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CASE REPORT

When a teratoma recurs differently: peritoneal neurogliomatosis in a 25-year-old patient

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Abstract

Background

Gliomatosis peritonei (GP) is a rare condition characterized by the peritoneal dissemination of mature glial tissue, most commonly associated with an immature ovarian teratoma. Its pathogenesis, clinical course, and long-term prognosis remain poorly understood.

Case presentation

We report the case of a 23-year-old woman initially diagnosed with a high-grade immature ovarian teratoma in 2021, who underwent surgical treatment without adjuvant therapy. Three years later, she presented with recurrence as a mature teratoma associated with gliomatosis peritonei. Histopathological examination demonstrated mature neuroglial proliferation without evidence of immaturity or malignant transformation. Given the benign nature of the lesions, no additional treatment was administered, and annual gynecological follow-up was recommended.

Discussion

GP is an exceptionally rare condition, most often diagnosed incidentally. Two main hypotheses have been proposed to explain its origin: implantation of glial tissue from the primary teratoma or glial metaplasia of submesothelial pluripotent cells. The available literature suggests a generally favorable prognosis, although recurrence may occur despite maturation of the lesions. Malignant transformation is exceedingly rare. Management should be individualized and relies on histological confirmation, multidisciplinary evaluation, and fertility preservation whenever appropriate.

Conclusion

This case highlights the importance of long-term surveillance following treatment of immature ovarian teratoma, the potential for benign recurrence associated with gliomatosis peritonei, and the need to avoid unnecessary treatment in the absence of malignant transformation.

Keywords: Gliomatosis peritonei, ovarian teratoma, recurrence, mature glial tissue, gynecological surveillance.

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Introduction

Gliomatosis peritonei (GP) is a rare condition most commonly associated with immature ovarian teratomas [1]. Its pathogenesis remains poorly understood. In the literature, GP is considered an extremely rare entity, with only approximately 100–150 cases reported worldwide. Given its rarity relative to the overall frequency of ovarian teratomas, the true incidence is unknown but is estimated to be substantially lower than 1 case per 1,000,000 women per year.

The present case illustrates the diagnosis and management of this rare condition. Given the limited number of reported cases, we also conducted a review of the literature to discuss the current evidence regarding its management and follow-up.

Case report

In February 2024, a 23-year-old nulliparous, virginal woman presented to the gynecology clinic with persistent right iliac fossa pain. Her medical history included a cystectomy by laparotomy in 2021 for a subtorsed right ovarian cyst. Histopathological examination revealed a high-grade immature ovarian teratoma, comprising 30% of the solid lesion, with negative peritoneal cytology. No adjuvant treatment was administered, and no postoperative follow-up was arranged.

In 2022, the patient experienced recurrent right iliac fossa pain. Abdominal ultrasonography suggested the presence of a new ovarian cyst, and ovarian suppression therapy was recommended for 3 months, followed by repeat ultrasonographic evaluation. However, the patient did not undergo the proposed treatment or attend the scheduled follow-up.

In February 2024, after several episodes of recurrent pain, the patient returned for evaluation. Physical examination of the abdomen was unremarkable, and abdominal ultrasonography was inconclusive. Given her history of an immature ovarian teratoma, pelvic magnetic resonance imaging (MRI) was performed. MRI revealed a recurrent right adnexal cyst measuring 32 mm in maximum diameter, consistent with a fat-containing mature cystic teratoma with benign imaging features (Figure 1).

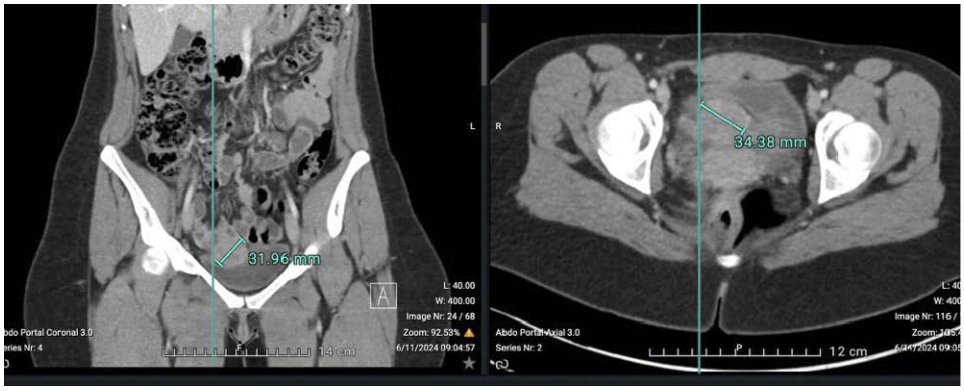

Figure 1: Thoraco abdominal CT.

A complementary evaluation, including a thoracoabdominal CT scan and serum tumor marker assessment (β-hCG, alpha-fetoprotein, LDH, CEA, CA125, and the ROMA score), was subsequently performed. The CT scan showed no evidence of metastatic disease and confirmed a 31-mm right adnexal lesion consistent with a teratoma (Figure 2). All tumor marker levels were within the normal range.

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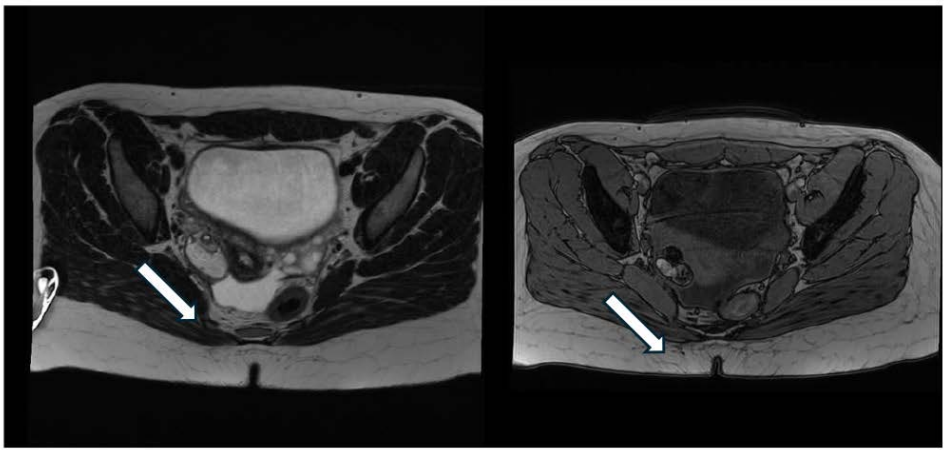

Figure 2: Pelvis MRI (arrow pointing the cyst).

During laparoscopy, the right adnexal mass was confirmed. Multiple whitish peritoneal lesions resembling miliary nodules were observed throughout the peritoneal cavity (Figure 3).

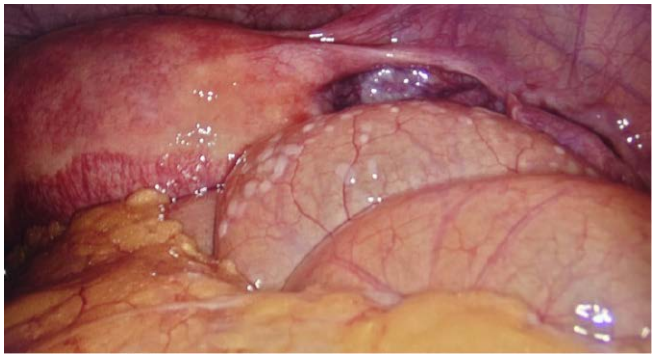

Figure 3: Miliary nodules on the colon.

Biopsies were obtained from the right hemidiaphragm, the round ligament of the liver, the omentum, the right parietal peritoneum, the supraumbilical anterior peritoneum, the left prevesical peritoneum, the lateral wall of the left iliac fossa, and the right ovarian fossa. Given the patient's history of an immature teratoma, a right adnexectomy was performed.

Histopathological examination confirmed a mature ovarian teratoma containing a prominent neuroglial component with no evidence of immature elements. Peritoneal cytology was negative. All peritoneal biopsies demonstrated mature neuroglial tissue, consistent with gliomatosis peritonei, which was confirmed by anti-glial fibrillary acidic protein (GFAP) immunostaining (Figure 4).

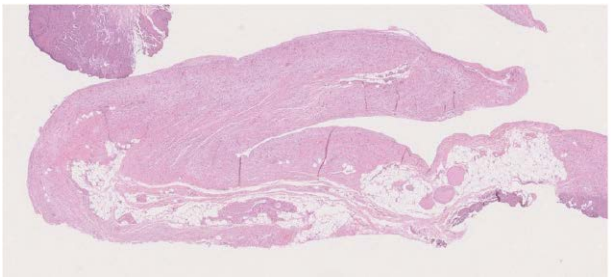

Figure 4a: at low magnification, normal fibro-adipose tissue is observed with a deposit of a mature glial tissue.

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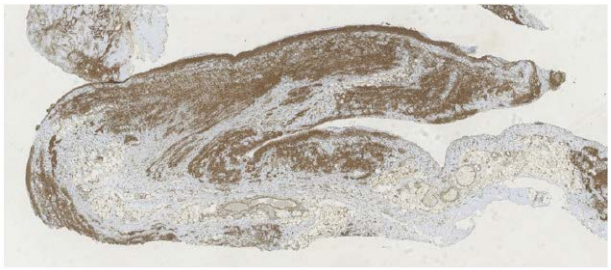

Figure 4b: Confirmation by anti-GFAP immunostaining.

Given the diffuse peritoneal involvement with miliary implants, the need for adjuvant chemotherapy was discussed at a multidisciplinary oncology conference; however, chemotherapy was not recommended because of the benign histopathological findings. Clinical surveillance alone was advised. At 1 year of follow-up, there was no evidence of disease recurrence.

Discussion

Gliomatosis peritonei (GP) is a rare condition that is most often diagnosed incidentally. It is characterized by the peritoneal dissemination of mature glial tissue and is typically associated with immature ovarian teratomas [1]. Its natural history and prognosis remain poorly defined.

The pathogenesis of GP remains incompletely understood. Nogales et al. [2] described two distinct mechanisms that may explain peritoneal dissemination. The classical hypothesis, which accounts for most reported cases, proposes that peritoneal implantation occurs following rupture of the capsule of an ovarian teratoma. This mechanism is supported by the frequent presence of other tissue components, such as keratin debris or hair, within the peritoneal implants, as well as by occasional lymph node involvement. According to this hypothesis, disseminated immature precursor cells subsequently differentiate locally into mature glial tissue [2].

An alternative hypothesis proposes that GP arises through peritoneal metaplasia. This theory emerged from genetic analyses showing that GP nodules may exhibit a heterozygous profile, identical to that of the patient's normal tissue, whereas the associated teratoma is homozygous. According to this model, GP results from the differentiation of peritoneal stem cells under the influence of growth factors. Nogales et al. concluded that, although implantation remains the most likely mechanism, metaplasia may explain cases characterized by a monomorphic astrocytic population [2].

Our case is characterized by the development of gliomatosis peritonei several years after the diagnosis of an immature ovarian teratoma, with recurrence as a mature cystic teratoma. To the best of our knowledge, this clinical presentation has not previously been reported in the literature.

An important differential diagnosis is growing teratoma syndrome (GTS). This condition develops during or after chemotherapy for an immature teratoma and is characterized by the appearance of enlarging mature teratomatous masses, even years after the initial treatment (reported intervals range from 2 to 78 months). Although these lesions may mimic malignant recurrence, they are paradoxically associated with normal or declining tumor marker levels, reflecting the phenomenon of chemotherapeutic retroconversion, in which chemotherapy eradicates the malignant components while allowing the mature benign elements to continue proliferating. GTS is rare, particularly when associated with gliomatosis peritonei, with only approximately 20 cases reported in the literature [3].

In a retrospective study, Wang et al. [4] reported a series of eight patients with gliomatosis peritonei, with a median age of 20 years. GP was associated with an immature teratoma in seven of the eight cases. All patients underwent fertility-sparing surgery, and five also received adjuvant chemotherapy. Complete excision of the peritoneal implants was considered impossible in seven patients, resulting in residual macroscopic disease. Nevertheless, the clinical outcomes were uniformly favorable. After a median follow-up of 60.5 months, all patients were alive and asymptomatic, and three spontaneous pregnancies had occurred, further supporting the benign nature of GP despite peritoneal dissemination.

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Although GP is generally associated with a favorable prognosis when accompanying an ovarian teratoma, the rare possibility of long-term malignant transformation should be recognized. This unfavorable outcome appears to have been reported exclusively in patients with immature teratomas and is supported by several published case reports. Schrefen et al. [5] described a patient who developed fatal malignant transformation more than 5 years after the diagnosis of a grade 3 immature teratoma, whereas Dadmanesh et al. [6] reported a similar progression to a malignant neuroectodermal tumor 7 years after the initial diagnosis.

While most cases of GP are associated with immature ovarian teratomas, its occurrence in association with a mature teratoma is much less common and is almost invariably associated with a favorable prognosis. This favorable clinical course is illustrated by two recent case reports. Bajracharya et al. [7] described a 21-year-old patient, whereas Khatiwada et al. [8] reported a 19-year-old patient. In both cases, histopathological examination demonstrated GP composed of mature glial tissue associated with a mature teratoma. Fertility-preserving surgery resulted in favorable outcomes, with no evidence of recurrence after 22 and 6 months of follow-up, respectively.

However, an unfavorable outcome has been reported in a patient with GP associated with a mature teratoma. Liu et al. [9] described a 17-year-old patient who had been followed since 2016 and who, after several incomplete surgical procedures and unsuccessful BEP chemotherapy, developed intestinal obstruction and massive ascites. Whole-exome sequencing identified a germline mutation in NF1 together with several somatic alterations, including MYC amplification, a TERT promoter mutation, and MGMT promoter methylation. Despite treatment with temozolomide, bevacizumab, and trametinib, the disease progressed rapidly, ultimately resulting in death from multiorgan failure. A summary of reported cases of GP associated with mature teratomas is presented in Table 1.

Author, Year (Country) [Reference]	Age (years)	Initial lesion	Interval to GP	Treatment	GP Characteristics	Evolution / Prognosis
Wang et al., 2016 (8 cases, China) [4]	15 - 25 (median 20)	7 immature, 1 mature	None	Conservative fertility-sparing surgery, completed by adjuvant chemotherapy in five cases.	Mature implants, peritoneal residue without malignancy (post-op)	Survival, asymptomatic, successful pregnancy
Bajracharya et al., 2021 (Nepal) [7]	21 years	Mature	None	Left adnexectomy, omentectomy, lymph node dissection and appendectomy via laparotomy.	Mature implants, peritoneal residue without malignancy (post-op)	Stable, no recurrence at 22 months
Khatiwada et al., 2024 (Nepal) [8]	19 years	Mature	None	Right adnexectomy and omentectomy via laparotomy.	- Nodular lesions on CT (pre-op) - Mature implants, peritoneal residue, without malignancy (post-op)	Stable, no recurrence at 6 months
Liu et al., 2022 (China) [9]	17 years	Mature	3 years	- Left adnexa resection, tumour cell reduction, right ovary biopsy, omentum resection, and appendectomy. - Chemotherapy (BEP type) - Targeted therapy (Temozolomide, Bevacizumab, and Trametinib)	Aggressive GP. NF1 mutation (post-op)	Aggressive progression, patient deceased
Chou et al., 2005 (Taiwan) [10]	36 years	Mature	None	Right adnexectomy, omentectomy	Mature implants, peritoneal residue without malignancy (post-op)	Stable, no recurrence at one year

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Author, Year (Country) [Reference]	Age (years)	Initial lesion	Interval to GP	Treatment	GP Characteristics	Evolution / Prognosis
Singla et al., 2017 (India) [11]	10 years	Mature	None	Right adnexectomy, partial omentectomy	- Subdiaphragmatic peritoneal deposits on CT (pre-op) - Mature implants, peritoneal residue without malignancy (post-op)	NA
Das et al., 2005 (India) [12]	20 years	Mature	None	Right adnexectomy, partial omentectomy	Mature implants, peritoneal residue without malignancy (post-op)	Stable, no recurrence at 26 months
Gocht et al., 1995 (Germany) [13]	13 years	Mature (for both lesions)	9 years	1st surgery: Left cystectomy with capsular rupture 2nd surgery: Right adnexectomy, partial omentectomy	Partial infiltration of the omentum intraoperatively	Stable, no recurrence at 18 months

Table 1. Cases of mature teratoma with gliomatosis peritonei (GP) found in the literature.

The “metastatic” implantation of mature glial tissue on the peritoneal surfaces can be extensive. Complete surgical excision is generally not feasible because these implants are often multiple and widely disseminated throughout the peritoneal cavity [4]. These implants may undergo fibrosis and eventually regress or persist without significant morphological changes. In rare cases, malignant transformation into glial or teratomatous tissue has been reported [14].

The indication for systemic therapy is determined primarily by the grade of the primary tumor. The presence of glial implants does not influence treatment decisions, provided that extensive histopathological sampling confirms their mature nature [15].

Given the rarity of this condition, there is no consensus regarding the optimal duration or modality of follow-up. Peritoneal implants have been reported to be readily detectable on CT imaging 43], although this was not the case in our patient. Therefore, CT could represent a useful modality for follow-up in selected cases. However, repeated CT examinations expose predominantly young patients to cumulative ionizing radiation, thereby increasing the lifetime risk of secondary malignancies. Given the very low rate of malignant transformation reported in GP, this risk may outweigh the potential benefits of routine CT surveillance.

Magnetic resonance imaging (MRI) represents an alternative imaging modality and can identify macronodular gliomatosis peritonei [16]. In our patient, however, the value of routine imaging surveillance is questionable, as no unfavorable outcomes have been reported in patients with mature ovarian teratomas associated with GP. Consequently, routine imaging follow-up was not recommended.

Conclusion

Because of the rarity of GP, no formal evidence-based guidelines are currently available. Consequently, management should be individualized and, whenever possible, undertaken in centers with expertise in rare peritoneal diseases. Our case, together with the literature review, allows us to propose the following management strategy for GP:

- Histopathological confirmation by multiple peritoneal biopsies.
- A multidisciplinary approach to diagnosis and management.
- No indication for systemic therapy or chemotherapy in patients with exclusively mature lesions.

The diagnosis should be confirmed by histopathological examination of multiple biopsies obtained from lesions identified during laparoscopy. Extensive sampling and expert pathological evaluation are essential to exclude the presence of immature or malignant implants.

A multidisciplinary approach is essential to avoid misclassification. An important differential diagnosis is

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growing teratoma syndrome (GTS), which develops during or after chemotherapy for immature teratomas. It is characterized by enlarging mature benign masses that mimic malignant recurrence despite normal or declining tumor marker levels. This phenomenon, known as chemotherapeutic retroconversion, reflects eradication of the malignant components while the mature benign elements continue to proliferate. GTS is rare, particularly when associated with gliomatosis peritonei, with only approximately 20 cases reported in the literature [3]. In our patient, GTS was excluded because she had never received chemotherapy.
Current evidence supports fertility-sparing surgery whenever feasible, with chemotherapy reserved according to the grade of the primary teratoma. In patients with exclusively mature lesions, systemic therapy or chemotherapy is generally not indicated.

There is currently no consensus regarding the optimal long-term follow-up of patients with GP. Therefore, repeated invasive investigations or routine imaging should be avoided in asymptomatic patients whenever possible. Only one case of malignant transformation in GP associated with a mature teratoma has been reported, involving a mutation in NF1. In all other reported cases, no malignant transformation has been observed. Although MRI and CT were considered in our patient, routine imaging surveillance was not recommended because the lesions were not visible on the initial imaging studies and the risk of malignant transformation appears to be extremely low. Instead, annual gynecological follow-up, including clinical examination and ultrasonography, was recommended.

Conflict of interest statement

None declared.

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