ISP-1-4 Small Endometrial Carcinoma 10 mm or less in Diameter : Clinicopathologic and Histogenetic Study of 131 Cases for Early Detection and Treatment

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[Objective] Natural history and clinicopathologic features of early endometrial carcinoma are not evident. Its knowledge is essential to make up strategies for prevention, early detection and treatment of endometrial carcinoma.[Methods] Clinicopathologically 131 patients with endometrial carcinoma measuring 10 mm or less in diameter (small endometrial carcinoma) were studied. Institutional Review Board approval was obtained with informed consent.[Results] The patients were, on average, 5 years younger than the controls whose carcinomas measuring greater than 10mm. Seventy-six% had the carcinomas located in the upper third section of the uterine corpus. Macroscopically 44% of the tumors were flat and 56% were elevated. Forty % of small endometrial carcinomas were associated with endometrial hyperplasia and 60% were not.[Conclusion] It is logical to believe that there are two pathways of endometrial carcinogenesis : carcinomas occurring from hyperplasia (40%) and carcinomas occurring from normal endometrium (60%). As hyperplasia-carcinoma sequence is not a main route we cannot probably prevent carcinomas only by treatment of hyperplasia. Effort must be focused on detecting early de novo carcinomas. As most small endometrial carcinomas arise in the upper third of the corpus, careful endometrial sampling is important for early detection.

ISP-1-5 Hormone therapy for women with atypical endometrial hyperplasia and stage Ia endometrial cancer

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[Objectives] The data from a 30-year, nationwide, population-based study in Taiwan by using records of 11558 women with uterine carcinoma from the Taiwan cancer registry provided important information on the secular trend of uterine cancer incidence. The number of cases of hormone-dependent type I endometrial cancer has increased rapidly in the past 30 years in Taiwan compared to other countries. The age-adjusted incidence rate of endometrioid adenocarcinoma increased from 0.83 per 100,000 women per year between 1979 and 1983 to 7.50 per 100,000 women per year between 2004 and 2008.The incidences of endometrioid adenocarcinoma increased more and more quickly in women with a younger age, especially for those born in recent cohorts. The incidence rates of endometrial cancer has also increased in Asian countries such as Japan and China, but with a relatively slower increase rate compared to that in Taiwan. Taiwan underwent rapid industrialization in the 1960s, and Taiwanese women born after the 1960s tend have an earlier menarche, delayed childbearing and reduced fertility rates. The body mass index of Taiwanese women has increased in past 30 years due to a westernization of lifestyles. All these risk factors can lead to prolonged, unopposed endogenous estrogen stimulation of the endometrium and increase the incidence of endometrial cancer. Uterine carcinomas are categorized as type I and type II carcinomas based on the pathogenesis of disease and clinical behavior of the patients. Endometrioid adenocarcinoma, regarded as type I carcinoma, accounts for about 80% of uterine carcinomas and follows a continuum of hyperplastic lesions that range from endometrial hyperplasia without atypia, to endometrial hyperplasia with atypia, to well-differentiated endometrial carcinoma. Atypical endometrial hyperplasia has been associated strongly with progression to endometrial carcinoma and the presence of concomitant endometrial carcinoma. Although atypical hyperplasia can be treated successfully with progestins, hysterectomy is recommended for postmenopausal women with cytologic atypia. From our previous review of 77 patients who had undergone hysterectomy for endometrial hyperplasia diagnosed by D&C. Fourteen out of 26 (54%) women with atypical endometrial hyperplasia were diagnosed with endometrial carcinoma as compared with six out of 51 (12%) women without cytologic atypia. In conclusion, the concurrent rate of endometrial carcinoma is patient with presumed endometrial hyperplasia preoperatively was 26%. Since the age of women diagnosed of endometrioid adenocarcinoma are younger in the recent years, fertility preservation might be an important issue in this subgroup. Because current standard treatment, including hysterectomy and bilateral salpingo-oophorectomy, might not be acceptable for women who want to retain their fertility, many of these women are being managed conservatively with oral progestin. Therefore we reviewed patients diagnosed of atypical endometrial hyperplasia and endometrial cancer and reported the clinical outcomes and pregnancy outcomes after progestin treatment.[Methods] Between January 2010 and August 2013, records of 18 patients with endometrial hyperplasia with atypia and 11 patients with endometrial cancer who received fertility preserving treatment were reviewed.[Results] We investigated the women by clinical parameters including age, menopausal status, obstetrical history, medical history of diabetes, hypertension and BMI. We also reviewed initial presentation, progestin treatment dose and duration, response duration, presence of recurrence and if patients underwent hysterectomy the presence or absence of malignancy.[Conclusions] Less than 10% women diagnosed of endometrial cancer are aged 40 or younger and are often associated with obesity, infertility or exogenous estrogens. With an increasing proportion in this subgroup of patients fertility preservation is an issue of concern therefore hormone/progestin treatment becomes promising. A systemic review of the contemporary literature by Camille C. Gunderson, endometrial hyperplasia has a significantly higher likelihood of response (66%) to hormonal therapy than grade 1 endometrial carcinoma (48%). Disease persistence is more common in women with carcinoma (25%) compared to hyperplasia (14%). Despite the good response of progestin treatment our previous study showed a significant proportion of women with atypical hyperplasia harbored underlying carcinoma.This probably explained why progestin treatment was less used in our hospital whether in patients of atypical endometrial hyperplasia or endometrial cancer and small case numbers in this review study. In our hospital, progestin agents administered included medroxyprogesterone acetate and megestrol acetate. Women underwent repeat endometrial sampling every three months with either hysteroscopy biopsy or endometrial curettage. In this review only one case achieved pregnancy and 8 of 11 patients with endometrial cancer did not achieve regression (either as persistent endometrial cancer or atypical hyperplasia). Half of the patients (9/18) of atypical endometrial hyperplasia regressed after progestin use however in the non-regression group two patients were found to have endometrial cancer after hysterectomy with presumed atypical hyperplasia.With increasing numbers of young endometrial cancer patients we hope to gain more experience in fertility preservation management of atypical hyperplasia and endometrial cancer whether in aspects of case selection, progestin administration protocol or follow-up guideline to achieve better clinical outcome.