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Case Report

Ovarian Carcinosarcoma: Case Report and Literature Review

Fernando Solarz, Janeth Lara Alcántara*, Iara Izquierdo M, M Florencia Arabena Bilbao, Facundo Salazar Molina, M Paula Pinto, Johanna Costa and Patricio Servienti

Oncology Service, General Acute Care Hospital, Dr. Teodoro Álvarez, Buenos Aires, Argentina

***Corresponding Author:** Janeth Lara Alcántara Oncology Service, General Acute Care Hospital, Dr. Teodoro Álvarez, Buenos Aires, Argentina.

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Abstract

Ovarian carcinosarcoma (OCS), also known as malignant mixed Mullerian tumor (MMMT), is a rare and aggressive tumor, accounting for less than 3% of ovarian cancers. It has a high morbidity and mortality rate, with an overall survival rate of less than two years.

There are factors that can increase the risk of OCS, as well as factors that contribute to a poor prognosis.

We present the case of a patient recently diagnosed with OCS and review the literature.

Introduction

Ovarian carcinosarcoma (OCS) is defined as a rare and highly aggressive epithelial neoplasm; it is also described as biphasic because it exhibits both mesenchymal and malignant epithelial components. Only 10% of cases are bilateral, and it affects postmenopausal women, with a mean age of presentation between 60 and 70 years.

Ovarian carcinosarcomas must be distinguished from pure ovarian sarcomas. OCSs are biphasic malignant neoplasms composed of high-grade carcinomatous and sarcomatous elements; they are classified into homologous and heterologous subtypes. The former comprises native ovarian tissue and consists of endometrial stromal sarcoma, fibrosarcoma, or leiomyosarcoma. Heterologous elements are tissue not native to the ovary, including bone, adipose tissue or striated muscle.

Clinical case

A 56-year-old female with a history of menopause at age 45. She presented to the emergency department with lower abdominal pain associated with constipation and dysuria of 10 days' duration. On physical examination, the abdomen was distended, depressible, and tender to deep palpation in the hypogastric region, with no signs of peritonitis. A hard, stony, mobile mass is palpable in the left flank. An abdominal ultrasound reveals a large, heterogeneous mass in the pelvic cavity, with a mixed, predominantly solid echotexture measuring approximately 160 mm × 110 mm × 160 mm; free fluid is present perihepatically, perisplenically, in both paracolic gutters, and surrounding the previously described mass in the hypogastric region. On gynecological examination, speculoscopy was normal, and on vaginal examination the impression of the abdominal mass was palpable. An ovarian tumor is suspected.

A transvaginal ultrasound was performed, revealing a heterogeneous uterus, enlarged to 180×97×80 mm, with multiple mixed lesions, the largest measuring 57×46 mm and exhibiting scant Doppler flow; the findings are suggestive of a neoplastic process, and the adnexa are not visible. Computed tomography with ascites: a large, mixed-density mass with heterogeneous enhancement, measuring 17×14×14 cm, appears to be of adnexal origin and extends from the pelvis into the abdomen, exerting mass effect on adjacent structures. The blood test shows a CA-125 level of 655 IU/mL.

An exploratory laparoscopy was performed, during which a large mass originating in the left ovary and adherent to the left mesocolon was found, along with multiple peritoneal implants on the liver capsule, both diaphragmatic domes, "omental cake," and at the vesicouterine fold. Since primary cytoreduction was not possible, multiple biopsies and ascetical fluid samples were taken.

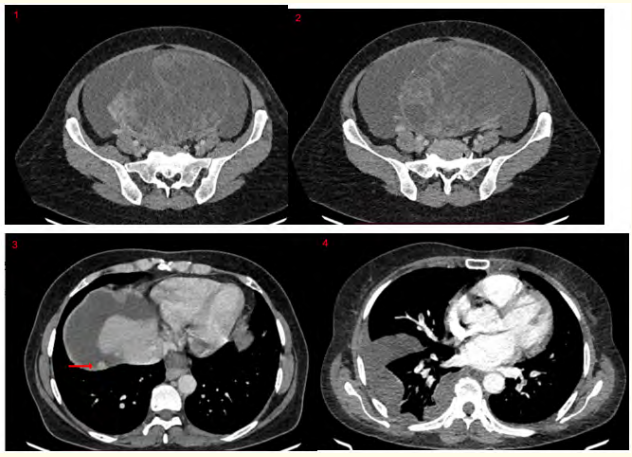
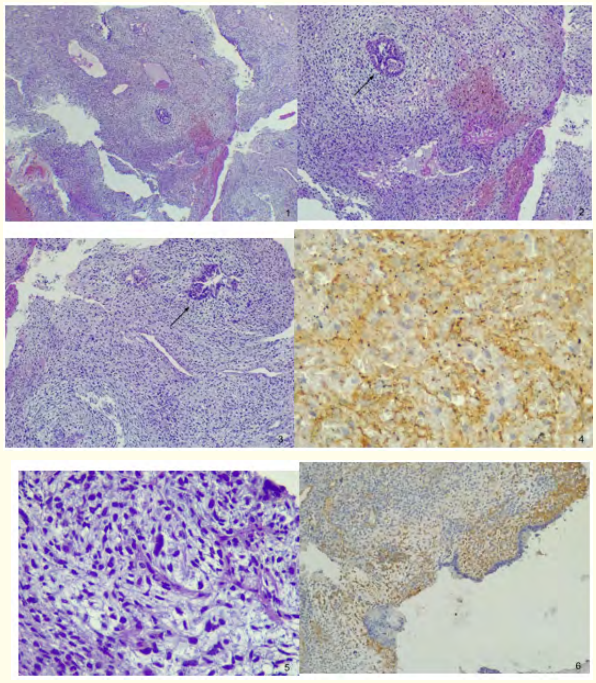


Image a: Axial tomography images: 1, 2: voluminous ovarian mass measuring 160x110x160 mm associated with ascites. 3: subdiaphragmatic peritoneal implant (arrow) 4: pleural effusion.

Pathological anatomy

Histopathological analysis of the biopsies showed atypical spindle cell proliferation including isolated tubular elements with extensive areas of haemorrhage and necrosis. Ascetical fluid showed abundant cells with anisokaryosis and cytoplasmic vacuoles. Pathological Immunohistochemistry demonstrated: ER (-), SMA (+), CK7 (+ focal), inhibin (-), PAX8 (-), desmin (+ focal), WT-1 (-), AE1/AE3 (-), vimentin (+ focal), DOG1 (-), CD117 (-), calretinin (-), chromogranin A (-), S100 (-), CD34 (+ focal), Ki67 (20%). Immunophenotype consistent with carcinosarcoma. (Biphasic atypical cell population composed of sarcomatous (90%) and carcinomatous (10%) elements, with clear demarcation between the two.).

Six days after surgery, the patient developed venous thrombosis of the right small saphenous vein and the left popliteal vein due to tumoral compression of venous return, requiring hospitalization and anticoagulation with enoxaparin. During hospitalization, she developed ascites and oedema syndrome requiring multiple therapeutic paracenteses. The disease was initially staged FIGO IVa due to pleural effusion without pathological confirmation. The patient had a performance score ECOG 2 and it was indicated to initiate cancer-specific treatment with carboplatin and paclitaxel chemotherapy for six cycles as definitive therapy. Bevacizumab could not be added to the therapeutic scheme due to thrombosis.



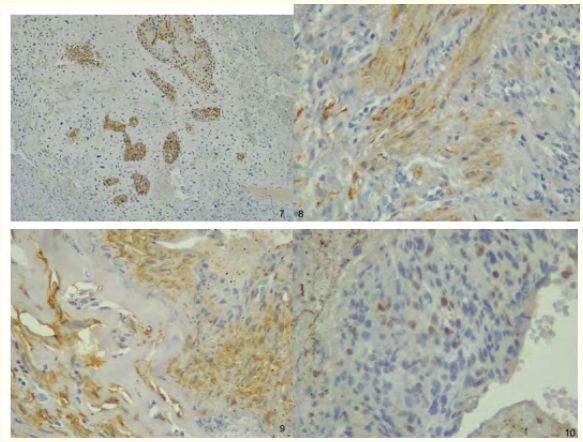


Image b: (1) H&E tumoral 4x picture. (2) y (3) H&E 10x, predominant sarcomatous element next to carcinomatous áreas (black arrow) (4) SMA Staining (5) H&E 20x, high grade sarcomatous element. (6) CD 34 (7) CK7 staining showcases carcinomatous elements. (8) Desmin staining. (9) Vimentin. (10) Ki67.

Discussion

OCS is an uncommon variant within the spectrum of ovarian tumors, accounting for less than 3% of malignant adnexal tumors [1]. They are biphasic neoplasms characterized by the presence of carcinomatous and sarcomatous elements, and are classified as homologous or heterologous based on the histology of the sarcomatous component [2].

There are multiple theories regarding the pathogenesis of this disease, including the collision theory, which posits two distinct neoplasms; the combination theory, which suggests that the tumor originates from pluripotent cells that subsequently differentiate into sarcomatous and carcinomatous elements; and the conversion theory, which proposes a purely carcinomatous origin with subsequent epithelial–mesenchymal transition [3]. Molecular studies of the tumor genome in 18 cases of ovarian carcinosarcoma conducted by Ho., *et al*. [4]. revealed a monoclonal origin of the tumor, thereby ruling out the collision theory. Additionally, they observed a high degree of epithelial transition mesenchymal in the cells of the carcinomatous component, which supports the postulate of the conversion theory.

OCS is a disease with a poor prognosis, with a 5-year survival rate of 28.2% compared to 38.4% for high-grade serous carcinoma [5], and poor prognostic factors such as advanced age at diagnosis, suboptimal cytoreduction, and diagnostic stage are consistent with those of other ovarian tumors [6].

We found no randomized clinical trials that included OCS; therefore, treatment recommendations are based on retrospective studies. Different series compared the response of clear cell ovarian carcinoma (CCOC) to platinum-based treatment with that of high-grade serous carcinoma and observed a lower response rate [7]. We know that platinum sensitivity is a prognostic factor for ovarian cancer, so this could contribute to the unfavourable outcome of clear cell ovarian carcinoma.

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