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Major Risk Factors Involved in Occurrence of Ovarian Cancer

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Short Communication

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ABOUT THE STUDY

Ovarian cancer rarely develops before the age of 40, with a median age of 63 years at diagnosis. With an incidence of 57 per 100,000 in the 70 to 74-year-old age group, the age-specific incidence peaks in the ninth decade as it rises with age. For the past three decades, incidence rates and the number of cancer-related deaths have remained largely steady. With a 5-year OS of 93%, the survival is noticeably improved for the 30% of patients with localised illness.

Yet, with an anticipated 5-year OS of 20% to 30%, roughly 70% of women receive a diagnosis after the cancer has spread. Women who were diagnosed before the age of 65 had a higher 5-year OS (65.8%) than women who were diagnosed after the age of 65 (32.9%). It is yet unknown what molecular processes are involved in the carcinogenesis of ovarian cancer. Nonetheless, epidemiologic studies have found environmental, hormonal, and genetic risk factors.

Environmental risk factors

Ovarian cancer risk has been linked to factors like race and country of residence. It is mostly seen in women in northern Europe and North America. Hispanic, Asian, and American Indian women's reported incidence rates are

the lowest. Several dietary factors have been examined in relation to the risk of ovarian cancer, such as a diet heavy in meat and calories, antioxidant intake (such as vitamins A, C, and E), and consumption of lactose or coffee, but none have consistently been demonstrated to significantly change the risk [1,2].

Hormonal factors

Many epidemiologic studies have demonstrated that hormonal factors, such as increasing parity, age at first birth before age 25, history of breastfeeding, usage of oral contraceptives, and tubal ligation, reduce the chance of developing ovarian cancer by 25% to 60%. The Million Women Study and a more recent Danish cohort study both identified hormone replacement treatment as a risk factor for ovarian cancer. Both studies showed that current hormone replacement treatment users had a 1.5-2 fold higher chance of getting ovarian cancer.

The "incessant ovulation" theory, which holds that each ovulatory cycle damages the ovarian epithelium and that the process of aberrant repair subsequently acts as the first step in carcinogenesis, is supported by the protective benefits of parity, multiple births, oral contraceptive use, and breastfeeding. Hence, a woman's lifetime chance of having ovarian cancer increases with the number of ovulatory cycles she has. According to a second theory, excessive gonadotropin secretion increases the risk. The elevated gonadotropin concentration causes estrogenic stimulation, which traps epithelial cells in inclusion cysts and promotes cell proliferation and malignant transformation. Pregnancy and oral contraceptive use both reduce gonadotropin levels in addition to halting ovulation; therefore both conventional risk-reducers are consistent with both theories. So, it would be expected that women receiving ovarian stimulation therapy for infertility would have a higher chance of developing ovarian cancer. Although extended usage of the ovulation stimulator, clomiphene citrate, in a group of women receiving treatment for infertility was associated with an elevated incidence of mainly borderline tumours, the data have not proven reliable. A significant Danish population-based cohort research as well as a retrospective cohort analysis of over 10,000 American women who took a variety of infertility drugs did not find any evidence to indicate a link between infertility drugs and ovarian cancer [3,4].

Genetic factors

Only 15% of all ovarian cancers are linked to known genetic abnormalities; this includes 2% of hereditary nonpolyposis colon cancer syndrome cases and 13% of cases of the hereditary breast and ovarian cancer syndrome. Colon, endometrial, and ovarian cancer are three cancers that are more likely to strike women with Hereditary Nonpolyposis Colon Cancer (HNPCC) syndrome. Women with HNPCC have a 15% to 25% lifetime risk of getting ovarian cancer. Several ethnic groups have different rates of BRCA mutations. Certain ethnic groups have founder mutations that have been identified; for instance, up to 40% of all women of Ashkenazi Jewish heritage with ovarian cancer carry one of three founder variants [5].

Women who are Polish and French-Canadian have been discovered to have more founder mutations. A BRCA1 mutation increases a woman's lifetime risk of getting ovarian cancer by 30% to 40%. Depending on where in the genes the mutation occurred, women with a BRCA2 mutation have a 15% to 25% lifetime risk of having ovarian cancer; a larger risk is associated with a mutation in the region of the genes that code for the ovarian cancer cluster. A BRCA1 or BRCA2 mutation in a woman increases her risk of developing fallopian tube cancer. For people with BRCA1 and BRCA2 mutations, the lifetime chance of developing breast cancer ranges from 50% to 85%.

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