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Original Article

Acquired uterine vascular abnormalities associated with persistent human chorionic gonadotropin: Experience at a Korean teaching hospital

Da Hye Ju, Sang Wook Yi*, Woo Seok Sohn, Sang Soo Lee

Department of Obstetrics and Gynecology, Gangneung Asan Hospital, University of Ulsan College of Medicine, Gangneung-si, Gangwon-do, South Korea

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ABSTRACT

Objective: The aim of this study was to describe our experience with the diagnosis and management of acquired uterine vascular abnormalities associated with persistent human chorionic gonadotropin (hCG). Through this case series, we sought to establish our protocol for the treatment and follow-up of uterine vascular lesions associated with persistent hCG.

Materials and methods: We examined the clinical presentations of 28 Korean women with acquired vascular uterine abnormalities associated with persistent hCG who were seen in the Department of Obstetrics and Gynecology of the Gangneung Asan Teaching Hospital, Gangneung-si, Korea between October 2006 and July 2012 and retrospectively reviewed their medical records.

Results: The mean patient age was 32.5 ± 6.4 years, and the mean parity was 1.4 ± 1.2. The mean size of the vascular lesions in color Doppler sonography and multidetector computed tomography with angiography was 3.1 ± 1.6 cm and 3.9 ± 1.6 cm, respectively. Multidetector computed tomography revealed arteriovenous malformation-like vascular lesions ($n = 15$) and pseudoaneurysms ($n = 3$). Treatments included clinical observation ($n = 11$), uterine artery embolization ($n = 11$), hysterectomy ($n = 4$), and chemotherapy, including single methotrexate (MTX) treatment and combination chemotherapy ($n = 9$).

Conclusion: When the uterine vascular lesion is not decreased, or if weekly clinical follow-up reveals that the serum β-hCG level is persistently elevated or sustained in conjunction with vaginal hemorrhage, a proper management strategy is required.

Introduction

Uterine vascular abnormalities are rare clinical findings. Consequently, only a few cases of uterine vascular abnormalities have been reported in the literature. Because these vascular lesions were often overlooked by obstetricians and gynecologists and a standard protocol for diagnosis and therapy has not yet been established, there are difficulties associated with the diagnosis and treatment of uterine vascular lesions [1].

Vascular abnormalities that affect the uterine arteries include congenital arteriovenous malformations (AVMs), acquired AVMs, arteriovenous fistulas, true aneurysms, and pseudoaneurysms [2]. Congenital uterine AVMs are rare lesions resulting from the abnormal embryonic development of primitive vascular structures that cause abnormal communications between arteries and veins [3]. We have encountered no cases of congenital AVM at our institution. Congenital AVMs usually have multiple feeding arteries, a tangle of vessels with histological characteristics of both arteries and veins, and many large draining veins [4]. Acquired uterine AVMs typically result from trauma, such as a prior dilation and curettage, a therapeutic abortion, uterine surgery, or direct uterine trauma [5,6]. Endometrial carcinoma, cervical carcinoma, and gestational trophoblastic diseases are other possible causes of acquired uterine AVMs [7,8]. If a hypervascular lesion with typical early venous filling within the myometrium is accompanied by a negative serum β-hCG level, a uterine AVM diagnosis should be considered [9]. However, uterine hypervascular lesions with turbulent flow have been observed with an elevated serum β-hCG level in patients with gestational trophoblastic disease (GTD) and

* Corresponding author. Department of Obstetrics and Gynecology, Gangneung Asan Hospital, University of Ulsan College of Medicine, 415, Bangdong-ri, Sacheon-myeon, Gangneung-si, Gangwon-do 210-711, South Korea.
E-mail address: buzzmi@naver.com (S.W. Yi).

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retained products of conception (RPOC) [10]. Uterine vascular abnormalities with elevated serum β-hCG levels may be distinguished from acquired AVM and associated with GTD and RPOC. However, there are some difficulties associated with the pathologic diagnoses of these lesions due to the limited availability of surgical specimens. Therefore, the lesions with elevated serum β-hCG could be considered to be acquired uterine vascular abnormalities associated with persistent hCG and distinguished from AVM.

The most important clinical manifestation of uterine vascular abnormalities is recurrent and/or profuse vaginal bleeding, which may result in hypovolemic shock and/or death. Vaginal bleeding arising from these vascular abnormalities may be aggravated by dilation and curettage performed for the purpose of biopsy and bleeding control, unlike excessive uterine bleeding due to other common causes [6].

The management of these vascular lesions ranges from a conservative approach, such as observation or medication, to invasive procedures, such as uterine artery embolization or emergency hysterectomy. However, most reports in the literature consist of only a few cases, and experience with vascular abnormalities at individual institutions is limited. In this study, we present the image patterns of uterine vascular abnormalities associated with persistent hCG using sonography with color Doppler and multidetector computed tomography with angiography and describe our findings regarding the immediate results and long-term outcomes of these lesions. By examining this case series, we sought to establish our protocol for the treatment and follow-up of uterine vascular lesions associated with persistent hCG.

Materials and methods

This study was approved by Gangneung Asan Hospital Institutional Research Ethics Committee (2010-056). We reviewed the medical records of patients with acquired uterine vascular abnormalities associated with persistent hCG diagnosed and treated at Gangneung Asan Hospital, Gangneung-si, Korea between October 2006 and July 2012. During the study period, a total of 3947 pregnancies, including abnormal pregnancies, were diagnosed at our outpatient department. At our institution, uterine vascular abnormalities associated with persistent hCG were diagnosed in 28 patients (0.71%). In this case series, patients with intrauterine vascular abnormalities with elevated serum β-hCG measurements were enrolled. According to our study design, patients with normal intrauterine pregnancy or ectopic pregnancy, in addition to those with uterine vascular lesions associated with negative serum β-hCG measurements, would be excluded from the case series because serum β-hCG measurements are elevated or sustained in normal intrauterine pregnancy and ectopic pregnancy regardless of the presence of uterine vascular abnormalities. Additionally, in cases of normal intrauterine pregnancy, multidetector computed tomography with angiography should be avoided due to the radiation hazard associated with this technique. For these reasons, we planned to exclude the cases of normal intrauterine pregnancy with uterine vascular abnormalities. However, there were no cases of uterine vascular abnormalities with normal intrauterine pregnancy or ectopic pregnancy.

When patients with vaginal spotting or bleeding and whose vital signs were stable visited our outpatient division, physical examinations including a vaginal examination, urine hCG test, and pelvic sonography with color Doppler were performed to detect uterine abnormalities. Three cases without symptoms were observed by chance during routine gynecological examinations. For suspected uterine vascular lesions, we performed serial serum β-hCG measurements and transvaginal sonography of the intrauterine cavity to rule out a normal intrauterine pregnancy or an ectopic pregnancy by additional image evaluation. In patients with stable vital signs, multidetector computed tomography with angiography was performed to evaluate the uterine vascular lesions.

The management options for these uterine vascular abnormalities consisted of observation, single chemotherapy with methotrexate (MTX), combination chemotherapy (EMA or EMA-CO), transcatheter uterine artery embolization, and hysterectomy. At our institution, the protocol for the treatment and follow-up of vascular abnormalities was established based on clinical case series and case reports in the literature (Figure 1). Based on our protocol for treatment and follow-up, if there was profuse vaginal hemorrhage with stable vital signs, transcatheter uterine artery embolization was performed. If the patients had unstable vital signs because of profuse vaginal bleeding, an emergency hysterectomy was performed. In cases with vaginal spotting or no specific symptoms, clinical follow-up, including transvaginal sonography with color Doppler and serum β-hCG measurements, was performed weekly. For cases in which two or three consecutive clinical follow-ups demonstrated a sustained or elevated level of serum β-hCG, chemotherapy was considered. For cases in which the size of the uterine vascular lesions increased or failed to regress during clinical follow-up, a transcatheter uterine artery embolization was performed. After treatment, we performed periodic physical examinations and measured the patients' serum β-hCG levels in the outpatient division to check for the recurrence of uterine vascular abnormalities.

Data are expressed as mean ± standard deviation (SD) unless stated otherwise. All statistical analyses were performed with SPSS version 13.0 for Windows (SPSS Inc., Chicago, IL, USA).

Results

The mean patient age was 32.5 ± 6.4 years (range, 19–42 years), and the mean parity was 1.4 ± 1.2 (range, 0–4). Twenty-five patients had vaginal hemorrhage, two patients had no specific symptoms, and one patient had amenorrhea. Regarding surgical history, nine patients had undergone a cesarean section one or more times, one patient had undergone an appendectomy, and one patient had undergone laparoscopy.

The antecedent pregnancy events of the patients were the following: missed abortion ($n = 11$), intrauterine curettage for artificial abortion ($n = 8$), hydatidiform mole ($n = 5$), cesarean scar pregnancy ($n = 2$), and unknown event ($n = 2$). With these antecedent pregnancy events, the patients had undergone surgical procedures such as dilation and curettage ($n = 18$), suction and curettage ($n = 5$), no procedure ($n = 3$), MTX injection ($n = 1$), and unknown ($n = 1$) before acquired uterine vascular abnormality was diagnosed. In two patients, uterine vascular lesions were diagnosed during a missed abortion (Figure 2).

The interval from the antecedent pregnancy event to the diagnosis of an acquired uterine vascular lesion ranged from 0 days to 192 days (mean ± SD: 48.9 ± 41.5 days). All patients had an elevated serum β-hCG level at the time of diagnosis, and the mean serum β-hCG level was 4,136.0 ± 10,399.9 mIU/mL, with a range from 3.9 mIU/mL to 48,427.0 mIU/mL. Therefore, we classified these lesions as acquired uterine vascular abnormalities associated with pregnancy because the serum β-hCG level is not elevated in uterine AVMs. A gray-scale transvaginal sonography with color Doppler scan was performed in 27 patients, and one patient underwent an emergency hysterectomy without a color Doppler scan because of unstable vital signs. Color Doppler sonography of the vascular lesions revealed a high-flow vascular lesion or tangles of vessels with multidirectional high velocity flow producing a color mosaic pattern. In 22 of 27 patients, multidetector computed tomography with angiography was performed. In five cases, uterine vascular lesions were diagnosed with color Doppler sonography without multidetector computed tomography.

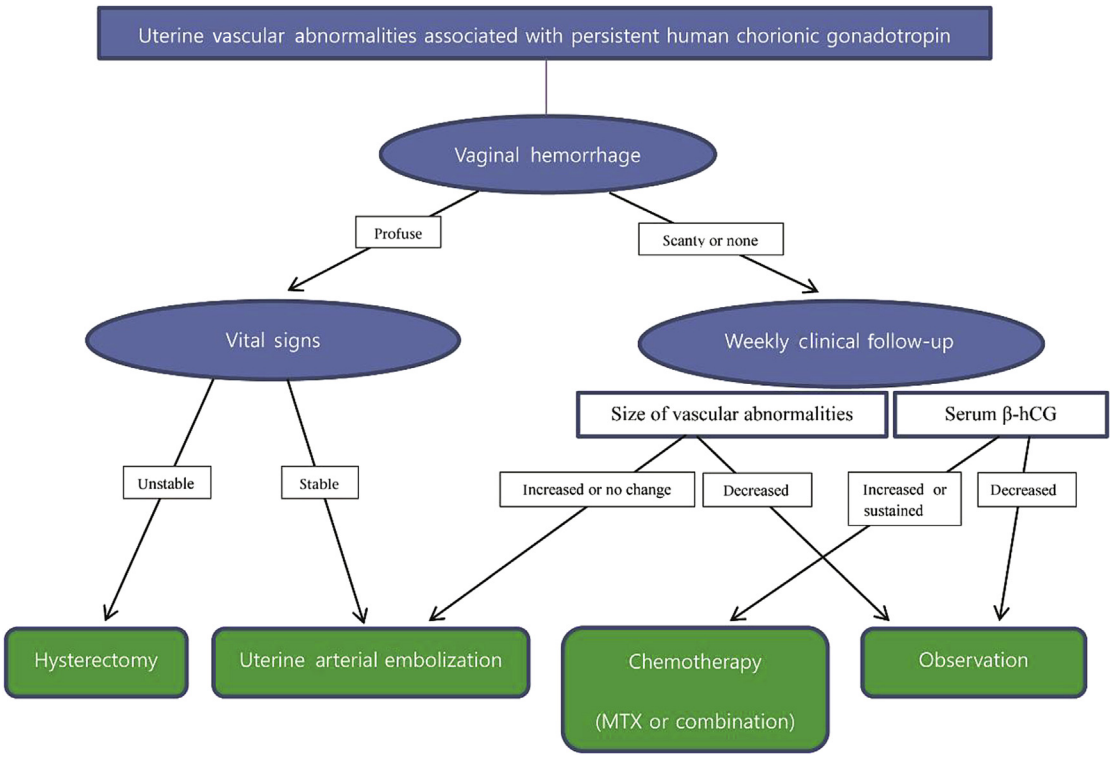

Figure 1. Protocol for the treatment and follow-up of uterine vascular lesions at our institute.

In one case, an emergency hysterectomy was performed without an imaging study because of profuse vaginal hemorrhage and unstable vital signs. The pathological diagnosis of the patient was confirmed as a uterine vascular lesion with an elevated serum β-hCG level, and the patient was enrolled in the case series.

Based on the multidetector computed tomography images, the vascular patterns of the lesions could be subdivided into AVM-like vascular lesions ($n = 15$) and pseudoaneurysms ($n = 7$; Figure 3).

The mean size of the vascular lesions on color Doppler sonography and multidetector computed tomography with angiography were 3.1 ± 1.6 cm (range, 1.5–8.0 cm) and 3.9 ± 1.6 cm (range, 1.5–6.5 cm), respectively.

With regard to the management of the patients, nine patients were placed under clinical observation. A uterine artery embolization was performed in five patients, and uterine artery embolization and chemotherapy, including single MTX and combination chemotherapy, was prescribed for four patients. A hysterectomy was performed in four patients. Chemotherapy alone, including single MTX and combination chemotherapy without any other intervention, was prescribed as a treatment for two patients. Following clinical observation and management, the uterine vascular lesions disappeared in 25 patients, and three patients were lost to follow-up (Figure 4).

Spontaneous regression of the vascular lesions was observed in nine patients without medical or surgical treatment. The mean size of the vascular lesions on color Doppler sonography in the spontaneous regression group was 2.4 ± 0.7 cm (range, 1.5–6.5 cm), and the mean serum β-hCG levels at diagnosis was 3371.9 ± 9041.7 mIU/mL (range, 4.0–27,410.0 mIU/mL). Serum β-hCG levels spontaneously returned to a normal level in all of these patients, and the

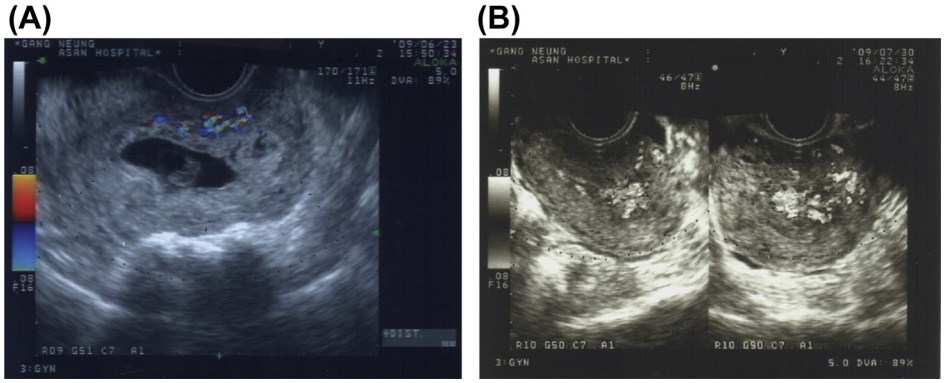

Figure 2. Color Doppler imaging of an arteriovenous malformation-like vascular lesion in a uterus with a missed abortion. A sagittal transvaginal color Doppler image of the uterus reveals (A) a tangle of vessels with multidirectional high-velocity flow producing a color mosaic pattern and (B) persistent vascular lesion of the uterus after dilation and curettage. In this case, massive vaginal hemorrhage during dilatation and curettage was expected, but vaginal bleeding was minimal. Thereafter, additional treatment was not required.

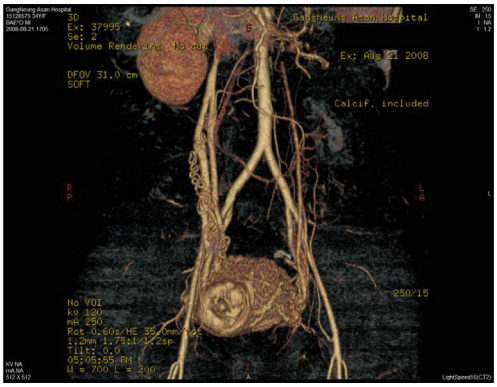

Figure 3. The volume-rendering multidetector CT image shows an arteriovenous malformation-like uterine vascular lesion feeding from the bilateral uterine arteries and right ovarian artery.

mean time to serum β-hCG level normalization after the lesion diagnosis was 42.9 ± 33.8 days (range, 7–120 days). The regression time for vascular lesions was 48.8 ± 37.5 days (range, 1–132 days). These results suggest that vascular lesions tend to persist after serum β-hCG level normalization in cases of spontaneous regression.

A uterine artery embolization was performed in five patients (Figure 5). The mean sizes of the vascular lesions on color Doppler sonography and multidetector computed tomography with angiography were 3.0 ± 1.1 cm (range, 2.0–4.9 cm) and 3.3 ± 1.3 cm (range, 2.0–5.6 cm), respectively. Despite the small lesion size (2 cm), the patient with a pseudoaneurysm had profuse vaginal hemorrhaging, and an emergent uterine artery embolization was performed.

Both uterine artery embolization and chemotherapy were performed in four patients. The mean size of the vascular lesions on color Doppler sonography and multidetector computed tomography with angiography were 3.4 ± 1.5 cm (range, 2.0–4.9 cm) and 4.6 ± 1.3 cm (range, 3.5–6.2 cm), respectively. The mean serum β-hCG level at diagnosis was 4972.0 ± 6636.4 mIU/mL (range, 3.9–14,261.0 mIU/mL). After treatment, the serum β-hCG levels returned to normal in all of these patients, and the mean time to normalization after lesion diagnosis was 62.5 ± 12.6 days (range, 48–74 days).

A hysterectomy was performed in four patients. Emergency hysterectomies were performed in two patients due to profuse vaginal bleeding with unstable vital signs. One patient was transferred to the emergency unit because of profuse vaginal hemorrhaging with unstable vital signs. Another patient underwent an emergency hysterectomy because of profuse vaginal hemorrhage after a diagnostic endometrial biopsy in a surgical setting. The other two patients underwent a hysterectomy because of treatment failure after single MTX chemotherapy and/or uterine artery embolization.

A pathological diagnosis of vascular lesions was confirmed in seven of the enrolled cases after a hysterectomy ($n = 4$) or dilation and curettage ($n = 3$). In the dilation and curettage cases, two patients had a missed abortion with uterine vascular lesions. Fortunately, no vaginal hemorrhage occurred after the dilation and curettage in either case. In the third patient, dilation and curettage was performed because of the presence of an intrauterine mass after uterine artery embolization. The pathological diagnoses were remnant conceptual tissues ($n = 4$), products of conception ($n = 1$), invasive mole ($n = 1$), and products of conception with hydropic villi ($n = 1$). Pathological diagnoses were not performed in the other 21 patients.

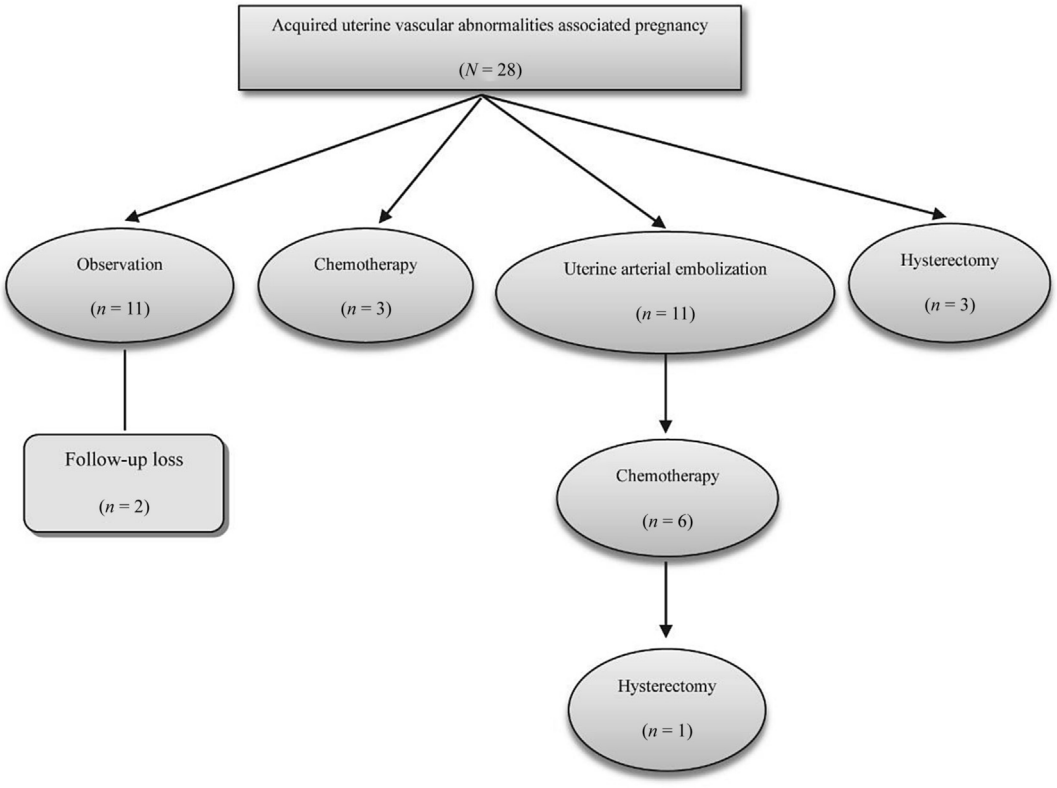

Figure 4. Management for our case series of acquired uterine vascular abnormalities associated with persistent human chorionic gonadotropin.

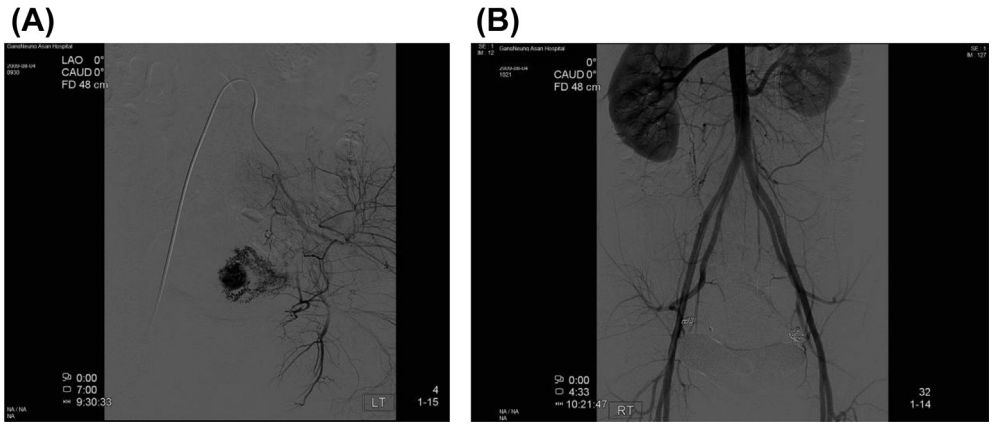

Figure 5. A selective internal iliac artery angiogram demonstrates (A) a uterine vascular abnormality associated with an arteriovenous malformation-like lesion, and (B) an aortogram after uterine arterial embolization with both coil and Gelfoam reveals complete obstruction of the vascular lesion.

The mean follow-up period was 8.2 ± 8.7 months, with the exception of the three patients for whom follow-up was not continued. During the clinical follow-up, the vascular lesions did not recur. A normal intrauterine pregnancy after management was confirmed in two cases that had been treated with uterine artery embolizations. Severe preeclampsia was diagnosed in one patient at a gestational age of 36 weeks and 5 days and who delivered a female infant weighing 2.48 kg via an emergency cesarean section. A histological examination of the placenta confirmed the diagnosis of a placental lake [11]. The second patient had fetal growth restriction at a gestational age of 38 weeks and 5 days. A male infant weighing 2.23 kg was delivered via an uneventful vaginal delivery after labor induction.

Discussion

Uterine vascular abnormalities are a rare gynecological condition; therefore, only a few cases have been reported in the literature. Vascular abnormalities that affect the uterine arteries include congenital arteriovenous malformations (AVMs), acquired AVMs, arteriovenous fistulas, true aneurysms, and pseudoaneurysms. A pseudoaneurysm is a contained rupture of an artery wall that arises from disruption of the wall caused by inflammation, vascular trauma, or iatrogenic injury, such as a surgical procedure, percutaneous biopsy, or drainage [12]. It has been reported that pseudoaneurysms of the uterine artery can be caused by cesarean sections, dilation and curettage, hysterectomies, myomectomies, oocyte retrieval for *in vitro* fertilization, and even uncomplicated vaginal deliveries [13]. Although pseudoaneurysms of the uterine artery can arise from traumatic causes, such as surgical procedures of the uterus, a pseudoaneurysm rupture at the fundal area of the uterus after cesarean delivery was reported in one study [14]. In this case, it can be assumed that placental tissues played some role in the disruption of the arterial wall.

Uterine AVMs are classified as congenital or acquired [15]. Acquired uterine AVMs tend to result from trauma, such as prior dilation and curettage, a therapeutic abortion, uterine surgery, or direct uterine trauma [5–8]. Histopathological investigations have demonstrated that AVMs occur because of errors in morphogenesis and are composed of stable endothelial cells that do not regress spontaneously. In addition, AVMs are associated with negative serum β-hCG levels [16]. In our study, all of the patients in whom uterine vascular abnormalities were diagnosed presented after a molar pregnancy or a miscarriage; most were followed by dilation and curettage. In our case series, all patients with vascular lesions had an elevated serum β-hCG level; therefore, we did not use the term AVM but instead used the terms “vascular abnormalities” and “AVM-like lesions”. The term “malformation” is generally used to describe defects in the structure of an organ or region of the body resulting from an intrinsically abnormal process of development. Therefore, the term “vascular abnormality” or “vascular lesion” appears to best describe hypervascular areas within the uterus on color Doppler ultrasound unless they are proved to be an AVM by angiography or pathological examination and negative β-hCG levels [17].

In a study of 21 AVM cases, O'Brien et al [9] suggested that a uterine AVM diagnosis cannot be made based on gray-scale findings alone, whereas color and spectral Doppler features of uterine AVMs are consistent and diagnostic. However, similar sonographic pictures can be observed in patients with a positive serum β-hCG, including those with persistently low levels of β-hCG. In these cases, physicians should consider a diagnosis of intrauterine pregnancy, ectopic pregnancy, RPOC, or gestational trophoblastic neoplasm. Kido et al [18] reported the presence of a vascular lesion within the uterus of a patient 6 weeks after surgical pregnancy termination. A hysterectomy was performed for a suspected AVM, but the pathological diagnosis revealed only necrosis of the chorionic villi. The authors hypothesized that necrosis of the chorionic villi left vessels with arteriovenous fistulas in the retained placental tissue. Another study described four patients with color Doppler imaging results that were consistent with arteriovenous malformations and who exhibited elevated serum hCG levels [10]. Some authors have reported the presence of uterine vascular lesions following pregnancy and suggested that the condition represents subinvolution of the placental bed [19].

In our case series, vascular abnormalities of the uterus associated with elevated serum β-hCG levels represent the subinvolution of retained placental tissue or postmolar gestational trophoblastic neoplasms. Although the serum β-hCG level returned to normal, uterine vascular lesions were persistent in six cases. The vascular lesions regressed spontaneously in five cases. In one case, the vascular lesion regressed after uterine artery embolization and MTX chemotherapy. In this case series, we presumed that the persistent hCG-associated vascular abnormalities may be a precursor to future acquired AVM and that placental tissues may have played a role in vascular disruption and neovascularization. Consequently, we suggest that the formation of vascular lesions in the uterus is associated with placental tissue invasion into the myometrium in addition to its association with trauma.

In nine patients, the uterine vascular lesions disappeared after conservative observation. The size of the vascular lesions in these patients was less than 3 cm on color Doppler ultrasound, and serum

β-hCG levels spontaneously returned to normal without treatment. After regression of the vascular lesions, β-hCG levels may be elevated or sustained at a persistently low level. In these cases, MTX chemotherapy or combination chemotherapy is required. Dar et al [20] reported on the long-term follow-up of arteriovenous malformations of the uterus. In four of their eight cases, sonographic resolution of the uterine AVM occurred. Based on these results, they suggested that conservative management should be considered as the primary approach.

Based on our observations, we recommend checking β-hCG levels in premenopausal women with vaginal bleeding. In addition, these patients should be considered for a color Doppler ultrasound and gray-scale transvaginal sonography. These diagnostic tools will help to determine the uterine pathology, including vascular abnormalities. Moreover, three-dimensional angiography using multidetector computed tomography displays fast and clear images that reveal the precise location of vascular lesions with their originating vessels and can help to identify suspicious vascular lesions through color Doppler ultrasound. A uterine arteriovenous malformation detected using ultrasonic and magnetic resonance imaging has been reported previously [21]. The authors of this previous report suggested that ultrasound, computed tomography, and conventional and dynamic magnetic resonance imaging (MRI) have the advantage of being noninvasive, readily available, and more accurately descriptive of AVM locations. Magnetic resonance angiography should be considered as an additional tool to make a definitive diagnosis.

When light vaginal bleeding is accompanied by decreasing vascular lesion size and decreasing serum β-hCG levels at a weekly follow-up, conservative management is recommended because of the probable spontaneous disappearance of the lesions. Because there is a possibility of sudden profuse vaginal hemorrhage during follow-up, the patient should be referred to a center with an interventional radiology unit. Vaginal hemorrhage occurs when the endothelial lining of the vessels in the vascular lesion is disrupted. Curettage can lead to heavy and even life-threatening bleeding when the diagnosis of a vascular abnormality has not been made prior to the intervention. In our case series, one patient underwent an emergency hysterectomy because of profuse vaginal hemorrhage after an endometrial biopsy in a surgical setting. In light of this experience, an endometrial biopsy should be performed in an operation room with available anesthetic personnel and equipment when a pathological diagnosis is needed.

If vaginal hemorrhage occurs, the uterine vascular lesion is not decreased in size, or the serum β-hCG level is sustained or elevated, a proper management strategy is required. When no other symptoms are observed, clinical follow-up should be considered because of the possibility of spontaneous regression of the uterine vascular lesions.

Vascular uterine abnormalities associated with pregnancy should be considered a disease entity separate from AVMs because of the spontaneous regression and positive serum β-hCG levels. As mentioned previously, we presumed that acquired AVM may follow earlier persistent hCG-associated uterine vascular abnormalities and that placental tissues may have been involved in vascular disruption and neovascularization. Furthermore, the placental tissue invasion into the myometrium associated with this distinct type of abnormality may disrupt the vasculature and promote neovascularization and therefore have long-term effects that include the development of acquired AVM.

We recommend checking β-hCG levels in premenopausal women with vaginal bleeding. In addition, these patients should be considered for a color Doppler ultrasound and gray-scale transvaginal sonography. These diagnostic tools assist in determining the uterine pathology, including vascular abnormalities. The protocol developed at our institution on the basis of the published clinical observations may be helpful in the treatment and follow-up of vascular abnormalities (Figure 1). Further case studies are needed to investigate the histopathological features and diagnostic criteria for vascular abnormalities of the uterus and to establish standard management guidelines.

Conflicts of interest

The authors have no conflicts of interest relevant to this article.

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