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Case Report

Pathology Section

Endometrioid Borderline Tumour of the Ovary: Pathological Analysis of a Rare Case

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ABSTRACT

Endometrioid Borderline Ovarian Tumours (EBOTs) are rare epithelial tumours with low malignant potential, also referred to as atypical proliferative endometrioid tumours. EBOTs exhibit glandular proliferation and nuclear abnormalities without invading surrounding tissue, which distinguishes them from invasive endometrioid carcinomas. The endometrioid subtype represents a rare category of Borderline Ovarian Tumours (BOTs). We present a case of a 45-year-old woman who had a right ovarian haemorrhagic cyst discovered during a routine ultrasound. The diagnostic workup included a pelvic Magnetic Resonance Imaging (MRI) scan, which showed a complex ovarian cyst with solid components, suggesting a neoplasm. Serum tumour markers, including Cancer Antigen (CA)-125, Carcinoembryonic Antigen (CEA), and CA 19-9, were all normal. Given the imaging results and clinical analysis, the patient underwent a Total Laparoscopic Hysterectomy (TLH) along with a Bilateral Salpingo-Oophorectomy (BSO). Histopathological Examination (HPE) of the removed ovary revealed glandular proliferation lined by stratified columnar cells with mild to moderate nuclear atypia and no stromal invasion. Immunohistochemistry (IHC) supported the diagnosis by showing positive results for Estrogen Receptor (ER), Progesterone Receptor (PR), and Cytokeratin 7 (CK7) with a lower proliferation index (Ki-67), which helped rule out invasive carcinoma and similar conditions. This case emphasises the need for a thorough diagnostic process, including imaging, tumour markers, HPE, and IHC for accurate classification. While EBOTs are less aggressive, careful pathological interpretation is necessary to prevent misdiagnosis and unnecessary treatment. Ongoing clinical monitoring is important due to the potential for recurrence or progression. Recognising EBOTs as a separate group allows for better patient management and improved outcomes.

Keywords: Atypical proliferative endometrioid tumour, Histopathology, Immunohistochemistry

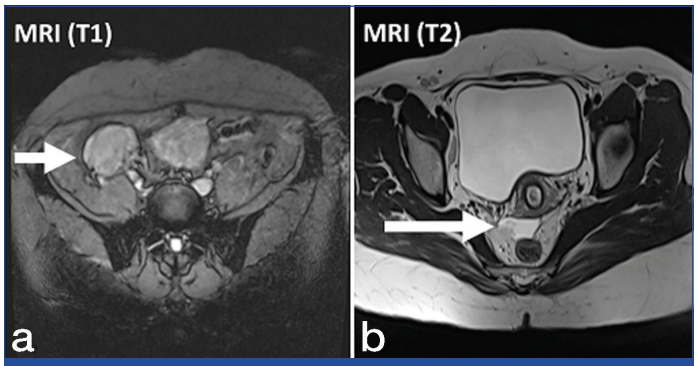
CASE REPORT

A 45-year-old female was incidentally found to have a right ovarian haemorrhagic cyst on ultrasound during a routine health check-up at an outside hospital and was referred to our hospital for further evaluation. The patient had a history of irregular menstrual cycles for the past year but no history of dysmenorrhoea. She reported no abdominal pain, loss of appetite, or weight loss. The patient denied any vaginal bleeding or abnormal discharge, and her bowel and bladder habits were normal. Her past medical history was unremarkable. In terms of obstetric history, she had one full-term delivery via caesarean section and had not undergone sterilisation. Her last childbirth was 15 years ago.

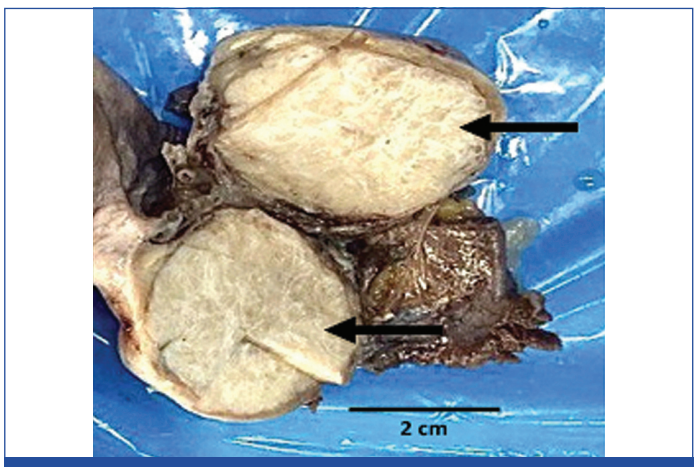
Pelvic examination revealed no significant abnormalities. Additional imaging studies were conducted to further evaluate her condition. An MRI of the abdomen and pelvis revealed a 3.9×3.4×3.2 cm lesion in the right ovary, displaying T1 hyperintensity along with tiny punctate foci of Gradient Recalled Echo (GRE) blooming [Table/Fig-1a]. These findings suggested the possibility of a cyst with a chronic haematoma or an organised haemorrhagic collection, although a neoplastic origin could not be excluded. Mild free fluid was detected in the pouch of Douglas [Table/Fig-1b], while the liver, gallbladder, pancreas, spleen, kidneys, and urinary bladder all appeared normal. The cervix, uterus, left ovary, and bilateral fallopian tubes were unremarkable.

The patient underwent diagnostic hysterolaparoscopy, followed by TLH with BSO under general anaesthesia. The procedure was uneventful, and the patient remained stable throughout her hospital stay. Her condition improved, allowing for her discharge.

The specimen, which included the uterus with cervix, bilateral tubes, and ovary, was sent for histopathological analysis. Grossly, the right ovary showed a nodular growth measuring 4×3.5×2.5 cm [Table/Fig-2]. The external surface was grey-white to grey-brown, encapsulated, and smooth. The cut surface was grey-white, solid, homogeneous, and firm to hard in consistency, appearing to arise from the ovarian stroma. The rest of the specimen, including the left ovary, bilateral fallopian tubes, endometrium, and cervix, appeared unremarkable.



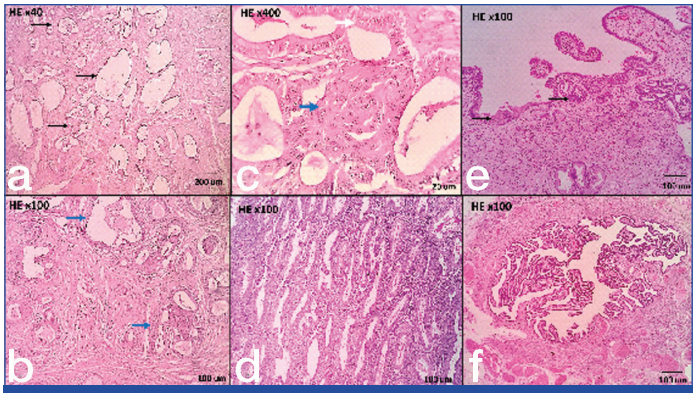
[Table/Fig-1]: MRI of the abdomen and pelvis. a) T1 hyperintense lesion with few tiny punctate foci of gradient recalled echo (GRE) blooming (white arrow) in the right ovary



[Table/Fig-2]: Gross image of solid nodular growth in the right ovary (black arrows).

Sections studied from the right ovary revealed a circumscribed neoplasm composed of endometrial glands embedded in fibrous stroma, with focal areas showing back-to-back glandular arrangements and glandular crowding [Table/Fig-3a]. The endometrial glands were tubular to cystically dilated and lined by columnar epithelium, with focal pseudostratification observed. The cells exhibited vesicular nuclei with a moderate amount of eosinophilic cytoplasm. Some glands showed nuclear crowding, enlarged nuclei, and loss of polarity. A few glands contained intraluminal eosinophilic secretions [Table/Fig-3b]. Areas of morules demonstrating squamous differentiation were also noted [Table/Fig-3b,c]. The fibrous stroma showed focal oedematous changes, and adjacent ovarian stroma contained thick-walled blood vessels. No confluent or destructive desmoplastic invasion was observed in the sections studied.

The sections from the endometrium demonstrated endometrial hyperplasia without atypia [Table/Fig-3d]. The cervix exhibited squamous metaplasia [Table/Fig-3e]. The left ovary and bilateral fallopian tubes [Table/Fig-3f] were unremarkable.



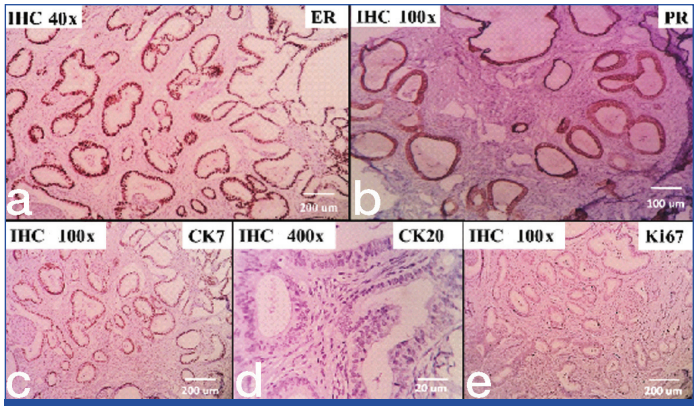
[Table/Fig-3]: Histological findings of nodular ovarian growth: (a,b) Back-to-back and crowded endometrial glands shown by black arrows in A {Haematoxylin and Eosin (H&E) stain, 40x} and blue arrows in B (H&E stain, 100x); (c) The glands displayed cells with bland vesicular nuclei, moderate eosinophilic cytoplasm, occasional nuclear crowding, nucleomegaly, loss of polarity shown by white arrow and squamous differentiation shown by blue arrow (H&E stain, 400x); (d) The endometrium showed hyperplasia without atypia (H&E stain, 100x); (e) The cervix exhibited areas of squamous metaplasia shown by black arrows; (f) The fallopian tube demonstrated normal histology (H&E stain, 100x)

Based on the above features, the differential diagnoses include EBOT, endometriosis, and well-differentiated endometrioid adenocarcinoma of the ovary. The presence of squamous morules further supports the possibility of an endometrioid-type lesion. Endometriosis requires identification of endometrial-type glands, stroma, or hemosiderin-laden macrophages. Given the glandular crowding and pseudostratification, the possibility of metastatic adenocarcinoma, especially from the endometrium or colorectal origin, must also be considered. However, metastatic colonic adenocarcinoma is less likely due to the lack of dirty necrosis and overt cytologic atypia.

Immunohistochemical analysis was performed to confirm the histopathological findings and establish the diagnosis. The expression of ER and PR showed strong nuclear positivity [Table/Fig-4a,b]. CK7 demonstrated both membranous and cytoplasmic positivity [Table/Fig-4c], while CK20 was negative [Table/Fig-4d], effectively ruling out the possibility of colonic malignancies. Additionally, the proliferation marker Ki-67 exhibited nuclear positivity in approximately 4-5% of cells in the examined glands [Table/Fig-4e], reflecting a low proliferative index and the less aggressive nature of the tumour. Based on the histopathological and immunohistochemical findings, a final impression of borderline endometrioid tumour of the ovary, with no desmoplastic or confluent invasion, was given. At present, the patient is doing well at six months post-operative period.

DISCUSSION

The BOTs are epithelial ovarian tumours characterised by atypical epithelial proliferation. However, unlike ovarian cancer, they typically do not exhibit extensive stromal invasion [1,2]. BOTs comprise about



[Table/Fig-4]: Immunohistochemical (IHC) examination of ovarian nodular mass. (a) Oestrogen receptor (ER) IHC showed strong nuclear positivity in the neoplastic endometrial glands (IHC, 40x); (b) Progesterone Receptor (PR) IHC showed strong nuclear positivity in the neoplastic endometrial glands (IHC, 100x); (c) CK7 showed strong membranous and cytoplasmic positivity in the neoplastic endometrial glands (IHC, 100x); (d) CK20 negative in the neoplastic endometrial glands (IHC, 400x); (e) Ki67 showed nuclear positivity in 4-5% of neoplastic endometrial glands (IHC, 100x).

15-20% of all epithelial ovarian tumours [1,3]. According to the recent 2020 World Health Organisation (WHO) classification, BOTs are categorised based on histopathological features into subtypes, including serous, mucinous, endometrioid, clear cell, seromucinous, and transitional (Brenner) types [1,4]. Serous and mucinous BOTs are the most frequently occurring histological subtypes, accounting for 53.3% and 42.5% of cases, respectively. Less common variants, comprising about 3%-4% of cases, include endometrioid, clear cell, transitional (Brenner), and mixed epithelial subtypes [1,5]. These tumours frequently occur in otherwise healthy young women of reproductive age, with about one-third of diagnoses being made in women under 40 years old [6]. Endometrioid Borderline Tumours (EBTs) are quite rare, accounting for approximately 0.2% of all epithelial ovarian tumours [7,8]. Their clinical behaviour is less aggressive compared to malignant endometrioid carcinoma [9-11].

After an extensive literature search through PubMed and other journals, we found two studies on EBOT in the Indian population. Surapaneni SL et al., studied 119 ovarian lesion specimens, comprising both non-neoplastic and neoplastic lesions from the Indian population in their institute. Among these neoplastic lesions, 47 cases (90.38%) were benign, two cases (3.84%) were borderline, and three cases (5.76%) were malignant. Of the two borderline cases, one (1.92%) was identified as a proliferative endometrioid tumour [12]. Similarly, Jetley S et al., reported a case of EBOT that was incidentally found in a 45-year-old woman after hysterectomy; however, their patient had irregular vaginal bleeding [13].

Among international studies, Ricotta G et al., analysed 48 EBOT cases, providing insights into clinical characteristics, prognosis, and management [14]. A retrospective study by Jia S et al., analysed 33 women with EBOTs. Among the 25 patients who underwent endometrial evaluation, 13 (52.0%) had endometrial disorders, including six cases of cancer, five with atypical hyperplasia, and two with non-atypical hyperplasia. Similarly, our case also revealed the presence of endometrial hyperplasia without atypia [15]. A review of 50 BOTs by Piura B et al., highlighted the distribution of BOTs in their institute. Serous BOTs were the most common (32 cases, 64%), followed by mucinous BOTs (17 cases, 34%). Endometrioid BOTs were rare, with only one case (2%), emphasising their rarity [16].

These tumours typically occur in premenopausal women, and the median age at diagnosis is usually between 40 and 50 years, similar to our patient's age. The aetiology of these tumours remains unclear [1]. They are associated with conditions such as endometriosis, endometrial hyperplasia, and endometrioid endometrial carcinoma in about 39% of cases [1,17]. However, due to their rarity, the risk factors and aetiology associated with borderline endometrioid tumours are not as well-defined as those of serous or mucinous borderline tumours [1].

Histologically, EBTs are characterised by two distinct growth patterns, with the adenofibromatous type being more prevalent compared to the intracystic type [1]. Approximately 50% of tumours displaying an adenofibromatous appearance contain endometrioid adenofibroma components. In these borderline tumours, the glands are densely packed and irregularly shaped, mimicking atypical endometrial hyperplasia with a lobular structure. The nuclei show mild to moderate atypia and low mitotic activity. Common features include squamous (particularly morular) metaplasia, with mucinous metaplasia also possible. The stroma is typically fibromatous [1]. Similarly, in our case, the tumour also displayed endometrial glands embedded in a fibromatous stroma with morules of squamous metaplasia. Intracystic tumours often present a simple papillary architecture extending into an endometriotic cyst. While microinvasion (< 5 mm) can occur, its diagnostic criteria can be ambiguous. A diagnosis of endometrioid carcinoma requires over 5 mm of continuous or destructive invasion; confluent morular metaplasia alone does not indicate carcinoma [1].

Surgery is the primary treatment for BOTs. In most instances, a total abdominal hysterectomy along with BSO is performed to completely remove the tumour and reduce the risk of recurrence [18]. For younger women seeking to preserve their fertility, the option of a unilateral salpingo-oophorectomy may be considered, provided that careful staging is performed and there is no evidence of invasive disease [18]. In our case, since the patient had completed childbearing, a total hysterectomy was performed. BOTs, including the endometrioid subtype, have a good prognosis compared to invasive ovarian carcinomas [1]. Extensive surgical staging is usually unnecessary, but uterine curettage is recommended if uterine preservation is planned to exclude concurrent endometrial pathology [19]. Regular ultrasound follow-up is essential, especially for conservatively treated cases, with prolonged monitoring due to the risk of late recurrence [20]. In our case, the patient is doing well six months after the postoperative period. The studies discussed above, along with a few others, are summarised chronologically in the table provided below [Table/Fig-5] [8,9,12-18,21,22].

Author (Year)	Study type	Sample/Patient details	Key findings/Conclusion
Bell DA and Scully RE (1985) [21]	Case series	27 cases	Atypical endometrioid adenofibromas have an excellent prognosis.
Piura B et al., (1992) [16]	Institutional review	50 BOTs (1 EBOT)	EBOT made up only 2% of borderline tumours, emphasising rarity
Bell KA and Kurman RJ (2000) [9]	Case series	33 patients	Defined histologic criteria of EBOT; emphasised absence of stromal invasion
Roth LM et al., (2003) [8]	Case series	30 patients	The prognosis of EBOTs were superior to that of well-differentiated endometrioid adenocarcinoma
Uzan C et al., (2012) [18]	ROL	16 patients	Described the treatment and follow-up of rare EBOT cases
Jetley S et al., (2016) [13]	Indian case report	1 patient	EBOT was incidentally found post-hysterectomy in a patient with irregular bleeding
Nakagawa E et al., (2017) [17]	Case reports	4 patients	Prognosis of EBOT was excellent. Concurrent endometrial lesions including endometrial cancer need to be considered and excluded
Jia S et al., (2018) [15]	Case series	33 patients	Endometrial sampling is recommended in EBOT cases undergoing conservative surgery, while hysterectomy is advised for those needing radical treatment
Khedr M et al., (2019) [22]	Case report	1 patient	EBOT with massive squamous differentiation; mimicked carcinoma
Surapaneni SL et al., (2022) [12]	Indian institutional case series	119 ovarian lesions (2 borderline; 1 EBOT)	Identified 1 case of proliferative endometrioid tumour in tertiary care centre emphasising rarity
Ricotta G et al., (2022) [14]	Case series	48 EBOT cases	Provided insights into clinical profile, prognosis, and treatment options
Present case (2025)	Case report	1 patient	Ovarian neoplasm incidentally detected on ultrasound. HPE and IHC confirmed EBOT diagnosis

[Table/Fig-5]: Summary of case reports and series describing clinical, pathological features and management of Endometrioid Borderline Ovarian Tumours (EBOTs) [8,9,12-18,21,22].

CONCLUSION(S)

Borderline endometrioid tumours of the ovary are rare and present unique diagnostic and management challenges. While their behaviour is less aggressive than that of invasive carcinomas, careful histopathological evaluation and appropriate surgical management are essential to ensure optimal outcomes. Our case highlights the importance of histopathological and immunohistochemical examination in accurately diagnosing and identifying such rare cases presented with ovarian masses. Given their rarity, further studies are needed to elucidate the clinical behaviour, risk factors, pathogenesis, and optimal treatment strategies for borderline endometrioid ovarian tumours.

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