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Uterine Carcinosarcoma Arising from Adenomyosis in a Postmenopausal Woman: A Case Report

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Abstract **Case Report**

Uterine carcinosarcoma is a rare malignancy composed of carcinomatous and sarcomatous components [1]. Its development from a focus of adenomyosis is exceptionally rare, and diagnosis requires careful histopathological assessment [2]. We report the case of a 58-year-old postmenopausal woman who presented with a six-month history of vaginal bleeding and pelvic pain. Imaging revealed a suspicious uterine mass associated with pelvic lymphadenopathy. Initial hysteroscopic biopsy showed a malignant mesenchymal proliferation without an identifiable epithelial component. Cytoreductive surgery was performed. Examination of the surgical specimen revealed a carcinosarcoma composed of a carcinomatous component and a sarcomatous component with leiomyosarcomatous differentiation. Foci of adenomyosis were present in the myometrium adjacent to the tumor. A lesional continuum containing areas of high-grade dysplasia was observed, while the eutopic endometrium showed no significant abnormality. These findings supported an adenomyotic origin. Five of the 24 pelvic lymph nodes examined contained metastases. The disease was classified as FIGO stage IIIC1. The patient received carboplatin and paclitaxel chemotherapy, followed by external-beam radiotherapy. Eighteen months after surgery, she was alive with persistent metastatic lymph node disease on PET/CT. This case illustrates the limitations of partial biopsy in the diagnosis of a heterogeneous uterine tumor. It also highlights the importance of examining the relationship between the tumor, adenomyotic foci, and eutopic endometrium when determining the origin of the lesion [7].

Keywords: Uterine carcinosarcoma; adenomyosis; postmenopausal woman; leiomyosarcoma; immunohistochemistry.

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INTRODUCTION

Uterine carcinosarcoma is a biphasic malignancy composed of an epithelial carcinomatous component and a mesenchymal sarcomatous component. Previously referred to as a malignant mixed Müllerian tumor, it can be difficult to diagnose when both components are not represented in the initial sample [1]. Adenomyosis is characterized by the presence of endometrial glands and stroma within the myometrium. The development of a malignancy within a focus of adenomyosis is rare. Establishing its origin requires distinguishing malignant transformation within an adenomyotic focus from secondary infiltration by a tumor arising in the endometrium or at another site [2]. Few cases of carcinosarcoma attributed to adenomyosis have been reported. Kim and Suh described a malignant mixed Müllerian tumor considered to have arisen in adenomyosis [3]. Other cases have since been published, with variable clinical and morphological presentations [4–6]. We report the case of a postmenopausal woman with uterine carcinosarcoma containing a sarcomatous component with leiomyosarcomatous differentiation and associated pelvic lymph node involvement. Foci of adenomyosis, a lesional continuum containing high-grade dysplasia, and an unremarkable eutopic endometrium supported an adenomyotic origin.

CASE REPORT

A 58-year-old postmenopausal woman, gravida 2, para 2, was referred for a six-month history of pelvic pain and postmenopausal bleeding despite treatment. Her personal and family medical histories were unremarkable. Abdominopelvic ultrasonography revealed a heterogeneous intrauterine mass with areas suggestive of hemorrhage and suspicious for malignancy. Cervical cytology was inconclusive because

of extensive cytolysis. Laboratory testing showed anemia and elevated lactate dehydrogenase levels. Diagnostic hysteroscopy with biopsy of the mass was performed. Initial histopathological examination showed an extensively necrotic malignant-appearing mesenchymal proliferation composed of undifferentiated spindle cells. Staging with computed tomography and pelvic magnetic resonance imaging demonstrated an intrauterine mass with cervical extension and suspicious pelvic lymphadenopathy (Figure 1). Abdominopelvic computed tomography showed no visceral or osseous secondary lesions. Following multidisciplinary discussion, maximal cytoreductive surgery was planned. The procedure consisted of total colpohysterectomy with bilateral salpingo-oophorectomy, pelvic and para-aortic lymphadenectomy, and peritoneal assessment including cytology, omentectomy, and peritoneal biopsies. Gross examination of the surgical specimen revealed a large, friable, extensively necrotic intrauterine tumor measuring approximately 7 × 6 cm and infiltrating the uterine wall. It occupied the entire uterine cavity and extended to the cervico-isthmic junction, with involvement of the cervical mucosa and stroma (Figure 2). Histological examination showed a poorly differentiated malignant tumor with a roughly lobulated architecture infiltrating the uterine wall. It contained two components: a predominant malignant spindle cell mesenchymal component and an epithelial carcinomatous component with a villoglandular architecture, arranged in solid clusters in some areas (Figure 3). Myometrial invasion measured 20 mm in a wall thickness of 24 mm. Foci of adenomyosis were identified within the myometrium adjacent to the tumor; some contained carcinomatous elements. A lesional continuum between the adenomyosis and the tumor was described, containing areas of high-grade dysplasia. The eutopic endometrium showed no significant abnormality. The adnexa were free of tumor. Immunohistochemistry showed strong and diffuse cytokeratin AE1/AE3 expression in the carcinomatous component (Figure 4), as well as estrogen and progesterone receptor expression. This component was negative for smooth muscle actin, desmin, and h-caldesmon. The sarcomatous component showed strong and diffuse expression of smooth muscle actin (Figure 5), desmin, and h-caldesmon, with focal CD10 expression. The combined morphological and immunohistochemical findings supported a diagnosis of uterine carcinosarcoma with a homologous sarcomatous component showing leiomyosarcomatous differentiation. The observed lesional continuum between the adenomyosis and the tumor, together with the absence of a reported abnormality in the eutopic endometrium, supported an adenomyotic origin. Five of the 24 pelvic lymph nodes examined contained metastases. The largest involved lymph node measured 2 cm in greatest dimension; lymphovascular tumor emboli from the carcinomatous component were also reported. The para-aortic lymph nodes were negative. Peritoneal cytology was negative, and the peritoneal biopsies and omentectomy specimen showed no tumor. The disease was classified as FIGO stage IIIC1 because of pelvic lymph node involvement. After surgery, the patient received carboplatin and paclitaxel chemotherapy, followed by postoperative external-beam radiotherapy to a total dose of 50 Gy in 25 fractions of 2 Gy, with the aim of reducing the risk of locoregional recurrence. Eighteen months after surgery, she was alive. Follow-up PET/CT demonstrated persistent metastatic lymph node disease despite chemotherapy.



Figure 1: Pelvic CT scan showing a heterogeneous endometrial mass infiltrating the uterine wall.



Figure 2: Gross examination of the total hysterectomy specimen showing a whitish endometrial mass with hemorrhagic changes infiltrating the myometrium.

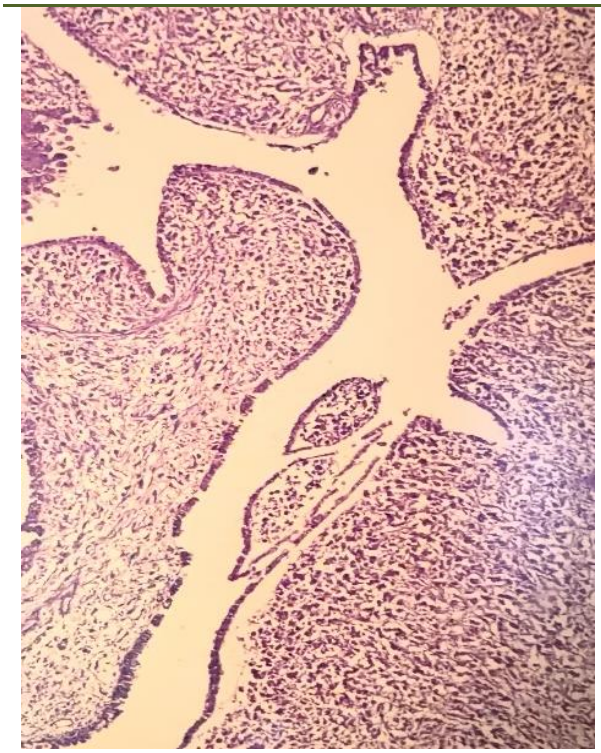


Figure 3: Tumor proliferation composed of epithelial and spindle cell components infiltrating the myometrium. Hematoxylin and eosin stain, original magnification × 20

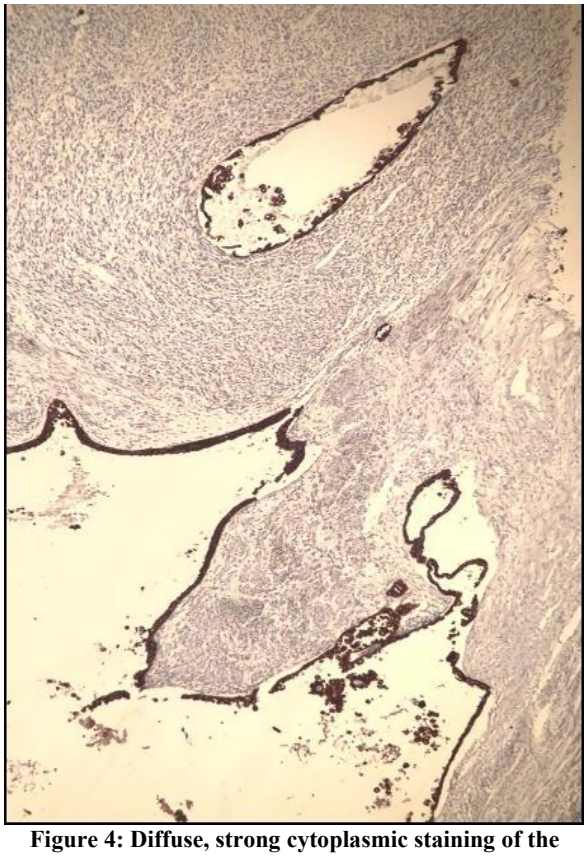


Figure 4: Diffuse, strong cytoplasmic staining of the epithelial cells for AE1/AE3 cytokeratins, with no staining of the mesenchymal cells for smooth muscle actin : immunohistochemistry



Figure 5: Immunohistochemistry showed strong, diffuse staining of the mesenchymal cells for smooth muscle actin, with no staining for AE1/AE3 cytokeratins: immunohistochemistry

DISCUSSION

This case raises two diagnostic challenges: recognizing carcinosarcoma when the initial biopsy shows only a malignant spindle cell proliferation, and determining its origin when adenomyotic foci are present. Distinguishing a tumor arising within adenomyosis from one that has secondarily infiltrated adenomyotic foci requires assessment of their histological relationship and a search for another primary site. The historical criteria of Colman and Rosenthal include the identification of adenomyotic tissue and exclusion of a carcinoma arising from the endometrium or another pelvic site [7]. These criteria were formulated for carcinomas and should be interpreted cautiously when the tumor is large and extensively altered. In our case, adenomyotic foci were present adjacent to the tumor, and a lesional continuum containing high-grade dysplasia was described. The eutopic endometrium was unremarkable. Together, these findings supported the proposed adenomyotic origin. A detailed description of the transition zones is particularly important to distinguish malignant transformation within adenomyosis from secondary infiltration. The mechanisms involved in malignant transformation of adenomyosis remain incompletely understood. Proposed mechanisms include the accumulation of molecular alterations and the influence of the tissue microenvironment. Their precise role in carcinosarcomas arising from adenomyosis remains to be established [8]. In the case reported by Kiuchi and

colleagues [4], the tumor was predominantly myometrial, with benign endometrial glands surrounded by stroma near the carcinosarcoma; the endometrium and adnexa were uninvolved. The systematic review by Raffone and colleagues [2], which included that case, did not report a transition in its summary table. Our case similarly included adenomyotic foci adjacent to the tumor and no reported abnormality of the eutopic endometrium. It also showed a described lesional continuum containing high-grade dysplasia. Kim and Suh [3] reported a lesion under the former designation of malignant mixed Müllerian tumor. It consisted of a well-differentiated adenocarcinoma and a sarcomatous component containing rhabdomyosarcomatous foci. The authors considered the tumor to have arisen in adenomyosis. Their report illustrates the value of including historical terminology in literature searches. Kiuchi and colleagues [4] described a tumor initially suspected to be a leiomyosarcoma, with final examination revealing an endometrioid carcinomatous component and a heterologous rhabdomyosarcomatous component. In our case, the sarcomatous component showed homologous leiomyosarcomatous differentiation, supported by strong and diffuse expression of smooth muscle actin, desmin, and h-caldesmon. Uchiyama and colleagues [6] reported a carcinosarcoma containing an angiosarcomatous component, with multiple adenomyotic foci in the myometrium and within the tumor. This case illustrates the morphological diversity of carcinosarcomas associated with adenomyosis. However, the available data do not establish its adenomyotic origin with certainty. The presentation reported by Hashizume and colleagues [5] differed from that of our patient: a ruptured subserosal cystic mass caused an acute abdomen and led to emergency surgery. In our case, postmenopausal bleeding and pelvic pain had been present for six months. These observations illustrate the range of presentations but do not establish clinical features specific to this rare proposed origin. Initial hysteroscopic biopsy showed an extensively necrotic malignant mesenchymal proliferation without an identifiable epithelial component. Examination of the surgical specimen identified both malignant components and established the diagnosis of carcinosarcoma. This discrepancy illustrates the limitations of partial sampling in a heterogeneous tumor [1]. Immunohistochemistry helped characterize the two components: cytokeratin AE1/AE3 was expressed in the carcinomatous component, whereas smooth muscle markers were expressed in the sarcomatous component. Focal CD10 expression adds to the description of the tumor's immunophenotype but does not by itself establish an adenomyotic origin. Molecular studies of uterine carcinosarcomas have identified features of epithelial-to-mesenchymal transition in some cases, potentially contributing to sarcomatous differentiation. These findings concern uterine carcinosarcomas as a group and do not establish the mechanism of transformation in our patient [9]. Pathological examination revealed five metastatic pelvic lymph nodes among 24 examined, while the para-aortic nodes were negative. Peritoneal cytology, peritoneal biopsies, and the omentum were free of tumor. FIGO stage IIIC1 was assigned under the 2023 FIGO classification on the basis of pelvic lymph node involvement [10]. The patient underwent cytoreductive surgery followed by carboplatin–paclitaxel chemotherapy and postoperative external-beam radiotherapy. In the absence of trials dedicated to carcinosarcomas attributed to adenomyosis, treatment decisions rely on evidence from uterine carcinosarcomas more broadly. In the randomized phase III trial by Powell and colleagues [11], carboplatin–paclitaxel was noninferior to ifosfamide–paclitaxel for overall survival in patients with uterine carcinosarcoma. In a retrospective analysis of patients with stage I–III uterine carcinosarcoma treated with surgery and chemotherapy, the addition of radiotherapy was associated with improved overall survival [12]. Eighteen months after surgery, our patient was alive with persistent lymph node disease demonstrated on PET/CT despite chemotherapy. Hashizume and colleagues [5] reported no recurrence approximately one year after surgery and adjuvant chemotherapy. Differences in clinical presentation, extent of disease, and follow-up preclude direct prognostic comparison.

CONCLUSION

Uterine carcinosarcoma arising from adenomyosis is a rare presentation whose diagnosis requires careful histological assessment. Our case also illustrates the difficulty encountered when an initial biopsy demonstrates only one of the two tumor components. Examination of the surgical specimen, supported by immunohistochemistry, established the diagnosis of carcinosarcoma with a sarcomatous component showing leiomyosarcomatous differentiation. Adenomyotic foci adjacent to the tumor, a lesional continuum containing high-grade dysplasia, and the absence of a reported abnormality in the eutopic endometrium supported an adenomyotic origin. The rarity of reported cases and the heterogeneity of criteria used to establish this origin limit conclusions regarding the specific prognosis of these tumors.

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