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Growing teratoma syndrome in pediatrics

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ABSTRACT

Growing teratoma syndrome (GTS), described by Logothetis in 1982, is characterized by the appearance of benign tumor lesions during or after chemotherapy for germ cell tumors, with normalized tumor markers. It is rare in pediatrics; in Mexico, there have been no previous reports of this condition in girls.

We present an 8-year-old girl with abdominal distension, colicky pain, and constipation; a CT scan revealed a mass in the right ovary, a liver lesion, and intra-abdominal fluid. Cytoreductive surgery revealed an immature teratoma with neuroglial implants. She was diagnosed with stage IV mixed germ cell tumor; chemotherapy was initiated. Given the persistence of a residual mass with normalized tumor markers, laparotomy confirmed mixed germ cell tumor. Since complete resection was not possible, close monitoring was decided upon; she remains stable at 11 months.

GTS should be considered in pediatric germ cell tumors with tumor growth and normalized markers. Surveillance is a valid alternative in unresectable cases.

Keywords: *germ cell and embryonic neoplasms; ovarian neoplasms; teratoma; adjuvant chemotherapy; pediatrics.*

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INTRODUCTION

Growing teratoma syndrome (GTS), described by Logothetis[1] in 1982, is a little-known condition characterized by the appearance of benign tumor lesions during or after chemotherapy for germ cell tumors. Its exact incidence is unknown; however, it has been estimated at up to 17.8%, and it primarily affects the ovary and retroperitoneum.

In pediatrics, it is rare and usually occurs in school-aged girls and adolescents.[2] This contrasts with the low incidence of ovarian tumors in this population, estimated at 2.2 per 100 000 girls aged 0 to 15, of which only 1% are malignant. In adults, it has been reported in nonseminomatous testicular tumors (1.9% to 7.6%) and in ovarian tumors (12% to 20%).[3]

Due to limited knowledge of this condition, it is often misinterpreted as disease progression. The etiology and pathogenesis are not well defined, and although tumor markers commonly return to normal levels, some malignant tumors never become elevated. GTS is characterized by resistance to chemotherapy and radiation therapy; therefore, complete surgical resection is the only curative treatment.[4]

We present a pediatric case of ovarian GTS with an unusual clinical course due to the inability to perform a complete resection of the tumor and treatment via close monitoring.

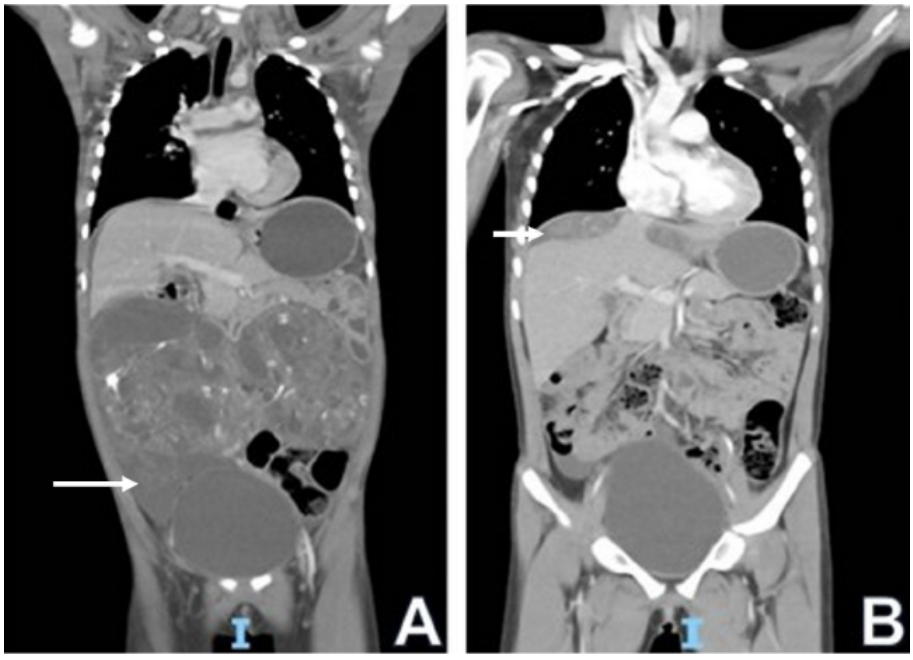
CLINICAL CASE

An 8-year-old girl presented with progressive abdominal distension lasting 20 days, colicky pain in the hypogastrium radiating to the right iliac fossa, and constipation. On physical examination, a stone-like, painless mass with poorly defined borders was palpated in the hypogastrium. Given the high suspicion of malignancy, an initial ultrasound was performed, which revealed a pelvic mass attached to the right ovary.

An abdominal CT scan revealed an abdominopelvic mass measuring 10.2 × 12.5 × 11.7 cm, a subcapsular lesion in the right liver, thickening of the right hemidiaphragm, and intra-abdominal fluid (*Figure 1*). Initial tumor markers were ordered: alpha-fetoprotein (AFP) 639.4 ng/ml and beta-human chorionic gonadotropin (β-hCG) <1.64 mIU/ml, consistent with a primary ovarian germ cell tumor with a probable endodermal sinus component.

Cytoreductive surgery with right salpingo-oophorectomy was performed; a right ovarian tumor with retroperitoneal implants in the abdominal cavity was found (*Figure 2*).

Figure 1. Contrast-enhanced computed tomography of the abdomen, coronal view



A) Initial study. Primary tumor of the right ovary (arrow).
B) Follow-up scan after chemotherapy. Liver metastasis (arrow).

Histopathological examination revealed an immature ovarian teratoma with mature neuroglial implants and an endodermal sinus component in the right and left paracolic gutters; the peritoneal fluid was negative for neoplastic cells. A diagnosis of a primary mixed germ cell tumor of the right ovary, stage IV due to liver metastasis, was established.

Four cycles of CISCA II chemotherapy (doxorubicin, cyclophosphamide, and cisplatin) were administered every 28 days. At the evaluation before the fourth cycle, the tumor markers had returned to normal: AFP 7.58 ng/ml and β-hCG <1.20 mIU/ml.

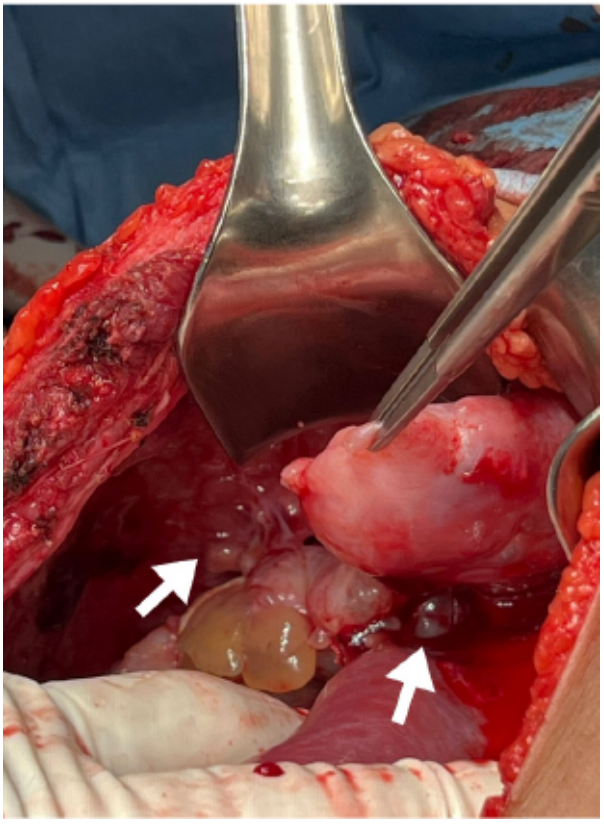
At the end of treatment, the CT scan showed that the tumor was still present, so a second exploratory laparotomy was performed. Multiple unresectable peritoneal and subdiaphragmatic implants were documented. Histological analysis confirmed a mature teratoma with peritoneal gliomatosis, consistent with GTS (*Figure 3*).

Given the disease's resistance to chemotherapy and the impossibility of complete resection due to the extent of the tumor, it was decided to discontinue chemotherapy and begin close monitoring. At 11 months, the patient remains stable, with no clinical progression or appearance of new lesions.

DISCUSSION

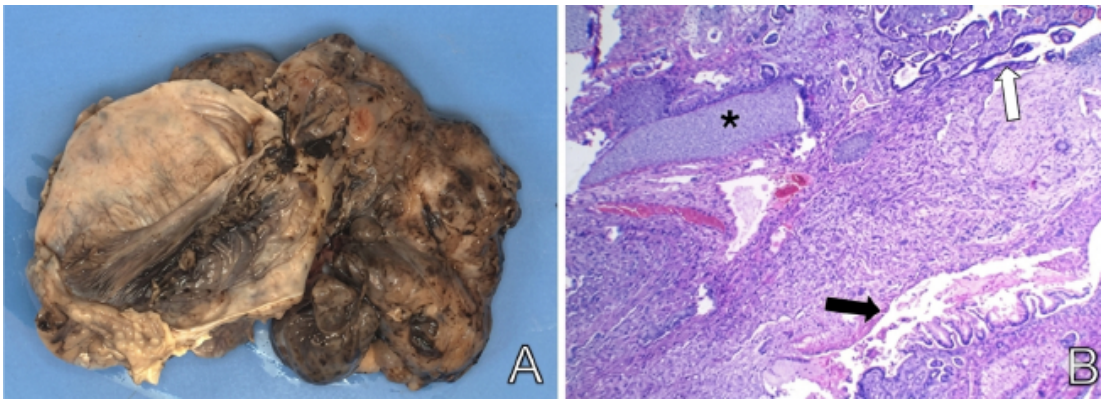
GTS poses a diagnostic challenge due to its similarity to neoplastic progression, which can lead to extensive surgical procedures and unnecessary chemotherapy. The key to diagnosis is the classic triad: tumor growth following chemotherapy, normalization of serum tumor markers, and histology consistent with a mature teratoma. The normalization of tumor markers occurs because chemotherapy eliminates the malignant components of the mixed germ cell tumor, leaving the mature teratoma, which continues to grow.[5]

Figure 2. Second-look surgery following chemotherapy



The white arrows indicate peritoneal implants.

Figure 3. Immature teratoma of the right ovary



A) Macroscopic. Specimen from an oophorectomy, showing a cystic area and a heterogeneous outer surface with smooth, opaque solid areas.
B) Microscopic. Histological section at 10× magnification, hematoxylin-eosin stain showing secretory epithelium (black arrow), mature cartilage (asterisk), and fragments of neuroepithelium (white arrow).

Most cases are reported in adolescents and young adults; in pediatrics, it is rare. The incidence has been estimated to be between 12% and 19% in patients with immature ovarian teratomas, with tumor growth that may occur even months after the initial surgery. In children, progression to GTS appears to occur more rapidly than in adolescents or adults.[2,6]

The reports available in the literature predominantly involve adult or adolescent patients. Pérez Gordillo et al.[7] described the case of an 18-year-old woman with a history of an immature teratoma of the right ovary, who developed retroperitoneal and hepatic lesions confirmed as a mature teratoma following resection. Scavuzzo et al.[8] documented two cases in adult males, both with non-seminomatous testicular tumors and a clinical course consistent with STC following chemotherapy. In contrast, our 8-year-old patient represents a pediatric case with an unusual clinical course, given that the inability to perform a complete tumor resection led to the decision to opt for close follow-up.

Internationally, Tao et al.[9] reported a case in China of ovarian GTS in a 20-year-old woman with multiple peritoneal implants following chemotherapy. The patient underwent complete resection via fertility-sparing surgery, with a favorable outcome and no evidence of disease at three months. Both cases share the classic diagnostic triad of GTS: tumor growth following chemotherapy, normalized tumor markers, and mature teratoma histology; however, they differ in terms of treatment. While complete resection led to a favorable outcome in that case, it was not possible in our patient.

The treatment for GTS remains surgical, as neither chemotherapy nor radiation therapy has proven effective. Bentivegna et al.[10] noted that chemotherapy can induce the “retroconversion” of immature cells to mature cells, but does not eradicate residual lesions. This is consistent with the course observed in our patient, in whom no benefit was evident after the administered cycles. The prognosis for GTS is generally favorable, with five-year survival rates close to 90% and a recurrence rate of less than 4% following complete resection. In cases of partial resection, the recurrence rate can reach 83%, underscoring the importance of complete surgery as the treatment of choice. Furthermore, although malignant transformation is rare (3% to 5%), it requires long-term follow-up.[9] Recurrences can occur even up to ten years after initial treatment, making long-term follow-up necessary.

In terms of spread, GTS most commonly affects the retroperitoneum and the abdominal cavity, although retrocrural (14%), pulmonary (6%), hepatic (6%), and cervical (4%) metastases have been reported. It has been observed that cases with rapid tumor growth tend to show a greater capacity for spread to other organs, which is associated with a less favorable prognosis.[11] In GTSs, tumor markers such as alpha-fetoprotein (AFP), beta-human chorionic gonadotropin (β-hCG), and lactate dehydrogenase (LDH) play

a limited role. Generally, they are elevated only when the germ cell tumor is mixed and contains components other than mature teratoma, such as yolk sac tumor, choriocarcinoma, or embryonal carcinoma. During chemotherapy, these malignant components are eliminated, leaving only the mature teratoma, which continues to grow despite the normalization of tumor markers. Therefore, these markers are not useful for the diagnosis or prognosis of GTS; however, monitoring them helps rule out malignant recurrences associated with other histological subtypes.[3]

In cases where resection is not possible, such as the one presented here, close monitoring is a valid strategy as long as tumor markers remain negative and there is no clinical progression. Furthermore, timely recognition of this syndrome helps avoid unnecessary treatments, such as additional cycles of chemotherapy, which do not alter the course of the disease and expose patients to significant adverse effects.[6,10]

In conclusion, this first Mexican pediatric report on ovarian GTS highlights the importance of timely identification of the diagnostic triad to distinguish it from tumor progression. It supports close monitoring as a valid alternative when complete resection is not feasible and tumor markers remain normal. ■

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