

POSTMENOPAUSAL BLEEDING IN PATIENT WITH INFANTILE UTERUS

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ABSTRACT

A 58 years old female patient married for 40 years, nulliparous, presented in outpatient department with the complaint of postmenopausal bleeding off and on for six months. Her total abdominal hysterectomy and bilateral salpingoophorectomy was performed. Uterus was small in size (infantile). Histopathology revealed endometrial hyperplasia with atypia.

KEYWORDS: Hypoplastic uterus, postmenopausal bleeding.

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INTRODUCTION

Hypoplastic uterus is an uncommon problem. There have been numerous classification systems for uterine malformations varying between those based on embryological development to those based on obstetric performance. However the most widely used classification in current use is that of Buttram & Gibbons¹ which is based upon the degree of failure of normal development.

CASE REPORT

Mrs. AB 58 years of age, married for 40 years nulliparous, admitted with complaints of postmenopausal spotting for the last six months. There was no associated history of any urinary or bowel complaints, drug intake, analgesic use

or use of anticoagulants, liver disease or bleeding disorder.

Her age of menarche was 19 years. She had oligomenorrhoea since menarche, bleeding for one day every 4-5 months. During investigations for infertility, she was diagnosed as having hypoplastic uterus. She is menopausal for the last ten years. Cervical smear was never done; and the couple had never practiced any contraception.

She was a known diabetic for the last 25 years taking Insulin NPH and known hypertensive for 25 years and taking ACE inhibitors. There was a history of ischaemic heart disease nine years back, no documentation was available. History of appendicectomy 20 years back. On examination, pallor was present; her blood pressure was 130/95mmHg, pulse was 90 bpm, regular. The breasts were normal. Examination of cardiovascular, respiratory system and central nervous system did not reveal any abnormality. Abdominal examination was unremarkable.

Pelvic examination: revealed atrophic vulva and vagina. Cervix was pin point and mild bleeding was seen on speculum examination. On bimanual examination size of the uterus could not be assessed.

Investigations: Blood group and Rh factor A +ve, Haemoglobin 11 gm/dl, Blood sugar random 220 mgs/ dl, coagulation profile, liver

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function tests and renal function test were within normal limit.

Abdominopelvic ultrasonography revealed an atrophic uterus 3x2x1.5 cm and endometrial thickness was 14 mm. the adnexa were normal.

Procedure: The patient was given general anaesthesia. Her total abdominal hysterectomy with bilateral salpingoopherectomy was performed. Uterus was infantile in size with the transverse diameter of 5 cm, length 4 cms and anterioposterior diameter of 2.5 cms. 1x1 cm subserous fibroid was seen on the anterior wall near the fundal area. Both ovaries were atrophic. A small clear cyst was seen in the left ovary. Cut section of uterus revealed thick endometrium. The specimen was sent for histopathology.

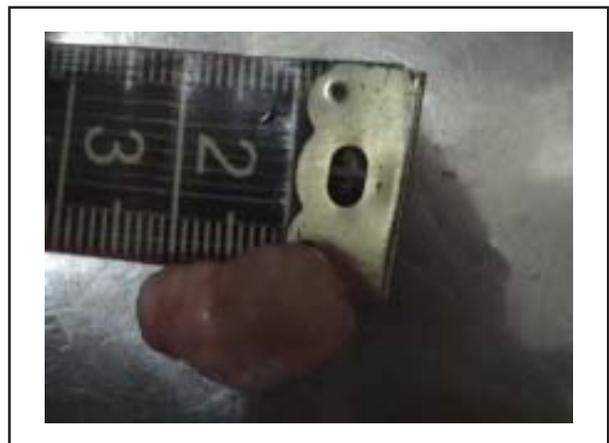
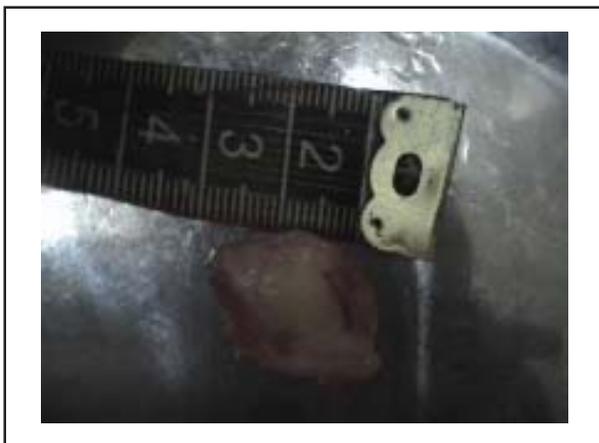
The histopathology report revealed benign serous cyst of the left ovary; atrophic right

ovary, leiomyoma and endometrial hyperplasia with moderate atypia.

DISCUSSION

The incidence of post menopausal bleeding in patients with hypoplastic uterus is rare obstetric series show an incidence of uterine abnormality ranging from 1/100 to 1/1000.² It is likely that the incidence of uterine malformation is greatly underestimated, as in the vast majority of patients no gynaecological or reproductive problems are ever experienced.

In an infertile population, the incidence increase to around 3%.³ Sorensen, investigated infertile women with oligomenorrhoea, 56% of patients were found to have mild uterine abnormalities. Oligomenorrhoea might be due to poor vascularization or steroid receptor development in the malformed uterus.⁴



Endometrial hyperplasia represents a spectrum of morphologic and biologic alterations of the endometrial glands and stroma, ranging from an exaggerated physiologic state to carcinoma in situ. Endometrial hyperplasia is important clinically because it may cause abnormal bleeding may be associated with estrogen-producing ovarian tumours, result from hormonal therapy, and precede or occur simultaneously with endometrial cancer.

Architecturally, hyperplasias are either simple or complex; the major differing features are complexity and crowding of the glandular elements. Atypical hyperplasia refers to cytologic atypia and can be categorized as simple or complex, depending on the corresponding glandular architecture.

The risk of endometrial hyperplasia progressing to carcinoma is related to the presence and severity of cytologic atypia. Kurman and colleagues found that progression to carcinoma occurred in 1% of patients with simple hyperplasia, 3% of patients with complex hyperplasia, 8% of patients with atypical simple hyperplasia, and 29% of patients with atypical complex hyperplasia.⁵

The most commonly recognized causes of endometrial hyperplasia result from excessive oestrogen stimulation unopposed by progesterone. This may arise from an endogenous source such as anovulatory cycles or an oestrogen-secreting tumour but exogenous,

unopposed oestrogen administration and the oestrogenic effects of tamoxifen are other common causes. Women with lesions showing cytological atypia who have completed their family should be offered hysterectomy.

CONCLUSION

Endometrial hyperplasia presenting in atrophic uterus is an uncommon condition. However, the management in this case would be for management of postmenopausal bleeding. Endometrial hyperplasia with atypia can potentially lead to development of endometrial cancer and total abdominal hysterectomy and bilateral salpingoopherectomy remains the mainstay of treatment.

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