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Opinion

Endometrial Cancer and its Epidemiology

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OPINION

Endometrial cancer is most commonly diagnosed at endometrial biopsy in symptomatic patients, after a postmenopausal patient reports vaginal bleeding. Unlike breast and prostate cancer, where screening tests are available to the general public, endometrial cancer is most commonly diagnosed at endometrial biopsy in symptomatic patients, after a postmenopausal patient reports vaginal bleeding. There is no universally relevant screening test. In the absence of vaginal bleeding, an increased endometrial stripe or other intrauterine abnormalities, such as a polyp, may trigger biopsy in individuals who have a pelvic ultrasound for another reason. Ultrasound is not suggested as a screening tool in asymptomatic individuals, according to the majority of experts. Both simple and complicated hyperplasia is common non-cancerous histology findings. Endometrial cancer can develop in 1–29 percent of cases if they are left untreated, depending on the kind of hyperplasia (simple vs. complicated) and the degree of cytologic atypia. A recent study conducted within the Gynecologic Oncology Group (GOG) revealed that a large percentage (42%) of patients with a biopsy diagnosis of atypical endometrial hyperplasia have a concurrent endometrial cancer at the time of hysterectomy, in addition to the risk of cancer progression with a diagnosis of endometrial hyperplasia made in the community setting. A comparable study at an academic medical facility looked at the frequency of endometrial cancer in hysterectomy materials from patients who had been diagnosed with atypical hyperplasia prior to surgery. Patients with a pre-operative diagnosis of endometrial hyperplasia had a slightly greater incidence (48%) of endometrial cancer, according to this study. In contrast, several smaller studies have shown rates of endometrial hyperplasia and endometrial cancer co-existence as low as 10% of the time. These findings imply that women with atypical endometrial hyperplasia should be closely monitored, with serious consideration given to hysterectomy in women who have finished childbearing or are not interested in reproduction, and progestin medication in women who want to keep their fertility. Depending on risk factors, adjuvant RT (vaginal brachytherapy or external beam), chemotherapy, or hormonal therapy may be indicated for patients who have undergone an adequate staging and treatment procedure. In the post-operative period, patients are classified based on risk stratification. Patients with low and low-intermediate risk may not require post-surgical therapy; however, if molecular risk factors such as p53 mutations are identified, this decision may be influenced. Given the potential negative effects of adjuvant therapy, it's critical to distinguish between patients who would benefit from adjuvant therapy and those who would be better served by just keeping a careful eye on their condition. Because 75 percent of recurrences occur in the pelvis, those with a high-intermediate risk should receive RT after surgery to minimise local recurrence. There is currently no well-established treatment procedure for people with advanced-stage illness, though clinical trials are underway. Adjuvant treatment is usually RT for high-risk cases confined to the uterus and chemotherapy for instances with extrauterine illness in high-risk patients. Post-operative pelvic radiation therapy reduces local recurrences but has little effect on overall survival, according to large prospective clinical trials. When treating patients with early stage endometrial cancer, many clinicians were concerned about the negative effects of whole pelvic radiation. Recent evidence from the PORTEC-2 trial shows that vaginal brachytherapy is no worse than whole pelvic radiation therapy, and as a result of this experiment, many hospitals in the United States have switched to vaginal brachytherapy for patients who require adjuvant radiation therapy. Patients treated with entire Pelvic Radiation Therapy (PORTEC-1) had higher urinary and bowel dysfunction, while patients who got vaginal brachytherapy had fewer side effects than those who received pelvic radiation, according to long-term follow-up studies for PORTEC-1 and PORTEC-2 (PORTEC-2). Obesity is certainly a risk factor for endometrial cancer development, but the processes by which this occurs remain unknown. While one theory is that estrone is produced in the adipose tissue and converted to estradiol in the endometrium locally, recent research suggests a genetic relationship between obesity and endometrial cancer. A recent study found a link between single nucleotide polymorphisms in genes linked to obesity and endometrial cancer. Much more needs to be learned about the link between obesity and endometrial cancer, and the National Cancer Institute (NCI) and other funding organizations are supporting these efforts, as seen by the NCI's recent request for proposals directly connected to obesity. The endometrium of the uterus is extremely susceptible to hormone stimuli. Progesterone causes epithelial differentiation, while oestrogen promotes epithelial proliferation. The use of progestin medication to treat endometrial cancer has been documented in various recent reviews. Progestins are hypothesised to induce tumour cell differentiation, as well as allow for the activation of apoptotic pathways or prevent active cell

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division, in order to produce the anti-tumor impact. Prognosis and response to progestin medication are both strongly correlated with PR expression, which is unsurprising. The overall response rate in patients with high PR expression is 72 percent, compared to 12 percent in patients with tumours without PR. It's worth noting, though, that patients who respond to progestin medication initially usually relapse. Progestins increase downregulation of ER and PR, which could be one reason for the lack of long-term effect. A pulse of oestrogen is hypothesised to either upregulate both ER and PR (allowing for more persistent responses to progestin therapy) or attracts neoplastic cells into the cell cycle in a synchronised manner, increasing chemotherapy susceptibility. Re-expression of PRB in PR-negative endometrial cancer cells, on the other hand, restored progestin regulation of cell proliferation, according to our findings. The GOG used an estrogen-like chemical like tamoxifen along with intermittent progestin use in trial 119 to try to avoid the progestin-dependent down-regulation of PR. In this study, the response rate in advanced disease was 33%, and it was separated by hormone receptor expression.