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Commentary

The Role of Hormones in Uterine Cancer: Causes and Preventive Measures

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DESCRIPTION

Uterine cancer is also known as endometrial cancer is one of the most common cancers affecting women, particularly in postmenopausal years. The majority of uterine cancers develop in the lining of the uterus, known as the endometrium. Hormones, particularly estrogen and progesterone, play a significant role in the regulation of the endometrium during a woman's reproductive years, but when hormonal imbalances occur, they can lead to abnormal growth of the endometrial tissue, increasing the risk of cancer. The endometrium undergoes cyclical changes throughout a woman's life, largely controlled by two key hormones: Estrogen and progesterone. During the menstrual cycle, estrogen stimulates the growth and thickening of the endometrial lining, preparing the uterus for a potential pregnancy. If pregnancy does not occur, progesterone facilitates the shedding of the endometrial lining during menstruation.

However, prolonged or unopposed estrogen exposure (when estrogen is not balanced by sufficient levels of progesterone) can lead to endometrial hyperplasia, an excessive thickening of the uterine lining. Hyperplasia increases the risk of abnormal cell growth, which can evolve into endometrial cancer over time. Estrogen dominance either due to high levels of estrogen or low levels of progesterone is thus a significant factor in the development of uterine cancer. There are several factors that can lead to hormonal imbalances, particularly increased estrogen levels, which in turn heighten the risk of developing uterine cancer. Fat tissue produces estrogen and women with higher body fat levels have increased circulating estrogen levels. This is particularly concerning for postmenopausal women, as their ovaries stop producing estrogen after menopause and fat tissue becomes the primary source of estrogen. Obesity can therefore lead to chronic estrogen exposure, increasing the risk of endometrial hyperplasia and cancer.

Estrogen replacement therapy, often prescribed to alleviate menopausal symptoms, can increase the risk of uterine cancer if not combined with progesterone. Unopposed estrogen therapy, which involves taking estrogen without progesterone, can stimulate excessive endometrial growth, leading to cancerous changes over time. Polycystic Ovary Syndrome (PCOS) is a condition characterized by hormonal imbalances, including elevated levels of androgens (male hormones) and irregular ovulation. Women with PCOS often have infrequent menstrual cycles, leading to prolonged periods of unopposed estrogen exposure. This increases the risk of endometrial hyperplasia and subsequent cancer development. Women who experience early menarche (the onset of menstruation) or late menopause have a longer reproductive lifespan and, consequently, more prolonged exposure to estrogen. This extended exposure can increase the risk of uterine cancer due to the cumulative effects of estrogen on the endometrium.

While certain risk factors for uterine cancer, such as genetic predisposition and reproductive history, cannot be altered, there are several strategies to reduce the risk of developing uterine cancer by addressing hormonal imbalances. Maintaining a healthy weight is one of the most effective ways to reduce the risk of uterine cancer, especially in postmenopausal women. Weight loss in overweight or obese women can reduce the levels of circulating estrogen produced by fat tissue, lowering the risk of endometrial hyperplasia and cancer. Studies have shown that the use of combined oral contraceptives (which contain both estrogen and progesterone) for five or more years significantly reduces the risk of uterine cancer. The protective effect of oral contraceptives lasts for many years, even after a woman stops taking them. This is thought to be due to the role of progesterone in preventing endometrial overgrowth. Regular exercise has been shown to reduce the risk of various cancers, including uterine cancer. Exercise helps regulate body weight, improve insulin sensitivity and lower estrogen levels, all of which contribute to reducing the risk of endometrial hyperplasia.

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