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PRIMARY OVARIAN CANCER PREVENTION WITH CONTRACEPTIVE PILLS

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KEYWORDS

INTRODUCTION

Though some theories include the cumulative consequences of repeated ovulation, the genesis of ovarian cancer is still poorly understood[1] With exposure to high gonadotropin concentrations in the ovary.[2] Reproductive factors that interrupt ovulation, such as pregnancies, oral contraceptive use, and lactation, consistently have been associated with a reduced ovarian cancer risk.[3-8] Carriers of the BRCA1 and BRCA2 mutations have a higher lifetime chance of getting ovarian cancer. In this high-risk demographic, oral contraceptives have been demonstrated to considerably reduce the risk of ovarian cancer by about 50%.[9]

In an effort to lower the frequency of negative side effects including thrombosis and cardiovascular events without sacrificing contraceptive efficacy, the estrogen and progestin concentration of oral contraceptive pills has steadily dropped during the past 50 years.[10,11]

Lower hormonal dosages, various steroid kinds, and non-oral methods of delivery have been introduced as a result of changes in contraceptive formulations and usage trends over time. It is unknown how these alterations will affect the likelihood of getting ovarian cancer.[9]

Prevalence Of Primary Ovarian Cancer

Among gynecological malignancies, ovarian cancer is the most common cause of cancer death. Because this tumor typically develops asymptomatically, most patients have advanced stages (III/IV) when they first show symptoms. For cases with these stages, the 5-year survival rates are projected to be between 20 and 25 percent.[12] Due to the disease's relatively high fatality rate—four out of five patients are identified with advanced disease—ovarian cancer accounts for 2.5% of all malignancies among females but for 5% of all cancer deaths.[13] A research objective is to increase early detection and prevention because local-stage illness has a 93% 5-year relative survival rate.[13]

Epidemiology of primary ovarian cancer: One in 75 women will get OC in their lifetime, and one in 1004 will pass away from the condition. The disease often manifests at an advanced stage, when only 29% of patients survive five years. When the 5-year survival rate is 92%4, only 15% of individuals have localized tumors (stage 1). Surprisingly, the global 5-year relative survival rate has only slightly increased (2%–4%) since 1995 and typically varies between 30% and 40%.[14] The developed world's regions, such as North America and Central and Eastern Europe, have the highest age-adjusted incidence rates, with rates typically above 8 per 100,000. Rates are lowest in Asia and Africa (3 per 100,000), and are intermediate in South America (5.8 per 100,000). Greater risk occurs from migration from nations with low rates to those with high rates.[15,16] Racial disparities in incidence and death mirror the documented global variance within the United States, with Whites experiencing the greatest rates, Hispanics experiencing intermediate rates, and Blacks and Asians experiencing the lowest rates.[17] The incidence and death rates are higher in developed, urban regions vs less developed, rural regions within major countries like China, which replicates international variance.[18] Epidemiologic studies have repeatedly demonstrated that women who use oral contraceptives have a lower chance of developing ovarian cancer.19

How OCP Works In The Prevention Of Ovarian Cancer

Progestin and estrogen, the hormonal components of oral contraceptives, are thought to influence the onset and/or progression of ovarian cancer. It has been suggested that progesterone can prevent the growth of ovarian tumors.[20]

Since women using oral contraceptives that contain only progestin are likewise at a lower risk of developing ovarian cancer, even if ovulation is only suppressed in around 40% of users, this protective effect may not be dependent on how progestin affects ovulation.[21] Contrary to progesterone, estrogens are thought to hasten the development of ovarian tumors.[22] Studies have revealed that estrogen-only hormone replacement therapy for women increases their risk of ovarian cancer.[23-25] To stop the hyperplasia or atrophy of the breast and endometrial, ovarian secretions must be effectively inhibited and there must be a balance between the hormones estrogen and progestin. While consecutive pills are excessively highly dosed in estrogen and also cause hyperestrogenism, the use of low dose progestins is frequently associated with insufficient suppression of gonadotrophins and relative hyperestrogenism.[26]

Review Of The Studies

Researchers discovered a protective relationship between oral contraceptives and ovarian cancer many years ago. This discovery has been confirmed by numerous observational studies utilizing OOCPs in various patterns and formulations in essentially unselected groups. Depending on the length of usage, many of these studies found that the risk was decreased by up to 50%. It is also well accepted that OCs may lower the risk of colorectal and endometrial cancer.[27] According to a study, using oral contraceptives regularly lowers the incidence of ovarian cancer in families, and the effect magnitude was determined to be statistically robust. Researchers found a statistically significant reduction in the risk of non-carriers when investigated this connection according to BRCA1/2 mutation status. [28] Oral contraceptives dramatically decreased the incidence of ovarian cancer in BRCA1/2 mutation carriers, according to a meta-analysis of 18 trials. In a case-control analysis including sister controls, it was found that ever use of oral contraceptives was associated with a 50% lower risk of ovarian cancer than never use, and usage for six or more years was associated with a 60% lower risk. They discovered a 60% reduced risk of ovarian cancer among those who had ever used the drug and a 70% reduced risk of ovarian cancer with six or more years of use when they limited their study to only cases and controls with a verified BRCA1/2 mutation.[29]

A study looked at whether using an oral contraceptive before receiving an ovarian cancer diagnosis was linked to better overall survival and progression-free survival. In univariate analysis, this protective connection was established, while multivariate analyses produced less reliable results. Prior oral contraceptive use in the latter was linked to better progression-free survival but not overall survival. Patients who were younger reported using oral contraceptives more frequently and living longer. This study offers additional proof that prior oral contraceptive use is linked to superior clinical outcomes in ovarian cancer patients, at least in terms of progression-free survival.[30]

The relative risk of ovarian cancer among users of all modern combination hormonal contraceptives, by progestogen type in combined preparations, and for all progestogen-only products, including non-oral preparations, was determined using Poisson regression. Women who had switched from one method of contraception to another and those with complete histories of contraception were also subjected to separate studies. The population prevented fraction was estimated after examining the duration, time since last use, and tumor histology. The study showed modern combination hormonal contraceptives are associated with a lower risk of ovarian cancer in women of reproductive age; this impact is related to the duration of use and declines after quitting use, according to Poisson regression, which was used to determine relative risk. These findings imply that progestogen-only products have no protective effect.[31] According to two studies from the late 1980s, combination oral

contraceptives (OCs) cut the risk of endometrial cancer by 50% and maintained that reduction in risk for up to 15 years after users stopped using OCPs. The risk was decreased by lower levels of estrogen and further decreased by higher levels of progesterone. Preparations with the lowest estrogen and highest progesterone levels produced the best level of protective effect.[32] In postmenopausal women who had ever used OCPs, a Seattle study from 1993 found a risk reduction of 52%. The risk climbed to 2.71 if OC use had begun between the ages of 20 and 24 though. The incidence of ovarian cancer was reduced by 36% (RR = 0.64), according to a meta-analysis conducted in 1992 on 20 big trials involving women who had used combined OCs for more than a year. Each year of OC consumption decreased the risk by 11%.[32] The risk was 54% lower when OCPs were used for more than 5 years. With continued OCP use, the risk was observed to decrease linearly. Ovarian cancer was prevented by using OCPs for more than ten years. The risk of ovarian cancer was dramatically raised by a favorable family history and nulliparity. In a 1994 meta-analysis, these parameters were examined in relation to the development of ovarian cancer, and it was discovered that usage of OC decreased risk in all groups (risk, age, and duration of use) in roughly identical amounts. The risk was decreased to a degree that was lower for women who had not used OCPs after ten years of use.[32] A research suggested that Using OCPs can prevent ovarian cancer for at least 15 years after stopping pill use. Inhibition of ovulation is the most plausible mechanism for this protective effect. Ovarian malignancies have been shown to have a substantial correlation between the total number of ovulations over the course of a person's lifetime and p53 gene mutation. It is possible that processes other than preventing proliferation-associated mutations contribute to the protective effect of ovulation inhibition because use of the pill for five years reduces the risk of ovarian cancer by 40% while reducing lifetime ovulations by only around 15%. The risk of endometrial cancer is also reduced by the pill by roughly 50%. From the standpoint of contraception and cancer prevention, OCPs currently offer US women the best choice.[33]

CONCLUSIONS

OCP use is linked to significant, duration-dependent decreases in ovarian cancer incidence in the general population.

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