Placental Disorders

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KEYWORDS

• Placenta previa • Abruptio placentae • Placenta accreta spectrum • Vasa previa
• Subchorionic hemorrhage

KEY POINTS

- Common placental disorders include subchorionic hemorrhage, placenta previa, abruptio placentae, placenta accreta syndrome, single umbilical artery, and vasa previa.
- Except for single umbilical artery, these entities may present risks to the gravid patient, primarily those of hemorrhage, caesarean delivery, and at times, cesarean hysterectomy.
- In pregnancies affected by these conditions, the fetus is at risk of demise and complications of prematurity.

INTRODUCTION

The placenta is remarkable for being a temporary and mammalian organ created by the trophoblastic tissue that has already attached to the endometrium less than 2 weeks after ovulation.[1] In addition to being the largest fetal organ,[2] it sustains a dynamic, increasingly complex, and metabolically demanding fetus throughout approximately 280 days of gestation. The expulsion of placenta seems to decrease estrogen and progesterone production[3] and thus signifies the hormonal conclusion of pregnancy. Owing to various factors, including but not limited to infertility and its management, abnormal placental development, maternal age, parity, use of stimulants and tobacco, and trauma, a variety of conditions can occur that threaten the ability of the placenta to continue to support fetal development and survival.

PLACENTA PREVIA

Placenta previa (PP) is diagnosed when the placenta lies over the internal os of the cervix (**Fig. 1**). When the placenta is within 2 cm of the internal os, it is considered a low-lying placenta. Low-lying placenta remains one of the most feared obstetric complications due to its potential for causing maternal hemorrhage, which remains a significant cause of maternal morbidity and mortality.[5,6] This condition is increasing in incidence, largely because of the dramatic increase of cesarean deliveries, and is estimated to affect between 0.5% and 1.3% of all pregnancies[7,8]; in addition to its

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Physician Assist Clin 7 (2022) 545–557
https://doi.org/10.1016/j.cpha.2022.02.010 **physicianassistant.theclinics.com**

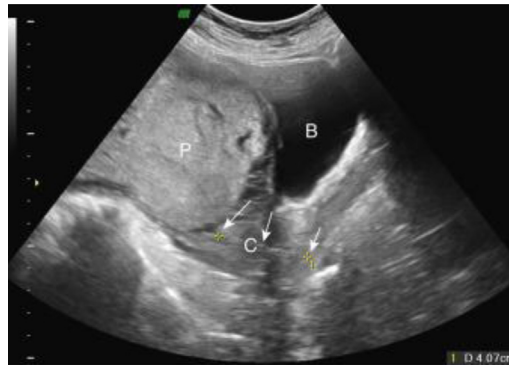

Fig. 1. Placenta previa.[4] (*From* Merriam A, D'Alton ME. "Placenta previa." In: In: Copel JA, D'Alton ME, Feltovich H et al. Obstetrical imaging: fetal diagnosis and care, 2nd ed. New York: Elsevier, 2018.)

association with hemorrhage, it is also associated with an increased risk of cesarean delivery, cesarean hysterectomy, succenturiate lobe, vasa previa (VP), prematurity, intrauterine growth restriction (IUGR),[9] and placenta accreta spectrum (PAS).[10] PP is believed to occur in patients with prior history of cesarean delivery primarily because of changes in blood flow within the endometrium and due to implantation of blastocysts in the vicinity of the scar.[7] Other factors that increase the risk of PP include advanced maternal age, higher-order parity, history of uterine instrumentation, infertility, and maternal use of stimulants and smoking.[11] Although the most common presentation is that of painless bright red bleeding in the third trimester, given the ubiquity of ultrasonography in developed nations today, most patients are diagnosed with PP via routine ultrasonography during the first or second trimesters. Clinicians must be mindful that approximately 66% to 98% of cases of PP and low-lying placentae are diagnosed before the third trimester; follow-up transvaginal ultrasonography should be obtained at 32 weeks.[12,13]

The use of transvaginal ultrasonography yields excellent sensitivity and specificity of the diagnosis of PP when compared with transabdominal ultrasonography.[9] Once the diagnosis has been made, outpatient management can usually be continued. However, patients with PP and other risk factors should be considered for admission. Patients at particular risk include those who live a significant distance from a tertiary care hospital with 24-hour obstetric anesthesia services or who have a concomitant history of antepartum hemorrhage, with short (<3 cm) cervical length, with a thick placental edge covering the internal os, or with history of prior cesarean delivery.[9] Bed rest is not recommended; the gravid patient may continue light exercise.[9] Although some authorities state that any vaginal or anal penetration be avoided (except for transvaginal ultrasonography),[9] others suggest that no evidence supports the avoidance of sexual relations that either do or do not result in orgasm in patients with PP.[14] Patients should be counseled about these modifications and should also be advised to seek immediate care in the case of bleeding or pelvic pain. Similarly, patients must be counseled about the need for cesarean delivery and of the potential for emergent delivery, blood transfusion, cesarean hysterectomy, prematurity, neonatal intensive care unit (NICU) admission, and maternal and fetal mortality.

In asymptomatic patients with confirmed PP with risk factors as noted earlier, cesarean delivery should be scheduled between 36 and 37 6/7 weeks gestational age (GA)[15]; those with a low-lying placenta within 1 cm of the internal os and with risk

factors should be delivered via cesarean delivery between 37 and 37 6/7 weeks GA, and those with a low-lying placenta and without risk factors may be delivered between 38 and 38 6/7 weeks GA.[9] Antenatal corticosteroids should only be administered for fetal lung maturity to patients at very high risk of preterm delivery.[9]

PLACENTA ACCRETA SPECTRUM

PAS (Fig. 2) is the term used to describe placental invasion that extends through the endometrium, into or through the myometrium, into or through the serosa, or beyond, usually due to a uterine defect due to prior surgical or interventional radiologic procedures.[17] Such invasion may result in catastrophic bleeding requiring hysterectomy, blood transfusion, and other procedures. Previously, PAS was defined as 3 distinct conditions: placenta accreta, in which the chorionic villi contact the myometrium; placenta increta, in which the placenta invaded the myometrium; and placenta percreta, in which the villi have traversed the serosa.[18] However, today it is known as one spectrum disorder due to a lack of international agreement concerning the terminology.[19] The incidence of PAS has dramatically increased from roughly 1:4000 deliveries in the 1970s to as many as 1 in 500 deliveries in 2018,[20] probably because of the rapid and sustained increase in cesarean deliveries. Coexisting PP is also significantly associated with PAS; the risk is as high as 3% in women who have never had a previous cesarean delivery, and for patients with a history of 5 or more cesarean deliveries, the risk is 67%.[17] The risk is also increased in patients with prior history of uterine artery embolization, manual removal of the placenta, endometrial ablation, or hysteroscopic adhesiolysis.[4]

To reduce the risk of maternal and neonatal morbidity and mortality, it is ideal to diagnose PAS antenatally, if possible, via transvaginal ultrasonography[18,21]; such an approach also allows identifying placental location to rule in or out PP.[17] Although such studies are usually performed in the second or third trimester, it is at times possible to identify PAS in the first trimester via the findings of a gestational sac in the lower uterine segment, or lacunae identified in the placental bed.[19] In the second and third trimesters, increased placental vascularity noted via color Doppler, multiple vascular lacunae, and abnormalities of the interface between the bladder and uterine serosa are all findings suggestive of PAS.[17,22] MRI is considered to be equally accurate[20] as and not superior to ultrasonography[18] in diagnosing PAS. Consultation with a radiologist with particular expertise in PAS is warranted. Repeat imaging is recommended at 18 to 20, 28 to 30, and 32 to 34 weeks GA.[20] Patients must be counseled about the need for cesarean hysterectomy as well as the potential for blood transfusion, intensive care unit admission, deep vein thrombosis, NICU admission, prematurity, and maternal and neonatal mortality.

Up to 50% of patients with PAS will require transfusion, and 7% die of this syndrome.[23] Because of these and other risks, including that of disseminated intravascular coagulopathy and the need for multiple transfusions,[22] it is essential that patients with PAS are cared for in a level 3 or level 4 center to minimize morbidity and mortality for the parturient and neonate.[20] The care team should include specialists in gynecologic oncology, urology, urogynecology, interventional cardiology, perinatology, obstetric anesthesiology, neonatology, critical care, trauma, and vascular surgery.[18,20] Delivery should be accomplished via cesarean delivery, followed by a hysterectomy, between 34 and 35 6/7 weeks GA in asymptomatic patients; the uterus should be exteriorized after delivery with closure of the uterine incision and retention of the placenta to reduce the potential for significant bleeding.[20] The blood bank should be notified before surgery, if feasible, to permit preparation for massive transfusion.[18]

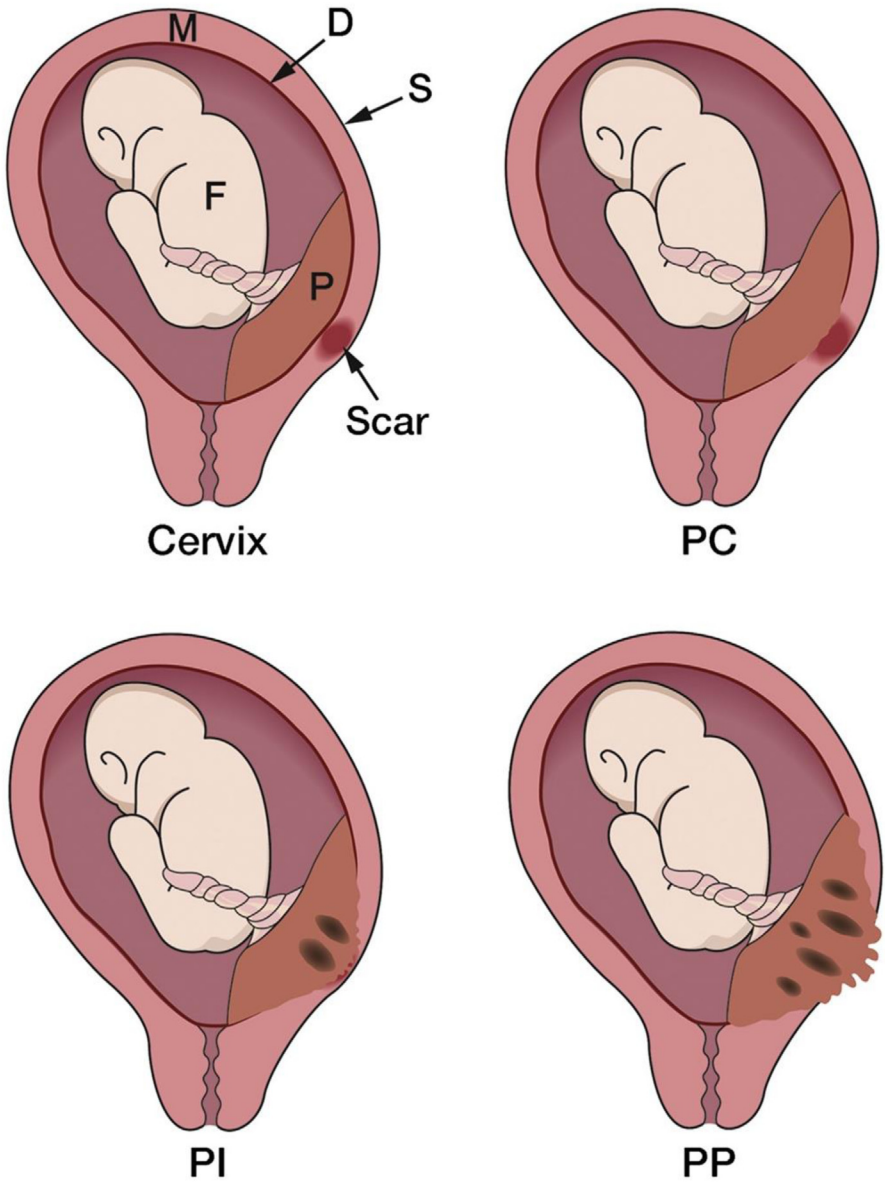

Fig. 2. Diagram showing normal and accreta placentation on a previous cesarean scar.[16] Anterior PP on a cesarean scar and different grades of PP accreta: creta where placenta villi adhere to myometrium without interposing decidual (D) tissue; increta where villi invade myometrium; and percreta where villi invade the entire myometrium and cross the uterine serosa. F, fetus; M, myometrium; P, placenta; PC, placenta creta; PI, placenta increta; PP, placenta percreta; S, serosa. Jauniaux E. Pathophysiology and ultrasound imaging of placenta accreta spectrum. Am J Obstet Gynecol 2018. (*From* Jauniaux E, Collins S, Burton GJ. Placenta accreta spectrum: pathophysiology and evidence-based anatomy for prenatal ultrasound imaging. Am J Obstet Gynecol 2018;218:75-87.)

ABRUPTIO PLACENTAE

Defined as the separation of the placenta from its uterine attachment after 20 weeks GA and before fetal delivery, this condition (**Fig. 3**) affects approximately 1% of all pregnancies[25] and results in perinatal mortality in approximately 10% of cases.[26] Abruptio placentae (AP) is the most common cause of bleeding after 20 weeks GA and most commonly occurs between 24 and 26 weeks GA.[27] In addition, AP can cause significant maternal morbidity, including antepartum or postpartum hemorrhage, sepsis, acute kidney injury, pulmonary edema, acute myocardial infarction, cardiomyopathy, disseminated intravascular coagulopathy death, and increased risk of transfusion and hysterectomy.[28,29] Neonatal complications include consequences of prematurity, hypoxia or asphyxia, IUGR, and congenital anomalies.[2,3]

AP most commonly occurs in patients who have had AP in a previous pregnancy. Other risk factors for AP include advanced maternal age, in vitro fertilization, thrombophilia, hypertension, preeclampsia, PP, chorioamnionitis, smoking, use of stimulants such as cocaine, trauma, multiple gestation, and African American race.[5,30–32] The pathophysiology of AP is uncertain but is believed to be due to abnormal trophoblastic invasion that leads to hemorrhage from the spiral arteries[33]; AP may be caused by an unknown event occurring early in pregnancy.[34] AP is diagnosed based on score on a scale of 0 to 3 in which 0 represents no symptoms with only a small retroplacental clot detected; 1 denotes vaginal bleeding, uterine irritability, and tenderness with no evidence of fetal or maternal distress; 2 is consistent with vaginal bleeding, uterine contractions, no signs of maternal shock, but with the presence of fetal distress; and 3 with evident or concealed severe bleeding, persistent abdominal pain, maternal shock, and fetal distress or death.[35]

Although the so-called classic presentation of AP includes dark red vaginal bleeding with passage of clots and tetanic uterine contractions, the accuracy of clinical findings alone is poor; one study found that only 38% of patients with AP had both pain and bleeding when diagnosed. One source reports that 35% fof present with occult abdominal bleeding, and 68% present with occult abdominal pain.[25] Patients may also present with decreased fetal movement, and cardiotocographic abnormalities are noted in most cases.[25] Obstetric ultrasonography demonstrates low sensitivity for AP.[36]

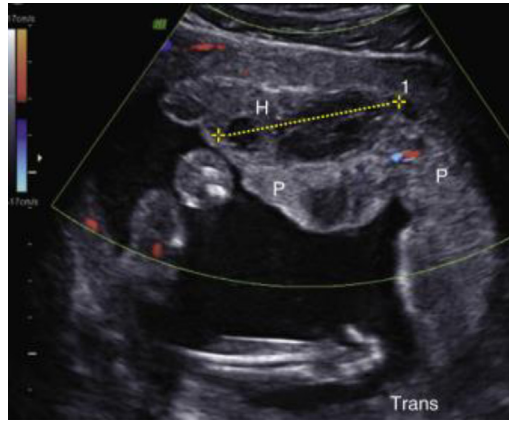

Fig. 3. Placental abruption.[24] (*From* Merriam A, D'Alton ME. "Placental abruption." In: Copel JA, D'Alton ME, Feltovich H et al. Obstetrical imaging: fetal diagnosis and care, 2nd ed. New York: Elsevier, 2018.)

Management of AP must first focus on maternal and fetal well-being. If the gravid patient is in shock, resuscitative measures must be undertaken expeditiously with large-bore intravenous (IV) access, IV fluids, and blood products. Once the mother is stabilized, plans for delivery will depend in part on the fetal status. In general, if the fetus is alive, emergent cesarean delivery is indicated; however, if there is fetal demise, vaginal delivery is preferable because it confers a lower risk of postpartum complications.[25] Aspirin greater than 100 mg daily instituted at or before 16 weeks GA may reduce the risk of AP in patients taking the drug for the prevention of preeclampsia.[37]

VASA PREVIA

VP (**Fig. 4**) is a rare entity affecting approximately 0.46 to 0.6 per 1000 pregnancies[39,40] that results from fetal vessels that are unprotected by Wharton jelly and that are placed through the membranes below the fetal presenting part and across the cervix.[41] VP either occurs when the vessel is connected to a velamentous cord

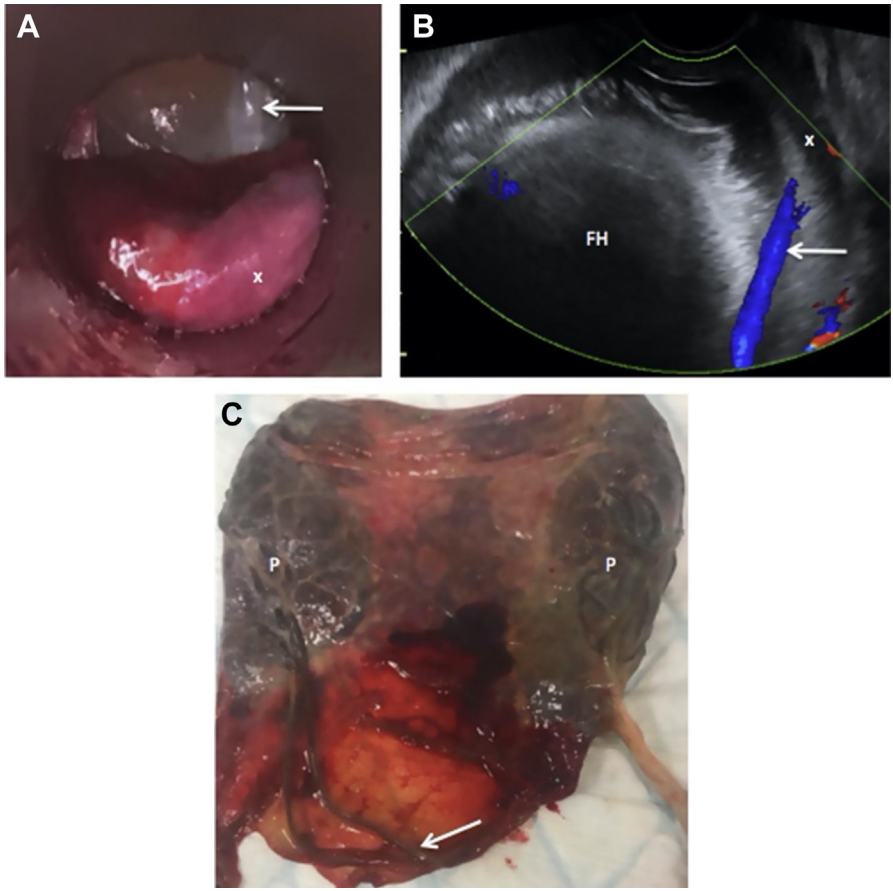

Fig. 4. Vasa previa[38] (*A*) amnioscopy showing vasa previa (*arrow*) in cervical dilatation area (X) (*B*) showing situation of vasa previa between fetal head (FH) and cervix (X) (*C*) showing two previa vessels connecting each part of bipartita placenta (P). (*From* Krief D, Naepels P, Chevreau J. Per labor vasa previa discovery: a simple clinical diagnosis. Eur J Obstet Gynecol reprod Biol 2018;231:284-285.)

(type I) or when it is instead connected with either a succenturiate or accessory placental lobe (type II).[4] These membranes can rupture and cause hemorrhage during spontaneous or artificial rupture of the membranes, or in the course of labor.[15] Because the fetal blood circulating volume is estimated to be less than or equal to 100 mL/kg, the fetus can exsanguinate rapidly[42,43]; when VP is not diagnosed antenatally, the risk of neonatal death is estimated at 60% or higher.[44] However, antenatal diagnosis and management can reduce this tragic statistic to nearly 0.[45]

Risk factors for VP include multiple gestation, velamentous cord insertion, presence of succenturiate lobe, and history of in vitro fertilization.[46] Clinically, VP should be suspected when a patient presents with painless vaginal bleeding, fetal distress, and rupture of the membranes.[47] However, today the diagnosis is usually made antenatally. Patients at high risk of VP, for example, those with a low-lying placenta, velamentous cord insertion, or succenturiate lobe should have a transvaginal ultrasonography with color Doppler; however, it is still possible for VP not to be identified with these modalities, and MRI remains an alternative to obstetric ultrasonography.[48]

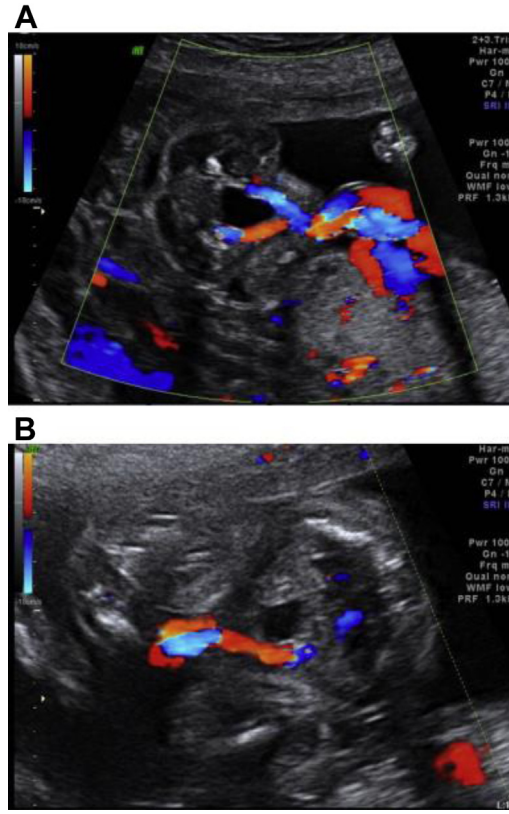

Fig. 5. Single umbilical artery[51] (*A*) Transverse view of fetal pelvis with color Doppler flow mapping showing bilateral umbilical arteries around the bladder in normal pregnancy (*B*) Transverse view of fetal pelvis with color Doppler ultrasound illustrating absence of color Doppler at the unilateral side of the umbilical artery in single umbilical artery. In this case, the right-side umbilical artery is missing. (*From* Wu Y-P, Tsai H-F, Cheng Y-C et al. Prenatal sonographic diagnosis of single umbilical artery: emphasis on the absent side and its relation to associated anomalies. Taiwan J Obstet Gynecol 2014;53:197-201.)

Patients diagnosed with VP antenatally should be considered for hospital admission at 30 to 32 weeks GA and should receive antenatal corticosteroids by 32 weeks; they should undergo scheduled cesarean delivery at 35 to 36 weeks, or emergently if spontaneous rupture of membranes occurs prior.[23]

SINGLE UMBILICAL ARTERY

The absence of 1 of the 2 umbilical arteries occurs in approximately 0.5% to 5% of pregnancies screened in the antenatal course,[49] in up to 1.6% euploid fetuses, but is more common in aneuploid fetuses, in which it is present in up to 11%.[50] Causes of single umbilical artery (SUA) (**Fig. 5**) include atrophy of a previously existing umbilical artery, primary agenesis, or persistence of the original allantoic artery of the body stalk.[52] Risk factors for this condition include advanced maternal age, smoking, multiple gestation, diabetes mellitus,[53] and in vitro fertilization. The term *isolated single umbilical artery* (iSUA) is used to denote cases in which there are no other fetal anomalies[54]; this entity is present in 80% of fetuses with SUA[55] and is also associated with an increased risk of PP, cord knots, and anomalous cord insertion.[56] In cases of SUA, fetal conditions include aneuploidy, structural malformations, low birth weight, NICU admission,[56,57] and IUGR,[58,59] with a higher incidence of such anomalies in cases of primary agenesis of the umbilical artery.[60] Thus, thorough anatomic scans are

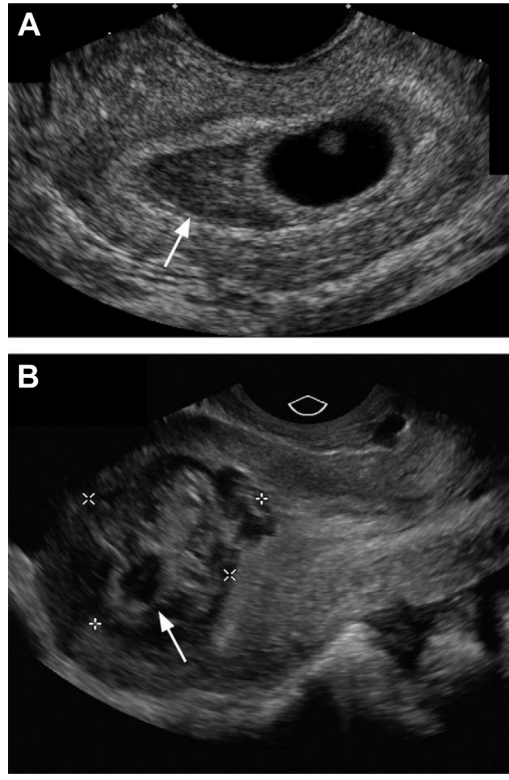

Fig. 6. Subchorionic hematoma[62] (*A*) Small, hypoechoic subchorionic bleed (less than one third of circumference of GS; *arrow*). (*B*) Large subchorionic bleed with heterogeneous echogenicity (between calipers) surrounding the GS (*arrow*). (*From* Mazzariol FS, Roberts J, Oh SK et al. Pearls and pitfalls in first-trimester obstetric sonography. Clin Imaging 2015;39:176-185.)

warranted when SUA is diagnosed during an obstetric ultrasonography in the mid-trimester. If iSUA is diagnosed, no other testing is indicated for aneuploidy.[61]

There is an increased risk of preterm birth, low birth weight, perinatal mortality, and hypertensive disorders in pregnancies affected by iSUA.[25,26] For fetuses with iSUA, the Society for Maternal-Fetal Medicine currently recommends a third-trimester ultrasonography for growth and weekly fetal surveillance at 36 weeks GA.[61]

SUBCHORIONIC HEMATOMA

Approximately 25% of all pregnant patients will experience first-trimester vaginal bleeding; a subchorionic hematoma (ScH) (**Fig. 6**) is a common diagnosis made in the patient with such vaginal bleeding[63] and is also the most common sonographic anomaly detected in the first trimester in the presence of a live fetus.[64] The incidence ranges significantly between 0.46% and 39.5% of pregnancies.[65] The condition is identified ultrasonographically by a hypoechoic or anechoic, crescent-shaped area between the chorion and myometrium.[66] The mechanism of this lesion is unknown but is believed to be due to a partial detachment from the uterine wall by the chorion.[67] AP may develop from an ScH, in particular, if it occurs in a retroplacental area.[68] Assisted reproductive technology is considered a risk factor for ScH[69,70]; however, it is also possible that causes of infertility, such as uterine pathology, obesity, and others, are also factors.[71] Although a causal association has not yet been established, associations between ScH and spontaneous abortion, abruption, preterm prelabor rupture of membranes, IUGR, hypertensive disorders of pregnancy, and placenta previa have been found.[72] However, in the first trimester, it remains unclear whether ScH is a risk factor for pregnancy loss, and retroplacental hematoma seems to be more commonly associated with such losses.[54]

CLINICS CARE POINTS

- PP is associated with the potential for maternal hemorrhage, caesarean delivery, and caesarean hysterectomy.
- Most patients today are diagnosed with PP via routine obstetric ultrasonography before presenting with bleeding.
- PAS is associated with a history of concomitant PP and prior history of caesarean delivery as well as with a maternal mortality rate of 7%.
- AP is the most common cause of vaginal bleeding after 20 weeks of gestation and is associated with a history of hypertensive disorders of pregnancy, maternal trauma, smoking, amphetamine use, and leiomyomata uteri.
- VP occurs when fetal vessels not protected by Wharton jelly are placed through the membranes below the fetal presenting part and across the cervix.
- Isolated SUA is associated with an increased risk of preterm birth, low birth weight, perinatal mortality, and hypertensive disorders of pregnancy.
- ScH is a common diagnosis made in the patient with first-trimester vaginal bleeding and is also the most common sonographic anomaly detected in the first trimester in the presence of a live fetus.

DISCLOSURE

The author has nothing to disclose.

REFERENCES

1. Kurman RJ, Ellenson LH, Ronnett BM, editors. Blaustein's pathology of the female genital tract. 7th edition. New York: Springer; 2019. p. 1225.
2. Turco MY, Moffett A. Development of the human placenta. Development 2019; 146:dev163428.
3. Feinshtein V, Ben-Zvi Z, Sheiner E, et al. Progesterone levels in cesarean and normal delivered term placentas. Arch Gynecol Obstet 2010;281:387–92.
4. Merriam A, D'Alton ME. Placenta previa. In: Copel JA, D'Alton ME, Feltovich H, et al, editors. Obstetrical imaging: fetal diagnosis and care. 2nd edition. New York: Elsevier; 2018.
5. Sebghati M, Chandraharan E. An update on the risk factors for and management of obstetric haemorrhage. Womens Health 2017;13:34–40.
6. Fan D, Xia Q, Liu L, et al. The incidence of postpartum hemorrhage in pregnant women with placenta previa: a systematic review and meta-analysis. PLoS One 2017;12:e0170194.
7. Jauniaux E, Moffett A, Burton GJ. Placental implantation disorders. Obstet Gynecol Clin North Am 2020;47:117–32.
8. Gibbins KJ, Einerson BD, Varner MW, et al. Placenta previa and maternal hemorrhagic morbidity. J Maternl Fetal Neonatal Med 2018;31:494–9.
9. Babayla J, Desilets J, Shrem G. Placenta previa and the risk of intrauterine growth restriction (IUGR): a systematic review and meta-analysis. J Perinat Med 2019; 47:577–84.
10. Jain V, Bos H, Bujold E, Society of Obstetricians and Gynaecologists of Canada. SOGC clinical practice guideline: diagnosis and management of placenta previa. J Obstet Gynaecol Can 2020;42:906–17.
11. Grönvall M, Stefanovic V, Paavonen J, et al. Major or minor placenta previa: does it make a difference? Placenta 2019;85:9–14.
12. Durst JK, Tuuli MG, Temming LA, et al. Resolution of a low-lying placenta and placenta previa diagnosed at the midtrimester anatomy scan. J Ultrasound Med 2018;37:2011–9.
13. Reddy UM, Abuhamad AZ, Levin D, et al. Fetal imaging: executive summary. Am J Obstet Gynecol 2014;210(5):387–97.
14. McPhaedran SE. Sexual activity recommendations in high-risk pregnancies: what is the evidence? Sex Med Rev 2018;6:343–57.
15. American College of Obstetricians and Gynecologists. Medically indicated late-preterm and early-term deliveries. ACOG Committee Opinion #831. Washington, DC: ACOG; 2021.
16. Jauniaux E, Collins S, Burton GJ. Placenta accreta spectrum: pathophysiology and evidence-based anatomy for prenatal ultrasound imaging. Am J Obstet Gynecol 2018;218:75–87.
17. American College of Obstetricians and Gynecologists and the Society for Maternal-Fetal Medicine. Obstetric care consensus. Placenta accreta spectrum. Obstet Gynecol 2018;132:e259–75.
18. Garmi G, Salim R. Epidemiology, etiology, diagnosis, and management of placenta accreta. Obstet Gynecol Int 2012;2012:873929.
19. Berkley EM, Abuhamad A. imaging of placenta accreta spectrum. Clin Obstet Gynecol 2018;61:755–65.
20. Silver RM, Branch DW. Placenta accreta spectrum. N Eng J Med 2018;378: 1529–36.

21. Cal M, Ayres-de-Campos D, Jauniaux E. International survey of practices used in the diagnosis and management of placenta accreta specrum disorders. Int J Gynaecol Obstet 2018;140:307–11.
22. Cahill AG, Beigi R, Heine RP, et al, Wax JR for the Society of Gynecologic Oncology, the American College of Obstetricians and Gynecologists, and the Society for Maternal-Fetal Medicine. Placenta accreta spectrum. Am J Obstet Gynecol 2018;219:B2–16.
23. Silver RM. Abnormal placentation: placenta previa, vasa previa, and placenta accreta. Obstet Gynecol 2015;126:654–8.
24. Merriam A, D'Alton ME. Placental abruption. In: Copel JA, D'Alton ME, Feltovich H, et al, editors. Obstetrical imaging: fetal diagnosis and care. 2nd edition. New York: Elsevier; 2018.
25. Li Y, Tian Y, Liu N, et al. Analysis of 62 placental abruption cases. Risk factors and clinical outcomes. Taiwan J Obstet Gynecol 2019;58:223–6.
26. Riihimäki O, Metsäranta M, Paavonen J, et al. Placental abruption and child mortality. Pediatrics 2018;142:e20173915.
27. Kinoshita Toshihiko, Takeshita Naoki, Takashima Akiko, Yasuda Yutaka, Ishida Hiroaki, Manrai Megumi. A case of life-threatening obstetrical hemorrhage secondary to placental abruption at 17 weeks of gestation. Clinics and practice 2014;4(1):605.
28. Downes KL, Grantz KL, Shenassa ED. Maternal labor, delivery, and perinatal outcomes associated with placental abruption: a systematic review. Am J Perinatol 2017;34:935–57.
29. Elkafrawi D, Sisti G, Araji S, et al. Risk factors for neonatal/maternal morbidity and mortality in African American women with placental abruption. Medicina 2020;56:174.
30. Fadi SA, Linnau KF, Dihe MK. Placental abruption and hemorrhage-review of imaging appearance. Emerg Radiol 2019;26:87–97.
31. Tikkanen M, Nuutila M, Hillesmaa V, et al. Clinical presentation and risk factors of placental abruption. Acta Obstet Gynecol Scand 2006;85:700–5.
32. Huls CK, Detlefs C. Trauma in pregnancy. Semin Perinatol 2018;l42:13–20.
33. Bräila AD, Gluhovschi A, Neaçsu A, et al. Placental abruption: etiopathogenic aspects, diagnostic and therapeutic implications. Rom J Morphol Embryol 2018;58:187–95.
34. Elsasser DA, Ananth CV, Prasad V, et al. Diagnosis of placental abruption: relationship between clinical and histopathological findings. Eur J Obstet Gynecol Reprod Biol 2010;148:125.
35. Qiu Y, Wu L, Xiao Y, et al. Clinical analysis and classification of placental abruption. J Matern Fetal Neonatal Med 2021;34:2952–6.
36. Jha Priyanka, Melendres Giselle, Bijan Bijan, Ormsby Eleanor, Chu Lisa, Li Chin-Shang, McGahan John. Trauma in pregnant women: assessing detection of posttraumatic placental abruption on contrast-enhanced CT versus ultrasound. Abdom Radiol (NY) 2017;42(4):1062–7.
37. Roberge S, Bujold E, Nicolaides KH. Meta-analysis on the effect of aspirin use for prevention of preeclampsia on placental abruption and antepartum hemorrhage. Am J Obstet Gynecol 2018;218:483–9.
38. Krief D, Naepels P, Chevreau J. Per labor vasa previa discovery: a simple clinical diagnosis. Eur J Obstet Gynecol Reprod Biol 2018;231:284–5.
39. Westcott JM, Simpson S, Chasen S, et al. Prenatally diagnosed vasa previa: association with adverse obstetrical and neonatal outcomes. Am J Obstet Gynecol MFM 2020;2:100206.

40. Villani LA, Pavalaganthrajah S, D'Souza R. Variations in reported outcomes in studies on vasa previa: a systematic review. Am J Obstet Gynecol MFM 2020; 2:100116.
41. Melcer Y, Maymon R, Jauniaux E. Vasa previa: prenatal diagnosis and management. Curr Opin Obstet Gynecol 2018;30:385–91.
42. Swank ML, Garite TJ, Maurel K, et al. Vasa previa: diagnosis and management. Am J Obstet Gynecol 2016;215:223e1–6.
43. Fischel Bartal M, Sibai BM, Ilan H, et al. Prenatal diagnosis of vasa previa: outpatient versus inpatient management. Am J Perinatol 2019;36:422–7.
44. Jauniaux E, Silver RM. Rethinking prenatal screening for anomalies of placental and umbilical cord implantation. Obstet Gynecol 2020;136:1211–6.
45. Oyelese Y. Vasa previa: time to make a difference. Am J Obstet Gynecol 2019; 221:539–41.
46. Pavalagantarajah Sureka, Villani Linda, D'Souza Rohan. Vasa previa and associated risk factors: a systematic review and meta-analysis. American Journal of Obstetrics and Gynecology MFM 2020;2(3):100117. In press.
47. Derbala Y, Grochal F, Jeanty P. Vasa previa. J Prenat Med 2007;1:2–13.
48. Gagnon R. No. 231: guidelines for the management of vasa previa. J Obstet Gynaecol Can 2017;39:e415–21.
49. Li T-G, Wang G, Xie F, et al. Prenatal diagnosis of single umbilical artery and postpartum outcome. Eur J Obstet Gynecol Reprod Biol 2020;24:6–10.
50. Blum M, Weintraub AY, Baumfeld Y, et al. Perinatal outcomes of small for gestational age neonates born with an isolated single umbilical artery. Front Pediatr 2019;7:79.
51. Wu Y-P, Tsai H-F, Cheng Y-C, et al. Prenatal sonographic diagnosis of single umbilical artery: emphasis on the absent side and its relation to associated anomalies. Taiwan J Obstet Gynecol 2014;53:197–201.
52. Kim HJ, Kim J-H, Chay DB, et al. Association of isolated single umbilical artery with perinatal outcomes: systematic review and meta-analysis. Obstet Gynecol Sci 2017;60:266–73.
53. Gersell DJ, Kraus FT. Diseases of the placenta. In: Kurman RJ, Ellenson LH, Ronnett BM, editors. Blaustein's pathology of the female genital tract. 7th edition. New York: Springer; 2019.
54. Ebbing C, Kessler J, Moster D, et al. Isolated single umbilical artery and the risk of adverse perinatal outcome and third stage of labor complications: a population-based study. Acta Obstet Gynecol Scand 2020;99:374–80.
55. Li Tian-Gang, Guan Chong-Li, Wang Jian, Peng Mei-Juan. Comparative study of umbilical cord cross-sectional area in foetuses with isolated single umbilical artery and normal umbilical artery. Journal of Obstetrics and Gynaecology 2021;28:1–6.
56. Ebbing Cathrine, Kessler Jörg, Moster Dag, Rasmussen Svein. Isolated single umbilical artery and the risk of adverse perinatal outcome and third stage of labor complications: A population-based study. Acta Obstetrica et Gynecologica Scandinavica 2019;99(3):374–80.
57. Luo Xiaohua, Zhai Shanshan, Na Shi, Mei Li, Shishong Cui, Yajuan Xu, Limin Ran, Lidan Ren, Teng Hong, Rui Liu. The risk factors and neonatal outcomes of isolated single umbilical artery in singleton pregnancy: a meta-analysis. Scientific Reports 2017;7:7396.
58. Hua Meiling, Odibo Anthony, Macones George, Roehl Kimberly, Crane James, Cahill Alison. Single umbilical artery and its associated findings. Obstetrics and gynecology 2010;115(5):930–4.

59. Mailath-Pokorny Mariella, Worda Katharina, Schmid Maximilian, Polteraurer Stephan, Bettelheim Dieter. Isolated single umbilical artery: evaluating the risk of adverse pregnancy outcome. European Journal of Obstetrics, Gynecology, and Reproductive Biology 2015;184:80–3.
60. Hasegawa J. Ultrasound screening of umbilical cord abnormalities and delivery management. Placenta 2018;62:66–78.
61. Society for Maternal-Fetal Medicine. Society for Maternal-Fetal Medicine consult series #57: evaluation and management of isolated soft ultrasound markers for aneuploidy in the second trimester. (Replaces Consults #10, Single umbilical artery, October 2010; #16, Isolated echogenic bowel diagnosed on second-trimester ultrasound, August 2011; #17, Evaluation and management of isolated renal pelviectasis on second-trimester ultrasound, December 2011; #15, Isolated fetal choroid plexus cysts, April 2013; #27, Isolated echogenic intracardiac focus, August 2013). Am J Obstet Gynecol 2021;32. https://doi-org.eresources.mssm.edu/10.1016/j.ajog.2021.06.079.
62. Mazzariol FS, Roberts J, Oh SK, et al. Pearls and pitfalls in first-trimester obstetric sonography. Clin Imaging 2015;39:176–85.
63. Karaçor T, Bülbül M, Nacar MC, et al. The effect of vaginal bleeding and nonspecific pelvic pain on pregnancy outcomes in subchorionic hematomas case. Ginekol Pol 2019;90:656–61.
64. Hashem A, Sarsam SD. The impact of incidental ultrasound finding of subchorionic and retroplacental hematoma in early pregnancy. J Obstet Gynecol India 2019;69:43–9.
65. Naert MN, Khadroui H, Rodriguez AM, et al. Association between first-trimester subchorionic hematomas and pregnancy loss in singleton pregnancies. Obstet Gynecol 2019;134:276–81.
66. Zhou J, Wu M, Wang B, et al. The effect of first trimester subchorionic hematoma on pregnancy outcomes in patients under went IVF/ICSI treatment. J Matern Fetal Neonatal Med 2017;30:406–10.
67. Sükür Yavuz, Göc Gösku, Köse Osman, Açmaz Gökhan, Özmen Batuhan, Atabekoglu Cem, Koç Acar, Söylemez Feride. The effects of subchorionic hematoma on pregnancy outcome in patients with threatened aboriton. Journal of Turkish-German Gynecological Association 2014;15(4):239–42.
68. Palatnik Anna, Grobman William. The relationship between first-trimester subchorionic hematoma, cervical length, and preterm birth. American Journal of Obstetrics and Gynecology 2015;213(3):403e1–4.
69. Reich J, Blakemore JK, Grifo JA. Comparison of subchorionic hematoma in medicated or natural single euploid frozen embryo transfer cycles. Fertil Steril 2020;114:595–600.
70. West BT, Kavoussi PK, Odenwald KC, et al. Factors associated with subchorionic hematoma formation in pregnancies achieved via assisted reproductive technologies. J Assist Reprod Genet 2020;37:305–9.
71. Rydze RT, Bosler J, Schoyer KD. Subchorionic hematoma and implantation: can better understanding the former help the latter? Fertil Steril 2020;114:509–10.
72. So S, Mochizuki O, Yamaguchi W, et al. Impact of subchorionic hematoma in early pregnancy on obstetric complications: a retrospective cohort study in women who had live births after frozen-thawed embryo transfer. Reprod Med Biol 2020;19:398–403.