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(CASE REPORT)

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Giant Immature ovarian teratoma with rupture and peritoneal dissemination mimicking carcinomatosis: Case report and brief literature review

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Abstract

Ovarian immature teratoma (IT) is a rare malignant germ cell tumor (GCT) that predominantly affects adolescents and young women and may present with rapid growth, rupture, and peritoneal dissemination, closely mimicking carcinomatosis. We report an 18-year-old nulligravid female who presented with a three-month history of progressive abdominal distension, pelvic pain, early satiety, weight loss, and dyspnea on exertion. Examination revealed a large abdominopelvic mass extending to the xiphoid process. Serum alpha-fetoprotein was markedly elevated (450 ng/mL), while beta-human chorionic gonadotropin (β-hCG) was normal. Contrast-enhanced CT demonstrated a large right adnexal solid-cystic mass with fat and calcifications, extensive peritoneal nodularity, omental caking, and hepatic surface implants.

The patient underwent fertility-sparing right salpingo-oophorectomy with comprehensive staging; intraoperative tumor rupture occurred, likely related to rapid tumor growth and compromised vascular supply. Histopathology confirmed a FIGO stage IIIC, grade 3 immature teratoma with immature neuroepithelium showing GFAP and Oct4 positivity and a high proliferative index (Ki-67 approximately 70%).

Following four cycles of bleomycin, etoposide, and cisplatin, complete remission with normalization of AFP was achieved. Recurrences involving the liver and peritoneum occurred at 28 months, necessitating secondary cytoreduction and second-line chemotherapy, after which the disease remained stable until loss to follow-up at 34 months. This case highlights the diagnostic challenges posed by ruptured giant immature teratomas, supports the feasibility of fertility-sparing management with BEP chemotherapy, and underscores the importance of long-term surveillance due to the risk of late recurrence.

Keywords: Ovary; Immature Teratoma; Germ Cell Tumor; Gliomatosis Peritonei; Peritoneal Carcinomatosis; Neuroepithelium

1. Introduction

Gliomatosis peritonei (GP), ruptured ovarian immature teratoma (IT) with peritoneal dissemination, and peritoneal carcinomatosis (PC) differ fundamentally in tissue composition, malignant potential, and clinical prognosis. They represent benign mature tissue seeding, malignant germ-cell spread, and epithelial cancer metastasis, respectively. [1,2]

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ITs are rare malignant ovarian GCTs that primarily affect adolescents and young women. [3,4] They are characterized by the presence of immature embryonic tissue, most commonly neuroepithelium, and their histologic grade is one of the strongest predictors of prognosis. [3] Patients typically present with abdominal pain, abdominal distension, or a rapidly enlarging pelvic mass. Diagnosis relies on imaging, serum tumor markers, and histopathologic examination. Treatment generally consists of surgical resection with fertility preservation when feasible, followed by platinum-based chemotherapy for advanced-stage disease. [3,4]

Although outcomes are generally favorable when diagnosed early, advanced diseases with tumor rupture and diffuse peritoneal spread remain uncommon and pose significant management challenges. We report the case of an 18-year-old woman with a giant grade 3 Ovarian IT complicated by spontaneous rupture, diffuse peritoneal dissemination, and hepatic surface implants. We report this case to highlight the aggressive presentation of advanced ovarian IT and the importance of early diagnosis, multidisciplinary management, and timely treatment in optimizing patient outcomes.

2. Case Presentation

2.1. Clinical Presentation

An 18-year-old nulligravid female presented with a three-month history of progressive abdominal distension, dull pelvic pain, and early satiety. She reported unintentional weight loss of 15 lb. over the preceding 2 months and increasing fatigue. The patient initially did not seek medical attention, but the rapid abdominal enlargement and new-onset dyspnea on exertion prompted evaluation. She had no significant prior medical history, had never undergone surgery, and her menstrual cycles were regular. There was no family history of ovarian or breast cancer. Her symptoms persisted despite over-the-counter analgesic use, leading to her emergency department visit.

2.2. Physical Examination, Laboratory, Imaging, and Differential Diagnosis

Physical examination revealed a firm, nontender, irregular abdominopelvic mass extending to the xiphoid process with visible abdominal distension. No ascites was clinically evident, and pelvic examination confirmed a palpable right adnexal mass. Laboratory studies demonstrated an elevated serum alpha-fetoprotein (AFP) level of 450 ng/mL, a normal β-hCG, and a mildly elevated CA-125 at 65 U/mL. Complete blood count and comprehensive metabolic panel were unremarkable.

Transabdominal and transvaginal ultrasonography revealed a large, predominantly solid, complex right ovarian mass measuring 18 cm with areas of calcification and cystic components, associated with moderate ascites and omental thickening. Contrast-enhanced CT of the abdomen and pelvis confirmed a 19 × 16 × 14 cm right adnexal mass with fatty and calcific densities, extensive peritoneal nodularity, omental caking, and several hepatic surface implants concerning for peritoneal carcinomatosis. Differential diagnoses included ovarian immature teratoma, primary epithelial ovarian carcinoma, gastrointestinal stromal tumor, or other germ cell tumor (e.g., dysgerminoma, yolk sac tumor). The combination of a large pelvic mass in a young woman with markedly elevated AFP strongly favored an immature teratoma.

2.3. Multidisciplinary Tumor Board Discussion

The case was presented at the multidisciplinary tumor board, which was attended by the gynecologic oncology, medical oncology, radiology, and pathology services. Discussion centered on the optimal surgical approach given the tumor's large size, the patient's age, and her strong desire for fertility preservation. The team recommended a fertility-sparing surgical approach via exploratory laparotomy with right unilateral salpingo-oophorectomy and comprehensive surgical staging, including peritoneal washings, infracolic omentectomy, and directed biopsies of peritoneal implants.

Complete cytoreduction was deemed essential as residual disease would require adjuvant chemotherapy. The board also addressed the surgical risk of intraoperative tumor rupture given its size and friable consistency. The board planned for immediate postoperative chemotherapy with bleomycin, etoposide, and cisplatin (BEP) given the high-grade and advanced-stage nature of the tumor. The patient was counseled regarding fertility preservation options prior to surgery.

2.4. Special Studies, Pathology, and Immunohistochemistry

Surgical exploration revealed a giant, solid-cystic right ovarian tumor with areas of necrosis and hemorrhage. Intraoperative tumor rupture occurred due to the mass outgrowing its blood supply, spilling its contents into the peritoneal cavity, and mimicking widespread carcinomatosis.

Peritoneal washings were positive for malignant cells. Histopathological examination of the tumor demonstrated immature neuroepithelial elements, primitive neural tubules, and immature mesenchymal tissue, consistent with a grade 3 immature teratoma (>3 low-power fields of immature neuroepithelial tissue per slide using Norris's grading system) (**Figure 1A, B, C, D**). Immunohistochemistry (IHC) showed diffuse glial fibrillary acidic protein positivity in the immature neuroepithelium, with a Ki-67 proliferation index of approximately 70%. Oct4 expression was also detected in the immature neuroepithelium, confirming the presence of high-grade, poorly differentiated components and supporting the diagnosis of a highly malignant immature teratoma. Surgical staging confirmed FIGO stage IIIC disease with positive omental and peritoneal biopsies, as well as hepatic surface implants.

2.5. Treatment, Outcome, and Follow-up

Postoperatively, the patient completed four cycles of BEP (Bleomycin, Etoposide, and Cisplatin) chemotherapy. At 18 months post-treatment, surveillance CT demonstrated complete radiographic remission, and serum AFP normalized to <5 ng/mL. However, at 28 months of follow-up, the patient presented with new-onset right upper quadrant pain and fatigue. Imaging revealed multiple new liver metastases and peritoneal implants consistent with disease progression. She underwent secondary cytoreductive surgery with resection of hepatic surface lesions and received second-line chemotherapy with vinblastine, ifosfamide, and cisplatin. She continues under surveillance with serial AFP monitoring and imaging every 3 months. She showed stable disease on her last follow-up visit 34 months from initial treatment, after which she was lost to follow-up.

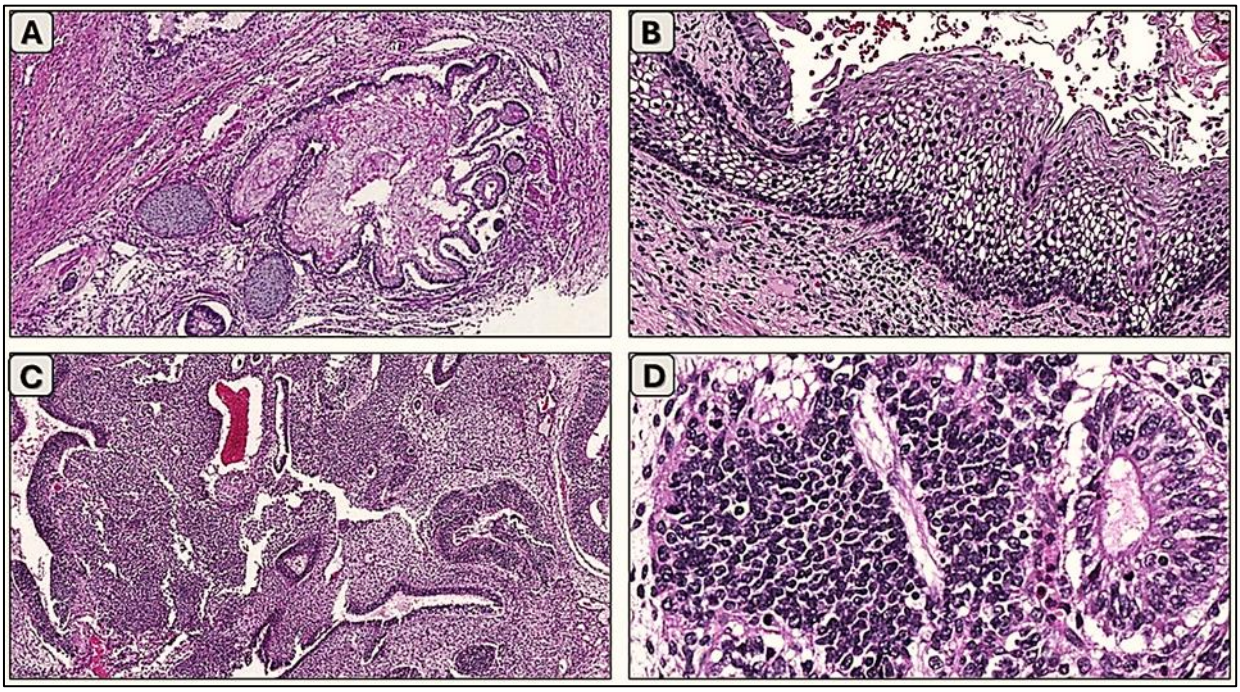

Figure 1 Microscopic features of mature elements and immature teratoma

- 1A**:** Low-power view showing mature glandular epithelium and mesenchymal elements (H&E X20)
- 1B**:** Intermediate-power view showing mature squamous and glandular elements (H&E X40)
- 1C**:** Low-power view showing immature primitive neuroepithelial elements (H&E X20)
- 1D: High-power view showing immature neural component with rosette formation (H&E X60)

3. Discussion

3.1. Background (History, epidemiology, risk factors, and WHO classification):

Ovarian IT is a malignant GCT arising from all three embryonic germ layers, distinguished from its benign counterpart by the presence of immature or embryonal tissue, most characteristically immature neuroepithelium in the form of primitive neural tubules and rosettes. [5] ITs are uncommon, accounting for roughly 1 to 3 percent of all ovarian teratomas and approximately 10 to 20 percent of malignant ovarian GCTs, ranking as the second most frequent

malignant germ cell tumor after dysgerminoma. [5,6] This stands in sharp contrast to the mature cystic teratoma, one of the most common ovarian neoplasms overall. [6]

The great majority of ITs arise in the first two decades of life, with peak incidence among adolescents and young women, a distribution that differs fundamentally from epithelial ovarian carcinoma, which predominates in the peri- and postmenopausal years. [3] Established risk factors remain largely undefined, and, unlike epithelial ovarian cancer, IT shows no consistent association with hereditary cancer syndromes such as BRCA-related disease. [6]

The tumor is graded by the quantity of immature neuroepithelium per low-power field, following the three-tier Norris system (grades 1 to 3), which the World Health Organization (WHO) classification of ovarian germ cell tumors incorporates within the teratoma category. [7] Higher grade correlates with a greater risk of extraovarian spread, a more advanced stage, and a poorer prognosis, and may accompany elevated serum alpha-fetoprotein. [7]

3.2. Pathogenesis, Pathophysiology

Two models explain how teratoma elements populate the peritoneal cavity after a breach or spill. The dissemination theory proposes direct mechanical seeding and extrusion of viable neuroglial/embryonal cells through a ruptured capsule. The metaplastic theory suggests that circulating growth factors prompt native, pluripotent submesothelial or Müllerian stem cells in the peritoneum to undergo direct neural/glial differentiation. [8,9,10,11] These giant tumors contain hypercellular, mitotically active primitive neuroectodermal tissues (such as immature glia and neuroblasts) which possess high proliferative potential when exposed to the peritoneal environment. [7,12]

Spilled immature elements or signaled stem cells frequently undergo "maturation arrest" or benign conversion, turning into mature nodules scattered across the omentum and peritoneal surfaces. [13,14] As the giant residual or seeded elements expand, they create localized compression, fibrous scarring, and dense chronic inflammatory adhesions between abdominal organs. [15] If the patient undergoes chemotherapy, malignant elements may regress while chemo-resistant mature teratomatous components paradoxically expand, resulting in a continuing mass effect despite normalized tumor markers (alpha-fetoprotein). [16]

3.3. Comparative Analysis of Our Case with Existing Literature

The published experience with IT spreading through the peritoneum falls into two distinct groups, and this case belongs to the more ominous one. Most reports of an immature teratoma "mimicking carcinomatosis" in fact describe GP the miliary implantation of benign mature glial tissue first characterized by Robboy and Scully in 1970. [17] Series such as the eight-case cohort published in the Journal of Ovarian Research in 2016 and the clinicopathologic study of 21 cases (median age approximately 19 years) confirm that GP typically follows an indolent course and does not itself worsen prognosis, despite an alarming intraoperative and radiologic appearance. [18,19]

Abbreviations

- Immature teratoma (IT)
- Germ cell tumor (GCT)
- Gliomatosis peritonei (GP;
- Peritoneal carcinomatosis (PC)
- Immunohistochemistry (IHC)

4. Conclusion, and What Have We Learned from This Case?

This case reinforces that serum alpha-fetoprotein is an indispensable early discriminator in a young woman presenting with a large, solid pelvic mass; its marked elevation redirected the working diagnosis toward a malignant germ cell tumor and away from the epithelial carcinoma suggested by the radiologic picture. That picture is the central pitfall. Fatty and calcific densities, omental caking, and hepatic surface implants can closely mimic peritoneal carcinomatosis, and disseminated teratomatous or ruptured tumor content compounds the illusion; therefore, imaging alone should not anchor the diagnosis in this age group.

The intraoperative course carries its own lesson: giant, necrotic, friable teratomas can outgrow their blood supply and rupture, seed the peritoneum and upstage the disease, which argues for meticulous surgical technique, avoidance of unnecessary manipulation, and comprehensive staging with washings, omentectomy, and directed biopsies whenever spillage occurs. The delayed hepatic and peritoneal recurrence at 28 months, after documented remission and normalization of AFP, is a reminder that high-grade, advanced-stage immature teratoma retains meaningful relapse

potential and demands sustained surveillance with serial AFP and imaging well beyond the first two years, rather than premature reassurance from an early complete response.

Compliance with ethical standards

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Disclosure of conflict of interest

All authors make the following declarations:

- Payment/services information: All authors have declared that they received no financial support from any organization for the submitted work.
- Financial relationships: All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might be interested in the submitted work.

Statement of ethical approval

This work did not involve any studies on human or animal subjects performed by the authors. It was a retrospective review of archival pathology material collected during routine clinical care, and all data were fully de-identified before analysis.

Statement of informed consent

The patient was lost to follow-up, and all attempts to reach the patient were unsuccessful. Therefore, the paper has been sufficiently anonymized to maintain patient confidentiality.

Data access statement

All relevant data are included in the paper.

Author contributions

All authors contributed equally to producing this manuscript.

References

[1] Fukuzawa T, Yamaki S, Irie M, Sasaki H, Kudo H, Nakamura M, Ando R, Okubo R, Endo Y, Hashimoto M, Tada K. Immature ovarian teratoma with pseudo-Meigs syndrome and gliomatosis peritonei. Journal of Pediatric Surgery Case Reports. 2022 Jun 1;81:102265.

[2] Gaillard F, Sharma R, Weerakkody Y, et al. Peritoneal metastases. Reference article, Radiopaedia.org (Accessed on Jul 28 2026)

[3] Marino G, Negri S, Testa F, Corti J, Giuliani D, Lugotti D, et al. *Immature Teratoma of the Ovary—A Narrative Review.* Cancers (Basel). 2025;17:3041.

[4] Alwazzan AB, Popowich S, Dean E, Robinson C, Lotocki R, Altman AD. *Pure Immature Teratoma of the Ovary in Adults: Thirty-Year Experience of a Single Tertiary Care Center.* Int J Gynecol Cancer. 2015;25(9):1616-1622.

[5] Travis LB, Feldman DR, Fung C, Poynter JN, Lockley M, Frazier AL. Adolescent and young adult germ cell tumors: epidemiology, genomics, treatment, and survivorship. Journal of Clinical Oncology. 2024 Feb 20;42(6):696-706.

[6] Norris HJ, Zirkin HJ, Benson WL. Immature (malignant) teratoma of the ovary. A clinical and pathologic study of 58 cases. Cancer. 1976 May;37(5):2359-72.

[7] Moraru L, Mitranovici MI, Chiorean DM, Coroș M, Moraru R, Oală IE, Turdean SG. Immature teratoma: diagnosis and management—a review of the literature. Diagnostics. 2023 Apr 23;13(9):1516.

[8] Bentivegna E, Gonthier C, Uzan C, Genestie C, Duvillard P, Morice P, Gouy S. Gliomatosis Peritonei: A Particular Entity With Specific Outcomes Within the Growing Teratoma Syndrome. International Journal of Gynecological Cancer. 2015 Feb 1;25(2):244-9.

[9] Li S, Su N, Jia C, Zhang X, Yin M, Yang J. Growing teratoma syndrome with synchronous gliomatosis peritonei during chemotherapy in ovarian immature teratoma: a case report and literature review. Current Oncology. 2022 Sep 4;29(9):6364-72.

[10] Zhang J, Ji H, Sun M, Wang J, Liu P, Feng L, Zhang P, Gao J. Ovarian Immature Teratoma with Gliomatosis Peritonei and Nodal Gliomatosis Metastasis: A Case Report. International Journal of Women's Health. 2025 Dec 31:5269-75.

[11] Anderson JE, Taylor MR, Romberg EK, Riehle KJ, Kapur R, Crocker ME, Crotty EE, Hergenroeder G, Greenberg SL. Mature mediastinal teratoma with tumor rupture into airway. Journal of Pediatric Surgery Case Reports. 2022 Jun 1;81:102270.

[12] Choi JH, Ro JY. Retroperitoneal sarcomas: an update on the diagnostic pathology approach. Diagnostics. 2020 Aug 27;10(9):642.

[13] Tarang P, Virendrakumar M. Gliomatosis peritonei and its relation to teratoma: role of imaging and histological aspects. Cureus. 2022;14(9).

[14] Türkmen O, Akar Inan S, Akbay S, Halilzade MI, Halilzade İ, Moraloğlu Tekin Ö. Coexisting growing teratoma syndrome and gliomatosis peritonei following ovarian immature teratoma: a case report and literature review. Turkish Journal of Medical Sciences. 2024;54(6):1192-7.

[15] Bahall V, De Barry L, Sookdeo R, Barrow M. Ovarian teratoma presenting with gliomatosis peritonei and a melange of symptoms mimicking ovarian cancer in a pediatric patient. Cureus. 2023 Dec 5;15(12).

[16] Song Y, Ge J, Wang J. Clinical insights from the management of a giant immature teratoma in an infant—a case report. AME Case Report. 2025 Jan 1;9:101.

[17] Robboy SJ, Scully RE. Ovarian teratoma with glial implants on the peritoneum: an analysis of 12 cases. *Human Pathology*. 1970;1(4):643-653.

[18] Wang D, Jia CW, Feng RE, Shi HH, Sun J. Gliomatosis peritonei: a series of eight cases and review of the literature—Journal of Ovarian Research. 2016 Jul 29;9(1):45.

[19] Liang L, Zhang Y, Malpica A, Ramalingam P, Euscher ED, Fuller GN, Liu J. Gliomatosis peritonei: a clinicopathologic and immunohistochemical study of 21 cases. Modern Pathology. 2015 Dec 1;28(12):1613-20

[20] Kumar A, Goyal P, Sehgal S, Ghosh S, Kumar V, Agrawal D, Singh S. Giant Immature Teratoma of the Ovary with Gliomatosis Peritonei in a 15-Year-Old Girl: A Case Report and Literature Review. Journal of Gynecologic Surgery. 2014 Aug 1;30(4):222-6.

[21] Pashankar F, Hanley K, Lockley M, Stoneham S, Nucci MR, Reyes-Múgica M, Elishaev E, Vang R, Veneris J, Rytting H, Olson T. Addressing the diagnostic and therapeutic dilemmas of ovarian immature teratoma: Report from a clinicopathologic consensus conference. European Journal of Cancer. 2022 Sep 1;173:59-70.

[22] Pashankar F, Hale JP, Dang H, et al. Is adjuvant chemotherapy indicated in ovarian immature teratomas? A combined data analysis from the Malignant Germ Cell Tumor International Collaborative. *Cancer*. 2016;122(2):230-237.