

Case report

Malignant Struma Ovarii with Papillary Thyroid Carcinoma Presenting with Concurrent Multinodular Goitre and Thyrotoxicosis: A Case Report

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Received 1-7-2026

Revised 19-7-2026

Accepted 1-8-2026

Published 4-8-2026

DOI: 10.67692/IODT.26.1002

How to cite: Elena Markovi, Ana Kovač. Malignant Struma Ovarii with Papillary Thyroid Carcinoma Presenting with Concurrent Multinodular Goitre and Thyrotoxicosis: A Case Report. Integr Oncol Diagn Treat. 2026;1(1):45-54. doi: 10.67692/IODT.26.1002

Abstract

Background: Malignant struma ovarii (MSO) is a rare monodermal ovarian teratoma in which thyroid tissue undergoes malignant transformation, most commonly to papillary thyroid carcinoma (PTC). Its rarity and nonspecific presentation make preoperative diagnosis difficult, and management is not standardised.

Case Presentation: A 38-year-old premenopausal woman presented with a two-month history of pelvic pain and thyrotoxic symptoms, a longstanding multinodular goitre, and a complex $9.5 \times 7.2 \times 6.8$ cm left adnexal mass. She underwent laparoscopic left salpingo-oophorectomy; histopathology revealed a 1.5 cm focus of classical PTC within a struma ovarii, with negative margins and peritoneal cytology. Given persistent thyrotoxicosis, multinodular goitre, and a wish to enable thyroglobulin-based surveillance, total thyroidectomy (revealing benign hyperplastic goitre) and adjuvant radioactive iodine (I-131, 120 mCi) were subsequently performed. Serial thyroglobulin was undetectable at 6 and 12 months, and the patient remained recurrence-free at 18 months of follow-up.

Conclusion: MSO warrants a high index of suspicion when pelvic and thyroid pathology coexist. Salpingo-oophorectomy remains the surgical cornerstone; total thyroidectomy and adjuvant radioactive iodine are best reserved for patients with high-risk features or a specific surveillance rationale, since adjuvant thyroid-directed therapy has not been shown to improve survival in unselected patients. Long-term thyroglobulin-based surveillance is warranted given the potential for late recurrence.

Keywords: Struma ovarii; Malignant struma ovarii; Papillary thyroid carcinoma; Ovarian teratoma; Thyrotoxicosis; Radioactive iodine therapy

INTRODUCTION

Struma ovarii (SO) is a rare, highly specialised monodermal ovarian teratoma defined by the predominance of thyroid tissue constituting more than 50% of the tumour mass. It accounts for approximately 2–5% of all ovarian teratomas and less than 1% of all ovarian tumours [1-3]. Malignant transformation is classically reported in 5–10% of SO cases, most frequently to papillary thyroid carcinoma (PTC), followed by follicular thyroid carcinoma (FTC) and its variants [4-7]; however, as discussed below, contemporary large cohorts suggest the true rate of malignant transformation may be considerably lower than this traditional estimate.

The clinical presentation of SO is often nonspecific, mimicking other ovarian neoplasms. Patients may present with pelvic pain, abdominal distension, menstrual irregularities, or, in a minority of cases, with signs and symptoms of hyperthyroidism due to autonomous hormone secretion by the ectopic thyroid tissue [8,9]. Hyperthyroidism is reported in 5–8% of SO cases and is even rarer in malignant cases [8,9].

Imaging findings are not pathognomonic, and definitive diagnosis relies on histopathological examination combined with immunohistochemical staining for thyroid markers, including thyroglobulin and TTF-1 [10,11]. Management of malignant struma ovarii (MSO) is not standardised, with options ranging from conservative fertility-sparing surgery to multimodal therapy including total thyroidectomy and adjuvant radioactive iodine (RAI), particularly in cases with high-risk features, extraovarian spread, or a clear surveillance rationale [12,13,14].

We report a case of MSO with papillary thyroid carcinoma in a 38-year-old premenopausal woman who presented with a complex pelvic mass, concurrent multinodular goitre, and mild thyrotoxicosis. This case is reported in accordance with the CARE (CAsE REport) guidelines [15] and ICMJE authorship criteria [16].

1. CASE PRESENTATION

A timeline of the principal clinical events is summarised in Table 1.

Table 1. Timeline of key clinical events.

Time Point	Event
Presentation (2-month symptom history)	Lower abdominal discomfort, intermittent pelvic pain, palpitations, heat intolerance, mild weight loss, increased sweating; longstanding multinodular goitre noted on examination
Initial work-up	Thyroid function tests and tumour markers (Table 2); pelvic and thyroid ultrasound; pelvic MRI
Surgery 1	Laparoscopic left salpingo-oophorectomy with peritoneal washings
Postoperative histopathology	1.5 cm focus of classical papillary thyroid carcinoma within struma ovarii; negative margins; peritoneal washings negative
Postoperative thyroid work-up	Persistent mild thyrotoxicosis; FNA of dominant thyroid nodule (Bethesda Category II); neck ultrasound/CT without suspicious features
Surgery 2	Total thyroidectomy; histopathology confirmed benign multinodular hyperplastic goitre
Adjuvant therapy	Thyroid hormone withdrawal and TSH stimulation, followed by RAI (I-131, 120 mCi); post-therapy whole-body scan showed thyroid-bed-only uptake; levothyroxine commenced at TSH-suppressive dose
6 months post-RAI	Thyroglobulin undetectable (<0.2 ng/mL)
12 months post-RAI	Thyroglobulin undetectable; anti-thyroglobulin antibodies negative
18 months (last follow-up)	Clinically well; no recurrence on examination, thyroglobulin, or imaging

1.1 Clinical History and Presentation

A 38-year-old premenopausal woman presented to the gynaecology clinic with a two-month history of lower abdominal discomfort, intermittent pelvic pain, palpitations, heat intolerance, mild weight loss, and increased sweating. Menstrual cycles were regular. She reported a longstanding anterior neck swelling that had gradually increased in size over several years. There was no personal or family history of thyroid malignancy or ovarian pathology.

On physical examination, the patient was alert, afebrile, and haemodynamically stable (blood pressure 118/74 mmHg, heart rate 92 bpm, temperature 36.8°C). Abdominal palpation revealed a firm, mobile, non-tender pelvic mass in the left adnexal region. Neck examination demonstrated a diffusely enlarged multinodular thyroid gland without cervical lymphadenopathy. Mild finger tremor and brisk deep tendon reflexes were noted; there was no ophthalmopathy, dermopathy, or pretibial myxoedema.

1.2 Laboratory Investigations

Initial laboratory evaluation revealed biochemical thyrotoxicosis with suppressed TSH and elevated free thyroid hormones (Table 2).

Table 2. Initial thyroid function tests and tumour markers.

Laboratory Test	Result	Reference Range	Interpretation
TSH	0.12 mIU/L	0.4–4.0 mIU/L	Suppressed
Free T4	22.5 pmol/L	9–19 pmol/L	Elevated
Free T3	6.1 pmol/L	2.6–5.7 pmol/L	Elevated
Thyroglobulin	45 ng/mL	<40 ng/mL	Mildly elevated
Anti-Tg antibodies	Negative	Negative	Normal
CA-125	68 U/mL	<35 U/mL	Elevated
CA19-9, CEA, AFP	Within normal limits	—	Normal

1.3 Imaging Investigations

Pelvic transvaginal ultrasound revealed a $9.5 \times 7.2 \times 6.8$ cm complex left adnexal mass with mixed solid and cystic components, internal septations, and areas of increased vascularity on Doppler imaging. The right ovary and uterus were unremarkable. No free fluid or lymphadenopathy was identified.

Thyroid ultrasound demonstrated a diffusely enlarged gland with multiple bilateral nodules, the largest measuring 2.1 cm in the left lobe, without suspicious echographic features or cervical lymphadenopathy.

Magnetic resonance imaging (MRI) of the pelvis confirmed a multilocular cystic-solid pelvic mass with low T2 signal intensity within solid components — consistent with colloid-rich thyroid tissue — and focal gadolinium enhancement after contrast administration. No extraovarian extension, peritoneal implants, or lymphadenopathy were demonstrated.

1.4 Surgical Management

Following multidisciplinary tumour board discussion, and given her wish for fertility preservation, the patient underwent elective laparoscopic left salpingo-oophorectomy rather than cystectomy alone, consistent with evidence that complete adnexal excision carries a lower recurrence risk than ovarian-sparing cystectomy ^[14]. Intraoperative findings included a well-encapsulated, lobulated left ovarian mass with intact capsule and no peritoneal or omental deposits. Peritoneal washings were obtained. The contralateral ovary appeared macroscopically normal.

Gross pathological examination of the excised specimen revealed a multiloculated cystic lesion ($10.2 \times 7.8 \times 6.5$ cm) with solid tan-brown areas resembling thyroid parenchyma. No macroscopic capsular breach or extraovarian involvement was identified.

1.5 Histopathology and Immunohistochemistry

Microscopic examination demonstrated a mature cystic teratoma with abundant thyroid tissue comprising more than 80% of the tumour volume. Within this thyroid-predominant component, a 1.5 cm focus of papillary structures was identified, lined by cells with enlarged, overlapping nuclei, prominent nuclear grooves, and optically clear ("ground-glass") chromatin. Psammoma bodies were present. No mitoses or necrosis were identified.

Immunohistochemical staining demonstrated strong and diffuse positivity for thyroglobulin, TTF-1, and PAX8, with focal positivity for HBME-1. The morphological and immunophenotypic features were diagnostic of papillary thyroid carcinoma (classical type) arising within struma ovarii. No capsular or lymphovascular invasion was identified, and all surgical margins were negative. Peritoneal washings were negative for malignant cells.

1.6 Postoperative Course and Thyroid Evaluation

Postoperative recovery was uneventful. Repeat thyroid function tests demonstrated persistent mild biochemical thyrotoxicosis. Serum thyroglobulin remained elevated at 42 ng/mL. Given the concurrent multinodular goitre and the need to exclude synchronous primary thyroid carcinoma, a comprehensive cervical thyroid evaluation was undertaken.

Fine-needle aspiration (FNA) cytology of the dominant left thyroid nodule was classified as Bethesda Category II (benign) using the Bethesda System for Reporting Thyroid Cytopathology ^[17]. Neck ultrasound and computed tomography (CT) demonstrated no suspicious nodal or parenchymal features. Nevertheless, total thyroidectomy was recommended, guided by: (1) the presence of multinodular goitre with persistent thyrotoxicosis; (2) the need to optimise postoperative thyroglobulin-based surveillance; and (3) the requirement for functional thyroidal clearance prior to RAI therapy. Total thyroidectomy was performed without complication. Histopathological examination confirmed multinodular hyperplastic goitre with no evidence of malignancy in either thyroid lobe.

1.7 Adjuvant Therapy and Surveillance

Following postoperative thyroid hormone withdrawal and TSH stimulation, the patient received adjuvant RAI therapy (I-131, 120 mCi), a decision individualised to her concurrent multinodular goitre, persistent thyrotoxicosis, and the goal of enabling reliable thyroglobulin-based surveillance (see Discussion, Section 2.8). Post-therapy whole-body scintigraphy demonstrated uptake restricted to the thyroid bed only, with no evidence of locoregional or distant metastatic disease. Levothyroxine was commenced at a TSH-suppressive dose (target TSH <0.1 mIU/L), consistent with American Thyroid Association guidance extrapolated from differentiated thyroid carcinoma management ^[18].

Serial serum thyroglobulin measurements at 6 and 12 months post-RAI were undetectable (<0.2 ng/mL), and anti-thyroglobulin antibodies remained negative. The patient remains clinically well at 18 months of follow-up, with no evidence of recurrence on clinical examination, thyroglobulin monitoring, or imaging surveillance. Given reported recurrences beyond several years in MSO, long-term surveillance for a minimum of 10 years is planned (Section 2.11).

Table 3. Summary of clinical features, investigations, management steps, and follow-up outcomes.

Clinical Feature	Finding / Action
Age and sex	38-year-old premenopausal female
Presenting symptoms	Lower abdominal discomfort, intermittent pelvic pain, palpitations, heat intolerance, mild weight loss, increased sweating, longstanding multinodular goitre
Thyroid function tests	TSH 0.12 mIU/L (suppressed); Free T4 22.5 pmol/L (elevated); Free T3 6.1 pmol/L (elevated) — biochemical thyrotoxicosis
Tumour markers	CA-125 elevated at 68 U/mL; thyroglobulin 45 ng/mL; CA19-9, CEA, AFP within normal limits
Pelvic imaging	Transvaginal ultrasound: 9.5 × 7.2 × 6.8 cm complex cystic-solid left adnexal mass. MRI pelvis: multilocular mass with low T2 signal (colloid), focal gadolinium enhancement; no extraovarian extension
Thyroid imaging	Ultrasound: diffusely enlarged multinodular thyroid; dominant left nodule 2.1 cm; no suspicious cervical lymphadenopathy
Surgical management	Laparoscopic left salpingo-oophorectomy; peritoneal washings obtained; intact capsule; no peritoneal or omental deposits
Histopathology	Mature cystic teratoma; >80% thyroid tissue; 1.5 cm focus of papillary thyroid carcinoma (classical type); no capsular or lymphovascular invasion; negative margins; peritoneal cytology negative
Immunohistochemistry	Thyroglobulin (+), TTF-1 (+), PAX8 (+), HBME-1 focal (+); confirms thyroid origin with PTC features
Thyroid evaluation	FNA of dominant nodule: Bethesda Category II (benign); total thyroidectomy: multinodular goitre, no malignancy
Adjuvant therapy	I-131 RAI 120 mCi; post-RAI whole-body scan: uptake in thyroid bed only; TSH suppression with levothyroxine commenced
Follow-up (18 months)	Serial thyroglobulin undetectable at 6 and 12 months; no recurrence on clinical, laboratory, or imaging surveillance

2. DISCUSSION

2.1 Epidemiology

Struma ovarii is a rare entity, classically representing approximately 2–5% of ovarian teratomas and less than 1% of all ovarian tumours [1-3]. The mean age at diagnosis is in the fourth to fifth decade, predominantly in premenopausal women [12,19]. Malignant transformation has traditionally been reported in 5–10% of SO cases, with PTC being the most common histological subtype [7,12,13]; however, this classic estimate derives largely from small historical series subject to selection and publication bias. A large single-centre retrospective cohort spanning more than four decades identified SO in only 2.14% of 12,864 ovarian teratomas, and malignant transformation in just 4.7% of these SO cases [19], suggesting that the true rate of malignancy in SO may be considerably lower than the traditionally cited 5–10% range. A recent multicentre analysis characterised the clinical spectrum of 17 MSO patients alongside a pooled review of 178 previously reported cases [12]. The rarity of MSO has limited development of standardised management protocols, and most recommendations are derived from case series, retrospective cohorts, and expert consensus rather than randomised evidence [12,13,14].

2.2 Clinical Presentation and Thyrotoxicosis

Common presenting features include pelvic pain, abdominal mass, and menstrual irregularities [10,12]. Hyperthyroidism is observed in 5–8% of SO cases and is even rarer in malignant cases [8,9]. The degree of thyrotoxicosis is thought to correlate with the volume and functional activity of ectopic thyroid tissue, an inference supported by reports of biochemical and clinical resolution of hyperthyroidism following surgical resection of the ectopic tissue [9,20]. In the present case, the patient exhibited mild biochemical thyrotoxicosis, most plausibly attributable to combined autonomous hormone production from both the ovarian struma and the coexisting

multinodular goitre. This concurrence introduced diagnostic complexity, as both the ovarian and cervical thyroid tissues may independently contribute to thyrotoxicosis, and neither source could be excluded as a contributor on biochemical grounds alone.

2.3 Imaging Characteristics

Preoperative diagnosis of SO on imaging is challenging due to nonspecific appearances. On ultrasound, SO typically presents as a complex multilocular cystic-solid mass with internal septations and areas of increased vascularity. Small, hyperechoic, richly vascularised nodular foci — sometimes referred to descriptively as "struma pearls" in the radiological literature, a sonographic pattern-recognition term that should not be conflated with psammoma bodies, which are a distinct histopathological finding — may suggest the diagnosis ^[10,21]. MRI characteristically demonstrates multilocular cysts with solid components and low T2 signal due to colloid-rich thyroid tissue ^[10]. In this case, imaging features typical of struma ovarii were present but could not reliably distinguish benign from malignant disease, nor definitively exclude other ovarian neoplasms — underscoring that histopathology remains the diagnostic reference standard.

2.4 Histopathology, Immunohistochemistry, and Molecular Pathology

SO is defined by mature thyroid tissue comprising >50% of tumour volume. MSO is diagnosed based on cytological and architectural features consistent with thyroid carcinoma, including papillary structures, nuclear grooves, optically clear chromatin, psammoma bodies, and, in FTC, capsular or vascular invasion ^[11,12]. Immunohistochemical staining for thyroglobulin, TTF-1, and PAX8 confirms thyroid origin, while HBME-1 and galectin-3 support a PTC diagnosis ^[11]. Molecular studies indicate that BRAF mutations, including but not limited to BRAF V600E, occur in a subset of MSO with papillary histology, but at a lower frequency than in eutopic primary thyroid PTC; RAS-family mutations (KRAS, NRAS) and other alterations have also been described, indicating a molecular landscape that only partially overlaps with that of primary thyroid PTC ^[22,23]. In this case, classical PTC morphology was confirmed, consistent with prior reports ^[7,11,12]; tumour molecular profiling was not performed, in keeping with the current absence of an established role for routine molecular testing in the management of MSO.

2.5 Differential Diagnosis

The principal differential diagnosis is between primary PTC arising within SO and metastatic thyroid carcinoma to the ovary. Ovarian metastasis from thyroid carcinoma is exceedingly rare and typically occurs in the context of a known primary thyroid malignancy, often without teratomatous elements ^[24]. In the present case, the absence of primary thyroid malignancy on thyroidectomy and the presence of abundant teratomatous tissue supported the diagnosis of primary ovarian PTC arising within SO. Other differential diagnoses include primary ovarian carcinoma, serous cystadenoma, and endodermal sinus (yolk sac) tumour, which were excluded on the basis of tumour marker profile, histology, and immunophenotype.

2.6 Surgical Management

Surgical excision is the cornerstone of treatment. For premenopausal women with disease confined to the ovary, unilateral salpingo-oophorectomy is generally sufficient and preserves fertility ^[14,25]. Ovarian-sparing cystectomy has been associated with a significantly higher recurrence risk than salpingo-oophorectomy and is discouraged when malignancy is confirmed or suspected ^[14]. Surgical staging should include peritoneal washings, omental inspection, and assessment for peritoneal implants ^[12,14]. In this patient, unilateral salpingo-oophorectomy with peritoneal washings was performed; no extraovarian disease was identified.

2.7 Role of Total Thyroidectomy

Total thyroidectomy is considered in MSO for three principal reasons: (1) to exclude synchronous primary thyroid carcinoma; (2) to enable uninterfered thyroglobulin-based surveillance; and (3) to facilitate effective RAI therapy, when RAI is indicated ^[12,13,14]. Because adjuvant thyroid-directed therapy has not been shown to improve survival

in unselected patients (Section 2.8), the decision to proceed to thyroidectomy should be individualised, weighing thyroid nodularity, imaging findings, thyroid function, and patient preference against the morbidity of an additional operation. In the present case, multinodular goitre with persistent thyrotoxicosis and the need for optimal surveillance justified thyroidectomy, which ultimately revealed benign multinodular hyperplasia.

2.8 Radioactive Iodine Therapy

Adjuvant RAI therapy has traditionally been offered in MSO with high-risk histological features, extraovarian spread, or when optimal long-term thyroglobulin-based surveillance is desired ^[12,13]. RAI ablates residual iodine-avid thyroid tissue and is intended to treat microscopic disease. However, a systematic review and meta-analysis of 376 surgically treated MSO patients found no demonstrable survival benefit from adjuvant thyroid-related therapy, including RAI, over surgery alone, raising the possibility that such therapy constitutes overtreatment in patients without high-risk features ^[26]. When RAI is pursued, the dose and protocol are extrapolated from guidelines for differentiated thyroid carcinoma, typically 100–150 mCi of I-131 ^[12,13,18]. Adjuvant RAI should therefore be applied selectively rather than routinely — reserved for patients with high-risk histology, macroscopic extraovarian or metastatic disease, or a specific surveillance rationale — rather than offered indiscriminately. In this patient, RAI (120 mCi) was administered following thyroidectomy, a decision driven primarily by the concurrent multinodular goitre and persistent thyrotoxicosis and by the intention to enable thyroglobulin-based surveillance, rather than by high-risk tumour features; post-therapy scanning showed no metastatic uptake, and serial thyroglobulin has remained undetectable, indicating complete biochemical remission to date.

2.G Peritoneal Strumosis and Patterns of Spread

Peritoneal strumosis refers to the presence of benign or highly differentiated thyroid tissue implants in the peritoneum, most classically described following rupture, spillage, or ovarian-sparing surgery for SO ^[27]. Malignant MSO may also metastasise to peritoneum, omentum, liver, lungs, or bone, though such extraovarian spread is uncommon at presentation ^[27]. In the present case, peritoneal cytology was negative and no implants were identified at surgery.

2.10 Tumour Markers

CA-125 may be elevated in SO and MSO, particularly in the presence of ascites or peritoneal irritation, but lacks specificity for malignancy ^[8,12]. Thyroglobulin is a sensitive and relatively specific marker for residual or recurrent disease after thyroidectomy and RAI ablation ^[11,12]. Anti-thyroglobulin antibodies can interfere with immunoassay measurement of thyroglobulin and should be monitored serially alongside it.

2.11 Long-Term Outcomes and Surveillance

The prognosis of MSO is generally favourable but declines gradually with longer follow-up. In a cohort of 194 patients, 5-, 10-, and 15-year overall survival rates were 91.4%, 87.7%, and 83.5% respectively, with corresponding disease-specific survival rates of 93.8%, 90.0%, and 85.7% ^[13]. Recurrence rates ranging from 7.5% to 35% have been reported across surgical series, with lower rates observed in more recent cohorts employing standardised staging and complete adnexal excision ^[12,14]. Risk factors for recurrence include extraovarian spread at diagnosis, larger tumour size, aggressive histological subtype, and incomplete surgical excision — particularly ovarian-sparing cystectomy rather than salpingo-oophorectomy ^[13,14]; reported tumour-size thresholds vary across series and should be interpreted in the context of the specific cohort cited rather than treated as a fixed cut-off. Given the potential for late recurrence, long-term surveillance with clinical examination, imaging, and serial thyroglobulin measurement is recommended for a minimum of 10 years ^[13,14].

2.12 Fertility Considerations

Fertility preservation is a key consideration in premenopausal women with MSO. Unilateral salpingo-oophorectomy is preferred when oncologically feasible; fertility-sparing surgery in malignant ovarian germ cell

tumours as a category is associated with favourable reproductive outcomes ^[25], although struma-ovarum-specific reproductive outcome data remain limited and this evidence is necessarily extrapolated from the broader malignant germ cell tumour literature. Oocyte or embryo cryopreservation may be discussed prior to RAI therapy, which can transiently impair ovarian reserve. Multidisciplinary counselling including reproductive medicine is essential.

2.13 Multidisciplinary Management and Guidelines

Optimal management of MSO requires coordinated input from gynaecologic oncology, endocrinology, pathology, nuclear medicine, and reproductive medicine. No universally accepted disease-specific guideline exists; recommendations are extrapolated from differentiated thyroid carcinoma protocols and general ovarian neoplasm guidelines. Multidisciplinary tumour board discussion ensures individualised, evidence-informed care balancing oncologic control with quality of life and fertility preservation ^[12,13].

2.14 Limitations

This report has several limitations inherent to single-case reporting. First, findings from one patient cannot be generalised, and management decisions were individualised rather than protocol-driven. Second, follow-up to date (18 months) is relatively short relative to the late recurrences (occasionally beyond several years) documented in larger MSO cohorts; longer surveillance is planned. Third, tumour molecular profiling (e.g., BRAF, RAS) was not performed, so the molecular subtype of this tumour remains uncharacterised. Fourth, evidence supporting fertility-sparing surgery outcomes and, to a lesser extent, adjuvant therapy decisions in MSO is drawn substantially from small case series, retrospective cohorts, or the broader malignant germ cell tumour literature rather than from MSO-specific prospective data, and should be interpreted accordingly.

3. CONCLUSION

Malignant struma ovarum is a rare and diagnostically challenging entity, particularly when presenting with concurrent multinodular goitre and biochemical thyrotoxicosis. This case underscores the importance of a high index of clinical suspicion, comprehensive preoperative evaluation, and multidisciplinary management. Surgical excision — favouring salpingo-oophorectomy over cystectomy — remains the cornerstone of therapy. Total thyroidectomy and adjuvant RAI are best reserved for selected cases with high-risk features, extraovarian disease, or a clear surveillance rationale, rather than applied routinely, since adjuvant thyroid-directed therapy has not been shown to confer a survival benefit in unselected patients. Serial thyroglobulin monitoring remains the most sensitive tool for detecting residual or recurrent disease, and surveillance should extend for at least 10 years given the potential for late recurrence. Individualised care, informed by oncologic risk stratification, patient preferences, and fertility considerations, is paramount. Further accumulation of multicentre case series and prospective data are necessary to refine management algorithms and clarify the true rate of malignant transformation in this rare tumour.

4. Declarations

Conflicts of Interest

The authors declare no conflicts of interest.

Funding

No funding was received for this work.

Patient Consent

Written informed consent was obtained from the patient for publication of this case report and any accompanying clinical details. All identifying information has been removed to ensure patient confidentiality, in accordance with the CARE guidelines and COPE recommendations.

Ethical Approval

This case report was prepared in accordance with the CARE guidelines and ICMJE authorship criteria ^[15,16].

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