OVARIAN CORTICAL STROMAL HYPERPLASIA IN ENDOMETRIAL ADENOCARCINOMA

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Introduction

Recent epidemiological studies suggest an association between endometrial carcinoma and estrogen use. A twelve fold increase in cancer risk in estrogen users as compared to the general population is reported by some (Horowitz and Feinstein, 1978). Also, there is an increased incidence of endometrial carcinoma in young women with Stein Leventhal syndrome or functional ovarian tumours which are known to be hyperoestrinic (Fechner and Kaufman, 1974). The ovaries are considered to be the major source of estrogen in premenopausal women. It is possible that ovaries in patients of endometrial carcinoma not on long term estrogen therapy may be the seat of strormal hyperplasia. Thus, we conducted this study to see the morphology of the ovary in patients with endometrial adenocarcinoma and compare them with age matched controls.

Material and Methods

Histologic preparations of ovaries removed from the study group of 25 patients with endometrial adenocarcinoma who underwent a hysterectomy and had no history of radiation or hormone therapy were available for analysis. Two groups of controls were taken. Control group I comprised of 20 age matched patients who underwent hysterectomy for estrogen related benign pelvic disease like adenomyosis or leiomyoma. Control group II consisted of 15 patients who underwent a hysterectomy and salpingo-oophorectomy for non-estrogen related conditions like prolapse of the uterus or carcinoma of the cervix.

The microscopic slides of 45 ovaries from patients with endometrial adenocarcinoma, 30 ovaries from control group I, and 18 ovaries from control group II were reviewed after randomisation. The degree of hyperplasia was classified according to Burt (1954) into 4 grades, grade 0—little or no hyperplasia in the cortex or medulla; grade I-hyperplasia of most of the cortex but no involvement of the medulla; grade II-hyperplasia of most of the cortex and partial medullary invasion; grade III-hyperplasia of most of the cortex and medulla. Other microscopic characteristics such as the presence or absence of primary follicles, follicular cysts, corpora albicantia, corpora lutea and stromal luteinisation were also determined. Luteinisation was seen as a focal alteration of stromal cells towards an epithelioid morphologic characteristic

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with a lipid laden cytoplasm and an enlarged nucleus.

Observations

The results of the degree of ovarian cortical hyperplasia in the three groups is illustrated in Table I. Both the ovaries from the same patient showed essentially the same morphology. The ovaries of endometrial carcinoma patients closely resembled the 2 control groups. Grade I (Fig. 1) and III (Fig. 2) hyperplasia was equally distributed in the three groups.

The other histologic features of the ovaries are illustrated in Table II. Corpora albicantia were observed in all the 3 groups and corpora lutea were conspicuous by their absence in endometrial carcinoma patients. Luteinisation was present in the 3 groups and appeared to be relatively more in patients with endometrial carcinoma and adenomyosis, but this was not confirmed by special stains.

No correlation was found between the pathologic grading of endometrial carcinoma and ovarian cortical stromal hyperplasia.

Discussion

Hormonal ovarian function may be a predisposing factor in the development of endometrial carcinoma. Several publications in regard to the increased incidence of ovarian cortical stromal hyperplasia in association with endometrial adenocarcinoma have appeared (Novak and Mohler, 1953; Marcus, 1963; Schneider and Bechtel, 1956). The stein-leventhal syndrome has also been linked in the development of endometrial carcinoma (Fechner and Kaufman, 1974). To the best of our knowledge except for 2 case reports (Jadhav and Deshpande, 1980) no

TABLE I

Degree of Ovarian Cortical Stromal Hyperplasia

Age (Years)	Patients with Endometrial carcinoma			Control Group I						Control Group II					
	Grade 0	Grade I	Grade II	Grade III	Grade 0	Grade I	Grade II	Grade III	Grade 0	Grade I	Grade II	Grade III	Grade 0	Grade I	Grade II	Grade III
30-39	—	—	1	—	—	2	—	—	—	2	—	—				
40-49	1	2	4	—	2	6	1	—	1	2	2	—				
50-59	4	5	3	1	1	4	4	—	2	—	5	1				
60-69	—	2	—	—	—	—	—	—	—	—	—	—				
70-79	—	1	1	—	—	—	—	—	—	—	—	—				
Total	5	10	9	1	3	12	5	—	3	4	7	1				
Percentage	20	40	36	4	15	60	25	—	20	26.6	46.6	6.6				

TABLE II
Histologic Features of Ovaries

Study Group	Total number of patients	Number of Patients with			
		Presence of follicular cysts	Presence of Corpora Albicantia	Presence of Corpora Lutea	Presence of Luteinisation
Patients with endometrial carcinoma	25	6 (24%)	22 (88%)	3 (12%)	5 (20%)
Control Group I	20	7 (35%)	20 (100%)	6 (30%)	4 (20%)
Control Group II	10	5 (33%)	13 (86%)	5 (33%)	4 (27%)

such studies are available in Indian women.

From our study it is obvious that there is no correlation between stromal hyperplasia of the ovaries and endometrial carcinoma. Also no hyperplasia was observed in benign conditions considered to be related to high estrogen levels. Ramzy and Nisker (1979) also found that the ovaries in young women with endometrial adenocarcinomas were similar to normal ovaries.

Gusberg and Kardon (1971) reported that out of 115 cases of theca-granulosa neoplasms, 21% had an associated endometrial carcinoma. This is a much larger incidence than the 3% reported by Emge (1953) and 10% by Larson (1954). In our study there were no estrogen producing neoplasm in the ovaries studied.

Jackson and Deckarty (1957) reported 16 cases of endometrial carcinoma in 43 women with the Stein-Leventhal syndrome. However, (Ramzy and Nisker, 1979) did not find any of the 15 ovaries examined from patients with endometrial carcinoma to have features suggestive of Stein-Leventhal syndrome. We also did not find any such incidence in our patients.

As the microscopic appearance of the ovaries compares with normal ovaries it is likely the abnormality in steroid function may be of nonovarian origin (Nisker *et al* 1978).

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