Case Report

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Tumor rupture and partial gut obstruction: Atypical presentations in a patient with adenomyosis

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Abstract:

A 49-year-old woman, Gravida 8 Para 8 (8007), came in due to vomiting and enlarging abdominal mass. Initial diagnosis was partial gut obstruction and acute kidney injury probably secondary to adenomyosis versus colonic pathology. Ultrasound showed adenomyosis but computed tomography scan showed a uterine mass with possible tumor rupture and mass effects. Emergency hysterectomy was done and showed an ill-defined endometrial mass with multiple areas of rupture. It was diagnosed with malignant but final histopathology revealed extensive adenomyosis with acute inflammation and necrosis with no malignancy identified. Unusual symptoms such as uterine rupture and mass effects can accompany adenomyosis, alongside typical signs like pain and bleeding. Ultrasound aided the diagnosis, although it missed uterine rupture, highlighting its limitations. Magnetic resonance imaging could have been useful. Ultimately, histopathology is the gold standard for diagnosing adenomyosis.

Keywords:

Adenomyosis, necrosis, tumor rupture

Introduction

The International Federation of Gynecology and Obstetrics classification system categorizes abnormal uterine bleeding (AUB) into polyp, adenomyosis, leiomyoma, malignancy (PALM) (structural) and Coagulopathy, Ovulatorydysfunction, Endometrial, Iatrogenicand Notyetclassified. COEIN (nonstructural) groups. Adenomyosis, a PALM entity, is designated as AUB–adenomyosis or AUB-A.[1] It is defined as the presence of endometrial glands and stroma in the myometrium. Diagnosis relies on histopathology, through imaging like ultrasound, computed tomography (CT) scan or magnetic resonance imaging (MRI) aids diagnosis. Ultrasound is recommended for structural abnormalities due to its noninvasiveness and real-time imaging.[2] Adenomyosis may mimic other uterine pathologies such as leiomyomas and uterine malignancies. The case presented involves tumor rupture from adenomyosis.

Case Report

We are presented with the case of a 49-year-old, Gravida 8 Para 8 (8007), who came in due to vomiting.

She is a known case of bronchial asthma and hypertension with no maintenance medications. She had no diabetes mellitus, cardiac diseases, and pulmonary tuberculosis. She has no previous surgeries or hospitalizations requiring intensive care. There are no heredofamilial diseases noted.

She is a nonsmoker, nonalcoholic beverage drinker and denies any illicit drug use. She had no known allergies.

She had her menarche at 12 years old, occurring at regular intervals lasting for

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7 days consuming 2 pads per day, moderately soaked, with no note of dysmenorrhea. Her last normal menstrual period is 2 weeks before admission (PTA).

She had her first coitus at 15 years old with 1 nonpromiscuous partner. They have been married for 13 years.

She is a Gravida 8 Para 8 (8007). All pregnancies were carried to term, delivered vaginally. She had unremarkable deliveries except for her eighth pregnancy where she was diagnosed with preeclampsia. Her fifth baby died a month after birth due to meningitis. She underwent bilateral tubal ligation in 2004.

Three months PTA, the patient complained of heavy menstrual bleeding, consuming 3 diapers per day lasting for 60 days with increasing abdominal girth. No other symptoms were noted. She consulted at our institution where internal examination was done which showed that the cervix measured 3.0 cm × 3.0 cm × 3.0 cm, and corpus enlarged to 16–18 weeks size with no adnexal masses nor tenderness noted. Transvaginal (TVS) with transabdominal ultrasound (TAS) were done which showed an enlarged uterus (20.0 cm × 16.3 cm × 11.0 cm) with adenomyosis and a left physiologic cyst [Figure 1]. She was given depot medroxyprogesterone acetate (DMPA) intramuscularly for three doses with resolution of the heavy menstrual bleeding.

Four days PTA, she had nonbloody, nonmucoid, postprandial vomiting which progressed to vomiting of bilous and fecaloid material the following day. There was no diarrhea, fever, or dysuria noted. However, she did not seek consult nor take any medications at this time.

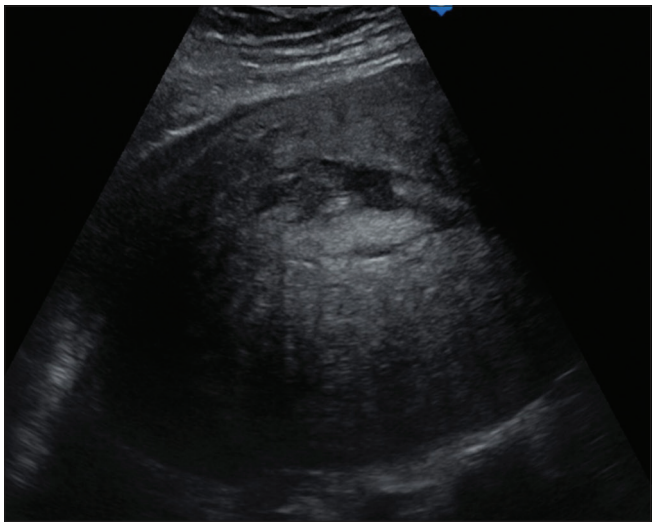

Figure 1: Transabdominal ultrasound showing asymmetrically thickened myometrial walls with venetian blind shadowing and endometrial masses

One day PTA, due to persistence of vomiting, now with anorexia, generalized body weakness and decreased urine output, she sought consult and was subsequently admitted to the surgery department of our institution. She had no fever, diarrhea, or melena.

On physical examination, she was conscious, coherent, in cardiorespiratory distress. She was tachycardic at 135 beats per min and tachypneic at 29/min, but normotensive and afebrile. She was orthopneic, complaining of thirst. Her body mass index was 28 kg/m^2. She had pale palpebral conjunctiva, anicteric sclera, and dry buccal mucosa. On chest examination, she had symmetric chest expansion with bibasal crackles. She had tachycardia, regular rhythm with no murmurs. On abdominal examination, the abdomen was globular, distended with tenderness on the upper quadrants. Pulses were full and equal with grade 3 bipedal edema. On digital rectal examination, there was intact rectal vault, good sphincteric tone, no blood per examining finger. On internal examination, she had normal external genitalia, parous vagina, smooth cervi × 3 cm × 3 cm × 3 cm, corpus and bilateral adnexa difficult to assess due to the enlarged abdomen and generalized abdominal tenderness.

The admitting impression was: (1) Hyperkalemia secondary to acute kidney injury (pre-renal from gastrointestinal (GI) losses on top of Chronic Kidney Disease (CKD) from hypertensive kidney disease; (2) Hyponatremia and hypochloridemia also from GI losses; (3) Sepsis from possible intraabdominal infection. There was also consideration for (4) Ileus or or partial gut obstruction, the latter probably due to an underlying colonic or gynecologic pathology. Included in the diagnoses also are (5) Complicated urinary tract infection; (6) AUB secondary to adenomyosis with adenomyoma; (7) Bronchial asthma, not in acute exacerbation; (8) Hypertension Stage II; (9) Obesity Class I; (10) Gravida 8 Para 8 (8007).

She was referred to nephrology and gynecology services. The patient was hydrated and serum electrolytes were serially monitored. She was placed on nothing per orem. Nasogastric tube showed bilous drainage and decreased urine output was noted. Omeprazole 40 mg intravenous (IV) once a day (OD), cefoxitin 2 g IV OD, and paracetamol 600 mg IV every 6 h were started.

Her laboratory examinations revealed an elevated serum creatinine with decreased glomerular filtration rate. Complete blood count showed anemia and leukocytosis. She was transfused with three units of packed red blood cells (RBCs) and 1 unit of fresh-frozen plasma. Urinalysis showed urinary tract infection.

TVS with TAS showed an enlarged uterus (measuring 18.2 cm × 18.5 cm × 15.2 cm) with findings suggestive

of adenomyosis and endometrial masses probably (1) blood clots and (2) endometrial polyp. The uterus had globular contour and heterogeneous echo pattern. The myometrium was asymmetrically thickened measuring 3.2 cm anteriorly and 8.4 cm posteriorly with ill-defined borders, casting posterior linear acoustic shadows. Within the endometrial cavity were two echogenic masses seen: Mass 1: 4.8 cm × 4.8 cm × 1.6 cm attached at the posterior mid-corpus and mass 2: 3.8 cm × 1.9 cm × 1.1 cm (volume: 4.0 cc) attached at the anterior mid-corpus, with cystic spaces within. Color flow mapping of the endometrial masses showed absent vascularity with a color score of 1 [Figure 2]. The right ovary was not visualized. The left ovary was normal and measured 3.2 cm × 1.8 cm × 2.3 cm (volume: 6.7 cc). There were no adnexal masses seen. There is moderate, septated anechoic free fluid in the abdominopelvic cavity.

CT scan with contrast of the abdomen was done on the second hospital day which revealed a large necrotic uterine mass with probable contained tumoral rupture and associated mass effects including partial jejunal obstruction; cannot rule out bilateral adnexal involvement [Figure 3]. At this time, she had distended tympanitic abdomen with no guarding. She had flatus with decreased caliber of stools.

The patient was referred to the Gynecologic Oncology Service and was scheduled for emergency laparotomy.

Intraoperatively, there was minimal serosanguinous ascites admixed with fibrin and purulent material. The parietal peritoneum was smooth. The liver, subdiaphragmatic surface, gall bladder, appendix, and spleen were grossly normal. The intestines and omentum were enveloped by a thin layer of fibrinous material. The uterus had irregular contour and measured 20.0 cm × 22.0 cm × 10.0 cm. There were multiple areas of rupture at the fundal and posterofundal area, which extruded purulent material. On cut section, the cream-white myometrium measures 4.0 cm anteriorly and 7.5 cm posteriorly. Within the endometrial cavity was a 10.0 cm × 10.5 cm × 2.0 cm friable and necrotic mass which on cut section exhibited full myometrial invasion at the fundus and posterior mid-corpus. The lower edge of the mass was 2.5 cm from the cervical OS. The uterine cavity was 12.5 cm deep, 2.5 cm of which was the endocervical canal. The cervix measured 4.0 cm × 3.0 cm × 2.5 cm, grossly normal [Figure 4]. The right ovary measured 4.0 cm × 3.0 cm × 1.5 cm while the right fallopian tube measured 12.0 cm × 0.5 cm and were grossly normal. The left ovary measured 5.5 cm × 3.5 cm × 1.5 cm, while the left fallopian tube measured 11.0 cm × 0.5 cm and was grossly normal. Estimated blood loss was 2500 ml. She was transfused with four units of packed RBC and 1 unit fresh-frozen plasma intraoperatively. The patient underwent exploratory laparotomy, extrafascial hysterectomy with bilateral salpingo-oophorectomy, selective lymph node sampling, biopsy of bladder, and Jackson-Pratt drain insertion under general anesthesia.

Postoperative diagnoses were partial gut obstruction secondary to uterine mass; uterine mass probably malignant, with tumor rupture, intraoperative stage IIIA; acute kidney injury from (1) obstructive uropathy, (2) sepsis, on top of CKD secondary to hypertensive kidney disease; sepsis from (1) infected endometrial mass and (2) complicated urinary tract infection; Bronchial asthma not in acute exacerbation; Hypertension stage II; Obese Class I; Gravida 8 Para 8 (8007).

She was referred to the Infectious Disease Service and was assessed with complicated urinary tract infection (no isolate) secondary to obstructive uropathy; to consider community acquired pneumonia; intra-abodominal infection not highly considered.

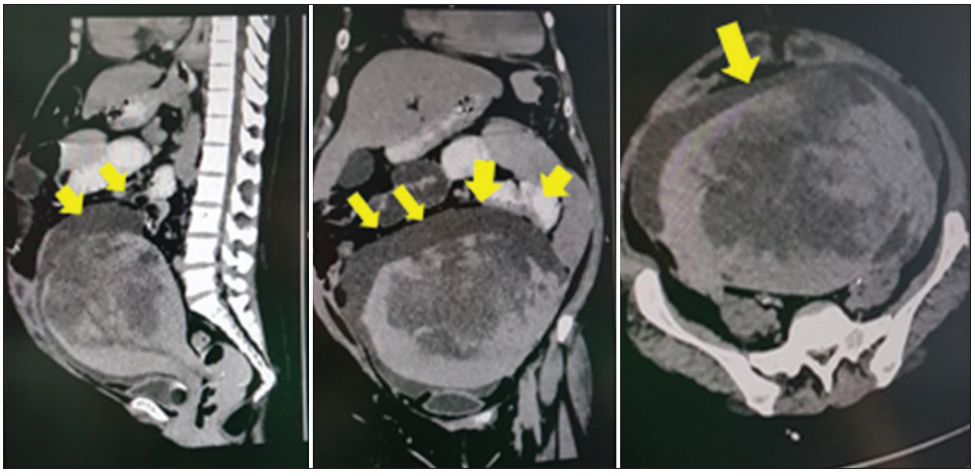

Figure 2: Computed tomography scan showing partial jejunal obstruction and tumor rupture (arrows)

Diet progression, control of hypertension, and completion of antibiotics were done. However, a superficial surgical site infection was noted on the seventh postoperative day. There was wound dehiscence measuring 2 cm × 2 cm, 5 cm below the umbilicus, with serosanguinous discharge. TAS showed intact fascia. Daily wound cleaning was done. Antibiotics were continued. Wound culture and sensitivity showed no growth on the 13th postoperative day. She was then discharged, stable, and improved.

The final histopathology report showed extensive adenomyosis with acute inflammation and areas of necrosis. No definite malignancy was identified. On histopathology, there was a tan-gray to brown, fleshy to friable, exophytic mass measuring 11.0 cm × 9.5 cm occupying mostly the posterior endometrium up to the uterine isthmus and is 2.5 cm away from the external OS. Serial sections of the mass showed that it has not infiltrated deep into the myometrium. The uninvolved tan-brown endometrium measures 0.3 anteriorly while the remaining cream tan, whorled, trabeculated myometrium measures 8.5 cm anteriorly and 8.4 cm posteriorly. Microscopic sections showed ectopic endometrial glands within the myometrium with areas of inflammation and necrosis [Figures 5 and 6].

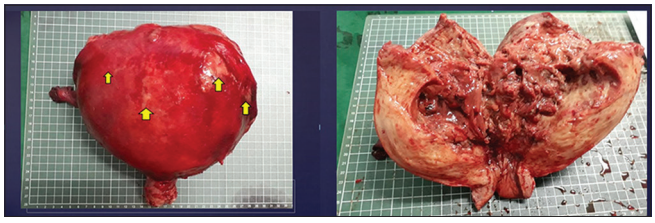

Figure 3: Gross specimen (left) showing globularly enlarged uterus with multiple sites of rupture (arrows) with thickened myometrial walls and irregular endomyometrial mass with necrosis (right)

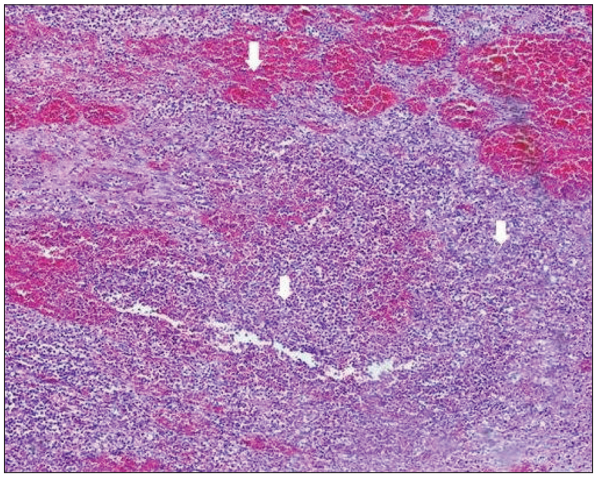

Figure 5: Scanning view of the myometrium showing diffuse inflammation and necrosis (white arrows)

Discussion

Adenomyosis is a pathological condition characterized by the presence of endometrial glands within the myometrium.[3] It is generally agreed that adenomyosis occurs when the normal boundary between the endometrial basal layer and the myometrium is disrupted.[4] It has two types: Diffuse and focal, which is also named "adenomyoma" in the literature. The posterior myometrial wall is more often affected as compared to the anterior and lateral sides of the uterus.[5]

Historically, the prevalence of adenomyosis varies from 5% to 70% in hysterectomy specimens.[6] A recent study in more than 300,000 women showed that the overall incidence adenomyosis was 1.03% or 28.9 per 10,000 woman-years. The incidence was the highest for women aged 41–45 years. At their respective peaks, it was higher for black versus white women (44.6 vs. 27.9 per 10,000 woman-years). The incidence in Asians was at 8% of the total population.[7]

The exact pathogenesis of adenomyosis is still unknown. Several theories have been proposed, but no theory has been experimentally proven as of yet. The most

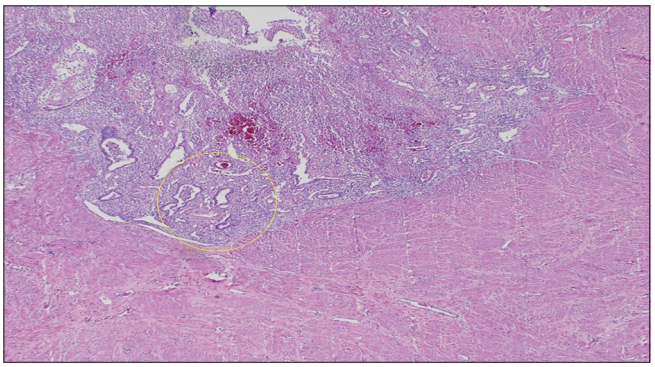

Figure 4: Histopathology showing adenomyosis

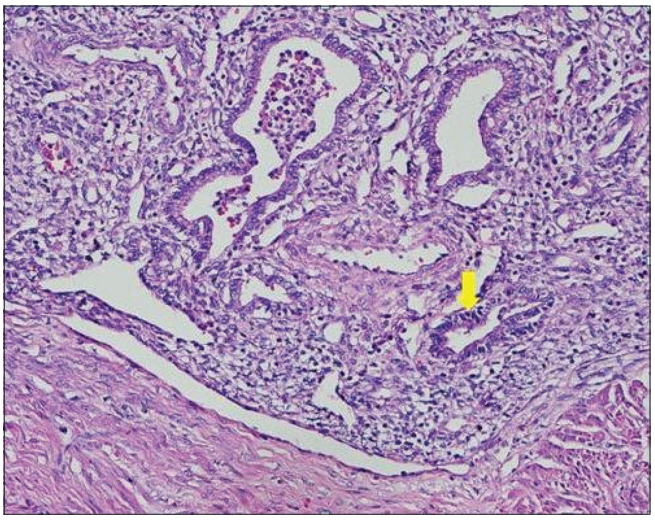

Figure 6: High power view showing endometrial glands and stroma (yellow arrow) within the myometrium

accepted theories are: (1) endometrial invagination of the myometrium via an altered or absent junctional zone (JZ);[8,9] (2) repeated hypercontractility causing uterine auto-traumatization and tissue injury and repair mechanism which damages the JZ and migration of ectopic endometrial tissue;[10] and (3) the role of embryonic or adult stem cells which may undergo metaplasia into the myometrium, as a *de novo* process.[11] Several studies have also implicated pathogenic mediators such as sex steroid hormone receptors aberrations, inflammatory molecules, extracellular matrix enzymes, growth factors, and neuroangiogenic factors.[12]

Unfortunately, there is no pathognomonic symptom that is characteristic of adenomyosis and up to 30% of women may even be asymptomatic.[13] The possible symptoms include AUB (such as heavy or prolonged menstrual bleeding, intermenstrual bleeding, or premenstrual spotting), infertility and recurrent miscarriage, local pressure symptoms, bladder and gastrointestinal symptoms (such as dysuria and dyschezia), and pain symptoms such as dysmenorrhea, dyspareunia, and chronic pelvic pain.[14]

There are multiple studies regarding the multiple risk factors for adenomyosis. However, most of the studies have not been correctly designed to identify significant increases in relative risk.[14] The suggested risk factors include parous middle-aged women ages 40–50 years old, multiparity, sex hormones, smoking, previous uterine surgical trauma such as dilatation and curettage and cesarean section.[5] Although adenomyosis is found more on the 5th decade due to higher hysterectomy rates, there is an ultrasound study on women aged from 18 to 30 years which showed that adenomyosis features were present in more than 30% of young women, correlating with dysmenorrhea and AUB.[15] A similar MRI study on women aged < 42 years showed that isolated diffuse adenomyosis occurred in one-third of the study population (34.6%).[16] Previous uterine surgical trauma increases the odds for adenomyosis, through the mechanical endometrial invasion of the myometrium. There was also increasing risk with a higher number of abortions and cesarean sections.[17-19]

Atypical presentations of adenomyosis include uterine rupture, which was seen in our index case, hemoperitoneum, and internal bleeding. Risk factors for uterine rupture in adenomyosis include trauma, striking force on the abdomen, multiparity, uterine distension with or without multiple pregnancies, prolonged steroid use, infection, and a scarred uterus owing to cesarean section or myomectomy. Peng *et al*. described uterine rupture and massive hemoperitoneum from uterine leiomyomas and adenomyosis in a nongravid and unscarred uterus.[20] In another case report of a primigravid, extensive adenomyosis with marked decidualization resulted to separating of the myometrial smooth muscle fibers and weakening of the myometrium, which lead to uterine rupture. Sixteen cases of uterine rupture in a gravid uterus related to adenomyosis have also been reported, with the locations of the rupture being the fundus, isthmus, or posterior cervical uterine junction.[21] During pregnancy, adenomyosis can adversely affect the obstetrical prognosis because of structural abnormalities of the myometrium caused by the hormonal changes of the pregnancy. Profuse decidual transformation of stromal cells in adenomyosis can lead to atrophy and necrosis of the myometrium with reduction of the uterine muscle mass, consequently causing uterine rupture.[22] The spontaneous uterine rupture of a twin pregnancy after laparoscopic adenomyomectomy has also been published.[23] Other complications of adenomyosis in pregnancy include higher rates of preterm delivery, preterm prelabor rupture of membranes, small for gestational age infants, fetal malpresentation, and caesarean section.[24]

TVS ultrasound remains to be the first line diagnostic tool for this disease. It is operator dependent and has a sensitivity ranging from 72% to 82% and a specificity that ranges from 81% to 85%.[25] Ultrasound findings include a globally enlarged uterus, heterogeneous myometrium, ill-defined lesions with no rim in diffuse adenomyosis, myometrial anteroposterior asymmetry, fan-shaped shadowing, myometrial cysts, hyperechogenic lines and buds, translesional flow on color flow mapping, thickened or irregular or ill-defined JZ, interrupted JZs even in the absence of localized lesions, and question mark form of the uterus.[26,27] The JZ is a highly specialized structure, identified in MRI studies of the uterus as the endometrial-myometrial junction or subendometrial halo in ultrasound as the hypoechoic tissue identified beyond the endometrial basal layer[14] For two-dimensional (2D) ultrasound findings, the presence of heterogeneous myometrium was the most sensitive at 86.0% and the question mark sign was the most sensitive at 92.3%. On three-dimensional ultrasound, poor definition of the JZ was the most sensitive at 87.8% and the hypoechoic linear striations were the most specific at 61.1%.[28] Color flow Doppler would show translesional flow while power Doppler studies would display vessels perpendicular to the endometrial interface.[8,25] For our index case, a globularly enlarged uterus, myometrial asymmetry with ill-defined JZ, fan-shaped shadowing and heterogeneous myometrium were noted on 2D ultrasound and minimal diffuse translesional flow on color flow mapping.

Furthermore, ultrasound was used to classify adenomyosis according to the involvement of the uterine layers. Type 1 when only the JZ is involved, Type 2 when the middle

myometrium (the layer between the JZ and the vascular arcade) is involved, and Type 3 if adenomyotic lesions are found in the outer myometrium.[29] In another study comparing the sonographic JZ and the histopathology results, the degree of invasion of adenomyosis were described as follows: (a) Adenomyosis of the inner myometrium defined as ≥2 mm myometrial invasion without contact to the basal endometrium, (b) serrated JZ defined as >3 mm myometrial invasion with contact to the basal endometrium or (c) linear JZ defined as no or marginal myometrial invasion ≤3 mm with contact to the basal endometrium.[30]

CT scan findings of adenomyosis may include globular uterine enlargement, thickened inner myometrium (>12 mm), and/or sub centimeter low attenuation myometrial lesions corresponding to myometrial cysts. Globular uterine enlargement is described as maximum transverse dimension on axial CT >6 cm, in the absence of or greater than that which could be explained by uterine leiomyomas alone. The normal size of the premenopausal adult uterus is approximately 4 cm × 5 cm × 8 cm in anteroposterior diameter, width, and length respectively. Parity increases the size by approximately 1 cm or greater in each dimension.[31] However, CT scan has poor diagnostic value due to similar images portrayed by adenomyotic foci and a normal myometrium.[32]

The CT scan of our index patient showed a large heterogeneously-enhancing, abdominopelvic mass arising from the uterine fundus with a large, irregular, central hypodense region, likely representing necrosis and appears to communicate inferiorly with the endometrial cavity. This may seem like the mass was communicating with the endometrium and malignancy could be a differential diagnosis. It also communicates anteriorly and superiorly through defects in the margins of the mass with a loculated hypodense abdominopelvic fluid collection that surrounds the mass and exhibits a maximum thickness of 2.8 cm; which seems to be the contained tumoral rupture.

MRI is also a noninvasive technique and a second-line tool in the diagnosis of adenomyosis. MRI has a sensitivity of 77% and a specificity of 89%.[25] Adenomyosis is evaluated using the T2 weighted sequence on MRI. The JZ is evaluated using these parameters: the thickening of the JZ at least 8–12 mm, the ratio of JZ maximum/total myometrium over 40%, and the difference between the maximum and the minimum thickness of the JZ (JZmax – JZmin) more than 5 mm.[33] A thickness of more than 12 mm is highly predictive of adenomyosis.[34,35] JZ <8 mm generally excludes the diagnosis of adenomyosis.[36]

Treatment of adenomyosis may be medical or surgical. Medical management includes management of symptoms such as nonsteroidal anti-inflammatory drugs or other analgesics for pain, hemostatic agents including tranexamic acid, levenorgestrel intrauterine device, and oral and injectable hormonal treatment such as oral contraceptive pills, progestins, GnRH agonist, and aromatase inhibitor. Surgical management includes fertility sparing surgery such as adenomyomectomy. Nonfertility sparing but uterus-sparing surgeries include endometrial ablation, high intensity focused ultrasound, and uterine artery embolization.[37] Finally, hysterectomy is the gold standard which immediately stops the bleeding and compression symptoms in patients who have no desire for future fertility.

In the index case, she initially presented with a history of profuse vaginal bleeding, uterine, enlargement and with sonologic findings of adenomyosis and was treated with DMPA. It then progressed to produce associated mass effects. Sonologic and CT scan findings all showed uterine enlargement causing compression of nearby abdominopelvic structures and eventually leading to acute renal failure and partial gut obstruction. Ultrasound findings include globularly enlarged uterus, myometrial asymmetry with ill-defined JZ, fan-shaped shadowing and heterogeneous myometrium. Since the JZ was already interrupted, there may be involvement of the basal endometrium. This is why there was a sonographic finding of endometrial masses which were not seen on histopathology. Although MRI allows for a better visualization of the JZ, it was not done due to the emergent nature of the disease in this patient.

Given that this patient was already perimenopausal who has completed her family size, with an enlarged uterus causing mass effects, with sonographic findings of adenomyosis and endometrial mass and CT scan findings uterine mass with possible tumoral rupture, the surgical team decided to do the definitive procedure which was emergency laparotomy, total abdominal hysterectomy. Grossly, a uterine mass with full myometrial invasion that involved the posterior endometrium up to the uterine isthmus with multiple points of rupture was seen, hence, uterine malignancy was considered and complete surgical staging was done. In a study by Teefey *et al*., gross inspection overestimated tumor invasion in 2 out of 15 cases.[38] In our case, there was full myometrial invasion on gross examination but no myometrial invasion on histopathology. The predisposing factor of this patient to adenomyosis was multiparity. The possible reason for the rupture is the uterine overdistention due to the presence of the adenomyosis. With continued growth of the mass, there was decrease in blood supply which led to uterine necrosis and eventual rupture.

Guilbeault *et al*. described an 18-year-old patient who had adenomyosis with massive uterine enlargement with necrosis. The patient presented with abnormal uterine bleeding and had multiple dilatation and curettage and

cervical polypectomy which showed polypoid endometrial hyperplasia without atypia and given hormonal treatments. Her pelvic sonograms showed rapid increase in uterine size in 2 months. With the rapid uterine enlargement, uterine sarcoma was considered. Emergency laparotomy showed a huge mottled necrotic uterus with a 5 mm perforation of its anterior left wall through which necrotic material protruded. The final histopathology result showed thickened myometrium with hemorrhagic and necrotic foci consistent with adenomyosis. The uterine enlargement was hypothesized to be due to prolonged exposure to hormonal treatment, upregulation of estrogen receptors resulting in increased metabolism, outgrowth of blood supply, ischemia, and disseminated intravascular coagulation.[39] These findings may also explain the presentation, imaging, and histopathologic picture of our patient.

The history of AUB and uterine enlargement coupled with the ultrasound finding helped in establishing the initial diagnosis of adenomyosis. CT scan finding of tumoral rupture and gross examination of the uterus led the surgeons to believe that the uterine mass was malignant. Endometrial carcinoma is reported to be found together with adenomyosis. A recent study showed that rate of co-existence of adenomyosis with endometrial cancer was 37.7% and as high as 40%–70% in older studies.[40-42] Although rare, adenocarcinoma has been reported to arise from adenomyosis.[43-47] Adenomyosis can mimic endometrial carcinoma at imaging and may result in staging errors when the 2 conditions co-exist.[38,48] In these cases, it is very difficult to determine whether the cancer is insinuating into pre-existing areas of adenomyosis or if there are areas of true myometrial invasion. This is important as myometrial invasion is used as a prognostic factor in endometrial cancer patients.[49] Even while using MRI, adenomyosis decreases the accuracy in assessing depth of invasion as it reduces the contrast between endometrial cancer adenomyosis-involved myometrium. Adenomyosis is therefore a co-founder making it difficult to assess myometrial invasion on imaging and challenging on histopathologic examination.[50]

Ultrasound was a useful tool in the pre-operative planning of this case, however it has its pitfalls too as the uterine rupture was not seen. MRI would have been an adjunct imaging modality for this case as CT scan may be unable to distinguish an adenomyotic foci from the normal myometrium. Histopathology remains to be the gold standard in diagnosing adenomyosis and hysterectomy is its definitive management.

Summary

We are presented with a 49-year old G8P8 (8007) who had uterine enlargement and heavy menstrual bleeding with eventual uterine necrosis and uterine rupture. Uterine enlargement led to a decrease in vascular supply consequently causing uterine necrosis and rupture. TVS with TAS helped in the preoperative planning and was superior to CT scan in diagnosing adenomyosis in this case. Adenomyosis is a benign pathology which can be co-existent with endometrial carcinoma. Distinguishing between these two entities may be difficult on imaging, gross examination, and histopathological examination. It is fortunate that the patient only had extensive adenomyosis. She underwent hysterectomy and bilateral salpingo-oophorectomy which was the treatment of choice. Although pelvic pain, AUB, and uterine enlargement are the usual manifestations of adenomyosis, we should always remember there may be other atypical presentations such as uterine rupture and compressive mass effects that may also be present, as was seen in the index case.

Authorship contributions

Dr. Ma. Patricia Grace O. Siao - Involved in the conceptualization, methodology, formal analysis, investigation, resources, data curation, writing of the original draft, review and editing, visualization, project administration, funding acquisition.

Dr. Izabelle Julienne A. Figueras-Prieto - Involved in conceptualization, formal analysis, review and editing of the draft, supervision.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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