OVARIAN CORTICAL STROMAL HYPERPLASIA

(Report of 2 Cases)

by

MRS. A. J. JADHAV,* M.S.

and

MRS. A. S. DESHPANDE,** M.D.

The manner in which the ovary may participate in the genesis of endometrial carcinoma, possibly through a sequence which includes endometrial hyperplasia, has continually aroused much interest. Various studies have explored the possibility that abnormal ovarian activity results in a hormonal overproduction or imbalance which may influence the incidence of endometrial cancer. Attention has been directed to the ovarian cortical stroma, especially in its hyperplastic state. Since the initial report of hyperplasia of the stroma of the ovarian cortex by Smith in 1941, there has subsequently been an increasing interest in this ovarian condition, particularly in its relevance to malignancies of endometrium, breast and the other more commonly encountered states of endometrial activity e.g., endometrial hyperplasia.

In the reported 2 cases of postmenopausal bleeding where endometrial pathology turned out to be endometrial hyperplasia in one and endometrial carcinoma in other, the ovaries being the primary source of estrogen, were studied in an attempt to correlate their pathology with the endometrial picture.

Case 1

Mrs. D. B. aged 60 years, para 7, menopause since 10 years was admitted to Government Medical College Hospital, Nagpur for postmenopausal bleeding since 8 days and leucorrhoea since 2 years.

On general and systemic examination, she was an obese woman with mild hypertension—160/100 mm of Hg. and was being treated for chronic asthma.

Abdominal examination revealed nothing abnormal. On vaginal examination, there was a small, soft flattened cervical polyp, ½ cm. in length arising from anterior cervical lip, uterus A. V. bigger in size for her age. No adnexal mass palpable.

Investigations—Hb 60%, urine showed sugar 0.5% in postmeal samples. Fasting and postprandial blood sugar levels were 105 and 132 mg% respectively, glucose tolerance test showed normal blood sugar levels with glycosuria 0.5% in all urine samples. Blood urea—28 mg%. Vaginal cytology showed the maturation index as 0/80/20.

On curettage good amount of endometrial strips were obtained. Histopathological examination showed it to be endometrial hyperplasia with features of both proliferative and secretory activity, latter predominating along with adenomatous polyp. Panhysterectomy was done. Gross examination revealed a bulky uterus and apparently normal tubes. Both ovaries were large for her age, yellowish grey in colour with marked convolutions. Microscopically, endometrium showed hyperplasia with marked progestational activity. Ovarian section revealed thickened cortex, prepondrance of spindle shaped stromal cells arranged in whorled and fascicles with scanty collagenous tissue.

Case 2

Mrs. R. M. aged 60 years, para 10, menopause since 20 years was admitted for postmeno-

*Reader,

**Associate Professor, Government Medical College Hospital, Nagpur.

pausal bleeding and leucorrhoea for 2 years. She had not received any hormone therapy.

General examination showed an averagely built woman with mild hirsutism, B.P. 170/100 mm of Hg. All laboratory investigations were within normal limits. E.C.G. showed right bundle branch block. Vaginal cytology showed maturation index as 2/46/52. Diagnostic curettage revealed the uterus to be larger for her age, no adnexal pathology was detected. A good amount of endometrial tissue was obtained which on histopathological examination turned out to be adenocarcinoma of endometrium.

She was subjected to laparotomy which revealed bulky uterus with normal tubes, both ovaries were larger in size for her age, with convoluted surface. In addition there was a thin walled cyst 2½" in diameter, apparently connected by thin fibrous band to right ovary. Histopathological examination confirmed adenocarcinoma of endometrium. Both ovaries showed features of cortical stromal hyperplasia. The cyst contained a small papule on inner surface. This also showed hyperplastic ovarian stromal element. It was thus concluded that the cyst must have arisen from embryonic ovarian rests.

Discussion

The recent finding of an association between estrogen therapy and endometrial carcinoma in postmenopausal women by Smith *et al* (1975), Ziel and Finkle (1975) adds to the evidence that estrogens are involved in the etiology of the disease. An association between ovarian stromal hyperplasia and endometrial carcinoma has been reported by many observers and ovaries with stromal hyperplasia have been shown to be estrogen secreting Procope (1969). A variety of ovarian tumors may be associated with endometrial hyperplasia and carcinoma, particularly when they secrete estrogens, Rome *et al* (1973).

Thus considering the role of estrogens in the genesis of endometrial carcinoma and its likely predecessor, endometrial hyperplasia, it seems logical to explore the possible source of estrogen production more so postmenopausally. In the present 2 cases, the ovaries obtained at operation were examined for histological evidence of recent or past excessive estrogenic activity. Persisting stromal activity or hyperplasia was indicated by hypercellularity, thickening and nodularity of stromal cells with additional leutinising features seen in case 1, in contrast to the atrophic appearance of normal postmenopausal ovary.

In an attempt to evaluate the estrogen level of these patients, vaginal cytohormonal study was carried out which in case 2 indicated some estrogen activity.

Thus correlating the clinical features and the pathological findings, it leads to speculate the possible source of hormonal hyperactivity as in case 1, the ovarian cortical stromal hyperplasia along with luteinisation of stromal cells and in case 2, hyperactive ovarian stroma along with additional extra ovarian source in the form of a papule in the cyst showing histological features of same hyperactive ovarian stroma.

Summary

Two post menopausal women having vaginal bleeding were investigated and treated by diagnostic curettage and subsequently hysterectomy. Histopathological diagnosis turned out to be endometrial hyperplasia in one and adenocarcinoma of endometrium in other. The ovaries obtained at operation were studied for evidence of recent or past excessive estrogenic activity. The implications of persistent stromal activity or hyperplasia in the causation of endometrial hyperplasia and adenocarcinoma are discussed.

Acknowledgement

We thank the Head of the Department of Obstetrics and Gynaecology and Dean,

Government Medical College Hospital, Nagpur, for allowing us to publish the hospital data.

References

1. Procope, B. J.: Acta Endocrinol. **60: 135**, 1969.
2. Rome, R. M., Laverty, C. and Brown, J. B.: J. Obstet. Gynec. Brit. C'wlth. **80: 984**, 1973.
3. Smith, G. V. S.: New Eng. J. Med. **225: 608**, 1941.
4. Smith, D. C., Prentice, R. Thompson, D. J. and Hermann, W. L.: New. Eng. J. Med. **23: 1164**, 1975.
5. Ziel, H. K. and Finkle, W. D.: New. Eng. J. Med. **23: 1167**, 1975.

See Fig. on Art Paper X