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Vaginal Bleeding in Pregnancy: Part II

This issue is the second in a two-part series covering vaginal bleeding in pregnancy. Part I discussed spontaneous abortion and ectopic pregnancy. Part II will discuss other causes of vaginal bleeding that typically present later in pregnancy.

Gestational Trophoblastic Disease. Gestational trophoblastic disease (GTD) refers to a group of benign and malignant tumors that arise from trophoblastic cells of the placenta. These tumors secrete high levels of beta-hCG and therefore cause symptoms similar to early pregnancy. Because of this, the diagnosis is often not made on initial presentation to the ED. The most common clinical manifestation is vaginal bleeding.[1] GTD is a rare complication of pregnancy found in 1 per 1,500 live births in the United States. The main risk factors are extremes of maternal age and a history of previous GTD.[2] GTD is divided into 4 main categories: hydatiform mole (molar pregnancy), persistent or invasive mole, placental site trophoblastic tumor, and choriocarcinoma. Despite differences in management, this group of tumors has an excellent prognosis even when metastatic disease is present.

Hydatiform mole accounts for 90% of GTD and is the most likely of this group of diseases to be encountered in the emergency department.[3] Molar pregnancies are caused by an abnormally fertilized ovum and overproliferation of trophoblastic tissue. They are benign tumors with a small but significant potential for malignant transformation. There are two genetically different types of molar pregnancies: complete and partial. A complete mole does not contain fetal tissue, while a partial mole does. Because fetal tissue is present with a partial mole, it is often indistinguishable from an incomplete or missed abortion by clinical examination and ultrasound.

Traditionally, a complete mole has been distinguished from other causes of GTD and vaginal bleeding in pregnancy by the following reported features:

- Uterine enlargement greater than expected for dates;
- Abnormally high beta-hCG. Levels often exceed 100,000 mIU/mL;
- Preeclampsia before the 20th week of gestation;
- Theca lutein cysts;
- Hyperthyroidism;
- Characteristic "snowstorm" pattern on ultrasound. (*See Figure 1.*)

These features are characteristic of "classic" disease in the second trimester and are uncommon in patients who seek medical attention earlier in their pregnancy.[4] Patients typically now present with vaginal bleeding in their first trimester when sonography is routinely performed and the diagnosis is made before these classic findings are detected.

The typical "grape-like" appearance on ultrasound is due to histological changes causing edema of the chorionic villi. An ultrasound done in the first trimester often misses both types of molar pregnancy, even with a high index of suspicion. A study of more than 1,000 patients referred to a trophoblastic disease center suspected of having hydatiform mole compared pre-evacuation

Executive Summary

- Ultrasound is not sensitive for two important causes of vaginal bleeding: molar pregnancy during the first trimester and placental abruption during the second trimester.
- Fetal monitoring should be instituted in the ED for women with vaginal bleeding and a potentially viable fetus.
- Painless bleeding in the second trimester suggests placenta previa.
- Painful bleeding in the second trimester suggests placental abruption.

ultrasound results with postoperative histology. The sensitivity and specifity of ultrasound examination in detecting histologically confirmed hydatiform mole was 44% and 74%, respectively (LR+ = 1.8 and LR- = 0.8).[5]

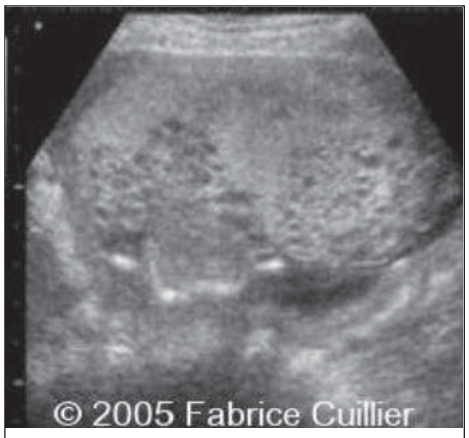
Molar pregnancies must always be treated surgically. In suspected cases, surgical evacuation of the uterus provides histological confirmation. The minimal lab work needed prior to surgery is a complete blood count and blood typing. Some authors also recommend a CXR and liver function tests to assess for metastasis, and a baseline beta hCG. RhoGHAM should be given if indicated, as described in part I of this article. Fifteen to 20% of women with a complete hydatiform mole and 2% to 3% with a partial mole will develop malignant sequelae.[6] For this reason, all patients with a molar pregnancy require close monitoring of beta hCG levels. A beta hCG that has plateaued or is rising should prompt the clinician to pursue a work-up for malignant GTD.

Malignant Gestational Trophoblastic Disease. Malignant GTD refers to the cancerous changes in gestational trophoblastic disease. There are three different types of malignant GTD: persistent or invasive mole, choriocarcinoma, and placental site trophoblastic tumors. Each of these may develop after a molar or nonmolar pregnancy and may present with local invasion or metastatic disease. The majority of malignant GTD, however, come from persistent disease after a complete molar pregnancy, and are not metastatic. Patients are usually asymptomatic, and the diagnosis is made when routine hCG monitoring shows a plateauing or increasing level of beta-hCG. Of patients who are symptomatic, vaginal bleeding is the most common complaint.[7] Additional characteristics are associated with the possible development of GTD after a molar pregnancy. (*See Table 1.*)

Choriocarcinoma is a very aggressive form of GTD that is often metastatic at the time of diagnosis. The most common sites of metastasis are the lung and vagina. The liver and brain are less commonly involved.[8] Choriocarcinoma is a very vascular tumor and typically presents with bleeding in the postpartum period. Choriocarcinoma follows a molar pregnancy in 1/40 women. It is uncommon after normal gestation or abortion, occurring in approximately 1/15,000-1/16,000 pregnancies.[8] Placental site trophoblastic tumors are extremely rare, accounting for about 1% of malignant GTD.[9]

In the absence of a known molar pregnancy, the diagnosis of malignant GTD is difficult to make on clinical grounds. The diagnosis is made by histology of a surgical specimen or after investigation of a high or abnormally rising beta hCG when normal pregnancy is excluded. It is important, therefore, to maintain a high index of suspicion for this disease in any patient after an abortion, ectopic pregnancy, or normal delivery with a beta hCG level higher than expected. Ultrasound in both invasive mole and choriocarcinoma may show an abnormal uterine mass that often is hypervascular on color Doppler. The treatment of malignant GTD is chemotherapy. In general, single-agent chemotherapy with methotrexate or dactinomycin may be used for low-risk disease, with good results. Combination chemotherapy is used for high-risk cases.

Figure 1: "Grape-like" Appearance of Molar Pregnancy on TVS



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Vaginal Bleeding in the Second Half of Pregnancy

Vaginal bleeding in the second half of pregnancy is also termed antepartum bleeding. It occurs in 4-5% of pregnancies and, by definition, is not related to labor and delivery.[10] Although it is much less common than bleeding earlier in pregnancy, it is more likely to be life-threatening. Fetal viability can occur as early as 23 weeks gestation, so any significant bleeding after this gestational age threatens the health of both the mother and the fetus. Bleeding in the second half of pregnancy is associated with a number of perinatal

Table 1: Characteristics Concerning for the Development of Malignant GTD After Molar Pregnancy

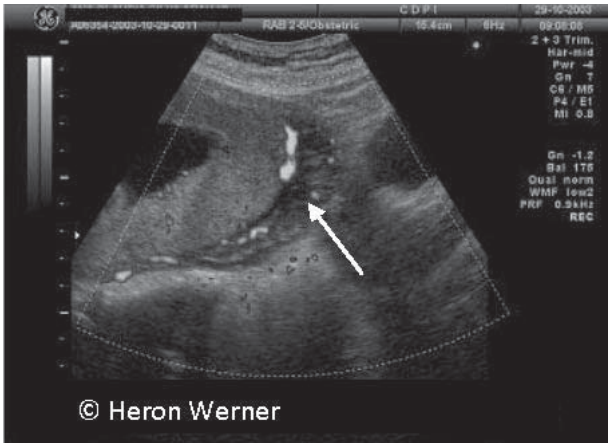
- Initial hCG > 100,000 mIU/mL
- Theca lutein cysts > 6 cm
- Excessively enlarged uterus
- Age > 40 years
- Histology revealing hyperplasia or atypia

complications, including preterm birth.[11] The ED evaluation involves stabilizing the patient, implementing fetal monitoring if obstetrical consultation is delayed, and obtaining timely obstetrical evaluation. Bedside ultrasound examination should be performed if it does not interrupt patient management. No patient with bleeding in this time period should be discharged from the ED without evaluation by the obstetrical service. Placentia previa, placental abruption, uterine rupture, and vasa previa are the major causes of obstetrical bleeding in the second half of pregnancy, and will be discussed here.

Placenta Previa. Placenta previa is a condition in which the placenta implants in the lower uterine segment near, or covering, the internal cervical os. (*See Figure 2.*) During labor, the dilation of the cervix can disrupt the vascular integrity of the placental attachment, and significant bleeding may occur. It occurs in approximately 1/300 deliveries in the United States and carries a maternal mortality rate of about 0.03%.[12] The etiology of placenta previa is unknown, but several independent risk factors have been identified: advanced maternal age, prior C-section, and infertility treatment.[12] In the first half of pregnancy, ultrasound shows placenta previa in up to 6% of patients. As the uterus grows, most of these migrate away from the internal os.[13]

Placenta previa characteristically presents with painless vaginal bleeding. A small percentage of patients also have uterine contractions.

Figure 2: Placenta Previa



The color Doppler shows vascularity within the placenta (arrow). Reprinted with permission from TheFetus.net. © Heron Werner.

Bleeding results from shearing forces on the placental attachments as the lower uterine segment grows and the cervix dilates. A woman may experience an initial episode of bleeding that resolves, only to return later in pregnancy. Placenta previa is associated with significant complications to mother and fetus at delivery, including postpartum hemorrhage and preterm delivery.[14] In the perinatal setting, recent research has found an association between short cervical length (< 30 mm) on transvaginal ultrasound and preterm delivery and hemorrhage in third-trimester patients with placenta previa.[15]

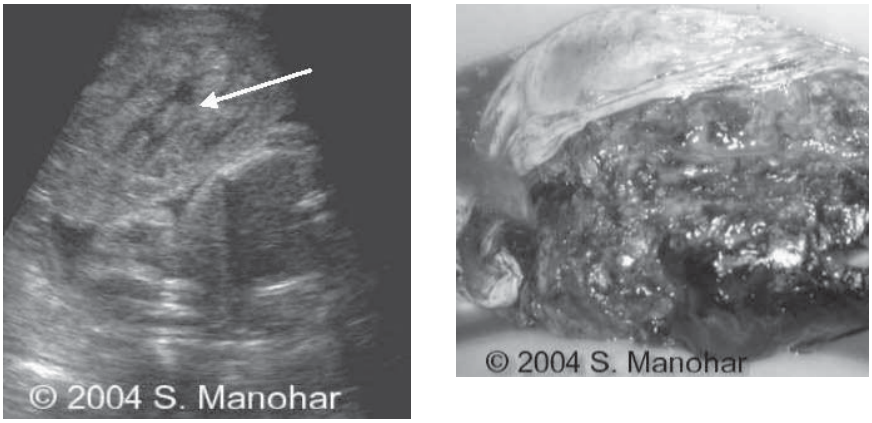
There are three types of placenta previa: total placenta previa, partial placenta previa, and marginal placenta previa. This classification is based on the proximity of the placenta to the internal os:

- Total placenta previa: the placental completely covers the internal os.
- Partial placenta previa: the placenta partly covers the internal os.
- Marginal placenta previa: the placenta is next to the internal os.

A patient who presents in the second half of pregnancy with painless vaginal bleeding is considered to have placenta previa until proven otherwise. ***Pelvic examination is contraindicated*** in these patients because of the risk of life-threatening hemorrhage caused by insertion of the examiner's digit into the cervical canal, impacting the highly vascular placenta. Emergency obstetrical consultation should be obtained, along with bedside fetal monitoring and preoperative laboratory studies. Ultrasound is the only safe method for reliably diagnosing placenta previa. TVS provides a better image of the location of the placenta to the internal os than transabdominal ultrasound, and is safe to perform.[16] Placenta previa is excluded if the placenta is greater than 2 cm from the internal os. There are several situations that make diagnosis of placentia previa difficult on ultrasound. An overdistended bladder may distort the anatomy to give the appearance of a placenta previa when it is not present. A fetal head low in the pelvis may obstruct visualization of the placenta, and it may be difficult to localize the placenta if uterine contraction occurs.[16]

Emergent obstetrical consultation is required in cases of placenta previa that present with vaginal bleeding. Indications for emergency cesarean section include hemodynamic instability, a stable patient who deteriorates, a mature gestation estimated by age greater than 36 weeks or weight greater than 2500 g, and evidence of fetal distress.[17] In the ED, start aggressive fluid resuscitation, send preoperative labs, and begin fetal monitoring. In the stable patient, if bleeding has slowed or stopped and the fetus is not mature, inpatient observation is reasonable.

Figure 3: Placental Abruption with Retroplacental Clot



Ultrasound shows retroplacental clot (arrow) caused by a placental abruption. Fetal parts are visualized underneath the clot. The surgical specimen is seen on the right with clot visible. Reprinted with permission from TheFetus.net. © 2004 S. Manohar.

There is currently no firm evidence to favor prolonged hospitalization over at-home observation for stable patients, and the decision should be made in conjunction with the patient and her obstetrician.[18]

Placental Abruption. Placental abruption refers to the premature separation of the placenta from its site of implantation in the uterus. It most commonly occurs around the 25th week of gestation, but this varies depending on the etiology of the abruption. Abruption occurs in approximately 1/100 births and is complicated by fetal death 15% of the time. The cause of abruption is unknown, but it has been associated with advanced maternal age, existing hypertension, cocaine use, trauma, smoking, and premature rupture of membranes.[19] Bleeding occurs into the decidual basalis, which then separates from the placenta. The separation may be large or small, and may dissect along the uterine placental junction and present as vaginal bleeding. Up to 20% of the time, the bleeding does not escape the placental margins and is concealed.[20] In this circumstance, vaginal bleeding is absent. The disruption of the maternal placental interface interferes with gas and nutrient exchange to the fetus with potentially devastating fetal compromise. If more than 50% of placental surface is disrupted, fetal death may occur.[21] Disseminated intravascular coagulation (DIC) is a serious complication of placental abruption that occurs in about 10% of patients. It is thought to be caused by leakage of a thromboplastin-like material from the placental system and is more common in cases associated with fetal distress and death.[22]

The classic presentation in placental abruption is painful vaginal bleeding and uterine contractions. The amount of vaginal bleeding is variable and does not correlate well with the extent or severity of the abruption and potential fetal risk.[23] The diagnosis is mainly a clinical one and should be suspected in any patient in the second half of pregnancy with painful vaginal bleeding, trauma, or preterm labor. If the placental separation is small, the physical findings may be minimal. In severe cases, hypotension and tachycardia, with uterine tenderness or palpable tetany are present on physical exam. Recent data from the New Jersey-Placental Abruption Study found a poor correlation between clinical predictors of abruption and a histopathological diagnosis.[24] Unfortunately, ultrasound is also often inconclusive in diagnosing abruption. The classic finding is a retroplacental clot (*see Figure 3*), but a normal ultrasound cannot exclude the diagnosis. Laboratory studies may reveal anemia and, in severe cases, evidence of DIC.

ED management of patients with placental abruption depends on the hemodynamic stability of the patient. In the unstable patient, the goal is to maintain hemodynamic stability and oxygenation of the patient. Aggressive volume resuscitation, supplemental oxygen, fetal monitoring, and emergent obstetric consultation should be instituted. Laboratory studies should include a complete blood count, blood type and crossmatch for two units of packed red blood cells, prothrombin time (PT), activated partial thromboplastin time (aPTT), and fibrinogen level. Transfuse blood emergently if bleeding is heavy and persistent, and administer fresh frozen plasma (FFP) if coagulopathy is present. If abruption is definitely diagnosed, and the fetus is alive and viable, preparation should be made for emergent C-section. If the fetus is dead, vaginal delivery should be attempted as soon as possible. In the stable patient, admit to the obstetrical service for continual fetal monitoring. Corticosteriods to accelerate fetal maturation and tocolytics to prevent labor are given at the discretion of the admitting service. In a minority of stable patients, vaginal delivery ultimately may be attempted.

Vasa Previa. Vasa previa is a rare condition in which the fetal blood vessels abnormally cross the internal os in the uterus. (*See Figure 4.*) Under normal circumstances the fetal vessels are housed in the umbilical cord, which inserts into the placenta. In vasa previa, the fetal vessels run through the fetal membranes without the protection of the cord and cross the internal os before they reach the placenta. Because they lie between the fetus and the cervix, there is no way to prevent trauma to these vessels when the membranes rupture or childbirth occurs. Since fetal circulation is involved, rupture of these vessels and loss of as little as 100 mL of blood can

quickly result in fetal death.[25] Risk factors associated with vasa previa include a low-lying placenta or placenta previa, velamentous insertion of the umbilical cord, multi-lobed placenta, multiple gestations, and in vitro fertilization.[25] Vasa previa occurs in about 1/2,500 pregnancies, but may be underrepresented if missed at ultrasound. In cases of velamentous insertion of the umbilical cord, rates increase to as high as 1/50.[26] Neonatal survival rates are greatly increased when the diagnosis is made antenatally.[27,28] Recent guidelines for the management of vasa previa published by The Society of Obstetricians and Gynaecologists of Canada showed a survival rate of 97% in cases diagnosed antenatally compared to a rate of 44% in those not diagnosed before symptoms appear.[29] Based on these findings, TVS screening should be performed with careful attention to the umbilical cord on all patients at risk.

The most common clinical presentation is vaginal bleeding at the time of rupture of membranes. Because this is a rare diagnosis that may be unknown to the patient, it is most commonly confused with placenta previa and placenta abruption.

The diagnosis of vasa previa is made by ultrasound and color Doppler prior to delivery or by inspection of the placenta after delivery. (*See Figure 3.*)

The optimal management of vasa previa is prenatal diagnosis and close monitoring, with elective C-section at about 35 weeks gestation. These patients often are hospitalized weeks earlier, when corticosteroids are administered to induce lung maturity in the fetus. The diagnosis is rarely made in the ED, and fetal survival is unlikely. In the rare instance of pre-term rupture of membranes with a live fetus, immediate C-section should be performed.

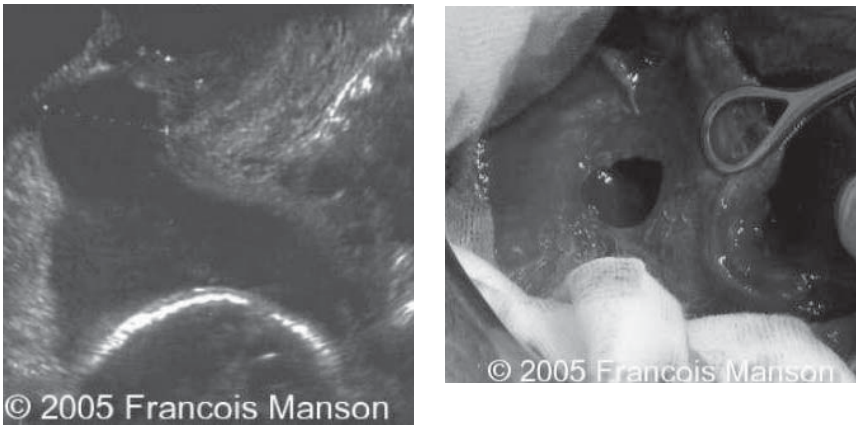
Uterine Rupture. Uterine rupture is caused by a complete tear in the uterine wall and is usually associated with labor. In rare instances, it occurs spontaneously in the second half of pregnancy. Risk factors in this circumstance include a previous classic cesarean section scar, uterine overdistension as seen in multiple gestations, cornual ectopic pregnancy, gestational trophoblastic disease, trauma, or placenta increta or percreta.[30] Most uterine ruptures studied occur in patients undergoing vaginal birth after cesarean section (VBAC). A recent review suggests the current at-risk population includes more women who have no history of previous uterine surgery and are not in labor than previously thought.[31] The true incidence is not precisely understood because the literature often fails to distinguish between true uterine rupture and scar dehiscence, and statistics often are limited to patients undergoing VBAC. In a large prospective observation study of almost 18,000 women who underwent a trial of labor after prior C-section, the rate of uterine rupture was 0.7%.[32]

Symptoms of uterine rupture not associated with labor can be misleading. Abdominal pain and vaginal bleeding is often minimal or even absent. In extreme situations, pain and bleeding may be severe, and hypovolemic shock may ensue. The fetus may protrude into the abdominal cavity, allowing fetal parts to be palpable on abdominal exam.

Mortality data, like prevalence data, are largely limited to information from VBAC births complicated by uterine rupture. Factors influencing morbidity and mortality include time to delivery after rupture occurs, the amount of blood lost, and the degree of placental separation from the uterus. In developed countries, maternal mortality has been reported to be as low as 0.2%, and fetal mortality, 5-6%.[33,34]

Ultrasound in the ED may reveal the rupture. (*See Figure 4.*) Once uterine rupture is suspected or diagnosed, steps should be taken to stabilize the mother and fetus in preparation for emergency cesarean section. Unfortunately, if complete uterine rupture occurs prior to arrival to the ED, fetal death usually has already occurred.

Figure 4: Uterine Rupture



Transvaginal ultrasound of uterine rupture in the lower uterine wall. The tear is clearly visualized interoperatively. Reprinted with permission from TheFetus.net. © 2005 Francois Manson.

References

1. Jauniaux E. Trophoblastic diseases and pregnancy. *The Obstetrician & Gynaecologist* 2003;5:130-135.
2. Tse KY, Chan KL, Tam KF. Gestational trophoblastic disease. *Obstetrics, Gynaecology & Reproductive Medicine* 2007;19:89-97.
3. Gerulath AH. Gestational trophoblastic disease. *SOCG Clinical Practice Guidelines* 2002;114:1-6.
4. Kirk E, Papageprgjiou A, Condous G. The accuracy of first trimester ultrasound in the diagnosis of hydatidiform mole. *Ultrasound in Obstetrics and Gynecology* 2007;29:70–75.
5. Fowler D, Lindsay I, Seckl M, et al. Routine pre-evacuation ultrasound diagnosis of hydatidiform mole: Experience of more than 1000 cases from a regional referral center. *Ultrasound in Obstetrics and Gynecology* 2006;27:56–60.

6. Gerulath AH. Gestational trophoblastic disease. SOCG Clinical Practice Guidelines 2002;114:1-6.
7. Berkowitz R, Goldstein D. Current management of gestational trophoblastic diseases. *Gynecologic Oncology* 2009;112:654-662.
8. Lurain J. Gestational trophoblastic disease I: Epidemiology, pathology, clinical presentation and diagnosis of gestational trophoblastic disease, and management of hydatidiform mole. *Am J Obstet Gynecol* 2010;doi:10.1016/j.ajog.2010.06.073.
9. Lan C, Li Y, He J, et al. Placental site trophoblastic tumor: Lymphatic spread and possible target markers. *Gynecologic Oncology* 2010;116:430-437.
10. Mukherjee S, Bhide A. Antepartum haemorrhage. *Obstetrics, Gynaecology & Reproductive Medicine* 2008;18:335-339.
11. Koifman A, Levy A, Zaulan Y, et al. The clinical significance of bleeding during the second trimester of pregnancy. *Arch Gynecol Obstet* 2008;278:47-51.
12. Oppenheimer L. *SOGC Clinical Practice Guideline* 2007;189:261-266.
13. Rosenberg T, Pariente G, Sergienko R, et al. Critical analysis of risk factors and outcome of placenta previa. *Arch Gynecol Obstet* 2010 [Epub ahead of print].
14. Zlatnik MG, Cheng YW, Norton ME, et al. Placenta previa and the risk of preterm delivery. *J Maternal-Fetal and Neonatal Medicine* 2007;20:719-723.
15. Stafford I, Dashe,J, Shivvers S, et al. Ultrasonographic cervical length and risk of hemorrhage in pregnancies with placenta previa. *Obstetrics & Gynecology* 2010;116:595-600.
16. Oppenheimer L. *SOGC Clinical Practice Guideline* 2007;189:261-266.
17. Dola CP, Longo SA. Diagnosis and safe management of placenta previa. *J Family Practice* 2006;18. http://www.jfponline.com/Pages.asp?AID=4488.
18. Neilson JP. Interventions for suspected placenta praevia. *Cochrane Database Syst Rev* 2003;(2):CD001998.
19. Oyelese, Y. Placental abruption. *Obstetrics & Gynecology* 2006;108:1005-1016.
20. Hladky K, Yankowitz J, Hansen WF. Placental abruption. *Obstet Gynecol Surv* 2002;57:299–305.
21. Oyelese, Y. Placental abruption. *Obstetrics & Gynecology* 2006;108:1005-1016.
22. Leunen K, Hall DR, Odendaal HJ, et al. The profile and complications of women with placental abruption and intrauterine death. *J Trop Pediatr* 2003;49:231-234.
23. Tikkanen M. Etiology, clinical manifestations, and prediction of placental abruption. *Acta Obstetricia et Gynecologica Scandinavica* 2010;89:732-740.
24. 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;148:125-130.
25. Gagnon R, Morin L, Bly S, et al. SOGC Clinical Practice Guideline: Guidelines for the management of vasa previa. *Int J Gynaecol Obstet* 2010;108:85-89.
26. Gagnon, R. Guidelines for the management of vasa previa. *JOGC* 2009;231:748-753.
27. Daly-Jones E, John A, Leahy A, et al. Vasa praevia: A preventable tragedy. *Ultrasound* 2008;16:8–14.
28. Oyelese Y, Catanzarite, V, Prefumo, F, et al. Vasa previa: The impact of prenatal diagnosis on outcomes. *Obstetrics & Gynecology* 2004;103:937-942.
29. Gagnona R, Morin L, Bly S, et al. Guidelines for the management of vasa previa. *International Journal of Gynecology & Obstetrics* 2010;108:85-89.
30. Vakim Z, Maymon R, Mendlovic, et al. Clinical, sonographic, and epidemiologic features of second- and early third-trimester spontaneous antepartum uterine rupture: a cohort study. *Prenatal Diagnosis* 2008;28:478–484.
31. Wuntakal R, Hollingworth T, Visvanathan D. Changing trends: Uterine rupture in the UK. *BJOG: An International Journal of Obstetrics & Gynaecology* 2010;117:1428-1429.
32. Landon M, Hauth J, Leveno K, et al. Maternal and perinatal outcomes associated with a trial of labor after prior cesarean delivery. *N Engl J Med* 2004;351:2581-2589.
33. Hofmeyr G, Say L, Gulmezogly AM. Systematic review: WHO systematic review of maternal mortality and morbidity: The prevalence of uterine rupture. *BJOG: An International Journal of Obstetrics & Gynaecology* 2005;112:1221-1228.
34. Lydon-Rochelle M, Holt V, Eastering T, et al. Risk of uterine rupture during labor among women with a prior cesarean delivery *N Engl J Med* 2001;345:3-8.

Physician CME Questions

91. Which of the following statements regarding gestational trophoblastic disease is true?
    A. It is a common complication of normal pregnancy.
    B. It is more commonly malignant than benign.
    C. It presents as a molar pregnancy most of the time.
    D. It has a mortality rate of 40% despite treatment.

92. Ultrasound is highly sensitive for diagnosing molar pregnancy in the first trimester.
    A. true
    B. false

93. All of the following suggest malignant gestational trophoblastic disease *except*:
    A. abnormal hypervascular uterine mass on color Doppler ultrasound
    B. rising serum beta hCG after an abortion
    C. persistent vaginal bleeding after treatment for an ectopic pregnancy
    D. persistent pelvic pain after a normal vaginal delivery

94. Which of the following statements regarding placenta previa is true?
    A. The bleeding is usually painless.
    B. Placenta previa is associated with a long cervical length.
    C. Bleeding usually presents during the first half of pregnancy.
    D. Transvaginal ultrasound (TVS) is not safe to perform in patients with suspected placenta previa.

95. Prolonged hospitalization is the standard treatment for patients with placenta previa, even if they are not bleeding.
    A. true
    B. false

96. Which of the following statements regarding placental abruption is true?
    A. Vaginal bleeding is usually painless.
    B. It most commonly occurs at 19 weeks of gestation.
    C. Disseminated intravascular coagulation is a severe complication that occurs in about 10% of patients.
    D. Vaginal bleeding is always present.

97. Which of the following statements regarding placental abruption is *false*?
    A. Ultrasound is a sensitive diagnostic test.
    B. Clinical predictors are not sensitive for the diagnosis.
    C. When diagnosed, emergent C-section should be performed if the fetus is viable.
    D. About 80% of cases present with vaginal bleeding.

98. Which of the following statements regarding vasa previa is *false*?
    A. Is it a common complication of velamentous insertion of the umbilical cord.
    B. Although a rare disease, it most commonly presents with vaginal bleeding.
    C. Patients are commonly hospitalized at 20 weeks for continuous monitoring.
    D. The diagnosis is usually made with color Doppler ultrasound.

99. Which of the following statements regarding uterine rupture is true?
    A. Maternal mortality exceeds fetal mortality.
    B. Maternal mortality increases with time from rupture to delivery.
    C. Classic symptoms of pain and vaginal bleeding are seen in more than 50% of patients.
    D. The most common risk factor is trauma from motor vehicle accidents.

100. Which of the following statements is true?
    A. Vaginal examination is contraindicated in a patient with placental abruption.

B. Patients with placenta previa usually present with uterine contractions.
C. The incidence of uterine rupture in women who undergo a trial of labor after previous C-section is less than 1%.
D. Choriocarcinoma occurs in about half of women with a molar pregnancy.

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13. Publication Name: Emergency Medicine Reports

14. Issue Date for Circulation Data Below: 08/17/10

15. Extent and Nature of Circulation			Average No. of Copies Each Issue During Preceding 12 Months	Actual No. Copies of Single Issue Published Nearest to Filing Date
a. Total No. Copies *(Net Press Run)*			3438	3395
b. Paid and/or Requested Circulation	(1)	Paid/Requested Outside-County Mail Subscriptions Stated on Form 3541. *(Include advertiser's proof and exchange copies)*	2218	2080
	(2)	Paid In-County Subscriptions *(Include advertiser's proof and exchange copies)*	0	0
	(3)	Sales Through Dealers and Carriers, Street Vendors, Counter Sales, and Other Non-USPS Paid Distribution	197	189
	(4)	Other Classes Mailed Through the USPS	92	83
c. Total Paid and/or Requested Circulation *(Sum of 15b(1) and 15b(2)*			2507	2352
d. Free Distribution by Mail *(Samples, Complimentary and Other Free)*	(1)	Outside-County as Stated on Form 3541	33	32
	(2)	In-County as Stated on Form 3541	0	0
	(3)	Other Classes Mailed Through the USPS	0	0
e. Free Distribution Outside the Mail *(Carriers or Other Means)*			20	20
f. Total Free Distribution *(Sum of 15d and 15e)*			53	52
g. Total Distribution *(Sum of 15c and 15f)*			2560	2404
h. Copies Not Distributed			878	991
i. Total *(Sum of 15g, and h.*			3438	3395
Percent Paid and/or Requested Circulation *(15c divided by 15g times 100)*			98%	98%

16. Publication of Statement of Ownership
Publication required. Will be printed in the 10/24/10 issue of this publication. ☐ Publication not required.

17. Signature and Title of Editor, Publisher, Business Manager, or Owner: President and CEO — Date: 9/27/10

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PS Form **3526**, September 1999 *(Reverse)*

CME Answer Key

91. C; 92. B; 93. D; 94. A; 95. B; 96. C; 97. A; 98. C; 99. B; 100. B

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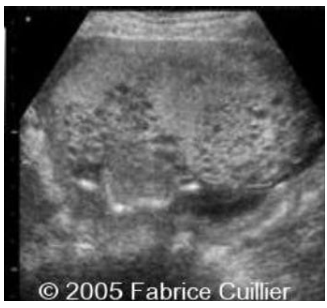
Emergency Medicine Reports
The Practical Journal for Emergency Physicians

Vaginal Bleeding in Pregnancy, Part II

"Grape-like" Appearance of Molar Pregnancy on TVS



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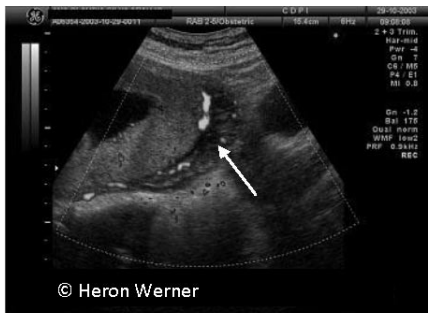
Characteristics Concerning for the Development of Malignant GTD After Molar Pregnancy

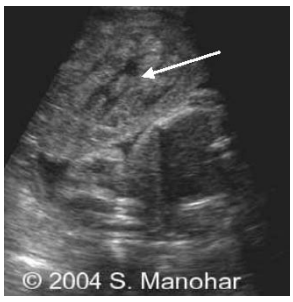
- Initial hCG > 100,000 mIU/mL
- Theca lutein cysts > 6 cm
- Excessively enlarged uterus
- Age > 40 years
- Histology revealing hyperplasia or atypia

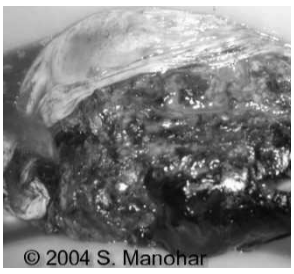
Placenta Previa



The color Doppler shows vascularity within the placenta (arrow). Reprinted with permission from TheFetus.net. © Heron Werner.

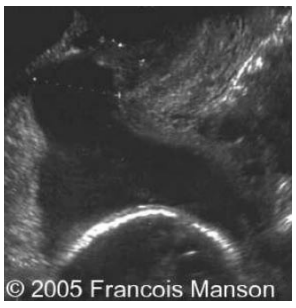
Placental Abruption with Retroplacental Clot

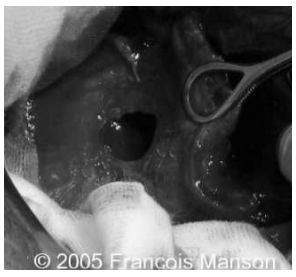




Ultrasound shows retroplacental clot (arrow) caused by a placental abruption. Fetal parts are visualized underneath the clot. The surgical specimen is seen on the right with clot visible. Reprinted with permission from TheFetus.net. © 2004 S. Manohar.

Uterine Rupture





Transvaginal ultrasound of uterine rupture in the lower uterine wall. The tear is clearly visualized interoperatively. Reprinted with permission from TheFetus.net. © 2005 Francois Manson.

Supplement to *Emergency Medicine Reports*, October 25, 2010: "Vaginal Bleeding in Pregnancy, Part II." *Author:* **Pamela Arsove, MD, FACEP**, Associate Residency Director, Department of Emergency Medicine, Long Island Jewish Medical Center, New Hyde Park, NY; Assistant Professor of Emergency Medicine, Hofstra North Shore — Long Island Jewish School of Medicine.