Case Report

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Pathology Section

Histopathological and Immunohistochemical Evaluation of Endometrial Polyp with Endometrial Intraepithelial Neoplasia in a Postmenopausal Woman: A Case Report

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ABSTRACT

Endometrial Intraepithelial Neoplasia (EIN) is a premalignant lesion recognised as a precursor to endometrioid endometrial carcinoma. Although endometrial polyps are predominantly benign, they may rarely harbour atypical or premalignant lesions, particularly in postmenopausal women presenting with abnormal uterine bleeding. A 60-year-old postmenopausal woman presented with postmenopausal bleeding. Pelvic ultrasonography demonstrated a bulky retroverted uterus with a diffusely thickened endometrium measuring 3 cm and no focal myometrial lesion. Magnetic Resonance Imaging (MRI) confirmed diffuse endometrial thickening and additionally revealed an intramural fibroid. Owing to persistent endometrial thickening and clinical suspicion of endometrial pathology, total abdominal hysterectomy with bilateral salpingo-oophorectomy was performed. Gross examination revealed a 2.5×1.5 cm endometrial polyp within a diffusely thickened endometrium. Histopathological examination demonstrated focal EIN involving both the endometrium and the endometrial polyp, confirming the lesion originated within the polyp. The myometrium showed adenomyosis without evidence of myometrial invasion or concurrent malignancy. The cervix exhibited chronic cervicitis, while both fallopian tubes and ovaries were histologically unremarkable. Immunohistochemical analysis for Bcl-2 (B-cell lymphoma 2) demonstrated intense staining in the endometrial glands and stroma with focal loss of expression, supporting the diagnosis. The final diagnosis was focal EIN arising within an endometrial polyp in a background of adenomyosis without associated endometrial carcinoma. No clinical evidence of recurrent disease was observed during the six-month follow-up. This case highlights the importance of meticulous histopathological evaluation of endometrial polyps in postmenopausal women with abnormal uterine bleeding, as premalignant lesions such as EIN may occur within polyps despite the absence of invasive carcinoma, thereby improving early detection, risk assessment, and optimal patient management.

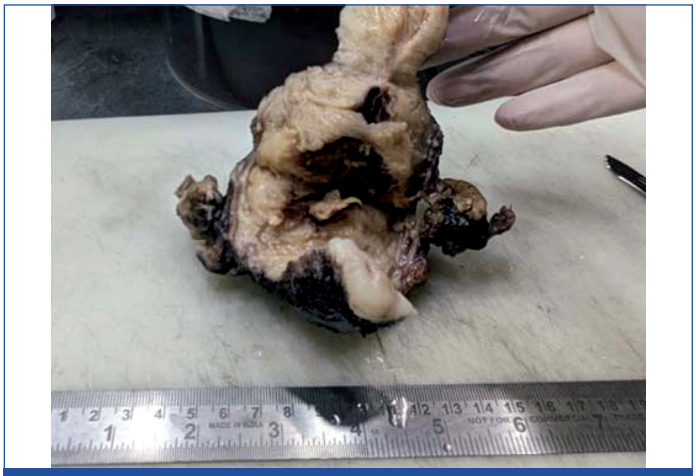
Keywords: Adenomyosis, Atypical hyperplasia, B-cell lymphoma 2, Hysterectomy, Premalignant lesion, Uterine neoplasms

CASE REPORT

A 60-year-old multiparous postmenopausal woman presented with heavy postmenopausal bleeding associated with the passage of blood clots for three months. She had a 10-year history of diabetes mellitus and hypertension and had been taking medications irregularly. There was no history of hormone replacement therapy or tamoxifen use, and no family history of gynaecologic malignancy.

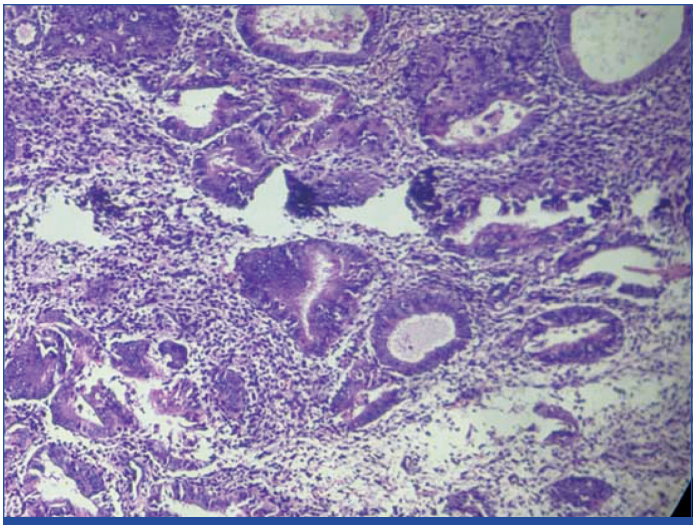
Initial evaluation included a Papanicolaou smear, which was reported as Negative For Intraepithelial Lesion or Malignancy (NILM), with reactive cellular changes secondary to inflammation. Pelvic ultrasonography revealed a bulky retroverted uterus with a diffusely thickened endometrium measuring 30 mm and no focal myometrial lesion. MRI confirmed diffuse endometrial thickening and additionally demonstrated an intramural fibroid.

In view of the persistent endometrial thickening and abnormal uterine bleeding, the patient underwent total abdominal hysterectomy with bilateral salpingo-oophorectomy. The surgical specimen was received in 10% neutral buffered formalin and subjected to gross examination according to standard pathological protocols. The uterus with cervix measured 9.7×7.5×2.7 cm. On opening the uterine cavity, a well-circumscribed pedunculated endometrial polyp measuring 2.5×1.5 cm was identified arising from the uterine fundus [Table/Fig-1]. The cut surface of the polyp was solid and homogeneous. The surrounding endometrium was diffusely thickened, measuring 3 cm. Both ovaries and fallopian tubes were grossly unremarkable.



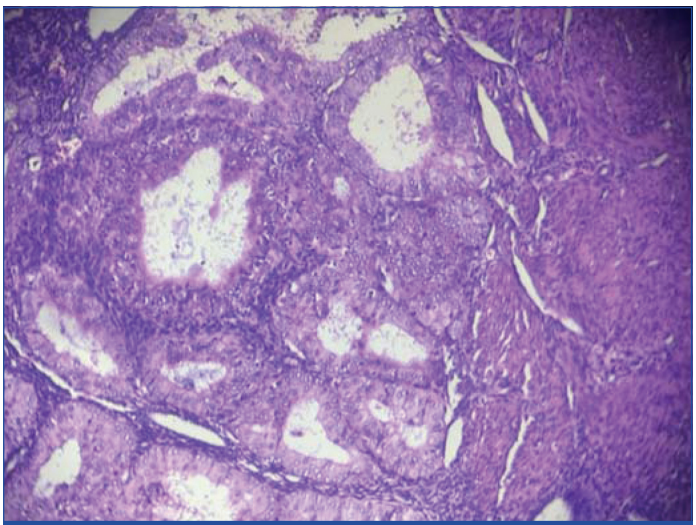
[Table/Fig-1]: Cut surface of uterus showing endometrial polyp and thickened endometrium.

Microscopic examination of the cervix revealed chronic cervicitis. Histopathological examination of the endometrium demonstrated a focal area of EIN. The associated endometrial polyp also showed focal EIN, confirming that the lesion arose within the polyp [Table/Fig-2]. The lesional glands exhibited an increased gland-to-stroma



[Table/Fig-2]: Microscopic picture showing Endometrial Intraepithelial Neoplasia (EIN) in the polyp showing back to back arrangement of endometrial glands with nuclear atypia. There is minimal stroma in between the glands (H&E,100x).

ratio (>1) with distinct cytologic demarcation from the adjacent benign endometrial glands. The surrounding endometrium showed hyperplastic changes [Table/Fig-3]. The myometrium exhibited features of adenomyosis, with no evidence of myometrial invasion or concurrent endometrial carcinoma. Both fallopian tubes and ovaries were histologically unremarkable.

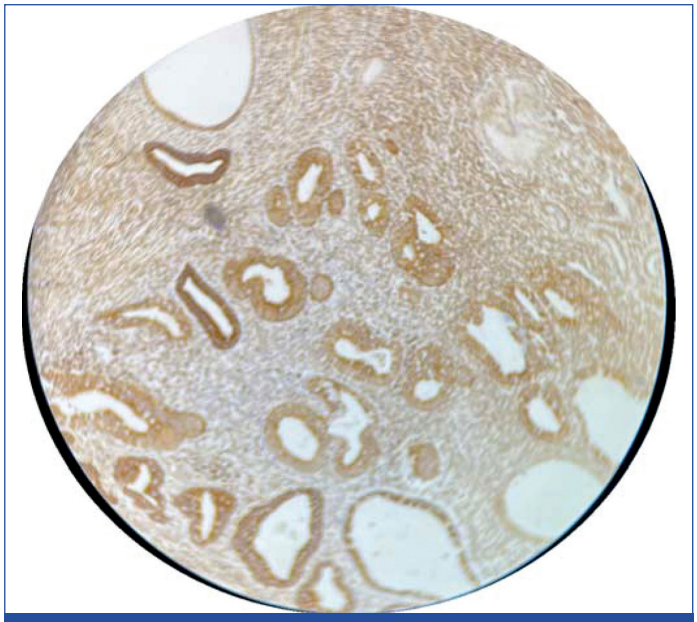


[Table/Fig-3]: Microscopy picture of endometrial hyperplasia in polyp with few glands showing nuclear atypia (H&E,400x).

Immunohistochemical analysis for Bcl-2 demonstrated intense staining in the endometrial glands and stroma with focal loss of expression, supporting the diagnosis of EIN [Table/Fig-4]. The postoperative course was uneventful, and the patient was discharged in stable condition. The final diagnosis was focal EIN arising within an endometrial polyp in a background of adenomyosis, with no evidence of concurrent endometrial carcinoma. No clinical evidence of recurrent disease was observed during follow-up period of six months.

DISCUSSION

Postmenopausal bleeding is a frequent gynaecological symptom that often prompts clinical evaluation because of its association with both benign and malignant endometrial conditions [1] and it is one of the manifestation of organic endometrial pathologies which includes endometrial polyp, endometrial hyperplasia, or carcinoma [2]. In the present case, 60-year-old female presented with the complaints of postmenopausal bleeding had an endometrial polyp which harboured EIN. The possible aetiological factors for postmenopausal endometrial pathology include hormone hypothesis, apoptosis, growth factors, ageing, selective oestrogen



[Table/Fig-4]: Immunohistochemistry for Bcl-2 showing variable expression (100x).

receptor modulator, hormone replacement therapy [3]. Alpha and beta oestrogen receptors in the glandular and stromal cell nucleus play an important role in the pathogenesis of the endometrial polyp [3]. During menstrual cycle, endometrial function and growth is regulated by the balanced expression of oestrogen and progesterone receptors at the functional and basal layer in the stroma as well as in the glandular epithelium of endometrium [3]. Focal oestrogen hypersensitive areas in the endometrium can cause the endometrium to grow leading to formation of endometrial polyp [3]. Loss of regulation of Bcl-2 expression will also lead to formation of endometrial polyp suggesting that the polyp development is not related to an excess endometrial growth rather due to loss of physiological mechanism of apoptosis [3]. Growth factors such as epidermal growth factor, transforming growth factor, platelet derived growth factor has a role in the pathogenesis of endometrial polyp formation as they function as mitogenic factors promoting the growth of endometrial basal layer cells [3]. All these growth factors are controlled by oestrogen during the proliferative phase [3]. Tamoxifen can influence healthy endometrium of postmenopausal women which results in an increase in the quantity of glandular oestrogen and stromal progesterone receptors [3].

Both conventional cervical cytology and Liquid-based Cytology (LBC) demonstrate moderate specificity for identifying uterine glandular abnormalities. However, their sensitivity for detecting glandular cell abnormalities remains relatively limited, which may result in missed diagnoses. Consequently, cytological findings should always be interpreted in conjunction with the patient's clinical history and presenting symptoms. In women with clinical features suggestive of a uterine glandular lesion, a negative Pap smear should not exclude further diagnostic evaluation, and a high degree of clinical suspicion should be maintained to ensure timely diagnosis and appropriate management [4].

EIN has histopathological criteria that distinguish it from benign hyperplasia [5]. The differentiation between atypical endometrial hyperplasia and well-differentiated endometrioid carcinoma is based on histopathological features such as the gland-to-stroma ratio and epithelial cytological abnormalities, including loss of cellular polarity, nuclear enlargement, and hyperchromasia. However, these cytological features may be encountered in benign metaplastic alterations such as tubal, mucinous, and eosinophilic metaplasia, which can pose diagnostic challenges [5]. EIN in polyp should be made in cytologic comparison of glands within the polyp and not in the non-polypoidal endometrial tissue [5,6]. Molecular studies further support the progression model from EIN to carcinoma by demonstrating shared genetic abnormalities, including microsatellite

instability and loss of PTEN and PAX2 expression [7-9]. Alterations in E-cadherin and catenin expression have also been documented in atypical hyperplasia and carcinoma [10]. These findings substantiate the premalignant nature of EIN.

Bcl-2 is an antiapoptotic protein and plays a pivotal point for the formation of endometrial polyp due to loss of regulation. The hormonal status of the patient with reference to oestrogen plays a role in control of Bcl-2 expression [3]. The presence of inflammation can stop apoptosis and influence polyp development [3]. Various studies have conflicting interpretation of Bcl-2 expression in EIN and endometrial carcinoma. Bcl-2 expression is generally strong in endometrial hyperplasia and may help inhibit progression to endometrial carcinoma [11]. In the present case, strong expression with Bcl-2 observed within the polyp, few glands with atypical features show complete loss of expression.

Postmenopausal bleeding with sonographic evidence of thickened endometrium has a significance. Transvaginal ultrasound, hysteroscopy, biopsy of endometrium by pipelle, dilatation and curettage are the most commonly used method for the endometrial evaluation. However tissue examination is considered as the gold standard method for diagnosis of the endometrial pathology. In the present case, the preoperative curettage performed at an outside institution showed endometrial hyperplasia without atypia and failed to identify EIN. This was likely due to sampling error because the EIN was focal and confined to the endometrial polyp. Preoperative endometrial polyps can be diagnosed with hysteroscopy or by radiological investigation. Polypoidal lesions may be missed by Pipelle's endometrial study like in the present case.

CONCLUSION(S)

This case highlights that postmenopausal bleeding with persistent endometrial thickening requires careful evaluation using clinical assessment, imaging, and histopathological correlation. A benign or non-atypical endometrial biopsy does not completely exclude focal premalignant lesions, particularly when they are confined to an endometrial polyp, as sampling error may occur during blind biopsy. Persistent clinical suspicion should prompt further assessment with hysteroscopy or definitive surgical management. Thorough examination of hysterectomy specimens is essential to identify focal EIN and to exclude concurrent endometrial carcinoma. The absence of carcinoma in this postmenopausal patient despite the presence of EIN illustrates the variable biological behaviour of these lesions. Variable Bcl-2 expression observed in this case further suggests the complex molecular characteristics of EIN and warrants additional investigation in larger studies.

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