Uterine carcinosarcoma with intestinal involvement: A case report and literature review

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Abstract. Uterine carcinosarcoma (UCS) is a high-grade endometrial cancer characterized by two components: Carcinomatous (epithelial) and sarcomatous (stromal tissue) elements. The present study describes a clinical case of this type of UCS and also provides a brief literature review of this type of tumor. A 72-year-old female visited the emergency department of the authors' hospital with pain in the hypogastric region, intestinal dysrhythmia since 3 months prior, fever and a palpable abdominal mass. Laboratory test results revealed sepsis and mild anemia and an imaging test revealed a large uterine tumor with wide areas of necrosis and adenopathies. As determined by the multidisciplinary committee, surgery was considered the main treatment option and this was performed with no incidences. Carcinosarcoma is a rare tumor, which most frequently occurs in older women. The diagnosis is based on symptoms and imaging tests, such as ecography and scans. The gold standard of treatment is surgery, although it is possible that other types of therapies, such as chemotherapy and radiotherapy may also be effective, depending on the tumor stage. On the whole, the prognosis of patients with this type of tumor is poor, with a low survival rate, even in earlier stages due to its malignant component and the possibility for metastasis. Surgery is the optimal treatment for this type of tumor, if this is possible, always individualizing patients.

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Introduction

Uterine carcinosarcoma (UCS) is a high-grade endometrial cancer characterized by a carcinomatous component (epithelial) and sarcomatous (stromal tissue) element (1). UCS is considered a rare tumor; however, its incidence is increasing (1). UCS is considered an aggressive tumor due to its components and it can exhibit metastasis at the time of diagnosis in many cases (2).

The present study describes the case of a patient with this type of tumor treated at the authors' hospital (2020-2021) and also provides a brief review of the published literature up to December, 2022, with the aim of offering an overview of the pathology, presentation and management of UCS from the point of view of General Surgery.

The limitation of the present study was there is not much homogeneity in UCS treatment as this is a rare tumor and has an aggressive pathology. In the search of published literature using PudMed, no article on UCS with intestinal involvement was found.

Case report

A 72-year-old female patient with no history of any serious conditions, visited the emergency service at Principe de Asturias Hospital due to a fever of 38˚C with 24 h of evolution, rectal bleeding, vesical tenesmus with an evolution of 3 months and occasional fecal incontinence that had become more severe over the last few days. During her stay in the emergency room, the patient was hemodynamically stable.

A physical examination revealed a soft and depressible abdomen, with a painful area in the hypogastric region, where a huge palpable mass was located. Laboratory test results revealed the following: Leukocytes, 12.600 μl; neutrophils, 90%; hemoglobin, 11 mg/dl; C reactive protein (CRP), 313 mg/l; procalcitonin, 8.15 ng/ml; lactate, 3.1 mg/dl; hematuria, leukocyturia and bacteriuria for *Escherichia coli*.

Computed tomography (CT) of the abdomen and pelvis with intravenous contrast demonstrated a large solid polylobulated mass with peripheral enhancement with the intravenous contrast and wide areas of necrosis. The mass was contacting,

compressing and moving the rectus and sigmoid colon to the posterolateral side with no intestinal obstruction signals. The tumor measured 12.5x8.23x8.87 cm (anteroposterior x transverse x craniocaudal diameters). There was a small edematous zone in the presacral fatty tissue, two small calcifications in the right mass zona and a significant growth in the vascular are around the described injury. Left paraaortic nodes with abnormal size, and a small adenopathy near the iliac bifurcation and around the fatty tissue of the mass were observed. The bladder and uterus were displaced anteriorly, and the ovaries were not visible (Fig. 1).

An ultrasound of the pelvis revealed a solid mass of 10 x6 cm in size in the pouch of Douglas with intense Doppler imaging. Due to the worsening of the patient's general condition and the laboratory test results during her hospitalization 4 days later, the authors' requested to perform an evaluation of the patient. A new CT scan was performed to compare the new findings with the previous ones. This also revealed a solid pelvic mass with wider areas of necrosis and pneumoperitoneum. Traces of free fluid were found in the pelvis and between the intestines. In a 3.5 cm segment of the jejunum, inflammation and and/or focal ischemia were observed (Fig. 2).

Immediate surgery was performed with medium laparotomy with intraoperative findings of purulent free fluid, small intestinal plastron that encompassed the superior rectus, left ovary and fallopian tube. A complete mesorectal dissection was made with the mass mobilization of the left ovary and tube. An oncological ultralow anterior resection and colostomy in left iliac fossa were performed with and appropriate splenic angle descent. There were no intraoperative and postoperative incidents.

The results of the pathological analysis revealed the following: Carcinosarcoma (mixed malignant Mullerian tumor) of probable ovarian origin that involved all the intestinal wall with large areas of necrosis. The resection margins were not assessable. Ki67 positivity was 90%, and immunohistochemical analysis (performed by the anatomic pathology service) yielded positive findings for CK AE1AE3, CK7, p63, Pax8, CD10 and vimentin (data not shown).

The clinical case was evaluated by the multidisciplinary oncological committee for oncological surgery enlargement, although after surgery, the patient was revaluated to determine whether she was a candidate for chemotherapy. Intraoperatively, there was a bleeding mass in the small intestine of 5 cm in size in the left iliac fossa. There are several tumoral implants in the omentum, intestine and presacral region. A simple hysterectomy with the excision of the right ovary was made, and also the resection of hepatic angle mesentery, transverse colon mesentery, the complete resection of the greater omentum, terminal colostomy of the antimesenteric border, and the resection of the injury in the proximal jejunum. Multiple lesions were observed in the terminal ileum; thus, a resection of 50 cm was made and 20 cm of the distal jejunum with all adenopathies visualized. Latero-lateral manual biplane anastomosis was performed between the terminal ileum and distal jejunum. In addition, there was an tumoral implant in the iliac artery bifurcation and left iliac vein, and thus there was a high risk of vascular damage. An R2 resection was made with a peritoneal carcinomatosis index of 18/39.

All the dissected sections were sent to the anatomic pathology service for analysis, which revealed carcinosarcomatous infiltration and 16 nodes of the ileum and 2 of the jejunum were reactive. The patient did not experience any incidences post-operatively, and was thus discharged from the hospital 9 days later.

After 1 month, the patient visited the emergency service with abdominal pain, a fever of 38.4˚C, with no functioning colostomy and peritoneal irritation. An examination revealed the following: Leukocytes, 13.100 μl; neutrophils, 90%; hemoglobin, 9.7 g/dl; CRP, 98 mg/l. A CT scan revealed cancer progression with new hepatic lesions in the VIII segment 2.6 cm in diameter, several tumoral implants in the sacral region next to the bladder, in the right iliac fossa, retroperitoneum (anterior to left psoas muscle) and between the small intestine. Following a consensus with the patient and her family, palliative care was commenced and in the following days, the patient succumbed to her condition.

Discussion

UCS is considered a rare tumor, comprising ~5% of endometrial cancer cases; however, its incidence is increasing (1). The annual incidence of UCS is 0.5-3.3 cases per 100,000 females (2). European statistics have revealed an incidence of 5.1-6.9 cases per 1.000.000 individuals per year, between the years 1989-2008 (1). UCS mainly affects individuals of 70-79 years of age, although in recent decades, younger patients have also been identified (1).

Women of African origin have the highest risk of developing this type of cancer, even though in recent years, the incidence among Hispanic women has increased compared with that in other races (1). UCS is considered to derive from a dedifferentiated sarcoma component (1). Recent research has demonstrated that carcinomatous and sarcomatous elements are derived from a common precursor with mutations that are typical of carcinomas (3).

A significant discovery was that epithelial-mesenchymal transition (EMT) plays a pivotal role in the pathogenesis of sarcomatous dedifferentiation and that heterologous sarcoma is associated with a higher EMT signature compared with homologous sarcoma (1). Mutations and genes in this type of tumor have not been extensively studied. The most common of these are probably PT53, PTEN and FBXW7 (1). The disease can also exhibit chromosomal instability and a complex karyotype (2).

Traditionally, UCS was considered a sarcoma, being the most frequent uterus sarcoma. It is also known as a malignant mixed mesodermal tumor or a mixed Mullerian malignant tumor (2). The carcinomatous epithelial element can be of low or high grade, while the sarcomatous part can be heterologous/no gynecological tissue (rhabdomyosarcoma, chondrosarcoma and osteosarcoma) or, homologous/gynecological tissue (leiomyosarcoma, fibrosarcoma and endometrial stromal sarcoma) (1,2).

The most frequent element is carcinomatous, highlighting high grade endometrioid and serous carcinoma (2). This type of element is the tissue that tends to metastasize and appeal (2). Carcinoma with no dominant homologous sarcoma is the most prevalent type of UCS (1). The main risk factors for UCS are presented in Table I.

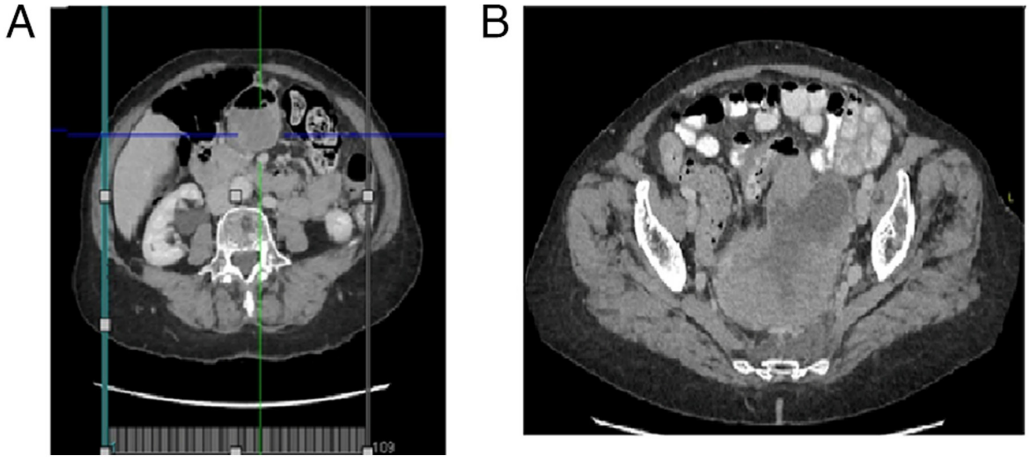

Figure 1. Computed tomography of the pelvic mass. (A) Tumor with intestinal involvement and adenopathies. (B) Polyglobulated mass in the pelvis with necrosis areas.

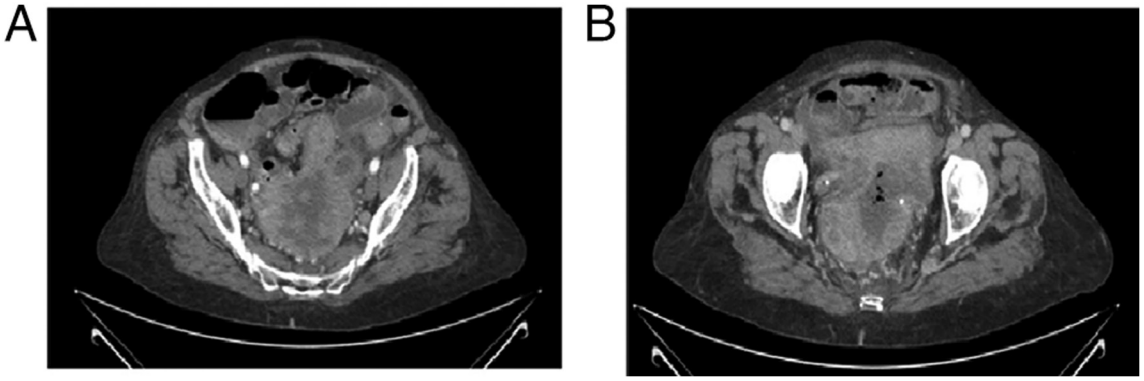

Figure 2. Posterior computed tomography scan revealing the worsening clinical condition of the patient 4 days later. (A and B) Pelvic mass with a wide necrotic mass in two different CT pelvic levels.

Clinically, UCS is similar other uterine adenocarcinomas, with symptoms such as vaginal bleeding (most common symptom), abdominal pain and a large uterus (2,4). It may present as a pelvic mass shown on an imaging test or as a prolapsed cervix (2). Due to this malignancy, the extrinsic compression of the bladder or intestines may occur, leading to urinary retention, bowel obstruction, constipation and tenesmus (5). Approximately 10% of patients exhibit metastasis at the time of diagnosis (4) and 30-40% are positive for adenopathies (6). Metastasis can occur in the lungs (49%), peritoneum (44%), bones (17%), liver (15%) and central nervous system (7).

The diagnoses of UCS is based on laboratory and imaging tests. Although there is no specific laboratory test to indicate this condition, ~10% of patients can present anemia due to vaginal bleeding (3). Ca125 levels can be elevated due to serous epithelial tissue and deep myometrial invasion (2).

The first step for imaging tests is a pelvic ultrasound, which may reveal hyperechoic lesions which are difficult to differentiate from adenocarcinoma (4). As patients frequently exhibit an extra-uterine pathology, CT scan is recommended for classification and/or magnetic resonance (MR) imaging prior to surgery (2). However, a CT scan is more effective than MR imaging for adenopathies (5). Positron emission tomography is more sensitive (68 vs. 50%), but less specific (88 vs. 93%) than MR imaging to detect adenopathies (7). The definitive diagnosis is anatomopathological (4). Due to the high risk of micrometastasis, a lymphadenectomy with posterior analysis is the gold standard (7).

Table I. Risk factors of uterine carcinosarcoma.

Risk factors for UCS (2)
• Elevated levels of estrogens
• Nulliparous
• Obesity
• African race
• Tamoxifen use
• Pelvic radiation

The number in parentheses denotes a reference.

The stages of carcinosarcomas are similar to those of the endometrial carcinoma system (5). The stages of stages of UCS

Table II. Stages of uterine carcinosarcoma (8).

Carcinosarcoma stage		
Stage	Description	Subgroup
I	Body uterine tumor	• IA: <50% myometrial invasion • IB: >50% myometrial invasion
II	Cervical stroma is invaded by tumor but no across uterus	
III	Local and/or regional dissemination	• IIIA: Serous and/or annexes • IIIB: Vagina and/or parametrium • IIIC1: Pelvic adenopathies • IIIC2: Paraaortic adenopathies
IV	Bladder, intestine invasion or metastasis	IVA: Bladder and/or intestine IVB: Metastasis or inguinal adenopathies

The number in parentheses denotes a reference.

are listed in Table II and in TNM staging (AJCC UICC 2017) is presented in Table III (8). Although treatment is multimodal, the prognosis of patients is poor, with a median survival rate of <2 years (1). More than half of the cases are diagnosed at an early stage, while the other half at an advanced stage (stage I, 43.9%; stage II, 8.7%; stage III, 22.9%; and stage IV, 24.4%) (1). The survival for rates of patients with different stages of the disease are generally as follows: Stage I, 78 months; stage II, 30 months; stage III, 19 months; and stage IV, 8 months (1). Factors associated with patient prognosis are presented in Table IV.

The concept of sarcomatous dominance is associated with a >50% decrease in survival (1). The worse survival subtype is carcinoma with heterologous sarcoma (1). Lymphatic invasion in recent years has increased the incidence of distant metastasis (1). The carcinoma element can metastasize to distant zones, while the sarcomatous element to regional zones (1). Approximately 60% of cases have disseminated illness, and 50% of tumors are treated using surgery and adjuvant therapy (2). The majority of recurrences occur outside the pelvis. Research has confirmed a poorer overall survival when compared to high grade endometrial carcinomas (9).

In addition, elevated levels of Ca125 after surgery are associated with a poor prognosis (2). In the case described herein, due to the rapid progression of the patient's tumor, the levels of Ca125 were not measured; however, these may be useful to evaluate prognosis and perform diagnosis, as these levels can be elevated due to serous epithelial tissue and the deep myometrial invasion. This is a limitation of the present study.

Recent research suggests that Aurora kinase expression is a poor prognostic marker due to positive adenopathies, vascular invasion and omental dissemination (6). The presence of lymphovascular invasion or >50% myometrial invasion is associated with a higher risk of positive adenopathies (10). The gold standard of treatment for UCS is hysterectomy, double adnexectomy with lymphadenectomy, even in patients with stage I disease, due to the high prevalence of micrometastasis (1,2).

In the early stages (stages I-II) of the disease, surgery with chemotherapy and pelvic radiotherapy or vaginal brachytherapy are used for local control. In the earlier stages, chemotherapy can improve tumor recurrence and progression-free survival, but not overall survival (4). Patients may have benefits from adjuvant multimodality therapy to reduce the chance of tumor recurrence with the potential to improve overall survival (11). However, it should be noted that, in stage I disease, radiotherapy does not improve survival (1). In patients with advanced stages of the disease, chemotherapy and cytoreduction is considered, with the possible inclusion of palliative radiotherapy (2).

Adjuvant chemoradiation is an independent good prognostic factor (12). The National Comprehensive Cancer Network guidelines (NCCN) recommends the use of carboplatin/paclitaxel (1). The use of radiotherapy with chemotherapy in the case of sarcomatous dominance is usually considered (1). Palliative radiotherapy can be used in non-resectable tumors or in cases in which surgery is not possible for clinical reasons, to treat endometrial bleeding or pelvic pain, always individualizing each patient (13).

In the case described herein, surgery was first performed due to severe illness (the results of the clinical analysis revealed sepsis, with radiological images indicating pneumoperitoneum; a physical examination did not reveal any notable comorbidities). This surgery was performed with the informed consent of the patient and her family. /the second surgery was performed following an evaluation by the multidisciplinary oncological team with an oncologist, gynecologist, general surgeon, radiologist, anatomic pathologist and a radiotherapy team, and following a discussion with the patient and her family. The second surgery was performed due to the consensus of the oncological committee in a patient with previously good quality of life, with intentions of resection the mass and completion of the treatment, always knowing the prognosis of this type of tumor. The results of the second surgery were not evident until after the surgery, as at the time of the first surgery, there was no distant metastases and neither in the previous CT scan. However, the second one revealed several metastatic implants. In the present study, the patient was operated on twice in a period of <2 months (urgent surgery was first performed and the second surgery was an attempt to complete the treatment); however, the tumor progressed rapidly.

Table III. TNM staging AJCC UICC 2017 (8).

Primary tumor, T category	FIGO stage	T criteria
Tx		Primary tumor cannot be assessed
T0		No evidence of primary tumor
T1	I	Tumor confined to the corpus uteri, including endocervical glandular involvement
T1a	IA	Tumor limited to endometrium or invading les than half the myometrium
T1b	IB	Tumor invading one half or more of myometrium
T2	II	Tumor invading stromal connective tissue of cervix but not extending beyond the uterus (not included endocervical tissue)
T3	III	Tumor involving serosa, adnexa, vagina or parametrium
T3a	IIIA	Tumor involving serosa and/or adnexa (direct or metastasis)
T3b	IIIB	Vaginal involvement (direct or metastasis) or parametrial involvement
T4	IVA	Tumor invading the bladder mucosa and/or bowel mucosa (bullous edema is not sufficient to classify a tumor as T4)
Regional lymph nodes, N category	FIGO stage	N criteria
Nx		Regional lymph nodes cannot be assessed
N0		No regional lymph nodes
N0(i+)		Isolated tumor cells in regional lymph nodes not >0.2 mm
N1	IIIC1	Regional lymph nodes to pelvic lymph nodes
N1m	IIIC1	Regional lymph nodes (>0.2 mm but not >2.0 mm) to pelvic lymph nodes
N1a	IIIC1	Regional lymph nodes (>2.0 mm in diameter) to pelvic lymph nodes
N2	IIIC2	Regional lymph nodes to para-aortic lymph nodes with or without positive pelvic involvement
N2Mi	IIIC2	Regional lymph nodes (>0.2 mm but not >2.0 mm in diameter) to para-aortic lymph nodes with or without positive pelvic involvement
N2a	I; IIC2	Regional lymph nodes (>2.0 mm in diameter) to para-aortic lymph nodes with or without positive pelvic involvement
Distant metastasis, M category	FIGO stage	M criteria
M0		No distant metastasis
M1	IVB	Distant metastasis - Included: inguinal lymph nodes, intraperitoneal disease, lungs, liver, bones - Excluded: pelvic and para-aortic lymph nodes, vagina, uterus serosa and adnexa

The number in parentheses denotes a reference.

Table IV. Prognosis of patients with carcinosarcoma.

Relevant factors in survival (1)	Aggressiveness factors (1)	Independent survival factors (2)
• Histology • Sarcomatous dominance[a] • Tumor size • Depth invasion • Lymphovascular invasion • Malignant peritoneal cytology • Positive adenopathies • Metastasis	• Size >5 cm (it is related to venous thrombosis) • Myometrial invasion • Lymphovascular invasion • Adenopathies (25% pelvic, 15% paraaortic)	• <40 years old • Caucasian race • Radiotherapy post-operatively • Lymphadenectomy x • Early stages

[a]Sarcomatous dominance is associated with a decreased survival rate.

Table V. Current possible treatments available for uterine carcinosarcoma.

Surgery	• Gold standard treatment is hysterectomy, double adnexectomy with lymphadenectomy
Chemotherapy	• It is an independent good prognostic factor • In earlier stages improve tumor recurrence and progression free survival, but not global survival • Recommended use of carboplatin/paclitaxel
Radiotherapy	• Using radiotherapy with chemotherapy to improve the effectiveness as long as sarcomatous dominance is present • The addition of radiotherapy for patients with stage I disease does not improve survival • Palliative radiotherapy can be used in non-resectable tumors, to treat endometrial bleeding or pelvic pain, always individualizing each patient.
Immunotherapy	Immunotherapies appear to be promising for patients with tumors positive for MMR-D and PD-L1, although further studies are required.
Other treatments	• The efficacy of hormone therapy for this tumor is not yet well known • Sentinel node efficacy is not well known

MMR-D, mismatch repair protein deficiency; PD-L1, programmed cell death-ligand 1.

Table VI. Treatment according to clinical stage.

Gold standard: Hysterectomy, double adnexectomy with lymphadenectomy. In all stages, it is important to individualize patient treatments.

Early stages (I-II)	Surgery + chemotherapy ± pelvic radiotherapy or vaginal brachytherapy for local control
Advanced stages (III-IV)	Important to evaluate all patient factors (comorbidities, mobility, patient's opinions, resectability of tumors and operative factors, possible complications, etc.) • Possible good prognosis: Chemotherapy + surgery (cytoreduction) + radiotherapy • Possible poor prognosis: Palliative treatment

It is well known in the literature that tumor progression is possible. For the patient described herein, after considering all the risks and benefits, the optimal treatment for the patient was offered; however, effective treatment was not possible for this case and the patient succumbed to the disease, most probably due to tumor progression. This type of tumor accounts for 16.4% of all deaths from uterine malignancies (14). This type of tumor progresses rapidly, even with treatment, as demonstrated in the case described herein. Consequently, this led to the death of the patient. However, in the patient in the present study, it was difficult to assess the main cause of death.

Sentinel node efficacy for this tumor is not yet well known (1), and hormonotherapy is not common treatment (6). There are currently studies available on treatment with tyrosine kinase inhibitors, Her2 and immune checkpoint inhibitors (5). Immunotherapies may hold promising outcomes for patients with tumors positive for mismatch repair (MMR) deficiency (MMR-D) and programmed cell death ligand-1 (PD-L1) (6). The most common mutations were observed are in *TP53* (86%), *PIK3CA* (34%), *FBXW7* (23%), *PTEN* (18%), *KRAS* (16%) and *PPP2R1A* (10%) (15). Microsatellite instability (MSI)-high is detected in 15-30% of endometrial cancer cases, and some studies have demonstrated that immune checkpoint inhibitors are effective for MSI-high solid tumors (16,17). High-frequency microsatellite instability is absent and is observed in ~5% of carcinosarcomas. The loss of heterozygosity for chromosome 11 is present in 17% of uterine sarcomas and 19% if carcinosarcomas (16).

Immunostaining for p53 defects is used in these types of tumors and is frequently between 67-85%, and is almost always consistent in the carcinomatous and sarcomatous component (18,19). Previous studies have revealed that deficiencies in MMR expression in UCS are rare, ~4% (15,19).

PD-L1 expression in these tumors may predict the response to checkpoint inhibitor therapies. The majority of cases of UCS exhibit at least a focal PD-L1 expression. Carcinosarcomas with an endometrioid morphology are significantly more likely to have high levels of PD-L1. MMR-deficient carcinosarcomas are also more likely to have high levels of PD-L1, although this has not reached statistical significance (P=0.2) (20).

The type of tumor has several mutations that are possible therapeutic targets; however, there are limited treatment options for this type of aggressive tumor with poor outcomes. Thus, further clinical trials are required to validate new treatments. The different treatments available for this type of tumor are presented in Tables V and VI.

Several clinical trials have examined the therapeutic efficacy in recurrent/metastatic UCS. The median response rates were shown to be 37.5% and 5.9 months for progression-free survival; however, after later therapies, the outcomes were worse (5.5% and 1.8 months, respectively) (1).

A limitation of the present study was that the Ca125 levels were not measured, as well as the rapid tumor progression and the worsening of the patient's clinical condition during the completion of treatment (chemotherapy, radiotherapy, etc.). No immunophenotypic analysis was performed for this tumor.

In conclusion, UCS is a rare tumor, comprised of two main elements, carcinomatous and sarcomatous. This type of tumor is usually more frequent in older-aged women of African origin. The most common symptom is vaginal bleeding, as with other uterine sarcomas. The first imaging test that needs to be performed is an ultrasound, followed by a CT scan in order to diagnose metastasis. UCS is a tumor with a poor prognosis due to its malignant component and the possibility for recurrence and metastasis. The gold standard of treatment is surgery, followed by adjuvant therapy based on the carcinosarcoma stages: Patients with the early stages of the disease may receive use chemotherapy and radiotherapy, and in those at the advanced stages, cytoreductive surgery may be considered with palliative radiotherapy to control symptoms, but always individualizing patients. There are several therapies in development that may be promising in future years.

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Availability of data and materials

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Authors' contributions

All authors (PLH, FMM, MBA, RAH, SSS, ESY, YAM, FMJ, RJM, LJA, DCG, MMDA and AGC) contributed to the diagnosis and treatment of the patient, and in the design of the study. PLH was a major contributor in the writing of the manuscript. PLH and FMM confirm the authenticity of all the raw data. All authors have read and approved the final manuscript.

Ethics approval and consent to participate

The present study followed international and national regulations and was in agreement with the Declaration of Helsinki, and ethical principles. The patient signed an informed consent form before the surgery was performed.

Patient consent for publication

The patient provided written informed consent for the publication of any data and/or accompanying images, before the surgery was performed. Patients have a right to anonymity and privacy, and authors have a legal and ethical responsibility to respect this right.

Competing interests

The authors declare that they have no competing interests.

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