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Late Recurrence of an Immature Ovarian Teratoma as a Mucinous Neoplasm

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Abstract **Case Report**

Immature ovarian teratomas are rare malignant germ cell tumors that typically occur in young women and have a favorable prognosis when treated with surgery and adjuvant chemotherapy. However, late recurrence with somatic-type malignant transformation is extremely uncommon. We report the case of a 48-year-old woman previously treated in 2002 for a FIGO stage IC2, grade 3 immature ovarian teratoma with left adnexectomy followed by four cycles of BEP chemotherapy. After a 20-year disease-free interval, the patient presented in 2022 with a hard parietal mass. Imaging and biopsy revealed an invasive mucinous adenocarcinoma of probable digestive origin. She received XELOX chemotherapy with partial response and underwent complete surgical resection. Histological analysis confirmed a low-grade mucinous tumor arising within a mature teratoma, consistent with a somatic-type malignancy. In 2024, the patient developed a large abdominopelvic mass with peritoneal involvement. Surgery revealed a mucinous carcinoma with signet-ring cells and peritoneal carcinomatosis. Molecular analysis showed an NRAS G12S mutation. She was started on palliative chemotherapy with FOLFIRI and bevacizumab. This case highlights a rare example of late relapse of an immature ovarian teratoma with transformation into mucinous adenocarcinoma of gastrointestinal phenotype. It underscores the importance of long-term surveillance and the role of immunohistochemistry and molecular profiling in guiding diagnosis and treatment.

Keywords: Immature ovarian teratoma; somatic-type malignancy; mucinous adenocarcinoma; NRAS mutation; late recurrence.

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INTRODUCTION

Immature ovarian teratomas are rare malignant germ cell tumors accounting for less than 1% of all ovarian neoplasms and approximately 20% of malignant ovarian germ cell tumors [1]. They primarily affect adolescents and young women and are characterized by the presence of immature neuroectodermal tissue [2]. The prognosis is generally favorable for early-stage disease, especially with complete surgical excision and, when indicated, adjuvant chemotherapy using platinum-based regimens [3].

Recurrence typically occurs within the first three years following initial treatment and is often associated with inadequate resection or high-grade tumors [4]. Late recurrence, defined as recurrence occurring more than five years after initial therapy, is exceedingly rare and may reflect either regrowth of microscopic residual disease or malignant transformation of mature teratomatous elements into somatic-type malignancies [5]. Among these, mucinous neoplasms represent a particularly unusual form of secondary somatic transformation [6].

Somatic-type malignancies arising from germ cell tumors, including adenocarcinomas, sarcomas, and primitive neuroectodermal tumors, have been well documented. Mucinous tumors, whether of intestinal or endocervical type, are a recognized but rare form of such transformation. The pathogenesis of these tumors is not fully elucidated but is thought to involve clonal evolution of epithelial elements within a mature or regressed immature teratoma [7]. Histological distinction between a de novo mucinous ovarian tumor and a mucinous neoplasm arising from a teratomatous component can be challenging, particularly in recurrent lesions [8].

This report describes a rare case of late recurrence of an immature ovarian teratoma, more than a

decade after initial diagnosis and treatment, presenting as a mucinous neoplasm. Through a detailed histopathological review and discussion of potential mechanisms, we aim to highlight the diagnostic complexity and the need for long-term surveillance in this subset of patients.

Case Presentation

A 48-year-old woman, with no significant medical history, had been treated in 2002 for a FIGO stage IC2, grade 3 immature ovarian teratoma. She underwent left salpingo-oophorectomy followed by four cycles of BEP (bleomycin, etoposide, cisplatin) chemotherapy. In May 2022, she presented with abdominal distension. Clinical examination revealed a firm, fixed right pubic wall mass (≈4 cm) along her surgical scar. Abdominopelvic ultrasound revealed three hypoechoic, solid masses (1.5–3.6 cm) and a fourth calcified lesion. Endometrial thickening (22 mm) was noted, with normal right ovary and no intra-abdominal effusion. Pelvic MRI showed an anterior midline mass overlying the bladder, suggestive of an incisional hernia (Figure 1).

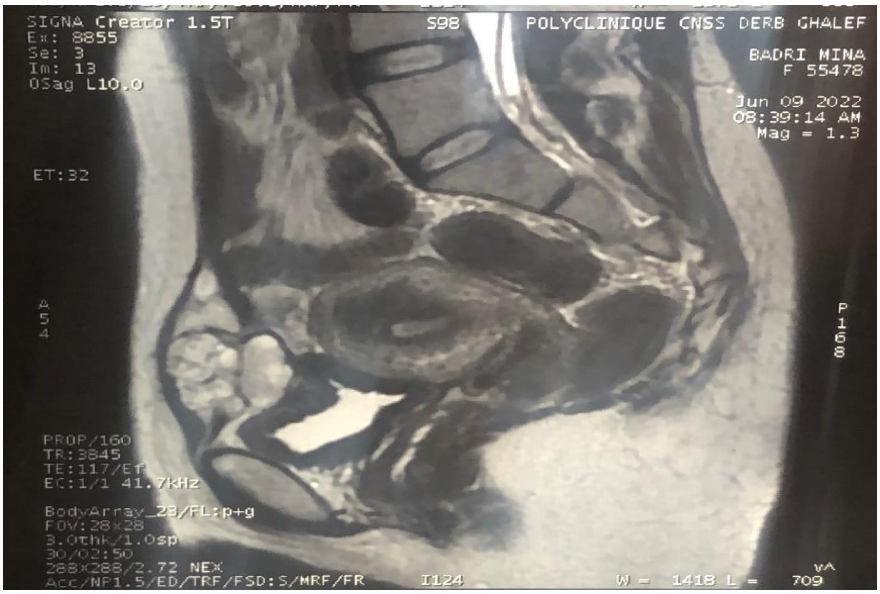

Figure 1: Pelvic MRI in sagittal T2-weighted sequence showing a large heterogeneous pelvic mass

Tumor markers (LDH, β-HCG, AFP, CA 125, CEA) were within normal limits. Multidisciplinary tumor board recommended biopsy of the pubic mass. Histology revealed an invasive mucinous adenocarcinoma with an immunoprofile consistent with gastrointestinal origin (PAX8 negative) (Figure 2).

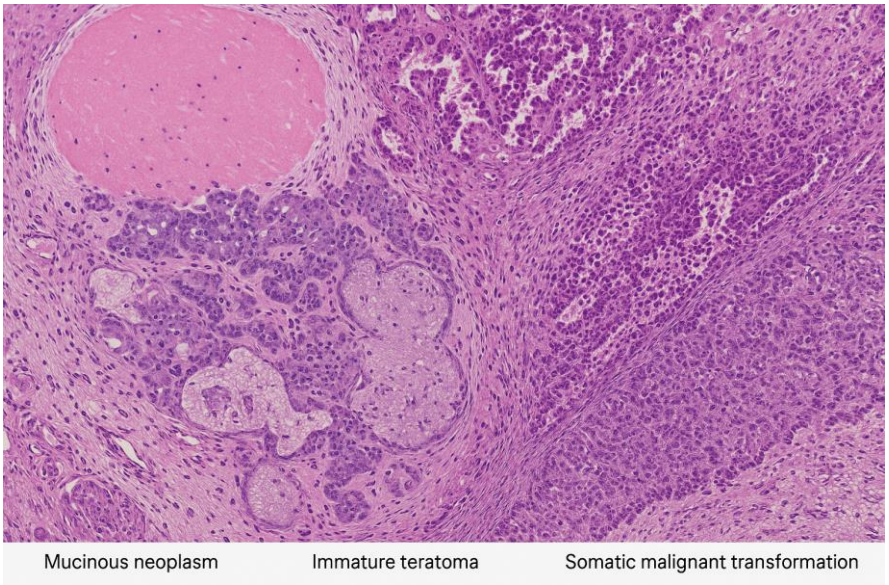

Figure 2: Histological section of a late recurrence of an immature teratoma with malignant transformation. H&E stain. On the left: mucinous neoplasm; in the center: immature teratomatous components; on the right: infiltrating mucinous adenocarcinoma arising from somatic-type malignant transformation

Repeat MRI showed a 79 × 39 mm pre-vesical mass with cystic components and a FIGO stage 0 intracavitary uterine lesion. PET-CT confirmed a suspicious 66 × 40 × 65 mm heterogeneous pre-vesical mass without distant metabolic activity. The patient received XELOX chemotherapy (capecitabine + oxaliplatin) over 3 months, achieving a 20% reduction in tumor volume. Cystoscopy was normal.

In March 2023, surgical resection of the mass was performed. Histology showed a low-grade mucinous epithelial neoplasm (appendiceal type) arising in a mature teratoma, without immature elements (Image 5). The ovary and appendix were benign; chronic inflammation was noted in the salpinx. The tumor was reclassified as a somatic-type neoplasm arising from a mature teratoma per 2019 WHO classification. Resection was complete (R0), and active surveillance was recommended. In August 2024, follow-up CT revealed a large complex abdominopelvic mass (154 × 155 × 143 mm), ascites, and possible peritoneal carcinomatosis. Two small indeterminate hepatic dome lesions were also noted. CA 125 and CEA were elevated (122 U/mL and 43 ng/mL, respectively). In September 2024, cytoreductive surgery was performed, including resection of the mass, partial removal of the utero-ovarian ligament, peritoneal biopsy, and cytology. Histology showed a colloid mucinous carcinoma with prominent signet-ring cell component, vascular emboli, and malignant cells in peritoneal fluid. Immunohistochemistry supported gastrointestinal origin (CK20+, CK7-, WT1-).

Upper and lower GI endoscopy were unremarkable. In November 2024, CT scan revealed progression of bilateral pulmonary nodules (largest 20 mm) and multiple peritoneal micronodules, suggesting metastatic spread. CA 19-9 was elevated (177.8 U/mL), while other markers remained normal. The case was reviewed in multidisciplinary conference and considered a metastatic recurrence of probable gastrointestinal origin. Molecular testing revealed an NRAS G12S mutation, with wild-type KRAS and BRAF. Palliative chemotherapy with FOLFIRI plus bevacizumab was initiated. As of now, the patient has completed three cycles.

DISCUSSION

Immature ovarian teratomas (IOTs) are rare malignant germ cell tumors that represent less than 1% of all ovarian cancers and approximately 20% of malignant germ cell tumors [9]. They typically affect adolescents and young women and are often diagnosed at early stages due to the presence of a palpable mass or abdominal symptoms. The prognosis for early-stage IOTs, especially following complete surgical excision and adjuvant chemotherapy (usually BEP: bleomycin, etoposide, cisplatin), is generally excellent, with 5-year survival rates exceeding 90% in stage I disease [10]. Recurrence, when it occurs, is most often observed within 2–3 years of initial treatment. Late recurrence, particularly after more than a decade, is exceedingly rare and poorly documented in the literature [11]. In this case, the patient presented nearly 20 years after initial treatment with a recurrent mass that histologically and immunohistochemically resembled a gastrointestinal mucinous adenocarcinoma. However, further surgical and pathological evaluation confirmed that the lesion arose within a mature teratomatous context, without evidence of immature neuroectodermal tissue. This points toward a diagnosis of somatic-type malignancy arising in a mature teratoma (STM-MT)—a rare but well-recognized phenomenon [12].

Somatic-type malignant transformation (SMT) occurs in approximately 0.2–2% of mature teratomas and includes transformation into carcinomas (e.g., squamous cell, adenocarcinoma), sarcomas, or neuroendocrine tumors [13]. The mucinous subtype is infrequent and typically mimics appendiceal or colonic tumors both histologically and immunohistochemically, as seen here with positivity for CK20 and negativity for CK7 and PAX8. This immunophenotype is more consistent with a lower gastrointestinal tract origin, raising the differential diagnosis of a primary GI tumor. However, the absence of any primary lesion on endoscopy and the presence of teratomatous elements support the diagnosis of a teratoma with secondary mucinous neoplasm [14]. Interestingly, the tumor in this case evolved over time, initially presenting as a low-grade mucinous neoplasm, later transforming into a colloid carcinoma with signet-ring cells and ultimately causing peritoneal dissemination and pulmonary metastases. This pattern of progression mirrors the behavior of mucinous tumors of the appendix or ovary that undergo peritoneal spread (pseudomyxoma peritonei). However, signet-ring morphology and NRAS mutation suggest a more aggressive phenotype and poorer prognosis [15].

Histologically, signet-ring cells are associated with aggressive behavior and chemotherapy resistance, particularly in gastric and colorectal carcinomas [16]. Their emergence in this case suggests clonal evolution under selective pressure, potentially exacerbated by prior chemotherapy exposure. From a molecular standpoint, the identification of an NRAS G12S mutation, with wild-type KRAS and BRAF, is notable. NRAS mutations are rare in colorectal and mucinous ovarian tumors but have been implicated in tumor progression and resistance to EGFR-targeted therapies [17]. This mutation likely limits the efficacy of anti-EGFR agents but opens the door to treatments like bevacizumab, which was included in the patient's regimen [18].

Therapeutically, there is no consensus on the optimal management of somatic-type malignancies arising from teratomas. Surgical resection remains the cornerstone of treatment. In recurrent or metastatic cases, therapy is often extrapolated from regimens used in

tumors that share histological features. In this case, XELOX (capecitabine/oxaliplatin) and later FOLFIRI (irinotecan, 5-FU, leucovorin) with bevacizumab were chosen based on the mucinous colorectal-like phenotype. The limited initial response followed by aggressive progression underscores the need for tailored treatment guided by molecular profiling [19].

This case also highlights the importance of lifelong follow-up in patients with a history of immature teratomas. Although current guidelines recommend surveillance for up to 5 years, this case demonstrates that recurrences—including with malignant transformation—can occur decades later. Periodic imaging and tumor marker monitoring may be warranted in selected high-risk patients, particularly those with high-grade or stage IC disease [20].

CONCLUSION

This case highlights an exceptionally rare instance of a late relapse of an immature ovarian teratoma, occurring nearly 20 years after initial treatment, and manifesting as a mucinous adenocarcinoma of somatic origin. Such an evolution underscores the unpredictable biological behavior of germ cell tumors, even in patients who have remained in complete remission for extended periods. Malignant transformation within a teratoma, although rare, should remain a differential diagnosis in patients presenting with a new abdominopelvic mass, regardless of the time elapsed since initial treatment. The immunohistochemical profile (CK20+, CK7−, PAX8−), along with the detection of an NRAS G12S mutation in this case, provided essential diagnostic and prognostic insights and informed treatment decisions. This case also emphasizes the lack of clear guidelines regarding long-term follow-up in patients treated for immature teratomas. It raises the question of whether extended surveillance might be appropriate in selected cases, particularly in those with high-grade tumors or complex histologic features. Finally, the therapeutic management of somatic-type malignant transformation remains a clinical challenge due to the absence of standardized protocols. Multidisciplinary decision-making is crucial, and treatment should be guided by both histopathological and molecular characteristics of the transformed tumor.

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