Access this article online

Quick Response Code:

Website: www.pogsjournal.org

DOI: 10.4103/pjog.pjog_59_23

A life-threatening benign vascular lesion of the uterus, cavernous hemangioma: A case report

Maria Lalaine Aviles Miranda[1], Eleyneth Ilagan Valencia[1]

Abstract:

The objective of this clinical report is to present the first local case of rare cavernous uterine hemangioma. This is a case of a 28-year-old G2P1 (1001) during her first trimester of pregnancy who was admitted to our institution for the second time due to profuse vaginal bleeding and severe anemia. The transvaginal scan revealed an embryonic demise of 8 weeks age of gestation noted at the endocervical canal. There is a posterofundal heterogeneous mass measuring 6.3 cm × 5.7 cm × 5.0 cm (volume: 94.2 ml) with multiple cystic spaces, which on Doppler studies showed abundant vascularity suggestive of uterine hemangioma. Antifibrinolytics were administered. The patient underwent emergency hysterectomy with bilateral salpingectomy due to profuse vaginal bleeding with histopathology result of cavernous hemangioma of the uterus. Cavernous uterine hemangioma is a rare vascular lesion that poses a great challenge in the diagnosis and management. There were limited published articles regarding cavernous hemangioma of the uterus. There were conservative treatment options such as uterine artery embolization, the use of hormonal oral contraception, intralesional glucocorticoid therapy, the use of interferon-α, laser therapy, and surgical excision. Hysterectomy is the definitive treatment for intractable bleeding. It is recommended to establish an international registry for this rare case. The experts in different specialties such as obstetrician-gynecologists, interventional radiologists, and vascular surgeons, can also formulate an algorithm for its diagnosis and treatment.

Keywords:

Case reports, hemangioma, vascular malformations

Introduction

Vascular anomalies (benign soft-tissue lesions) involving the adult female genital tract are extremely uncommon. The International Society for the Study of Vascular Anomalies reclassified vascular lesions into tumors and malformations.[1] According to this classification, vascular anomalies referred to as hemangioma are now separated into true hemangioma and vascular malformations on the basis of physical findings, clinical behavior, histological findings, and cellular kinetics.

Cited in different published articles, there were fewer than 50 reported cases of uterine hemangioma worldwide. As of July 2022, this is the first reported local case of uterine hemangioma in the Philippines based on the Philippine Society of Obstetrics and Gynecology Virtual Library and the Philippine Society of Ultrasound in Obstetrics and Gynecology collection of interesting case reports and journals. After a thorough search of literature using online platforms such as PubMed, National Guideline Clearing House, and National Institute for Health and Care Excellence, to date, there are no published guidelines on the diagnosis and management of this benign vascular lesion of the uterus, cavernous uterine hemangioma.

[1]Jose R. Reyes Memorial Medical Center, Manila, Metro Manila, Philippines

Address for correspondence:
Dr. Maria Lalaine Aviles Miranda, Jose R. Reyes Memorial Medical Center, Rizal Avenue, Santa Cruz, Manila, Metro Manila, Philippines.
E-mail: lainemirandamd@gmail.com

Submitted: 25-Aug-2023
Revised: 26-Sep-2023
Accepted: 15-Oct-2023
Published: 27-Dec-2023

How to cite this article: Miranda ML, Valencia EI. A life-threatening benign vascular lesion of the uterus, cavernous hemangioma: A case report. Philipp J Obstet Gynecol 2023;47:283-90.

Cavernous uterine hemangioma was first described in 1897 and was an incidental discovery from an autopsy of a young woman who developed anemia and dyspnea and died 24 h after delivering twins. Its exact incidence still remains unclear owing to the extremely small number of case reports in the past century.[2]

The objective of this case report is to describe a rare case of cavernous hemangioma of the female reproductive tract that poses as a life-threatening vascular lesion. This will present the history and progression of the disease, the diagnostic dilemma with sonographic description, as well as the discussion of the different treatment plans and outcomes.

Case Report

This is a case of a 28-year-old G2P1 (1001), Filipino, Roman Catholic, married from Calamba, Laguna, who came in due to profuse vaginal bleeding and was admitted for the second time in our institution.

She had menarche at the age of 9 years. Past medical and family history were all unremarkable. She works as a machine operator in a company in Calamba, Laguna, but had occasions that she cannot report to work due to heavy menstrual bleeding.

History of the present illness started at the age of 18 years, the patient experienced having increased menstrual volume compared to her regular menstrual flow. On the interim, there were intermittent and multiple consults to the emergency room due to profuse vaginal bleeding, which was temporarily relieved by IV antifibrinolytic agents.

She was noncognizant on her first pregnancy due to intermittent vaginal bleeding. She only had a self-pregnancy test done when she noticed abdominal enlargement and perceived fetal movements at 7 months age of gestation. She delivered through normal spontaneous delivery, attended by a midwife and denied having had any complications. She was started on combined oral contraceptive pills as her family planning method, which she took for 3 years and was asymptomatic.

After 3 years, she had a recurrence of intermittent heavy menstrual bleeding amounting to two adult diapers per day lasting for 3 days. She sought consultation with another private doctor with consideration of pelvic congestion syndrome, probably a uterine hemangioma, due to the presence of multiple tortuous adnexal and uterine veins in the posterior myometrium; hence, she was referred to a tertiary hospital for possible hysterectomy.

She was admitted for the first time in our institution due to profuse vaginal bleeding. Transvaginal ultrasound revealed a heterogeneous mass at the posterior myometrium extending to the posterior isthmus measuring 6.0 cm × 4.1 cm × 5.9 cm (volume: 78.7 ml). On the 3D scan, the volume was 81.2 ml. There were some cystic structures traversing the endometrial canal, which on Doppler studies showed abundant intratumoral flow (score of 4), probably a uterine hemangioma [Figure 1a-e]. She was eventually

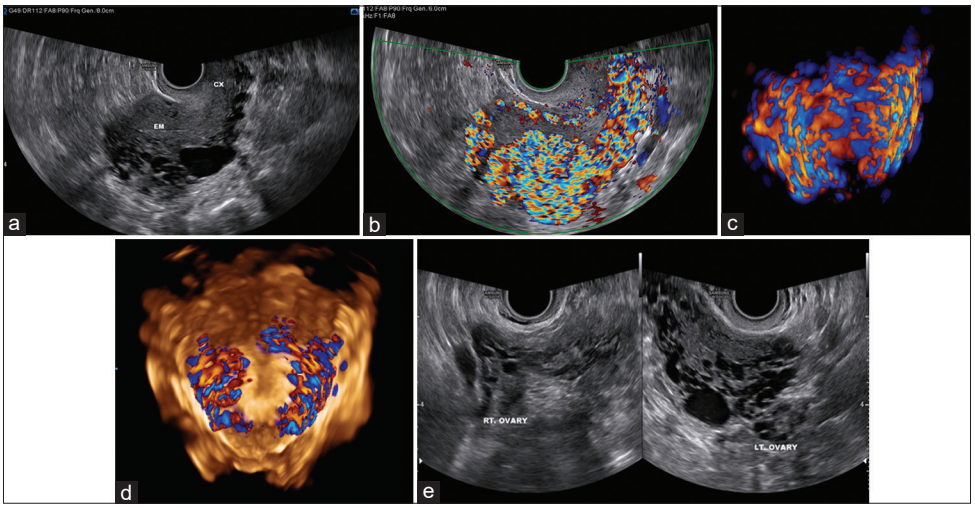

Figure 1: (a) Transvaginal sonography in sagittal view of the prepregnant uterus showing the posterior myometrium from isthmus up to the fundus showing a heterogeneous cystic structure approximately measuring 6.0 cm × 4.1 cm × 5.9 cm (volume: 78.7 ml). There are some cystic structures traversing the endometrial canal. The endometrium was described as thin measuring 0.3 cm, uniform, hyperechoic, not defined endometrial midline with regular endometrial junction and no color flow (score of 1), (b) Same ultrasound image with Figure 1a on color Doppler. There was diffuse hypervascularity noted more on the posterior aspect of the uterus, which on color flow showed abundant intratumoral flow (score of 4), (c) 3D scan of the uterus, the volume is 81.2 ml, (d) 3D scan of the uterus showing vascularity of the myometrium both the anterior and the posterior walls, (e) Sagittal view of normal looking ovaries

discharged and improved after the correction of anemia.

One of the differential diagnoses was an arteriovenous malformation (AVM). A vascular magnetic resonance imaging (MRI) was requested on outpatient basis, which showed an AV malformation [Figure 2A and B]. Possible referral to an interventional radiologist for possible embolization was also considered. The plan was to refer also to the service of reproductive medicine for the trial of gonadotropin-releasing hormone (GNRH) treatment. GNRH is an aromatase inhibitor that can help minimize the estrogen surge. Due to poor compliance, the patient could not undergo the treatment.

The patient was admitted for the second time in our institution at 8 weeks gestation, presenting with profuse bleeding. She was conscious, coherent but weak looking. She had pale palpebral conjunctiva, pale nail beds, palms, and soles. The vital signs revealed blood pressure of 90/70 and mild tachycardia at 104 bpm. Internal examination revealed an open cervix at 2 cm dilatation with palpable placental-like tissue plugging the cervical os. She had severe anemia with hemoglobin of 7.6 mg/dL and hematocrit of 23.0%.

Transvaginal ultrasound showed a heterogeneous mass at the endocervical canal measuring 4.8 cm × 4.7 cm × 3.9 cm (volume: 48.0 ml) representing the gestation with an irregular sac with no yolk sac but with an embryo (crown-rump length measuring 1.6 cm equivalent to 8 weeks age of gestation) with no cardiac activity. There was also note of an anteverted uterus measuring 7.2 cm × 6.5 cm × 6.5 cm with a posterofundal heterogeneous mass measuring 6.3 cm × 5.7 cm × 5.0 cm (volume: 94.2 ml) with cystic spaces of varied sizes that on color flow and spectral Doppler study was suggestive of uterine hemangioma [Figure 3a-d].

During her first hospital stay at the ward, she had an episode of profuse vaginal bleeding and the hemoglobin dropped to 4.0 mg/dL with a hematocrit of 15.0%. The preoperative diagnosis was G2P1 (1001) abortion incomplete 8 weeks age of gestation, nonseptic, noninduced, to consider uterine hemangioma versus AVM, anemia severe – ongoing correction. An informed consent was given by the patient and the family members. She underwent a total abdominal hysterectomy with bilateral salpingectomy.

Intraoperatively, there was no ascitic fluid noted. The surfaces of palpable pelvoabdominal organs were smooth and with intact surfaces. The corpus measures 6.5 cm × 8.0 cm × 3.5 cm with a smooth and intact serosal surface. On the cut section, the endometrial canal measures 6.5 cm × 0.2 cm endometrial stripe. The products of conception were noted attached at the posterofundal area measuring 4.5 cm × 5.0 cm × 2.5 cm. On the cut section of the mass, there were diffusely dilated vascular spaces noted [Figure 4a-c]. The cervix

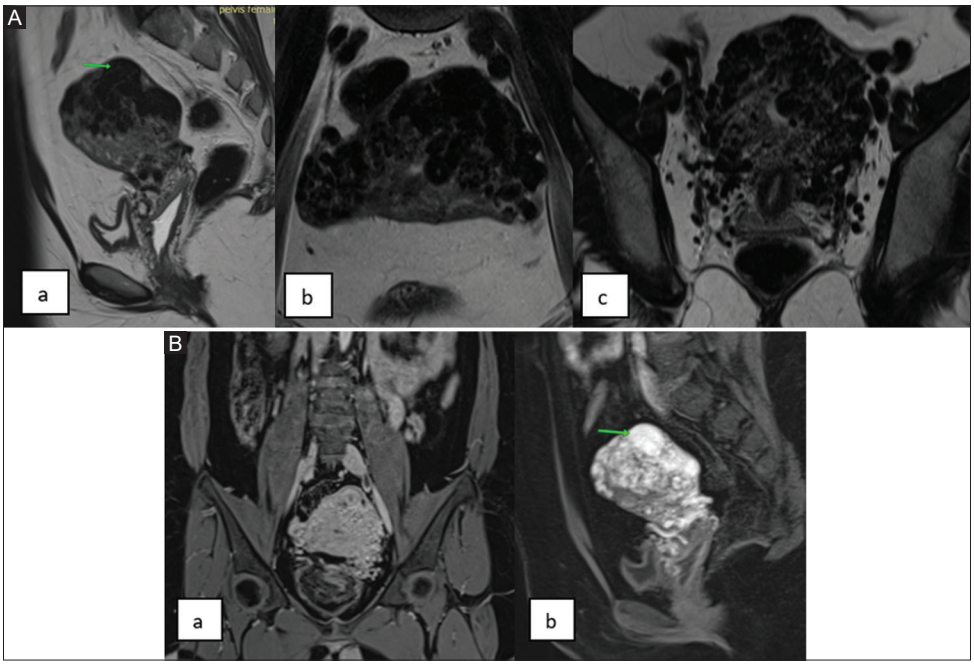

Figure 2: (A) T2-weighted images in sagittal (a), coronal (b), and axial (c) plane showed multiple vascular-like serpentine flow-related signal voids (green arrow) involving the myometrium and parametrium, more significantly at the posterofundal portion of the uterus, (B) T1 -weighted postcontrast images in coronal (a) and sagittal (b) plane shows the complex serpentine vessels involving the myometrium with intense enhancement. A central nidus (green arrow) measuring 1.8 cm in its greatest diameter is shown. The bilateral parametrial vessels are prominent. The vaginal canal is distended

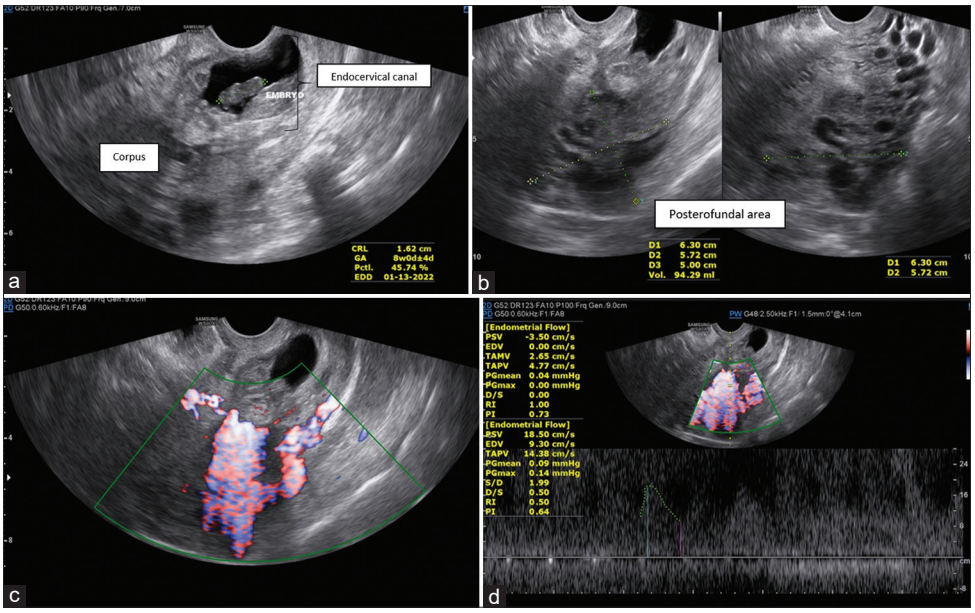

Figure 3: (a) Sagittal view of the uterus during pregnancy. Within the endocervical canal is a heterogeneous mass measuring 4.8 cm × 4.7 cm × 3.9 cm (volume: 48.0 ml) and an irregular gestational sac with no yolk sac and an embryo with no cardiac activity at the time of scan, (b) The uterus is anteverted measuring 7.2 cm × 6.5 cm × 6.5 cm with a posterofundal heterogeneous mass measuring 6.3 cm × 5.7 cm × 5.0 cm (volume 94.2 ml) with cystic spaces of varied sizes, (c) Same picture as above in color Doppler showed abundant vascularity (score of 4), (d) Spectral Doppler interrogation of the vascular lesion with peak systolic velocity of 18.50 cm/s and resistive index of 0.50

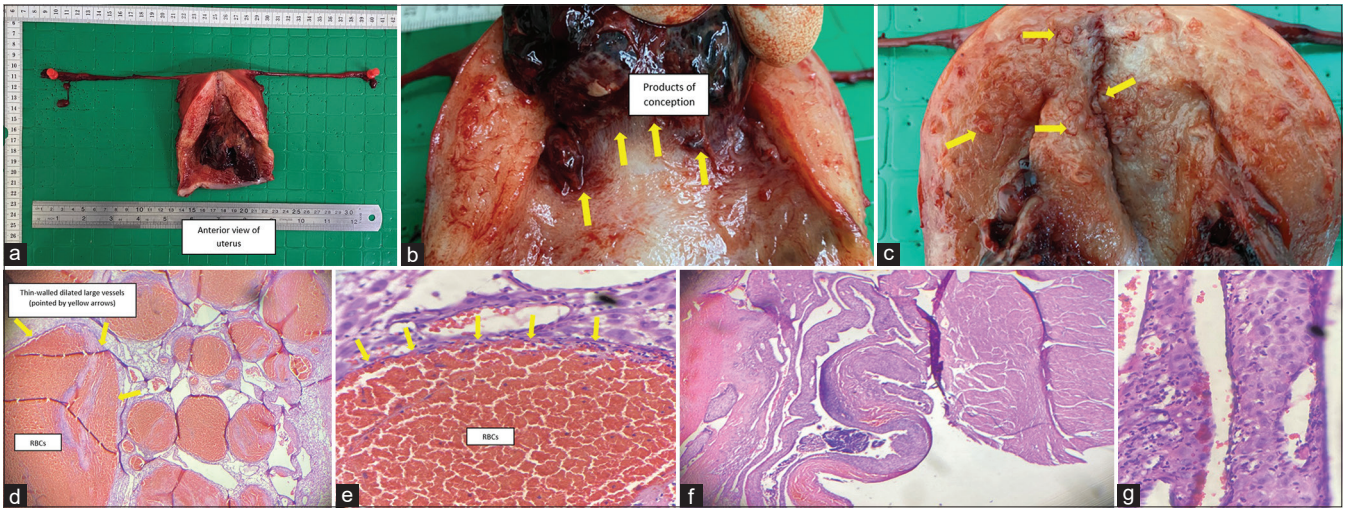

Figure 4: (a) Gross specimen of the uterus with products of conception and the fallopian tubes (b) Gross specimen of the uterus with the mid posterior attachment of the products of conception (Yellow arrow), (c) On cut section, there were dilated vascular spaces extending up to the myometrium (some of the dilated vessels are labeled using the yellow arrow), (d) Microscopically, on low power field, there were numerous large vessels with cystically dilated lumen with thin walls filled with red blood cells. The single layer of endothelium are pointed by the yellow arrows, (e) On high power field, the dilated vascular channels are lined by a single flat layer of endothelium (yellow arrows) and are filled with red blood cells, (f) On scanner view, seen are the myometrium, endometrium with gestational endometrium (with increased number of glands and blood vessels with areas of decidualization). Likewise seen are chorionic villi and areas of hemorrhages, (g) On high power view of the gestational endometrium, there are decidual cells with large, irregularly shaped cells with pale nuclei and abundant granular eosinphilic cytoplasm

was smooth, measuring 4.5 cm × 3.0 cm × 1.5 cm, with blood clots plugging the cervical os. On the cut section, the endocervical canal was smooth, measuring 3.5 cm. The right and left fallopian tubes were grossly normal, measuring 13.5 cm × 0.4 cm × 0.3 cm and 13 cm × 0.4 cm × 0.2 cm, respectively. On the right fallopian tube were paratubal cysts noted, the largest of which measured 1.7 cm × 0.9 cm × 0.5 cm.

The patient tolerated the procedure well. She was sent home improved after 3 days of postoperative care.

Histopathologic report was signed out as cavernous hemangioma; gestational endometrium with retained placental tissues; chronic cervicitis with squamous metaplasia; acute salpingitis, bilateral; and paratubal cyst, bilateral [Figure 4d-g].

Discussion

Hemangiomas are categorized as congenital or acquired. It is further classified on the basis of size of vascular spaces and histology, such as capillary and cavernous hemangiomas.[3] Capillary hemangiomas are small thin-walled blood-filled vessels composed of a single layer of flattened and plump endothelial vessels, containing pericytes, and reticular fibers. On the other hand, cavernous hemangioma is deep, irregular, dermal tangles of large, thin-walled vessels separated by scanty connective tissue. Capillary hemangiomas have a proliferative phase and involution phase, whereas cavernous hemangiomas are more stable and usually fail to regress.

The most common cause of uterine hemangioma among individuals may be due to the influence of estrogen.[4] Any conditions leading to increased estrogen levels may play a role in the development of uterine hemangioma among reproductive age women. Uterine hemangioma in nonpregnant women can be explained by the gradual increase in size of a preexisting lesion following pregnancy. Physical changes, including hypoxia, endometrial curettage, previous pelvic surgery, tissue injury, and increased blood volume during pregnancy, as well as some conditions leading to hormone alterations, especially high estrogen levels such as menarche, pregnancy, endometrial cancer, and trophoblastic diseases, have been proposed as probable causes of acquired uterine hemangioma.

There are few reported cases of uterine cavernous hemangioma worldwide. Benjamin *et al*.[2] in 2010 described a rare case of abnormal vaginal bleeding of a 27-year-old Malay, who was transferred to their hospital due to heavy vaginal bleeding 11 weeks postcesarean section. The patient underwent uneventful low cesarean section, but soon presented with heavy vaginal bleeding with hypovolemic shock. An emergency laparotomy followed by hysterectomy with salpingectomy was done, which revealed a diffuse ramifying hemangioma of the cervix and uterus with left hematosalpinx on histopathologic examination. Patient's torrential bleeding was attributed to the ramifying hemangioma described as endothelial-lined vascular spaces between the uterine musculature.

In 2016, there was a detailed case report of uterine cavernous hemangioma by Knight *et al*.[5] where sonographic features of uterine cavernous hemangioma in pregnancy were described. Ultrasound examination revealed an anatomically normal and appropriately grown fetus with a normal placenta. There were extensive cystic spaces noted within the myometrium confirmed by MRI. Their considerations were trophoblastic disease concomitant with pregnancy and uterine hemangioma. Unremarkable lower segment cesarean section was done to the patient without complication. 6 months postoperatively, on repeat scan, the uterus showed small cystic spaces involving the anterior wall. The patient was managed with hormonal contraception.

A case report of Yu *et al*.[6] in 2017, a 30-year-old primiparous woman had massive bleeding from the episiotomy site. A deep punch biopsy in the upper episiotomy site of the vaginal wall revealed a cavernous hemangioma confirmed by the presence of large dilated blood vessels lined by flattened endothelium. They did a successful transarterial embolization to treat the intractable bleeding from the vaginal hemangioma. Other treatment options for hemangioma include intralesional glucocorticoid therapy, the use of interferon-α, laser therapy, and surgical excision.

A very recent systematic review of literature by Bauters *et al*.[7] regarding uterine hemangioma in pregnancy. There were 15 case reports that uterine hemangioma in pregnancy was detected either before, during, or directly after pregnancy. More than half of the women developed a postpartum hemorrhage, necessitating hysterectomy for control of bleeding. They concluded that current knowledge on uterine hemangioma was limited, including the treatment options.

In our case, we can only postulate that the vascular lesion our patient might have been present during childhood or a congenital type of uterine hemangioma, though not detected earlier because she was asymptomatic. Following menarche at the age of 9 years, she started to have exposure to estrogen, which is a known factor to influence the growth of uterine hemangioma, thereby a component of an acquired type cannot be completely disregarded. Hence, we can surmise that the cause of her uterine hemangioma may be multifactorial.

Gray scale ultrasound can detect the presence of multiple tubular or "spongy" anechoic or hypoechoic areas within the myometrium of a normal endometrium seen in both hemangioma and AVM. We can use color and Doppler ultrasound to differentiate AVM from uterine hemangioma. A normal myometrial signal of a myometrial vessel will show a peak systolic velocity (PSV) of 9–44 cm/s and resistive index (RI) of 0.6–0.8.[7]

On spectral Doppler interrogation during the second scan, the RI is at 0.50 and the PSV is at 18.50 cm/s [Figure 3d]. These findings are more compatible with uterine hemangioma than AVM, wherein the low-velocity flow is related to the stagnant deep and larger blood-filled vessels. This is expected in uterine hemangioma, where

the caverns are filled with red blood cells that are slow flowing.

On the other hand, the most commonly reported uterine vascular lesion, AV malformation, is described as cystic and hypervascular lesions in the uterine wall. According to Müngen,[8] AVMs are errors of morphogenesis with stable cellularity and do not show spontaneous regression, unlike hemangiomas. Gray-scale sonography has a limited role in the diagnosis of uterine AVMs and often shows an ill-defined uterine mass consisting of mildly echogenic tissue that is interspersed with multiple small hypoechoic spaces of varying sizes. In line with the color Doppler interrogation, spectral analysis of the vessels within the lesion will show low-impedance, high-velocity flow, with resistance index values ranging from 0.25 to 0.55, and peak velocity values ranging from 40 cm/s to 96 cm/s in previously reported cases. This is due to the turbulent flow within the hypervascular lesions within the myometrium.

MRI is also an important diagnostic tool wherein uterine AVMs are manifested as a bulky uterus, a focal uterine mass, disruption of the junctional zones, serpiginous flow-related signal voids, and prominent parametrial vessels.

On our case, the MRI with contrast was described as multiple vascular-like serpentine flow-related signal voids involving the myometrium and parametrium, more significantly at the posterofundal portion of the uterus. A central nidus measuring 1.8 cm in its greatest diameter was noted. The bilateral parametrial vessels were prominent and the vaginal canal was distended. The case was signed out on the MRI as highly considering an AVM. In AVM, the central nidus is the tightly packed structures of enlarged feeding arteries. The difference between AVM and uterine hemangioma cannot be fully committed. Both AVM and uterine hemangioma may present as vascular serpentine flow-related intense enhancement. The velocity of flow and RI cannot be measured on MRI unlike on Doppler studies using ultrasound.

AVM, even if it is rare, still has a handful of experienced treatment options. One of the treatments is uterine artery embolization (UAE). According to Katano *et al*.,[9] bilateral UAE is a safe and effective first-line therapeutic option for the management of bleeding uterine AVMs. However, incomplete embolization due to unembolizable feeders or difficult access into the uterine artery may lead to suboptimal treatment. Trial of UAE was also a consideration in this case to decrease the volume of menses and possibly resolve the vascular lesion.

Another dilemma in management was also encountered since she is a young patient. A family conference was prepared to ensure that the family and the patient were informed about the rare condition and the outcome of management.

On gross pathology, uterine hemangioma presents with dilated vascular spaces with adjacent brownish color changes [Figure 4c], and microscopically, there are dilated irregular venous type channels lined by a single flat layer of endothelium containing blood vessels [Figure 4d]. The histopathologic report on our case was consistent with cavernous hemangioma of the uterus. Microscopically, the subserosal region of the myometrium showed irregularly shaped cavernous vascular spaces infiltrating between the myometrial fascicles and located predominantly in the outer portion of the myometrium. The large vascular spaces are walled by flat endothelial cells and distended by blood. No endothelial cell atypia is seen.

In AVM, there is a gradual replacement of normal myometrium. In comparison with hemangioma that is lined by flat endothelial cells, AV malformation have large tortuous arteries, thick-walled veins, and disruption of the arterial internal elastic lamina.[4]

To cite other differential diagnosis is the Klippel–Trenaunay syndrome (KTS). It is believed to be a congenital type of uterine hemangioma. It is also a rare entity characterized by the triad of capillary malformations, atypical varicosities or venous malformations, and bony or soft-tissue hypertrophy most commonly affecting unilateral lower limbs.[10] In a case report, ultrasonography demonstrated tubular echolucent spaces throughout the myometrium and color Doppler showed blood flow within some of the cystic lesions. Histological examination of the myometrial biopsy showed numerous, variably sized, and thin-walled vessels distributed throughout the myometrium. The endothelial lining on the vessel wall was confirmed by strong immunoreactivity for CD31 and CD34, and a lack of elastic fiber layer was observed by immunostaining with Elastica van Gieson. Although vascular malformation is also seen in the histopathology of KTS, it can be ruled out because the patient in our case report did not present the triad of the syndrome.

Other differential diagnosis includes lymphangioma. It is also a vascular disease; however, very few literature have described uterine lymphangioma. The sonographic features are multilocular cystic masses, internal septa with varying sizes, usually anechoic content, and without color flow. This lesion is generally thought to be of congenital origin. They can also be described as capillary, cavernous, or cystic tumors. Grossly, they are clusters

of thin-walled vesicles with clear fluid. Microscopically, there are thin-walled vessels that does not contain blood vessels, unlike the cavernous hemangioma.

Gestational trophoblastic disease (GTD) can also be one of the differential diagnoses. The ultrasound findings of GTD show multiple cystic spaces of varied sizes noted within the endometrial cavity. Color flow mapping will help differentiate the cystic structures from vascular lesion. There is no or minimal color flow within the cystic structures compared to vascular lesions such as uterine hemangioma and AVM, which have abundant vascularity. In light of the present pregnancy of our case, GTD can also be ruled out.

Summary

The case presented a life-threatening condition of a benign vascular lesion of the uterus. It is worthwhile to consider abnormal vascular lesions as possible causes of heavy vaginal bleeding.

Due to the rarity of cavernous hemangioma of the uterus, the recommended diagnostic tests are very nil. The patient presented with several years of intermittent heavy menstrual bleeding. An earlier detection would have saved a lot of expenditures, not to exclude emotional and psychosocial burdens to the patient and to her family.

The use of ultrasonography with 2D, color Doppler, and spectral interrogation was proven to be a helpful imaging modality because it may help differentiate uterine hemangioma from other uterine lesions. Doppler studies will also determine the velocity of blood flow within the lesions. Uterine hemangioma will present a low velocity flow compared to AVM with a high velocity flow.

MRI was done in this case in preparation for possible UAE; however not very helpful in the differentiation of uterine hemangioma from AV malformation since they may appear the same using this modality.

Management of uterine hemangioma is also a dilemma among clinicians. If the bleeding can be controlled medically, what medications or interventions must be given to avoid the recurrence of symptoms. Preserving the uterus and fertility of the patient with uterine hemangioma is also a factor to consider.

Most of the time, surgical intervention is warranted for cavernous hemangiomas due to the persistence of heavy menstrual bleeding. Some authors describe conservative treatments such as carbon dioxide laser excision, knife excision, cryotherapy, radiotherapy, electrocauterization, internal artery ligation, UAE, local excision, conization, and laser ablation.[4] Corticosteroids are utilized in the treatment of some vascular proliferative diseases, this may be considered in the management of future cases.[3]

Even if the treatments were suggested, after a thorough search from online publications such as Pubmed, there were no written discussions or case reports of the procedure done to a case of uterine hemangioma yet. Moreover, these treatment modalities are not available in all institutions. Moreover, finally, if presented with intractable bleeding, hemodynamic instability, hysterectomy is the most plausible definitive treatment.

Definitive diagnosis relies on the final histologic examination. The likely complication of torrential bleeding, such as hypovolemic shock, can be abated by proper preparation of a medical and surgical team, the availability of blood products, and an intensive care unit in a tertiary hospital.

The author recommends creating an algorithm by experts in the field, like obstetrics and gynecology, vascular surgeons, and interventionists, wherein the diagnostics and management can be clearly stated. Through case reporting of such rare cases, the possibility of starting an international registry of uterine hemangioma is highly anticipated.

Authorship contributions

Maria Lalaine A. Miranda, MD - Involved in the conceptualization, methodology, data curation, writing of the original draft, review and editing.

Eleyneth I. Valencia,MD - Involved in review and editing of the draft.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

References

1. Kunimoto K, Yamamoto Y, Jinnin M. ISSVA classification of vascular anomalies and molecular biology. Int J Mol Sci 2022;23:2358. doi: 10.3390/ijms23042358.
2. Benjamin MA, Yaakub HR, Telesinghe P, Kafeel G. A rare case of abnormal uterine bleeding caused by cavernous hemangioma: A case report. J Med Case Rep 2010;4:136.
3. Çorbacıoğlu Esmer A, Özsürmeli M, Yüksel A, Kalelioğlu İ, Has R, Bakar N. Antenatal sonographic diagnosis of diffuse cavernous hemangioma of the uterus. Marmara Med J 2014;27:134-7. Retrieved from https://dergipark.org.tr/en/pub/marumj/issue/45477/571021. [Last accessed on 2023 Jul 20].
4. Vijayakumar A, Srinivas A, Chandrashekar BM, Vijayakumar A. Uterine vascular lesions. Rev Obstet Gynecol 2013;6:69-79.
5. Knight P, Robertson M, Paoletti D. Uterine cavernous haemangioma in pregnancy. Australas J Ultrasound Med 2016;19:37-41.

6. Yu BR, Lee GE, Cho DH, Jeong YJ, Lee JH. Genital tract cavernous hemangioma as a rare cause of postpartum hemorrhage. Obstet Gynecol Sci 2017;60:473-6.
7. Bauters E, Aertsen M, Froyman W, van der Merwe J. Uterine hemangioma in pregnancy: A case report and review of the literature. J Gynecol Obstet Hum Reprod 2022;51:102401.
8. Müngen E. Vascular abnormalities of the uterus: Have we recently over-diagnosed them? Ultrasound Obstet Gynecol 2003;21:529-31.
9. Katano K, Takeda Y, Sugiura-Ogasawara M. Conservative therapy with a gonadotropin-releasing hormone agonist for a uterine arteriovenous malformation in a patient with congenital heart disease. Clin Case Rep 2015;3:479-82.
10. Yara N, Masamoto H, Iraha Y, Wakayama A, Chinen Y, Nitta H, *et al*. Diffuse venous malformation of the uterus in a pregnant woman with klippel-trénaunay syndrome diagnosed by DCE-MRI. Case Rep Obstet Gynecol 2016;2016:4328450. doi: 10.1155/2016/4328450.