequently to carcinoma, whereas serous carcinomas are thought to be the result of fallopian tube epithelial cells. High-grade serous, endometrioid, and clear cell carcinomas account for 74%, 13%, and 6% of EOC, respectively, and an even greater proportion of ovarian carcinoma fatalities. This theory is founded on several significant discoveries: a) no precursor or intermediary lesion has been found on the ovary or peritoneum; b) the majority of fallopian tubes from surgical specimens of clinical "primary" ovarian and "primary" peritoneal serous carcinoma harbour occult and precursor lesions with the same genetic mutation and p53 signature as the cancer; c) pelvic serous carcinoma expresses biomarkers more similar to tubal epithelium cells (Mullerian origin) (urogenital origin). Furthermore, specimens from risk-reducing salpingo-oophorectomy (RRSO) in healthy women with BRCA mutations contain early tubal carcinoma in 5% of cases and pre-cancerous lesions (STIC or STIL) in 15% of cases; no precursor lesions in the ovaries or peritoneum have been observed.

Endometrioid and clear cell carcinomas are thought to be caused by endometrial cells and endometriosis lesions, according to histopathologic research. PTEN and ARID1A mutations are seen in atypical endometriosis lesions in women with endometrioid and clear cell ovarian carcinomas. Observational clinical studies show that preventing retrograde menstruation, whether through tubal ligation or hysterectomy, reduces the risk of endometrioid and clear cell cancer. A pooled analysis of 1.3 million women from 21 prospective cohort studies is the largest examination of prospectively acquired data on diverse exposures and the related ovarian cancer risk [4].

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The recommended timing of RRSO in women at increased hereditary risk of epithelial ovarian cancer (EOC) is based on the age distribution of cancer incidence specific to the gene mutation and the individual patient's reproductive desires, family history, and breast cancer prevention and screening strategy. BRCA gene mutations a germline BRCA1 or BRCA2 mutation affects between 1 in 300 and 1 in 800 women. When compared to BRCA2 carriers, BRCA1 carriers have a higher degree and earlier incidence of cancer risk. Breast cancer occurs more frequently and at a younger age among both mutation subgroups than ovarian cancer. The initial incidences of breast cancer occur between the ages of 21 and 30 (5 per 1000 person-years), and the cumulative risk until age 50 is 43% and 35%, respectively, for BRCA1 and 2. The first incidences of ovarian cancer appear in the early thirties (2 per 1000 person-years), and the cumulative risk until age 50 is 8% and 1%, respectively, for BRCA1 and 2 carriers. Despite the fact that the median age of breast cancer diagnosis is younger, only one-third of people who develop ovarian cancer have previously been diagnosed with breast cancer. Furthermore, BRCA mutation carriers are more likely to die from ovarian cancer than breast cancer.

The features of ovarian cancer tumours are comparable across BRCA mutant subtypes, whereas breast cancer tumours diverge. EOC accounts for nearly all ovarian malignancies, with high grade serous carcinoma accounting for over 70% of cases. Breast tumours with BRCA1 carriers, on the other hand, have a higher grade and act more aggressively than those in BRCA2 carriers. This is most likely owing to subtype differences in the tumor's hormone-receptor status. Seventy-eight percent of cases in BRCA1 carriers are ER-negative, compared to only 23% of cases in BRCA2 carriers. The majority of ER-negative tumours are triple negative. This has consequences for prognosis and treatment because triple negative tumours are more aggressive and resistant to hormone therapy [5,6].

Premenopausal RRSO increases overall survival in women with BRCA mutations by lowering the risk of ovarian and, most likely, breast cancer. The degree of protection is determined by the age at which RRSO is performed. Occult cancer is found in 4-8% of specimens when conducted before the age of 45. When surgery is postponed until the patient is 45 or older, the risk rises to 20%. Surgery performed before the age of 50 reduces mortality by 53-79%, with the degree of benefit increasing as the age of surgery is reduced to 30 years. Risk-reducing surgery reduces the risk of ovarian cancer by 72-96% and of breast cancer by up to 64%, while other research and biostatisticians challenge the breast cancer protective connection.

The NCCN advises that women with BRCA1/2 mutations have their ovaries and fallopian tubes removed once childbearing is complete and by the age of 35-40. Given the later age of beginning of ovarian cancer in BRCA2 mutation carriers, the NCCN believes it is fair for BRCA2 mutation carriers to wait until 40-45 years of age if the woman has had bilateral mastectomy [7]. This guideline emphasises the necessity of multidisciplinary planning and management of BRCA mutation carriers, since the approach to breast cancer prevention influences ovarian cancer prevention techniques and vice versa.

There is very limited data on the use of hormone treatment (HT) after RRSO in patients with a greater hereditary risk of breast and ovarian cancer. As a result, investigations in women at high risk who undergone premenopausal bilateral salpingo-oophorectomy recommend practical usage of HT in this cohort (BSO). The data in this population are reassuring, and most doctors are comfortable using it in the high-risk population up to natural menopause; expert opinions differ on which formulations are favoured [8]. Large prospective studies demonstrate that using HT helps to mitigate the negative health implications of premature surgical menopause.

CONCLUSION

Many BRCA mutation carriers decline the recommendation of premenopausal RRSO. Thirty-six percent of the 785 BRCA mutation-positive women who enrolled in the National Ovarian Cancer Prevention and Early Detection Study chose the investigational cancer screening arm over the standard of care RRSO arm. RRSO uptake varies widely from 32-74% and appears to be decreasing. For women who decline, prophylactic salpingectomy with delayed oophorectomy (PSDO) is an option. In a 2013 practice statement on Salpingectomy for Ovarian Cancer Prevention, the SGO recommended that physicians of such patients discuss the option of salpingectomy while awaiting oophorectomy. In contrast, the NCCN discourages PSDO outside of a clinical trial. Ongoing studies are evaluating the uptake, safety, impact on quality of life, and effectiveness of PSDO.

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Not applicable.

CONFLICT OF INTEREST

The author has no conflicts of interest to declare.

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