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Case Report

Cystic degeneration of a fibroid in the context of abnormal uterine bleeding in young patients: a case report

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ABSTRACT

Uterine fibroids are the most common uterine tumors in perimenopausal women; their presence and growth are associated with elevated estrogen levels and stimulation of progesterone receptors. Clinically, fibroids are associated with abnormal uterine bleeding, anemia, recurrent pregnancy loss, infertility, and/or chronic pelvic pain. Ultrasound imaging faces challenges when fibroids present atypical features. Degenerated fibroids in their cystic variant are related to edema and multiple intrauterine cysts. In young patients, obstetric pathology should not be overlooked. The easy accessibility and low cost of ultrasound allow for universal screening of patients with abnormal uterine bleeding. The pathognomonic presence of a "cluster of grapes" pattern raises suspicion of molar pregnancy in this patient population. It is essential to establish an accurate diagnosis to initiate appropriate therapeutic management, prognosis, and follow-up. Gynecologists and sonographers should consider the β-hCG hook effect or receptor saturation, which may produce false-negative results, as well as recognize the degenerative variants of uterine fibroids to accurately raise diagnostic suspicion.

Keywords: Abnormal uterine bleeding, Uterine fibroid, Hydatidiform mole, Ultrasound, Beta-HCG, Pelvic mass

INTRODUCTION

Uterine leiomyoma, commonly known as uterine fibroid, is the most frequent gynecological tumor during the reproductive age. Although benign, its significant morbidity results in substantial costs for the public health system. Its growth can lead to degenerative changes due to excessive edema and ischemia, which may produce heterogeneous or unusual presentations, posing a diagnostic challenge. Currently, ultrasound constitutes the first-line imaging modality in the evaluation of pelvic pathology due to its availability and low cost. The finding of hypoechoic areas with a “cluster of grapes” appearance during this examination in a reproductive-aged patient presenting with abdominal pain and vaginal bleeding raises suspicion for hydatidiform mole. However, definitive diagnosis is established through histopathology, which guides the diagnosis, therapeutic strategy, prognosis, and/or probability of disease recurrence.[1-5]

Pelvic masses in the evaluation of gynecological pathology have become a diagnostic challenge for sonographers and gynecoobstetricians due to their close relationship with abdominal organs. In the initial assessment of a patient presenting with abdominal pain and abnormal uterine bleeding, cervical pathologies such as polyps and/or endocervical fibroids, as well as exophytic lesions with suspicion of malignancy in advanced stages must be considered. However, obstetric

causes in reproductive-aged patients should be the primary suspicion, including first-trimester incomplete abortions, ectopic pregnancy, and gestational trophoblastic disease, also known as hydatidiform moles, which can be classified as complete or partial varieties.[1]

A clinical case is presented of a young woman with abnormal uterine bleeding and abdominal pain. A qualitative immunological pregnancy test was performed, yielding a negative result. Supportive imaging by ultrasound was inconclusive, complicating the diagnostic and therapeutic approach.

Leiomyomatosis, myomatosis, adenomyosis, or uterine fibroids are the most common benign tumors among women of reproductive and perimenopausal age (30-50 years), affecting approximately 80% of women throughout their lifetime. They originate in the myometrium under the influence of estrogen and progesterone, with estrogen acting as an inducer of progesterone receptors, which are related to the growth of uterine fibroids. Despite this, these hormones may mitigate clinical symptoms associated with endometrial shedding.[2]

Uterine fibroids are primarily composed of smooth muscle cells without atypia or mitotic activity, organized in interlacing fascicles within a fibrous stroma made up of collagen, fibronectins, laminins, and proteoglycans. Their etiology is not fully understood but is considered multifactorial, arising from mutations in monoclonal smooth muscle cells of the uterus as a response to inflammation and hormonal stimulation, influenced by genetic, epigenetic, and environmental factors. Altered stem cells transform into smooth muscle stem cells, promoting the formation of an abnormal extracellular matrix around these cells, leading to the development of uterine fibroids.[1]

Most cytogenetic abnormalities lead to alterations in growth cytokines, such as mutations in MED12 found in up to 70-80% of cases. MED12 mutations appear to be inversely correlated with the size of uterine fibroids, although MED12 inactivation results in upregulation of transforming growth factor beta (TGF-β), a promoter of the extracellular matrix.[1,3] Various studies have demonstrated the involvement of genetic factors in the formation of uterine fibroids, as well as the significant epigenetic role of exposure to endocrine disruptors.

Products containing phthalates (DEHP), bisphenol A (BPA) plasticizers, diethylstilbestrol (DES), genistein (GEN), perfluoroalkyl substances (PFAS), and parabens found in personal care items are widely associated with the development of uterine fibroids. In vitro and in vivo exposure to DEHP has been shown to reduce apoptosis and increase cell viability, proliferation, and collagen production by inducing the expression of HIF-1α, COX-2, PCNA, and BCL2.[4] Uterine fibroids can be classified based on their location within the uterine wall into submucosal/sub endometrial, intramural/myometrial, or subserosal types, according to the International Federation of Gynecology and Obstetrics (FIGO) system.

Their presence is associated with significant morbidity, including dysfunctional uterine bleeding, anemia, infertility, recurrent pregnancy loss, and/or chronic pelvic pain. Uterine fibroids reduce their blood supply, leading to ischemic changes associated with different types of degeneration: hyaline (60%), cystic (4%), red (3%), myxoid (1-3%), calcific (4%), and sarcomatous (0.1-0.8%). The typical appearance of fibroids is easily recognized on ultrasound imaging; however, atypical appearances resulting from these degenerative changes can cause diagnostic confusion.[5]

Hyalinization is the most common type of degeneration, occurring in approximately 60% of degenerated fibroids. In contrast, cystic degeneration is rare but not uncommon. Cystic degeneration results from rapid growth that outpaces blood supply, which can be considered an extreme form of edema. Macroscopically, areas of liquefaction of the hyaline regions of varying sizes and shapes are observed, reported as findings in up to 4% of all uterine fibroids. The clinical presentation of patients with uterine fibroids is variable; around 50% are asymptomatic and detected incidentally during routine gynecological examinations.

Conversely, symptomatic patients often experience episodes of abnormal uterine bleeding, dyspareunia, intermittent abdominal pain, urinary discomfort, constipation, and infertility. In young reproductive-age patients, obstetric causes should not be excluded. Ultrasound findings suggestive of multiple cysts in "grape-like" clusters, in the context of uterine bleeding and abdominal pain, should raise suspicion for molar pregnancy. This condition is characterized by abnormal fertilization due to chromosomal anomalies, where one or two sperm fertilize an empty ovum (complete hydatidiform mole; mainly 46, XX) or two sperm fertilize a normal ovum (partial hydatidiform mole; mainly 69, XXY), resulting in villous edema and trophoblastic hyperplasia with or without embryonic development.

Complete molar pregnancy is considered a benign tumor with metastatic and malignant potential, occurring with an incidence of approximately 1 in 1,000 pregnancies. Currently, many patients are asymptomatic at diagnosis due to the widespread use of ultrasound in the first trimester.[6,7] Clinically, patients with a complete molar pregnancy may present with an enlarged uterus, often with a uterine height greater than expected for the gestational age, accompanied by elevated β-hCG levels frequently exceeding 100,000 mIU/ml.[8] However, elevated β-hCG levels can cause saturation of antibodies bound to β-hCG, leading to a "false negative" or "Hook Effect" in the diagnosis. This phenomenon occurs when extremely high concentrations of β-hCG oversaturate the antibodies used for detection, resulting in falsely low readings despite high actual hormone levels.[9] The initial study is usually ultrasound due to its accessibility and

low cost. In complete molar pregnancy, ultrasound reveals anechoic cystic spaces resembling "grape clusters" with an echogenic mass and a characteristic "snowstorm" appearance.[10]

Diagnosis

Currently, the differential diagnosis of cystic tumors includes complex adnexal masses, molar pregnancy, and uterine fibroids with cystic degeneration. However, due to their low prevalence, diagnosis remains challenging as there are no specific biomarkers to differentiate these entities. The elevation of certain markers, such as CA-125, commonly used for ovarian cancer evaluation, further complicates the diagnostic dilemma. It has been demonstrated that peripheral CA-125 levels can be elevated in large uterine fibroids due to stretching and irritation of the overlying peritoneum.[11] Through studies on genomic imprinting, aberrant gene expression such as the absence of p57 protein (encoded by the NLRP7 gene) has been isolated and used as a valuable supportive marker complementing genetic evaluations of molar disease.

Approximately 50 mutations in the NLRP7 gene and around 10-14% involving KHDC3L are implicated in the development of hydatidiform moles.[12] Pelvic ultrasound is usually the first imaging modality performed in women with pelvic symptoms. It is useful for detecting a pelvic mass but has significant limitations in evaluating tumors located deep within the pelvis or near gas-containing organs. This is because ultrasound waves can be interrupted or scattered by air or gas, reducing image quality and limiting the evaluation of structures obscured by these factors.[13] On ultrasound, fibroids typically appear hypoechoic with posterior acoustic shadowing, known as the "Venetian blind artifact," which is considered a very characteristic sonographic feature. On Doppler ultrasound, they may show a peripheral blood flow pattern.[14,15]

Computed axial tomography (CT) has limited value in the pelvis due to its low contrast resolution with soft tissues. Various authors such as Winarto H. and colleagues have reported cystic fibroids that, during ultrasound examination, simulated ovarian malignancy.[16] Preoperative sampling of uterine tumors is often difficult due to their variable location; therefore, magnetic resonance imaging (MRI) is currently used for preoperative evaluation of tumor size and location. MRI offers multiplanar capability and excellent soft tissue contrast.[17] Its role is fundamental when ultrasound characteristics are inconclusive or atypical. However, the high cost and limited accessibility of MRI restrict its use in routine gynecological practice.[18] Cystic fibroids typically show low signal intensity on T1-weighted images (T1W) and high signal intensity on T2-weighted images (T2W), with no enhancement of the cystic areas. During the study, they behave like fluid with increased signal on T2 and absence of enhancement.[19,20] Macroscopically, cystic lesions can be differentiated.

Molar pregnancy, specifically hydatidiform mole, is characterized by clusters of vesicles that develop from the transformation of poorly vascularized chorionic villi with hydropic swelling and myxomatous and edematous stroma. This creates a fluid-filled cistern. It is associated with increased expression of the endothelial growth factor receptor in trophoblastic cells and absence of p57 expression, reflecting the androgenic origin of complete moles.[21] The definitive diagnosis of uncertain pelvic tumors is established through histopathology. Complete and partial hydatidiform moles are characterized by an overgrowth of villous trophoblasts with cystic, swollen villi. In contrast, fibroids are characterized by abundant muscle cells associated with loose connective tissue.[6]

CASE REPORT

Female, 22 years old, presents to the emergency department reporting heavy transvaginal bleeding with a progressive course over two weeks, associated with abdominal pain and increased abdominal girth. The vital signs were normal, with a blood pressure of 120/80 mmHg, a heart rate of 70 beats per minute, a temperature of 36.5°C, and a respiratory rate of 16 breaths per minute. Physical examination reveals increased abdominal diameter and a palpable intrapelvic mass located in the hypogastric region and right iliac fossa, mobile and non-tender.

Laboratory tests show hemoglobin of 9.0 g/dl, leukocytes 6,000 cells/mm³, neutrophils 74%, negative hCG, creatinine 0.6 mg/dl, C-reactive protein 0.1 mg/dl, INR 1.1, and prothrombin time 10.1 seconds. On physical examination, a pelvic mass approximately 18 cm in size is palpable in the hypogastric region and left iliac fossa, non-mobile and non-tender. Transvaginal ultrasound reveals a multilobulated cystic image with lacunae and solid areas, located subendometrially, with scarce vascularity on color Doppler (Figure 1).

Due to the presence of a complex pelvic mass, a computed axial tomography (CT) scan was requested, revealing a uterus with a cystic lesion originating from the anterior and left lateral wall, displacing the endometrium. Multiple prominent septa and some solid areas enhancing with contrast during the portal venous phase were observed, measuring 10.1×9.4×9.1 cm (Figure 2). The atypical finding of heterogeneous cystic septa prompted magnetic resonance imaging (MRI), which confirmed an oval-shaped lesion with regular borders located in the anterior uterine body, measuring 443 cm^3 in volume. The lesion exhibited heterogeneous signal intensity compared to the myometrium on fluid-sensitive sequences, with thin and thick septa up to 5.6 mm, isodense to myometrium on T1 and T2 sequences, hyperintense on diffusion-weighted imaging, and showing enhancement (Figure 3).

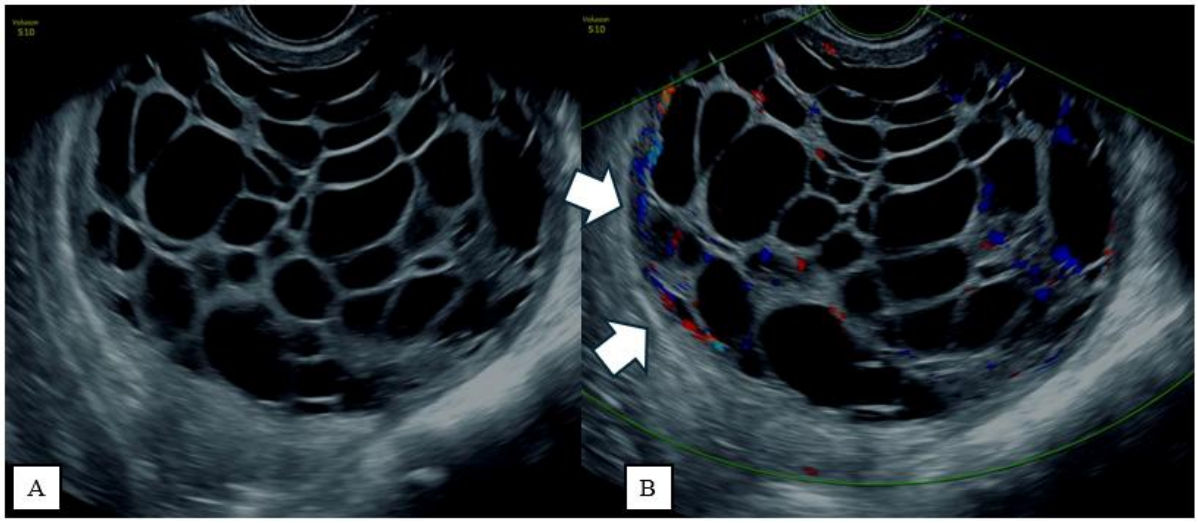

Figure 1: Endovaginal ultrasound.

(A) Multiloculated cystic image with solid areas, located subendometrially on the left anterior wall, entirely intramural, contacting both the endometrium and serosa and (B) low vascularity is observed on color Doppler imaging. White arrows indicate the hyperechoic endometrium.

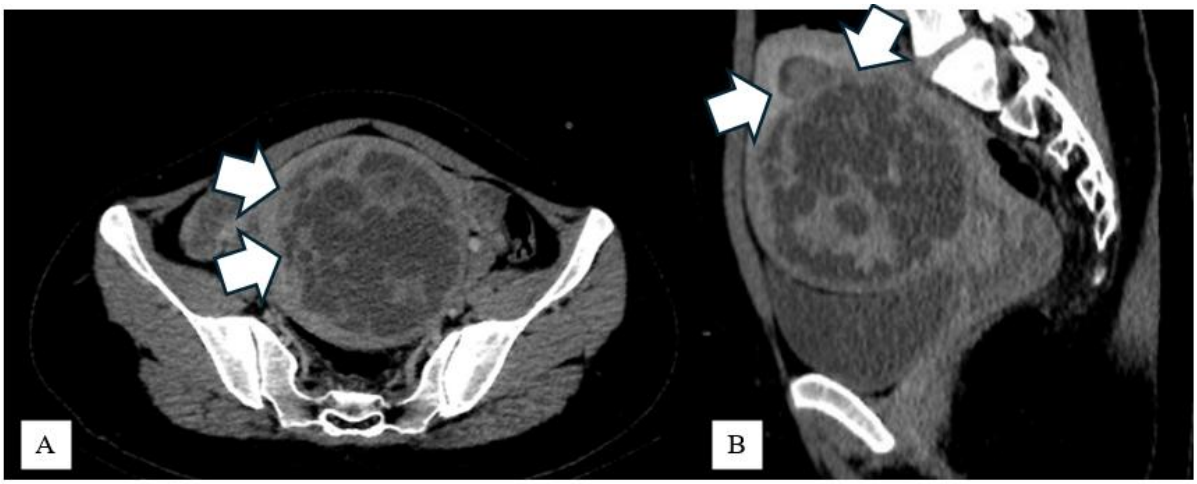

Figure 2: Computed tomography.

(A and B) Pelvic cystic image dependent on the anterior and left lateral wall, displacing the endometrium, 100% intramural from endometrium to serosa, with multiple septa and some solid portions. It measures 10.1×9.4×9.1 cm. Endometrium contains hyperdense material; white arrows indicate the endometrium.

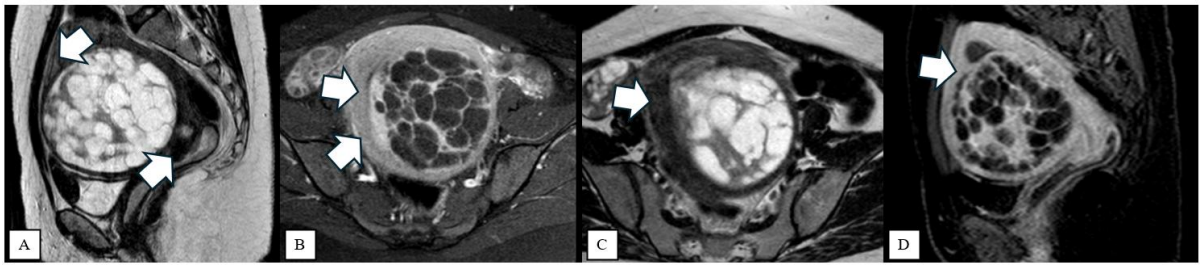

Figure 3: Magnetic resonance imaging.

(A and D) Heterogeneous appearance of the myometrium secondary to an oval-shaped image with regular and well-defined borders, located in the anterior body (left parasagittal) and fundal body, extending to the isthmo-cervical junction, measuring 9.5×9.4×9.5 cm with a volume of 443 cc and (B-D) lateral and posterior displacement of the endometrium as well as of the anterior and posterior pelvic structures. The lesion shows heterogeneous signal intensity in myometrium-susceptible fluid sequences, associated with fine and thick septa isodense to myometrium on T1 and T2, measuring up to 5.6 mm, hyperintense on diffusion sequences with enhancement. White arrows indicate the hyperintense endometrium.

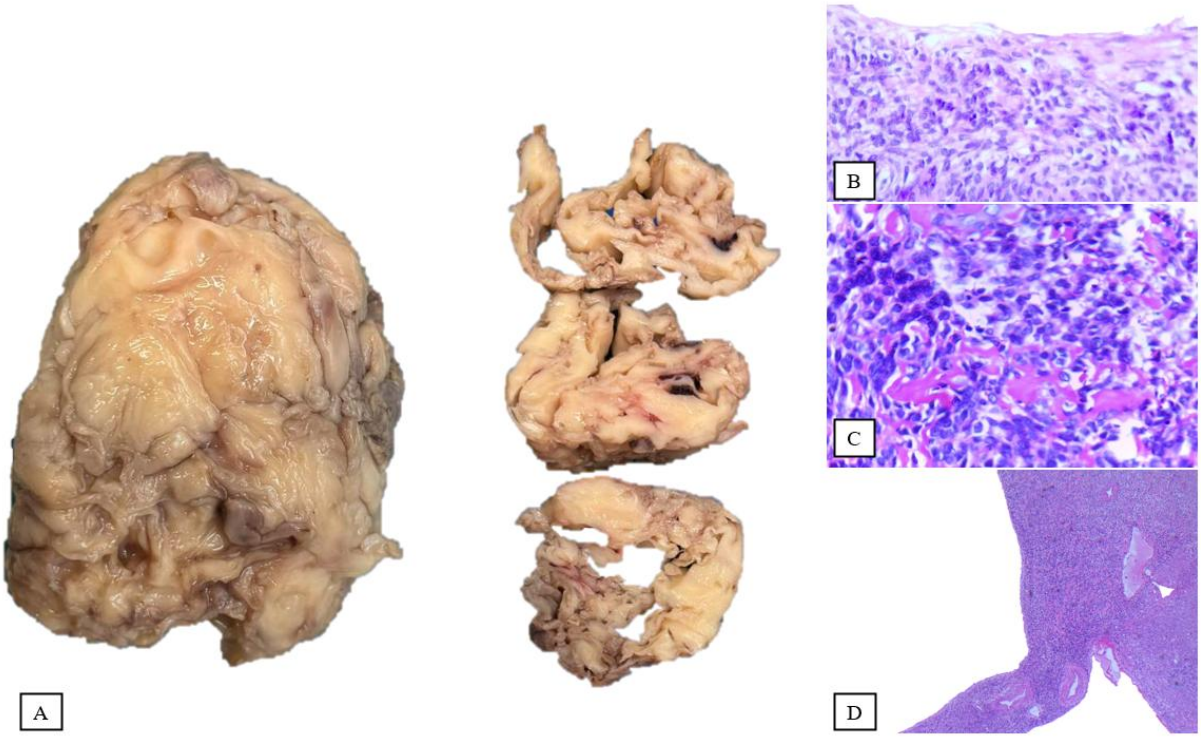

Figure 4: Histopathology.

(A) An irregularly ovoid specimen measuring 7.0×5.0×2.5 cm; light brown, rubbery consistency, with a white, pearly, swirled, and solid appearance on cut surface. Histopathological diagnosis: Common leiomyoma with cystic degeneration measuring 7.0×5.0×2.5 cm and (B-D) sections stained with hematoxylin and eosin. Samples from solid portions and septa showing spindle-shaped cells consistent with smooth muscle morphology.

The diagnosis of cystic degenerative fibroid was established. An exploratory laparotomy was performed, and a degenerative fibroid measuring 7×4.2 cm was excised by myomectomy using standard technique. The fibroid specimen was sent for pathological examination, confirming an irregular ovoid leiomyoma measuring 7.0×5.0×2.5 cm, with solid and heterogeneous content. On sectioning, it appeared pearly white, swirling, and solid in texture. Histopathological diagnosis: Leiomyoma with cystic degeneration measuring 7.0×5.0×2.5 cm (Figure 4). Microscopic examination with hematoxylin and eosin staining showed endometrial cells and abundant extracellular matrix. The solid portions and septa contained spindle-shaped smooth muscle cells confirming the diagnosis. Postoperative follow-up was conducted according to international guidelines, including antibiotic therapy, analgesia, and thromboprophylaxis. The patient was discharged 24 hours post-surgery without complications.

DISCUSSION

The treatment is determined by the diagnostic suspicion. Hysterectomy or myomectomy, through various approaches, is the treatment for uterine fibroids. Hysterectomy involves the complete removal of the uterus and is clearly superior in controlling bleeding and pain compared to myomectomy (showing increased hemoglobin levels and pain control in 70-90% of patients at 2 years follow-up). It is recommended as a definitive treatment in patients who do not desire future pregnancies and understand the risks associated with major surgery.[21] Molar pregnancies can be evacuated under ultrasound guidance and general anesthesia. However, in complete molar pathology, recurrence is frequent, and future pregnancy desire must be considered. Hysterectomy with salpingectomy is recommended therapeutically and to prevent gestational trophoblastic neoplasia in 80% of cases.[20]

To monitor gestational trophoblastic neoplasia, an hCG assay capable of detecting all forms of hCG should be used, including beta-hCG, core hCG, C-terminal hCG, free beta, core beta, and preferably the hyperglycosylated forms.[22] The risk of gestational trophoblastic neoplasia following complete mole is reported at 25%. Beta-hCG levels should be monitored every 1–2 weeks until normalized. Once normalized, monthly monitoring for 6 months is recommended in women with complete hydatidiform mole. For partial mole cases, an additional normal monthly measurement is advised, according to the recommendations of the International Federation of Gynecology and Obstetrics (FIGO).[23] In clinical case, treatment is performed according to the international recommendations for managing uterine myomatosis by FIGO. They suggest that in women with reproductive desire, myomectomy consists of the selective removal of the degenerated myoma via laparoscopic or open

approach, depending on the size, number, and location of the fibroid. For women without reproductive desire, comorbidities must be considered due to the high risk of bleeding, chronic anemia, injury to adjacent organs, and/or pelvic thrombosis from venous steatosis.

CONCLUSION

Gynecological pathology often presents a diagnostic challenge for the ultrasonographer and the field of gynecology. The study of abnormal uterine bleeding in reproductive-aged patients usually begins with ultrasound imaging. The presence of a solid mass with acoustic shadowing raises the possibility of a uterine fibroid diagnosis. However, atypical presentations with cystic and multiple lesions can mimic entities such as complex adnexal cysts or hydatidiform mole, considering the saturation of beta-HCG receptors. Currently, magnetic resonance imaging enhances diagnostic certainty and anatomical relationships with various structures, allowing for therapeutic strategy planning. In the presented clinical case, establishing a definitive diagnosis is fundamental to determine the risk of recurrence, metastasis, and to plan follow-up.

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REFERENCES

1. American College of Obstetricians and Gynecologists. ACOG Practice Bulletin No 96: Alternatives to hysterectomy in the management of leiomyomas. Obstet Gynecol. 2008;112(1):387–400.
2. Anyanwu M, Gassama K, Kandeh M. Diagnostic dilemma of hyaline cystic degeneration of uterine fibroids. Obstet Gynecol Int J. 2019;10(3):202–5.
3. Babacan A, Kizilaslan C, Gun I, Muhcu M, Mungen E, Atay V. CA 125 and other tumor markers in uterine leiomyomas and their association with lesion characteristics. Int J Clin Exp Med. 2014;7(4):1078–83.
4. Coyle C, Short D, Jackson L, Sebire NJ, Kaur B, Harvey R, et al. What is the optimal duration of human chorionic gonadotrophin surveillance following evacuation of a molar pregnancy. A retrospective analysis on over 20 000 consecutive patients. Gynecol Oncol. 2018;148(2):254–7.
5. Ghassemzadeh S, Farci F. Hydatidiform Mole. In: StatPearl. Treasure Island (FL): StatPearls Publishing. 2025.
6. Cunningham RK, Horrow MM, Smith RJ, Springer J. Adenomyosis: a sonographic diagnosis. Radiographics. 2018;38(5):1576–89.
7. Dibaba AD, Ljani N, Mustafa A, Khanbhai H, Ntungi A, Secha A, et al. Complete cystic degeneration of a uterine myoma posing a diagnostic dilemma. Case Rep Radiol. 2025;2:5627017.
8. Gonzalez J, Popp M, Ocejo S, Abreu A, Bahmad HF, Poppiti R. Gestational trophoblastic disease: complete versus partial hydatidiform moles. Diseases. 2024;12(3):159.
9. Hui P. Germline NLRP7 mutations: genomic imprinting and hydatidiform mole. Virchows Arch. 2020;476(2):175–6.
10. Kaushik C, Prasad A, Singh Y, Baruah BP. Case series: cystic degeneration in uterine leiomyomas. Indian J Radiol Imaging. 2008;18(1):69–72.
11. Kim HJ, Lee WY, Kim YH, Jin HJ, Jeong MJ, Cho HJ, et al. Effects of phthalate esters on human myometrial and fibroid cells: cell culture and NOD-SCID mouse data. Reprod Sci. 2021;28(2):479–87.
12. Lin Y, Wu RC, Huang YL, Lin CR, Yu MH, Liu YH, et al. Uterine fibroid-like tumors: spectrum of MR imaging findings and their differential diagnosis. Abdom Radiol (NY). 2022;47(6):2197–208.
13. Lobo Antunes I, Curado J, Quintas A, Pereira A. Negative β-hCG and molar pregnancy: the hook effect. Acta Med Port. 2017;30(9):656–8.
14. Lukinovic N, Malovrh EP, Takac I, Sobocan M, Knez J. Advances in the diagnosis and treatment of gestational trophoblastic disease. Radiol Oncol. 2022;56(4):430–9.
15. Madueke-Laveaux OS, Elsharoud A, Al-Hendy A. What we know about the long-term risks of hysterectomy for benign indication: a systematic review. J Clin Med. 2021;10(24):5335.
16. Mension E, Calaf J, Chapron C, Dolmans MM, Donnez J, Marcellin L, et al. An update on the management of uterine fibroids: personalized medicine or guidelines. J Endometr Uterine Disord. 2024;7(1):100080.
17. Morena López M, Vallejo Ribera SR, Valbuena Durán EJ, Bonilla López L, Jiménez Relimpio C, López Ruiz L, et al. Assessment of uterine fibroids: what the radiologist should know. SERAM. 2024.
18. Ngan HYS, Seckl MJ, Berkowitz RS, Xiang Y, Golfier F, Sekharan PK, et al. Update on the diagnosis and management of gestational trophoblastic disease. Int J Gynaecol Obstet. 2015;131(2):123–6.
19. Nougaret S, Nikolovski I, Paroder V, Vargas HA, Sala E, Carrere S, et al. MRI of tumors and tumor mimics in the female pelvis: anatomic pelvic space-based approach. Radiographics. 2019;39(4):1205–29.
20. Okizuka H, Sugimura K, Takemori M, Obayashi C, Kitao M, Ishida T. MR detection of degenerating uterine leiomyomas. J Comput Assist Tomogr. 1993;17(5):760–6.
21. Pavone D, Clemenza S, Sorbi F, Fambrini M, Petraglia F. Epidemiology and risk factors of uterine fibroids. Best Pract Res Clin Obstet Gynaecol. 2018;46:3–11.
22. Baasanjav B, Usui H, Kihara M, Kaku H, Nakada E, Tate S, et al. The risk of post-molar gestational trophoblastic neoplasia is higher in heterozygous

than in homozygous complete hydatidiform moles. Hum Reprod. 2010;25(5):1183–91.
23. Winarto H, Simatupang ONN, Calvin D, Siregar TP, Andrijono. Diagnostic challenge: distinguishing uterine fibroid with cystic degeneration vs. ovarian cystic malignancy. J Radiol Case Rep. 2023;17(4):1–12.

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