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Screening of Ovarian Cancer in Hong Kong

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From March 1993 to March 1998, 35,581 women attended the Well Women Clinic, Kwong Wah Hospital for breast, cervical and ovarian cancer screening. They were self referred, asymptomatic and had no known gynaecologic abnormalities. Transvaginal ultrasonography with a high-resolution vaginal transducer was performed for ovarian cancer screening in 6,022 women. The ovarian morphology, outline and volume were studied. An ovarian volume of at least 20 cm in premenopausal women and 10 cm in postmenopausal women was considered abnormal. 286 patients had abnormal vaginal ultrasonograms (4.7%). Four patients were confirmed to have ovarian cancers on laparotomy: one with serous cystadenocarcinoma; one with mucinous cystadenocarcinoma; one with immature teratoma; one with serous cystadenoma of borderline malignancy. All carcinomas were stage I disease. Two of these women had abnormal findings on pelvic examination and all women had normal CA-125 levels. Moreover, increased CA-125 level was detected in a significant number of patients with benign pelvic pathology. In contrast to the traditional teaching, it appears that it is not cost-effective to use transvaginal ultrasonography for screening ovarian cancer and CA-125 is not useful at all.

I S—38 Correction of CDDP resistance in Mismatch Repair Deficient Cells by Chromosome Transfer

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CDDP resistance is an important problem for improvement of long-term prognosis in the ovarian cancer.
DNA mismatch repair (MMR) deficiency has been reported as one of the factors for CDDP resistance mechanisms.
To examine the role of MMR deficiency in the CDDP resistance mechanisms, we have done the analysis of the function of G2 cell cycle check point and the instability of microsattelite loci using hMLH1 mutated cell line and the chromosome 2 transferred isolated clone.
In the results, chromosome 2 transfrred isolated clone was restored the function of G2 cell cycle check point and was completely stabilized the microsattelite loci compared as the parental cell line.
Our data suggest that loss of the G2 cell cycle checkpoint function is the major causes of CDDP resistance mechanisms in MMR deficient tumors.