

Adult-Type Granulosa Cell Tumour of the Ovary Presenting with Hyperoestrogenic Manifestations and Mimicking an Endometrioma on MRI: A Case Report with Staged Surgical Management

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Abstract

Case Report

Background. Adult-type granulosa cell tumours (AGCTs) are rare sex cord–stromal neoplasms accounting for fewer than 5% of ovarian cancers. Their hallmark is autonomous oestradiol secretion, and they recur late. Where molecular testing is unavailable, the diagnosis rests on morphology and immunohistochemistry. **Case Presentation.** A 50-year-old woman was referred for a pelvic mass, abnormal uterine bleeding and a palpable right breast nodule. Abdomino-pelvic MRI showed a multiloculated left ovarian lesion with haemorrhagic content, T2 shading, restricted diffusion and non-enhancing contents, scored O-RADS MRI 4 but reported as suggesting an endometrioma; the endometrium measured 17 mm and was reported as probable hyperplasia, with diffuse adenomyosis. CA-125 was normal. Laparoscopic left salpingo-oophorectomy removed a mass with a 9 × 8 cm solid component; the specimen reached the laboratory in seven fragments, upstaging the tumour to FIGO IC1. Histology showed a microfollicular and trabecular proliferation of monomorphic grooved cells without necrosis, with intraluminal tumour clusters in the tube. Immunohistochemistry was positive for inhibin, calretinin, oestrogen and progesterone receptors and negative for PAX8, CK7, CK20, WT1, chromogranin A, synaptophysin and GATA-3. Completion hysterectomy with omentectomy showed no residual tumour and no endometrial hyperplasia. Four cycles of etoposide–cisplatin were delivered without modification, and imaging confirmed complete morphological remission at eight months. **Conclusion.** A haemorrhagic AGCT can reproduce the MRI signature of an endometrioma; where the narrative report and the O-RADS score diverge, the score must govern management, including the surgical route. Intraoperative fragmentation upstages the disease and is avoidable. Endometrial thickening requires histological confirmation before it is called hyperplasia.

Keywords: Granulosa cell tumour; hyperoestrogenism; O-RADS MRI; intraoperative fragmentation; FOXL2; immunohistochemistry; EP adjuvant chemotherapy; inhibin B.

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1. INTRODUCTION

Sex cord–stromal tumours (SCSTs) of the ovary constitute a heterogeneous group of neoplasms, biologically distinct from epithelial carcinomas in histogenesis, natural history and endocrine activity. They represent approximately 5% of ovarian malignancies, and their defining feature is the capacity to synthesise sex steroids, generating early endocrine manifestations that are typically absent in epithelial ovarian cancer.[1] Adult-type granulosa cell tumour (AGCT) is the commonest malignant member of the group.

The landmark *FOXL2* c.402C>G (p.Cys134Trp) somatic mutation, described by Shah and

colleagues in 2009,[2] is the molecular signature of the disease; in the largest contemporary series profiled by next-generation sequencing (227 tumours, 786 genes) it was present in 99% of cases, *TERT* promoter mutations were found in 43% — more frequently in recurrences — and *FOXO1* mutations were associated with poorer overall survival.[3] Molecular confirmation is nevertheless unavailable in many centres, including ours, and the diagnosis must then rest on morphology and a purposefully constructed immunohistochemical panel.

AGCT also recurs late. In a long-term cohort of 97 patients the median time to relapse was 53 months, 47% of recurrences occurred beyond five years and the

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longest interval was 27.6 years, which led the authors to advocate lifelong follow-up.[4] We report a case in which four issues converged in a single patient: a lesion scored O-RADS MRI 4 that was reported as suggesting an endometrioma; an intraoperative fragmentation of the specimen that migrated the stage from IA to IC1; a diagnosis secured by immunohistochemistry alone; and an endometrial thickening reported as probable hyperplasia that was not confirmed on the hysterectomy specimen. The report follows the CARE guideline for case reports.

2. CASE PRESENTATION

2.1. Clinical History and Presentation

The patient, a 50-year-old woman (para 5), with menarche at 14 years and approximately 15 years of combined oral contraceptive use, was referred to Mohammed VI University Hospital, Marrakech, for the management of a symptomatic pelvic mass. She had no personal or family history of gynaecological malignancy and no other relevant medical or surgical history.

From October 2024 she reported progressive pelvic heaviness, lower abdominal discomfort and repeated episodes of low-volume abnormal uterine bleeding, her general condition remaining preserved. She was first assessed locally, where symptomatic treatment was given; a thoraco-abdomino-pelvic CT scan and a pelvic ultrasound were requested but their reports were not available for review. She was referred to the gynaecology department in May 2025. On examination there was a firm, mobile hypogastric mass and a firm nodule in the upper quadrants of the right breast; no lymphadenopathy was palpable.

2.2. Diagnostic Workup and Imaging

Abdomino-pelvic MRI was performed on 4 July 2025 (Figure 1), with T2 sequences in three planes, axial T1, diffusion-weighted imaging and fat-saturated T1 sequences before and after gadolinium.

The uterus was enlarged (11×5 cm), retroflexed and retroverted, with a preserved central cavity line. The junctional zone was diffusely thickened to a maximum of 19.5 mm, hypointense on T2, with irregular external contours and microcystic areas that were hypointense on T1 and hyperintense on T2 — one of them hyperintense on T1 and of intermediate T2 signal, indicating haemorrhagic content — none of which enhanced after contrast. The endometrium was thickened to 17 mm at the uterine corpus, mildly heterogeneous on T2, and enhanced after gadolinium.

The left ovary was enlarged and contained a thin-walled lesion that was hyperintense on T1, markedly hyperintense on fat-saturated T1, heterogeneously hyperintense on T2 with shading, and heterogeneously hyperintense on diffusion-weighted imaging with restricted ADC; its contents did not enhance after contrast. Superiorly the lesion lay in contact with the

sigmoid colon and small-bowel loops; medially it displaced the uterine corpus, with preservation of the intervening fat plane. An adjacent cystic structure of 24×16 mm, hyperintense on T2 and on fast T1 and containing a single thin septum, did not enhance and was considered simple. There was no infiltration of pelvic fat, no pelvic effusion, no pelvic lymphadenopathy, no dilatation of the urinary excretory cavities and no suspicious bone lesion on the slices obtained; the bladder was normal and the right ovary was not visualised and presumed atrophic.

Abdominal sequences showed no dilatation of the intra- or extrahepatic biliary ducts, a free main bile duct with homogeneous content, a liver of normal size and homogeneous signal, and a normal spleen, pancreas and kidneys. An upper-polar right renal cyst measuring 64×75.8 mm contained thin septa and was hypointense on T1, markedly hyperintense on T2 and non-enhancing after contrast.

The radiological conclusion was a multiloculated left ovarian lesion with haemorrhagic content, classified O-RADS MRI 4[5], suggesting an endometrioma, although a mucinous cystadenoma could not be excluded and correlation with tumour markers was recommended; a thickened endometrium probably corresponding to endometrial hyperplasia; and adenomyosis. A sex cord–stromal tumour was not raised as a possibility before surgery. No numerical measurement of the adnexal lesion is given in the report; the only documented dimension is the 9×8 cm solid component recorded at laparoscopy.

Serum CA-125 was within normal limits, which is characteristic of hormone-secreting sex cord–stromal tumours but which, in this context, also reinforced the benign radiological hypothesis. Serum oestradiol, inhibin B and anti-Müllerian hormone were not measured before surgery. Mammography and breast ultrasound characterised the palpable right nodule as a well-circumscribed oval hypoechoic lesion of 6.8×8.8 mm with posterior enhancement, alongside an incidental left subcutaneous lipoma and benign punctate calcifications; the overall assessment was ACR 3, with no suspicious lesion.

2.3. Surgical Management

First procedure (4 August 2025).

After multidisciplinary tumour board review, a laparoscopic left salpingo-oophorectomy was performed. Exploration found a cystic mass with a 9×8 cm solid component arising from the left ovary, normal right adnexa, no ascites and no peritoneal carcinomatosis; peritoneal lavage was carried out.

The specimen reached the pathology laboratory fragmented, in seven pieces weighing 31.3 g in total, the largest measuring $8 \times 2 \times 1$ cm.

Loss of specimen integrity during extraction constitutes surgical spill, and the tumour was therefore

staged FIGO IC1 rather than IA,[6] which changed the adjuvant strategy.

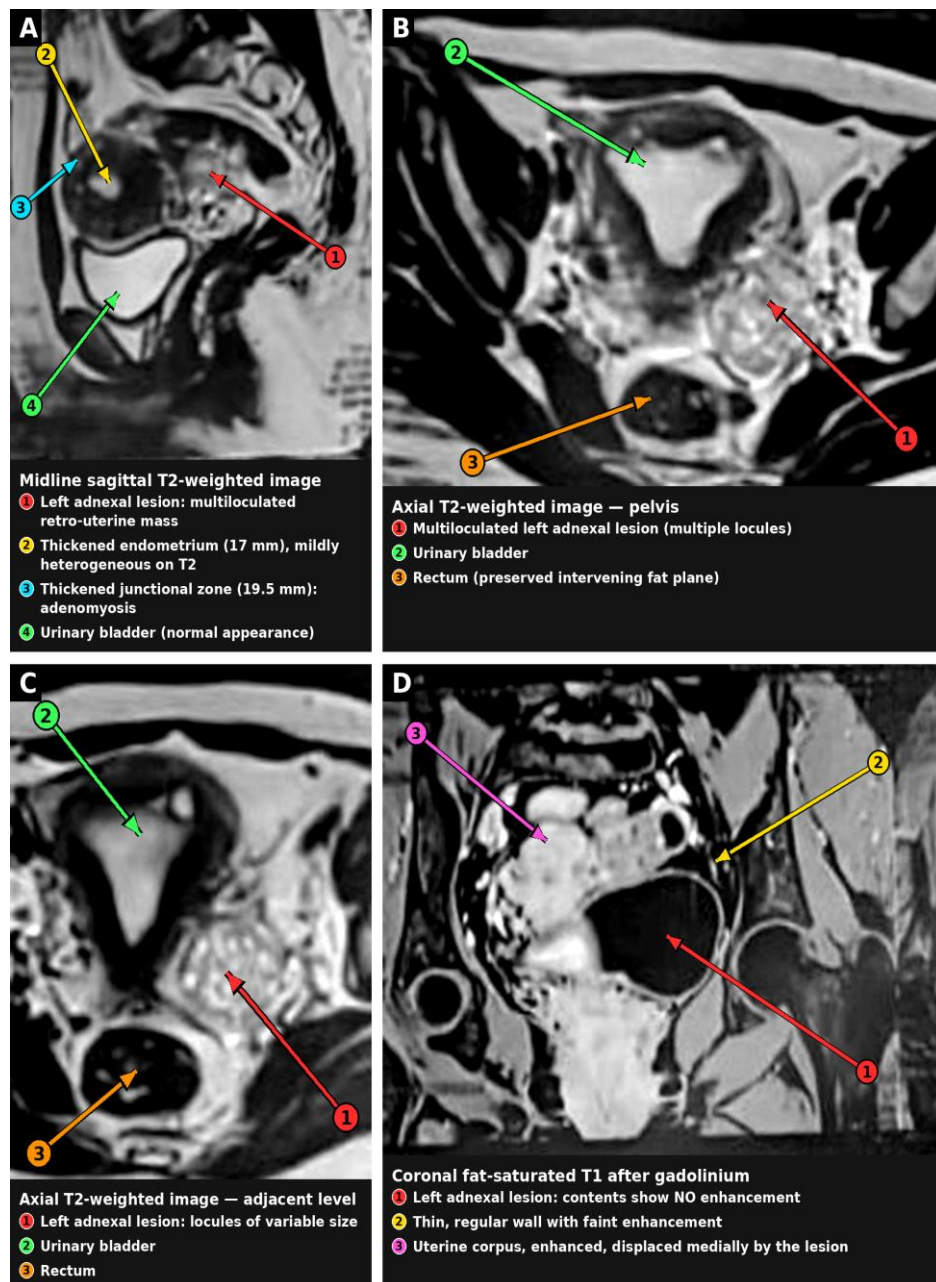


Figure 1: Diagnostic abdomino-pelvic MRI at initial work-up (4 July 2025); the anatomical key is embedded in each panel. (A) Midline sagittal T2: retroverted and retroflexed uterus, junctional zone thickened to 19.5 mm (adenomyosis), endometrium thickened to 17 mm, and a multiloculated retro-uterine left adnexal lesion. (B, C) Adjacent axial T2 slices: the lesion is multiloculated, with locules of unequal size and signal separated by thin regular septa, without mural nodule or papillary projection, and with a preserved fat plane from the uterus and rectum. (D) Coronal fat-saturated T1 after gadolinium: the lesion contents do not enhance; only the thin, regular wall enhances faintly, and the enhancing uterine corpus is displaced medially. The lesion was classified O-RADS MRI 4 and reported as suggesting an endometrioma, a mucinous cystadenoma not excluded

Second procedure (22 September 2025).

Surgical completion via midline laparotomy comprised total hysterectomy without adnexal conservation, omentectomy, multiple peritoneal biopsies and peritoneal washing. Exploration again found no ascites and no peritoneal carcinomatosis, with a normal liver and appendix.

2.4. Histopathology and Immunohistochemistry

Microscopic examination of the salpingo-oophorectomy specimen showed ovarian parenchyma replaced by a proliferation with diffuse, trabecular and microfollicular architecture containing occasional papillae with fibrovascular cores. The cells were monomorphic, with angular nuclei showing longitudinal grooves and eosinophilic cytoplasm. Mitotic figures

were scanty and there was no tumour necrosis; the stromal reaction was densely fibrous with congested, thick-walled vessels. The tubal wall showed congestive change and contained intraluminal clusters of tumour cells of identical appearance. Ki-67 was not performed. On morphology alone the lesion was reported as a poorly differentiated tumour requiring immunohistochemical characterisation.

Immunohistochemistry proved decisive (Table 1, Figure 2). Inhibin and calretinin were positive, establishing sex cord–stromal, granulosa cell lineage; oestrogen and progesterone receptors were expressed,

consistent with a hormonally active tumour. The panel was negative for PAX8, CK7, CK20, WT1, chromogranin A, synaptophysin and GATA-3. The combination of morphology and immunophenotype established adult-type granulosa cell tumour without recourse to molecular testing.

Final pathology of the completion specimens showed mild non-specific subacute cervicitis with squamous metaplasia, intramural leiomyomas and a dystrophic right ovary. No endometrial hyperplasia, no atypia and no residual or metastatic tumour were identified in any specimen, including the omentum.

Table 1: Immunohistochemical panel — results and diagnostic significance

Marker (clone)	Result	Diagnostic significance
Inhibin (R1); calretinin (RBT-CALB2)	Positive	Establish sex cord–stromal, granulosa cell lineage
Oestrogen receptor (SP1); progesterone receptor (SP2)	Positive	Hormonally active tumour
PAX8 (BC12); CK7 (OV-TL 12/30); WT1 (6F-H2)	Negative	Exclude Müllerian epithelial, high-grade serous and endometrioid carcinoma
CK20 (Ks20.8)	Negative	Excludes metastatic gastrointestinal carcinoma
Chromogranin A (LK2H10); synaptophysin	Negative	Exclude neuroendocrine tumour
GATA-3 (L50-823)	Negative	Excludes metastatic breast carcinoma — relevant given the palpable breast nodule

Positivity was reported qualitatively by the referring laboratory, without quantification of stained nuclei. *FOXL2* sequencing was not available. ER: oestrogen receptor; PR: progesterone receptor.

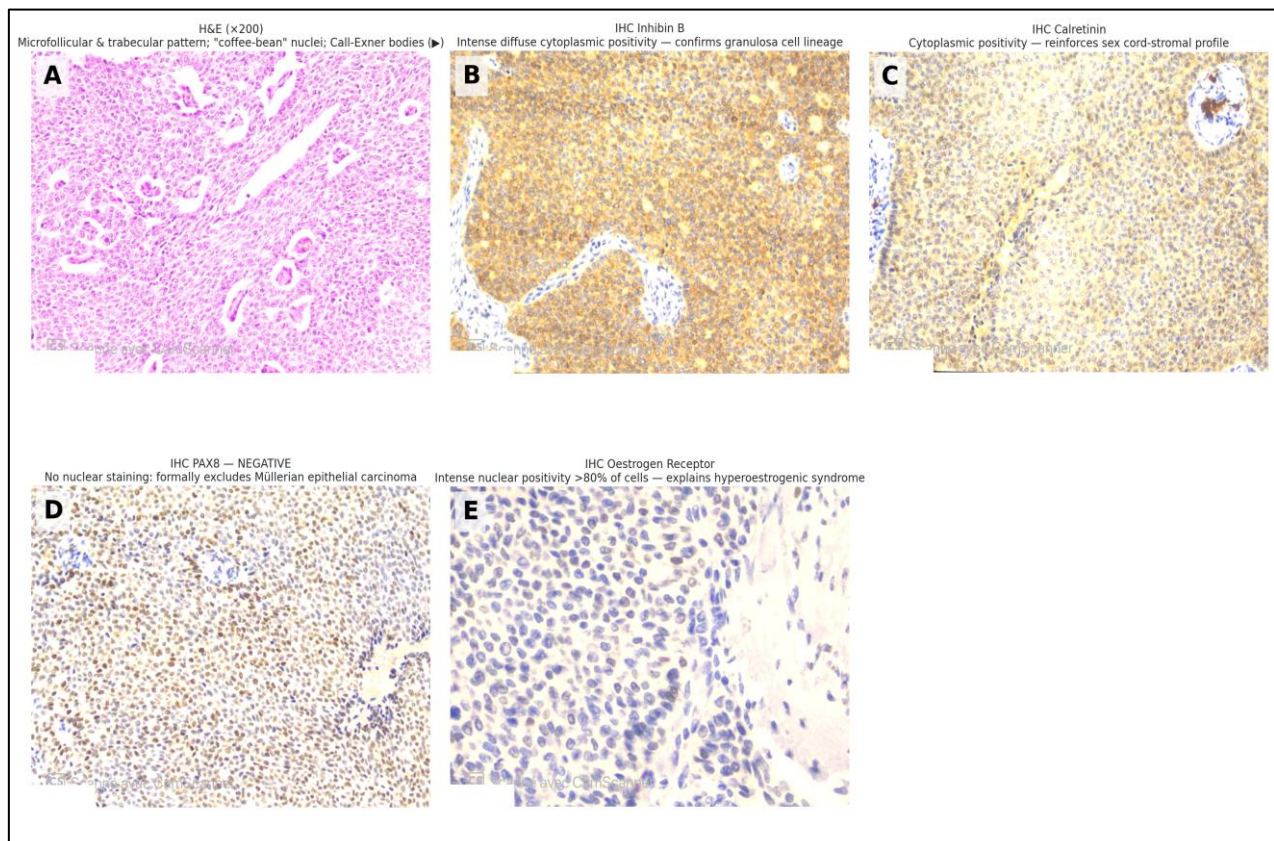


Figure 2: Histopathological and immunohistochemical analysis of the left ovarian tumour. (A) Haematoxylin and eosin (×200): microfollicular and trabecular architecture; monomorphic cells with angular, grooved "coffee-bean" nuclei. (B) Inhibin: intense diffuse cytoplasmic positivity, confirming granulosa cell lineage. (C) Calretinin: cytoplasmic positivity, reinforcing the sex cord–stromal profile. (D) PAX8: complete nuclear negativity, formally excluding Müllerian epithelial carcinoma. (E) Oestrogen receptor: nuclear positivity, consistent with a hormonally active tumour

2.5. Postoperative Staging Workup

Thoraco-abdomino-pelvic CT performed on 15 October 2025 showed no pleuropulmonary lesion, no pleural effusion, no abnormality of the mediastinal great vessels and no mediastinal lymphadenopathy. In the abdomen and pelvis it showed the expected postoperative appearances, millimetric lumbo-aortic, iliac and inguinal nodes of no pathological significance, a normal liver, spleen, pancreas, kidneys and adrenals, patent hepatic and portal veins, and no retroperitoneal or intraperitoneal lymphadenopathy. The right renal cyst measured 72×61.7 mm, contained septa and thick calcifications, and was classified Bosniak IIF. No hepatic focal lesion, pulmonary metastasis or peritoneal carcinomatosis was identified, and the conclusion was the absence of any evident secondary lesion.

2.6. Adjuvant Chemotherapy and Outcome

At multidisciplinary tumour board review, two concurrent adverse features — FIGO stage IC1 following intraoperative fragmentation, and tumour size — indicated adjuvant chemotherapy. Four cycles of etoposide–cisplatin (EP) were administered at three-weekly intervals. Treatment was well tolerated, with toxicity not exceeding grade 2 (NCI-CTCAE), and without dose delay or reduction. Pelvic MRI on 27 February 2026, after completion of chemotherapy, showed a free hysterectomy bed, no visible pelvic mass, millimetric inguinal and iliac nodes and no fluid in the pouch of Douglas, and concluded that there was no sign of local recurrence.

2.7. Surveillance Imaging at Eight Months (May 2026)

At the June 2026 follow-up consultation, the patient brought a contrast-enhanced thoraco-abdomino-pelvic CT performed on 21 May 2026, eight months after completion surgery (Figure 3). She remained asymptomatic, with an unchanged performance status.

Thoracic assessment showed no pulmonary parenchymal lesion, no mediastinal or hilar lymphadenopathy and no pleural or pericardial effusion (Figure 3A–B). The liver, spleen, pancreas, kidneys and adrenals were normal, without focal lesion (Figure 3C). The pelvis was free, with no residual or recurrent mass in either adnexal fossa, no deep lymphadenopathy, no peritoneal effusion and no suspicious bone lesion (Figure 3D).

The only reported abnormality was incidental and non-tumoural: bilateral renal cysts, the largest on the right measuring 7 cm and containing septa and thick calcifications, classified **Bosniak IIF**. This lesion was already present on the diagnostic MRI (64×75.8 mm) and on the staging CT (72×61.7 mm), and has therefore been morphologically stable over ten months. Bosniak IIF is not equivalent to a simple cyst and requires its own imaging surveillance pathway, which we have arranged separately from the oncological follow-up.

The examination therefore revealed no new or progressive abnormality, confirming maintained complete morphological remission eight months after completion surgery. Clinical review and serum inhibin B measurement are performed three-monthly.

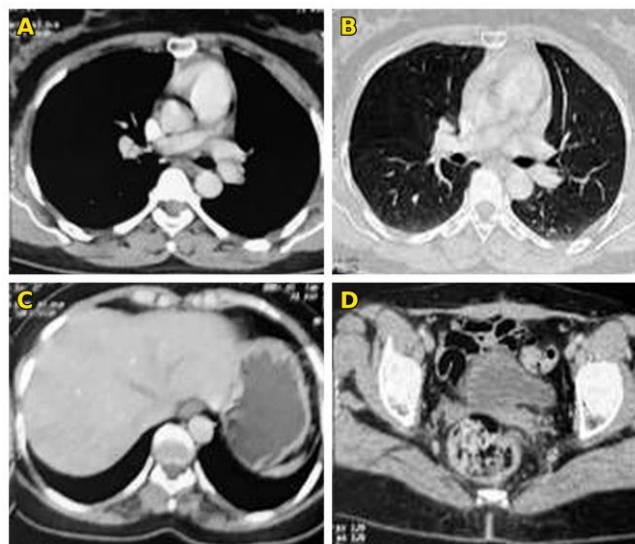


Figure 3: Surveillance contrast-enhanced thoraco-abdomino-pelvic CT (21 May 2026), reviewed at the June 2026 follow-up consultation — eight months after completion surgery. (A) Axial thoracic slice, mediastinal window at the level of the pulmonary trunk and hila: no mediastinal or hilar lymphadenopathy, no pleural or pericardial effusion. (B) Axial thoracic slice at the same level, lung window: normal pulmonary parenchyma, no nodule or secondary lesion. (C) Axial slice at the hepatic dome: homogeneous liver of regular contour without focal lesion, normal spleen, no perihepatic effusion. (D) Axial pelvic slice: free operated pelvis, no residual or recurrent mass in either adnexal fossa, no deep lymphadenopathy, no peritoneal effusion. Overall assessment: no new or progressive abnormality — maintained complete morphological remission

3. DISCUSSION

3.1. Diagnostic Robustness: from a Misleading MRI to a Decisive Immunoprofile

Every feature of the adnexal lesion pointed away from a malignant sex cord–stromal tumour: T1 hyperintensity persisting on fat-saturated T1, T2 shading, restricted diffusion and non-enhancing contents — the classical signature of an endometrioma, reinforced by a normal CA-125. Two lessons follow. First, the numerical score and the narrative impression pointed in opposite directions, and the score was right: an O-RADS MRI 4 lesion carries an intermediate risk of malignancy and, by design, mandates management by a gynaecological oncology team.[5] Where the two diverge, it is the score that must govern the pathway, including the choice of surgical route. Second, a newly diagnosed endometrioma is less likely at the menopausal transition, since endometriomas tend to regress once oestrogen support is withdrawn; the combination actually present - a haemorrhagic adnexal cyst, an endometrium thickened to 17 mm and enhancing, adenomyosis and abnormal uterine bleeding — is better read as one endocrine syndrome than as coincidental findings.

The immunohistochemical panel then carried the diagnosis in the absence of molecular pathology. Our profile reproduces the phenotype described in the largest immunohistochemical series of molecularly confirmed AGCTs.[7] Its decisive contribution was nonetheless negative rather than positive: PAX8, CK7 and WT1 negativity excluded high-grade serous and endometrioid carcinoma and thereby excluded a carboplatin–paclitaxel strategy that would have been inappropriate; chromogranin A and synaptophysin negativity excluded a neuroendocrine tumour; and GATA-3 negativity excluded metastatic breast carcinoma, a relevant differential in a patient with a palpable breast nodule. This is the transferable message: without molecular pathology, the panel should be built around the differential diagnoses that would change treatment.

3.2. Hyperoestrogenism as a Multi-Organ Alarm Signal

Systematic evaluation of oestrogen target organs is justified in hormone-secreting ovarian tumours: in a population-based cohort of 1031 women with granulosa cell tumour, endometrial hyperplasia and endometrial carcinoma were both substantially over-represented.[8] Our patient met the usual criteria for uterine assessment, with abnormal bleeding, an endometrium of 17 mm that enhanced after contrast and an explicit radiological conclusion of probable hyperplasia.

The hysterectomy specimen nonetheless showed no hyperplasia and no atypia. The thickening may have regressed during the seven weeks that separated removal of the oestrogen source from the completion procedure, which would make it a reversible functional response rather than an established lesion;

alternatively, the measured thickness may in part have reflected the adjacent adenomyosis. The corollary is practical: if the endometrial effect of hyperoestrogenism is to be documented, sampling must be obtained before the hormone source is removed, and hysterectomy should be indicated on its own merits rather than on an imaging measurement — a distinction that matters most for younger women in whom uterine preservation is at stake. The palpable breast nodule justified the same systematic approach; it proved benign, and no causal link with the tumour can be drawn from a single case.

3.3. Intraoperative Fragmentation: An Avoidable Event with Quantifiable Consequences

Loss of specimen integrity at extraction constitutes surgical spill and reassigns a tumour otherwise confined to the ovary from stage IA to IC1.[6] The consequence is not merely nomenclatural. In a long-term cohort of 43 granulosa cell tumours, intraoperative rupture occurred exclusively during laparoscopic procedures, and median recurrence-free survival was 42 months in the rupture group against 158 months when the specimen remained intact ($p < 0.001$), with median overall survival of 94 against 246 months ($p = 0.015$); recurrence occurred in 66.7% after minimally invasive surgery against 25.8% after laparotomy ($p = 0.031$).[9]

The corollary is surgical and actionable. The route should be decided preoperatively on tumour volume and capsular quality, with a low threshold for laparotomy or for protected in-bag extraction whenever a large, tense, thin-walled cyst is anticipated. The intraluminal tumour clusters found within the tubal wall in our specimen add a further argument for en-bloc salpingo-oophorectomy rather than cystectomy in any adnexal mass suspected of being a sex cord–stromal tumour. Completion laparotomy confirmed the absence of residual disease; in a cohort of 333 stage IA and IC AGCTs, completion of staging surgery was associated with a lower recurrence rate (34.4% versus 51.2%, $p = 0.006$) and independently predicted disease-free survival,[10] although a prospective multicentre study of 208 patients found that staging surgery did not by itself reduce recurrence.[11]

3.4. A Reasoned De-escalation: the EP Doublet in Practice

Adjuvant chemotherapy was given for surgical spill and tumour size, in line with recommendations for platinum-based treatment in higher-risk non-epithelial ovarian cancer.[1] The evidence base is nonetheless weak: a systematic review and meta-analysis of adjuvant chemotherapy in stage IC granulosa cell tumours found no clear survival advantage over observation,[12] and the decision rests on multidisciplinary judgement rather than demonstrated benefit.

The choice of regimen deserves the same candour. Bleomycin-related pulmonary toxicity has long motivated interest in bleomycin-free platinum doublets,

but the only randomised trial conducted in chemotherapy-naïve stromal ovarian tumours compared paclitaxel–carboplatin with bleomycin–etoposide–cisplatin: median progression-free survival was 27.7 against 19.7 months with overlapping confidence intervals, and the response rate among patients with measurable disease favoured the bleomycin-containing arm (50% against 18%).[13] No randomised comparison of etoposide–cisplatin with BEP exists. Omitting bleomycin where serial pulmonary function testing is not routinely available is therefore a pragmatic extrapolation from the platinum backbone, not a demonstrated equivalence. We report the EP doublet as feasible and well tolerated in one patient — four cycles delivered on schedule, without dose modification — and make no claim of non-inferiority.

3.5. A Surveillance Protocol in Accordance with Current Evidence

Late recurrence is the defining feature of the natural history of AGCT, with a median time to relapse beyond four years and relapses recorded as late as 27.6 years.[4] Given the surgical spill and the tumour size, we intend to maintain structured surveillance well beyond the conventional five years.

Biochemical surveillance is nonetheless constrained by the absence of a preoperative baseline. Serum anti-Müllerian hormone and inhibin B are both sensitive markers of recurrence and are complementary when used together,[14] but their interpretation depends on comparison with a preoperative value, which was not obtained. We therefore rely primarily on clinical assessment and cross-sectional imaging, using the first postoperative inhibin B as an internal reference. This is a lesson for practice: in any adnexal mass with clinical or radiological signs of hyperoestrogenism, oestradiol, inhibin B and AMH should be drawn before surgery.

3.6. Strengths and Limitations

The strengths of this report are the completeness of the diagnostic pathway, documented against the primary source records at each step, the explicit reporting of a surgical complication and of its staging consequences, and the histological verification — negative — of an assumption that is usually left untested.

The limitations are substantial. This is a single case, from which no conclusion about comparative effectiveness can be drawn. *FOXL2* sequencing was unavailable, so the diagnosis rests on morphology and immunophenotype, which is nevertheless the reality of practice in most centres worldwide. Serum oestradiol, inhibin B and anti-Müllerian hormone were not measured before surgery, so hyperoestrogenism is inferred from the clinical and radiological findings rather than demonstrated biochemically. The mitotic index and Ki-67 were not quantified, and the operative record does not document the moment at which the specimen lost its

integrity, which was established on receipt in the laboratory. Follow-up remains short at eight months.

4. CONCLUSION

Adult-type granulosa cell tumour of the ovary presents a multifaceted diagnostic and therapeutic challenge. In this patient the tumour reproduced the MRI signature of an endometrioma, and the narrative radiological impression diverged from the O-RADS score of 4 that accompanied it; the score was the reliable guide, and following it would have changed both the preoperative hormonal work-up and the surgical route. The diagnosis was ultimately established by a focused immunohistochemical panel in which the negative markers carried more decisional weight than the positive ones — an approach that is transferable to any centre without access to molecular pathology.

Surgically, prevention of intraoperative tumour fragmentation must guide the preoperative choice of approach according to tumour volume and capsular quality, since spill migrates the stage from IA to IC1 and is associated with markedly shorter recurrence-free survival. Where adjuvant chemotherapy is indicated, a bleomycin-free platinum doublet was feasible and well tolerated, and remains a defensible pragmatic option where bleomycin monitoring is difficult, but it has never been compared with BEP in a randomised trial. The endometrial thickening that motivated part of the surgical plan was not confirmed histologically, which argues for sampling the endometrium before the hormone source is removed.

Finally, the potential for late recurrence justifies extended follow-up incorporating serial inhibin B and anti-Müllerian hormone measurement — markers whose value depends on a baseline that must be obtained before surgery, not after.

Patient Consent

informed consent was obtained from the patient for the anonymised publication of this case report and of the associated imaging and histological material. Patient identity has been fully de-identified.

Ethics Approval

In accordance with institutional policy, anonymised single-case reports do not require formal ethics committee approval.

Conflicts of Interest: The authors declare no conflicts of interest.

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Author Contributions

F. El Haou and W. Hassar: conception, data collection, original draft, clinical review. T. Igarramane, A. Elomrani and M. Khouchani: supervision, critical

revision, final approval. All authors read and approved the final manuscript.

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