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CASE REPORT

Uterine Adenomyosarcoma Complicated by Uterine Prolapse and Necrosis: A Case Report

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Abstract: In this case, the patient had uterine adenocarcinoma with a huge necrotic mass prolapsed from the vagina, complicated by necrotic infection and massive bleeding. Based on ultrasound results preoperatively, uterine prolapse with infected necrosis was considered due to significant vaginal bleeding, prompting emergency surgery and blood transfusion. Postoperatively, pathology review indicated a misdiagnosis. This article aims to analyze the reasons for misdiagnosis through case review and literature review.

Keywords: uterine adenosarcoma, UA, müllerian adenosarcoma of the uterus, case report

Introduction

Uterine adenosarcoma (UA), also known as müllerian adenosarcoma of the uterus, is a relatively rare malignant tumor of the uterus. It constitutes about 8% of uterine sarcomas and merely 1% of all uterine malignancies, characterized by a combination of benign glandular epithelial and malignant stromal components. Its histopathological and biological features lie between benign adenofibroma and highly malignant sarcomas.[1]

Medical Case Report

A 58-year-old postmenopausal woman presented with a one-year history of irregular vaginal bleeding and a two-month history of a protruding vaginal mass. Initially, the vaginal bleeding was scant, irregular, and dark red, with no apparent triggers or associated symptoms such as lower abdominal pain. Despite an increase in bleeding frequency and volume over time, which caused noticeable abdominal heaviness, the patient attempted self-management with traditional Chinese medicine and did not seek medical attention. Two months before admission, she noticed a mass at the vaginal opening that became more pronounced during standing or squatting, approximately the size of an egg, requiring daily manual repositioning. Two days before the presentation, the bleeding intensified, with bright red blood and large clots, and the mass became irreducible, prompting her to seek medical care. She reported menarche at age 13, menopause at age 56, and a history of regular menstrual cycles with no dysmenorrhea. She had no sexual activity for 7 years following a divorce and underwent cervical screening (HPV + TCT) 5 years ago, which reportedly showed no abnormalities, although the results were unavailable. The patient is divorced and has a history of G5P1 (one full-term delivery in 1989), two ectopic pregnancies, and two early miscarriages following IVF attempts. Menarche occurred at 13 years of age with regular menstrual cycles every 30 days lasting seven days, normal menstrual flow, and no dysmenorrhea. She has been postmenopausal for two years. In 2000, she underwent right salpingectomy due to "tubal pregnancy" at an external hospital with a transfusion during hospitalization. In 2005, she received conservative treatment for "ectopic pregnancy" at another hospital, with successful management. The patient was referred to our hospital with a suspected uterine tumor (possibly sarcoma) for further treatment.

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Upon admission, physical examination revealed slight abdominal distension symmetrically. A palpable firm mass was detected in the lower abdomen with moderate tenderness but no rebound tenderness. The upper edge of the mass reached below the umbilicus. External genitalia appeared normal, but a protruding mass was noted at the vaginal introitus measuring approximately 10×8 cm, purplish-black with purulent discharge on the surface, positive for blood on palpation, and emitting a foul odor suggestive of necrotic tissue. The mass completely filled the vagina without any gaps, making bimanual examination impossible. Dark red blood was observed flowing from the vagina. Anorectal examination indicated a large mass within the vagina, measuring about 15×13 cm, mildly tender. The anterior rectal wall felt smooth, and no blood was on the examining glove (Figure 1).

Following admission, additional investigations were conducted. Coagulation parameters showed a prothrombin time of 15.1 seconds, a D-dimer level of 1.69 mg/L, and fibrinogen degradation products of 5.11 mg/L. Tumor markers CA-

Figure 1 Tumor photograph (pre-operation).

125 and CA-199 were within normal ranges. Squamous cell carcinoma-related antigen was 2.97 ng/mL, and human epididymis protein 4 was 246.8 pmol/L. A blood routine examination revealed a white blood cell count of 7.14 × 10^9/L, with 85.1% neutrophils and 7.9% lymphocytes. The hemoglobin level was 41 g/L, and the erythrocyte sedimentation rate was 0.15 L/L. C-reactive protein was elevated at 28.37 mg/L. Abdominal gynecological ultrasound showed a heterogeneously echogenic mass in the pelvic uterine bed area, measuring approximately 17.8×9.7 × 8.9 cm. The boundaries were relatively straightforward and had a complete and somewhat regular shape. The capsule appeared smooth, but internal echogenicity was irregular and chaotic. A hyperechoic area was noted on the abdominal side toward the central region, showing abundant blood flow signals and extending downwards in a band-like pattern toward the perineum. The lower part of the mass had irregular echogenicity with sparse to absent blood flow signals and scattered small fluid areas. Structures below the symphysis pubis were indistinct. Conclusion: A heterogeneous echogenic mass in the pelvic region suggests uterine prolapse complicated by infarction, with the associated mass not ruled out. Further investigations are recommended (Figure 2).

Upon evaluation, significant vaginal bleeding, anemia, and findings from imaging and clinical examination suggested a large pelvic mass with infection. Differential diagnoses included uterine malignancy with infection (primary consideration), cervical malignancy with infection (given elevated squamous cell carcinoma antigen levels and lack of recent cervical screening), uterine prolapse with infection, and severe anemia secondary to chronic blood loss. Upon admission, the patient received urgent measures, including blood transfusion, fluid replacement, and hemostasis. Despite initial management with transfusion, intravenous fluids, and hemostatic measures, persistent heavy bleeding and hemodynamic instability necessitated emergency exploratory laparotomy under general anesthesia on September 1, 2023. Intraoperatively, the following findings were noted: the cervix and cervical fibroid protruded from the vaginal introitus, showing signs of infection and necrosis, cylindrical in shape, approximately 8×10 cm. Upon abdominal exploration, the uterus was enlarged, distorted, and showed degenerative necrosis. It extended into the vaginal prolapsed tissue, with a total size of 15×17 cm, Intraoperatively, the ovaries were of normal size and appearance, with no visible abnormalities or lesions, and the fallopian tubes were unremarkable, Both ovaries and fallopian tubes were removed during the surgery. During surgery, the uterus, vaginal mass, and bilateral adnexa were completely removed (Figure 3).

Postoperative pathology report: (Uterus + Bilateral Adnexa) Malignant tumor of the uterus with extensive hemorrhage and necrosis. Immunohistochemical analysis confirmed adenomyosarcoma with sarcomatous overgrowth. The tumor showed fragmentation, with dimensions approximately 14×6×2.5 cm and 17×14×6 cm, involving the superficial muscle layer. No intravascular tumor thrombus was identified. Immunohistochemistry results on paraffin sections: CK (epithelial +), CD10 (+), SMA (+), Desmin (partially +), CD34 (-), ER (+), PR (+), S-100 (-), Ki67 (approximately 30% +), Cyclin D1 (-), H-caldesmon (partially +), CD68 (histiocytes +) (Figure 4). Postoperative pelvic MRI revealed no enlarged lymph nodes in the pelvic cavity and no abnormal signals in the bladder or rectum. Considering clinical, pathological, and MRI findings, the final diagnosis for the patient was stage IB adenomyosarcoma of the uterus. After

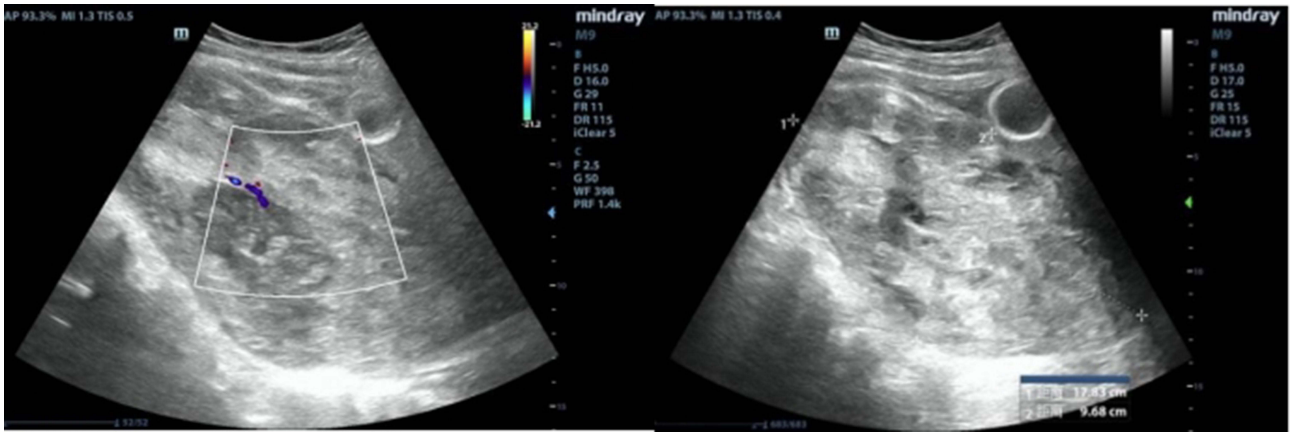

Figure 2 Ultrasonoscop.

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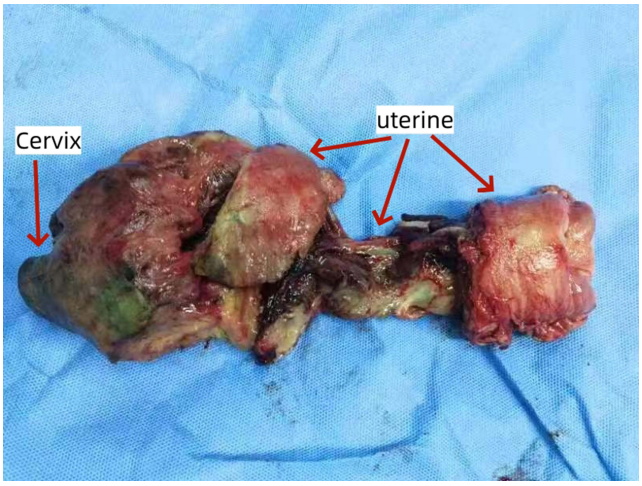

Figure 3 Excision specimen photograph.

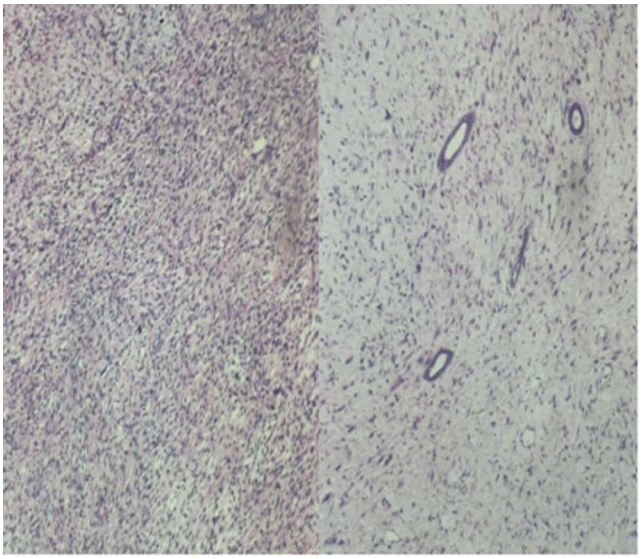

Figure 4 Pathological report.

surgery, the patient was transferred to our oncology department for further treatment, where she underwent two cycles of chemotherapy with intravenous epirubicin and oral megestrol acetate (0.16 g daily). After two cycles of chemotherapy, the patient has voluntarily transferred to a hospital in Beijing for further treatment. Telephone follow-ups were conducted, and after completing the third cycle of chemotherapy at the previous hospital, no further treatment was pursued. One month later, there was a recurrence of a large mass prolapsing from the vagina. Subsequently, the patient underwent tumor reduction surgery at the previous hospital. Currently, the patient is receiving concurrent radiotherapy and chemotherapy in the oncology department.

Discussion

Clinical Presentation

This condition predominantly affects postmenopausal women. Studies indicate that 51.5% of UA patients are between 40–65 years old, 38.4% are over 65, and those under 40 constitute about 10%[2] In this case, the patient is 58 years old and has been postmenopausal for two years.

Clinical manifestations of uterine adenosarcoma lack specificity. The primary symptoms include vaginal bleeding (76%), lower abdominal pain (5%), symptoms related to uterine prolapse (4%), and abnormal vaginal discharge (2%).[3] Common physical signs include protrusion of masses through the cervix or into the vagina (53%), uterine enlargement (39%), and pelvic masses (5%). The patient, in this case, presented mainly with uterine enlargement, cervical and partial uterine body prolapse through the vaginal introitus, and recurrent irregular vaginal bleeding. Auxiliary examinations indicated a pelvic mass, which is consistent with these symptoms. The clinical and radiological features of uterine adenosarcoma lack specificity, making it challenging to distinguish from conditions such as endometrial carcinoma. Consequently, there is a relatively high rate of misdiagnosis in initial assessments, necessitating confirmation through postoperative pathological examination.

Auxiliary Examinations

In the auxiliary diagnosis of UA, gynecological ultrasound is commonly used and typically shows cervical or uterine cavity-occupying lesions with heterogeneous hypoechogenicity. Some cases exhibit rich blood flow signals.[4] CT scans often detect lesions within the endometrium, with a minority affecting the cervical canal. Preoperative CT scans provide insights into lesion location, size, growth pattern, and potential metastasis. Contrast-enhanced CT scans can reveal internal blood flow within the tumor, contributing to diagnostic accuracy.[5] MRI examinations frequently reveal sizeable soft tissue masses within or outside the uterine cavity, often with ill-defined borders. Lesions in the cervical canal may penetrate through the cervix.[6] On T1-weighted imaging, lesions typically display slightly low or mixed signal intensities, while on T2-weighted imaging, they appear predominantly hyperintense with irregular cystic signals. Diffusion-weighted imaging shows low signal intensity.[7,8] T1-weighted MRI offers high tissue contrast resolution, surpassing CT scans in visualizing lesions and surrounding tissue structures. Therefore, MRI is functional for confirming suspicious malignant lesions identified on ultrasound and assessing infiltration relationships between the tumor and adjacent tissues.[9,10] The role of serum tumor markers in aiding UA diagnosis remains inconclusive, as specific tumor markers have not been identified.

In some cases, CA-125 levels may rise, albeit with low specificity. In this instance, the patient's carbohydrate antigens CA-125 and CA-199 levels were within normal ranges. Squamous cell carcinoma-related antigen was 2.97 ng/mL, and human epididymis protein 4 was 246.8 pmol/L.

Pathological Features

Under microscopic examination, uterine adenosarcoma (UA) exhibits characteristic pathological features of dense stromal cells surrounding glandular formations, often forming distinctive "cuff-like" structures.[11] Research indicates that 15% to 28% of UA cases show infiltration into the myometrium, with 72% to 98% involving infiltration depth less than 50% of the myometrial thickness. Increased depth of myometrial infiltration correlates with reduced 5-year overall survival rates.[12] In this case, postoperative pathological findings indicated UA stage IB (tumor infiltrating less than half of the myometrium), accompanied by sarcomatous overgrowth. After surgery, the patient was transferred to our oncology department for further treatment, including oral administration of medroxyprogesterone acetate 0.16g daily and chemotherapy with doxorubicin 50mg on Day 1 and 60mg on Day 2. After two cycles of chemotherapy, the patient opted to continue treatment at another hospital.

Pathological risk factors for UA include tumor cell heterologous differentiation, sarcomatous overgrowth, deep myometrial invasion, lymph node metastasis or vascular invasion, and tumor necrosis.[13,14] UA lacks typical immunohistochemical markers but commonly expresses CD10 (71%) and WT1 (79%). The expression of estrogen receptors (ER) and progesterone receptors (PR) correlates with sarcomatous overgrowth.[15]

Differential Diagnosis

Rhabdomyosarcoma

Originating from embryonic mesenchymal cells, rhabdomyosarcoma shares common features with uterine adenosarcoma (UA), such as active mitotic figures and mild to moderate cytological atypia in stromal cells around glands or beneath the epithelium. However, UA is characterized by its typical polypoid and papillary structures, which are absent in

rhabdomyosarcoma.[16] Rhabdomyosarcoma primarily affects children and adolescents, accounting for 4% to 6% of malignant tumors in this age group. Common clinical manifestations include irregular vaginal bleeding (50%) and polypoid masses in the cervix (78%). Diagnosis relies on pathological examination.

Uterine Adenofibroma

Distinguishing between adenofibroma and adenosarcoma is challenging, with some suggesting that adenofibroma represents a better-differentiated form of UA. The 2014 WHO classification defines *adenofibroma* as having benign endometrial epithelial cells covering papillary stromal components, with cystic and cribriform glands in each case. Adenofibroma lacks peripheral condensation, stromal mitotic activity, and a mitotic count index ($\geq$ 2 MF/10 hDF), which are criteria distinguishing it from UA.[17]

Endometrial Polyp and Cervical Polyp

Compared to UA, benign endometrial polyps are typically smaller (<3 cm) and exhibit minimal stromal cytological atypia. They lack morphological features in UA, such as stromal cytological atypia, prominent stromal cell proliferation, cuff-like changes around glands, and myometrial invasion.[18]

Uterine Carcinosarcoma

Also known as a malignant mixed müllerian tumor, the primary distinction from UA lies in the malignant nature of the epithelial component. A hypothesis suggests that a portion of uterine carcinosarcomas originate from malignant transformation of the epithelium in UA. Studies have observed that 8% to 16% of uterine carcinosarcomas exhibit growth patterns similar to uterine adenocarcinoma, supporting this hypothesis.[19]

Endometrial Stromal Sarcoma

Composed of endometrial stromal cells, endometrial stromal sarcoma lacks the glandular formations and lacy projections of stromal tissue within glands seen in UA. It also lacks distinct tissue borders typical of UA and exhibits a diffuse infiltrative growth pattern within lymphatic vessels. These differential diagnostic considerations emphasize the importance of histopathological examination in confirming UA and distinguishing it from other uterine pathologies with overlapping clinical and imaging features.

Treatment

Treatment for uterine adenosarcoma (UA) primarily involves surgery, with recommended procedures including total hysterectomy with bilateral salpingo-oophorectomy.[20] It is generally advised that young patients with fertility desires and without high-risk factors (which require specific evaluation) may consider tumor excision alone after assessment. Close postoperative follow-up is essential, and in cases where high-risk pathological factors are present postoperatively, prophylactic total hysterectomy after childbearing may be considered to reduce recurrence and metastasis.[21]

Due to the meager rate of lymph node metastasis (0–6.5%), lymphadenectomy is not recommended unless there is clear evidence of lymph node involvement.[22] For patients with stage III–IV disease, the presence of any high-risk pathological factors, or age <18 years, adjuvant chemotherapy after surgery may be effective. For recurrent patients, treatment options include surgery, chemotherapy, and possibly bevacizumab therapy,[23] though routine adjuvant chemotherapy is currently not recommended for UA patients.[24]

Recurrence and metastasis of UA commonly remain confined to the pelvic and abdominal cavities, with a distant metastasis rate of only 2%. Studies have shown that pathological stage, presence of tumor cell heterologous differentiation, sarcomatous overgrowth, deep myometrial invasion, lymph node metastasis, vascular invasion, tumor necrosis, primary tumor size, ethnicity, and age differences all impact prognosis.[13]

Treatment for recurrence and metastasis typically involves surgical resection of lesions and/or systemic chemotherapy, with hormone therapy also considered. Particularly for isolated recurrent lesions, surgical resection with appropriate adjuvant chemotherapy is recommended.[6] Radiation therapy is not advocated due to its severe complications and lack of significant survival improvement in advanced-stage patients. Research by Clement and Scully[3] indicates a 5-year survival

rate of 60.2% among 100 UA patients, with a median survival time of 5.9 years and approximately one-third experiencing recurrence within ten years, highlighting the need for long-term follow-up in UA management.

Misdiagnosis Analysis

Based on the preoperative assessment, several key findings guided the initial diagnosis. First, the patient experienced postmenopausal vaginal bleeding persisting for one year. Second, clinical examination revealed uterine enlargement and a massive vaginal prolapsed tumor, exhibiting signs of infection and necrosis. Third, the absence of cervical cancer screening results in the past five years further raised suspicion of malignancy. Given these factors, the primary considerations upon admission were uterine malignancy with infection and cervical malignancy with infection. Unfortunately, due to the patient's critical condition with heavy vaginal bleeding and severe anemia at the time of admission, there was insufficient time for comprehensive imaging and pathological biopsy. Intraoperative findings of the vaginal tumor and uterine characteristics confirmed that prioritizing a diagnosis of malignancy was the correct approach.

Currently, both uterine adenosarcoma (UA) and uterine infarction lack typical features on ultrasound imaging. The initial diagnosing physician lacked experience in interpreting such cases. Color Doppler flow imaging (CDFI) revealed sparse blood flow signals in the uterine bed area, accompanied by apparent necrosis and prolapsed cervix clinically, leading to an initial diagnosis of uterine infarction. Upon reviewing postoperative pathology and related literature and considering the growth characteristics of adenosarcoma, the following reconsiderations were made: The uterine bed area tumor in the patient was significantly larger than average, with sparse blood flow signals, suggesting that superficial infarction was unlikely. A more significant occupying lesion was considered more likely, especially since the tumor displayed heterogeneous echogenicity throughout, making it challenging to distinguish regular uterine layers, which raised suspicion of malignancy.

Moreover, a hyperechoic area lateral to the tumor, with internal blood flow signals extending downward, suggested intracavitary tumor distribution with a polypoid growth pattern. At the same time, the thicker stalk structure indicated a higher likelihood of malignancy. The chaotic echogenicity and sparse or absent blood flow signals in most areas of the tumor, combined with the foul odor and evident necrosis of the prolapsed cervical tissue, suggested a possible malignant lesion complicated by infectious infarction necrosis. Considering the polypoid growth and pronounced uterine prolapse, a diagnosis of uterine adenosarcoma could not be ruled out. Through this experience, the author gained deeper insights: imaging examinations must not overlook the disease's growth characteristics and clinical pathology. Postoperative analysis and review are crucial in determining whether the correct diagnosis was made.

Based on the above, uterine adenosarcoma is a rare and low-incidence condition with non-specific clinical presentations and a lack of characteristic tumor markers, leading to a high rate of misdiagnosis. However, postmenopausal vaginal bleeding and an enlarged uterus remain significant symptoms of uterine adenomyosarcoma. Patients presenting with similar symptoms should first consider the possibility of a malignant uterine tumor. If conditions permit, it is important to complete imaging and pathological examinations to avoid misdiagnosis or inadequate treatment. Currently, there is no standardized approach for diagnosing and treating this disease. Further accumulation of cases, clinical observations, and identification of specific tumor markers are necessary to standardize diagnostic and therapeutic protocols, aiming to reduce misdiagnosis and missed diagnoses.

Consent Statements

The patient has approved and written informed consent for the publication of this case report including the images. The Medical Ethics Committee of Qingdao Central Medical Group has also approved the publication of this article.

Disclosure

The authors report no conflicts of interest in this work.

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