Mu'tah University || Medicine college

OBS & GYN SHEET

Antepartum Hemorrhage

Dr. Ahlam Alkharabsheh

Done by:
Obadah alayied

Designed by:
Bayan Mahmoud
Raina khattab
Raghad abu khalaf
Aman abu saqout
Hiba makkawi

Color code :

الأخضر :تبييض و تين + روح

الأزرق :ملاحظات خارجية

Nulliparous: no any pregnancy
complete 24 week ore more (may be miscarriage).
Nulligravida: no any pregnancy before.

Ante-partum hemorrhage (APH):
hemorrhage.. Birth canal = cervix, vagina & vulva
Any bleeding from the birth canal occurring after the 24th week
of gestation (some authors define this as the 20th week) and until the second stageof labor is complete.
APH complicates 3–5% of pregnancies.

Causes of early pregnancy bleeding as:
miscarriage, molar, ectopic ...etc.
Bleeding from any part other than birth canal (as bleeding from anus, urethra .. Etc) not considered antepartum
Ante-partum hemorrhage (APH):
hemorrhage.. Birth canal = cervix, vagina & vulva

Causes of APH
1) Placental abruption (30%)
Placenta Previa (20%)
3) Uterine rupture (rare)
4) Vasa Previa (rare)
5) Cervical and vaginal pathologies : Ectropion, polyps, tumors, varicosity. [local causes of bleeding]
Local causes of APH presented like : (early bleeding of pregnancy , spotting with minimal amount , post intercourse bleeding)

In the remaining cases, the exact etiology of the ante-partum bleeding cannot be determined.

Show = bloody stained mucous
Show is the term used to describe the small amount of blood with mucous discharge that may precede the onset of labor by as much as 72 hours. The amount of bleeding varies (minimal – heavy)

In antepartum hemorrhage, how to differentiate if this caused by local lesion or more serious causes (abruption & previa)??
- intialy, you have to think in any cause that may increase maternal mortality and rule out it.

- Associated symptoms: all abruption patient and 20% of placenta previa has abdominal pain, but in local patient (as cancer and others) never come with abdominal pain.
- Onset of bleeding: local causes of bleeding may start as early pregnancy bleeding (bedor 24 weeks), while abruption or previa not start befor week 24 (antepartum hemorrhage only)
- Amount of bleeding: in abruption and previa it is large amount, but in local it is minimal or spotting (vaginal spotting)
- Provocative factors: in local lesion it may related to intercourse (bleeding occur after intercourse), also intercourse may case bleeding in placenta previa patients.

Why show cause?

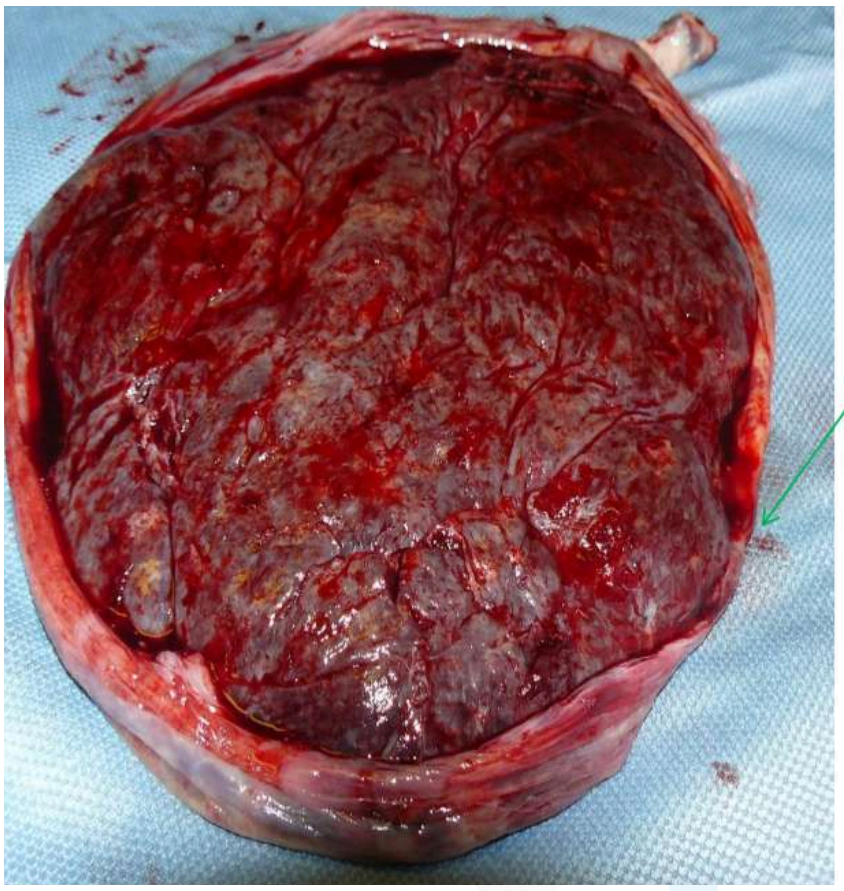
Because of cervical changes (dilation and effacement) during labor, which cause disruption of small blood vessels which cause rupture and bleeding, and mucus come from endocervix (layer of cervix, columnar epithelial lining, which secret mucous)

- Note: show is benign cause of bleeding occur at labor or before 72hrs

Placental abruption

- Bleeding at the decidual-placental interface that causes partial or total placental detachment , after 24 weeks of gestation and prior to delivery of the fetus.
- Placental abruption is the premature separation (partial or complete) of a normally situated placenta from the uterine wall, resulting in hemorrhage before the delivery of the fetus.
- a significant cause of maternal and perinatal mortality/morbidity.
- Incidence : 0.4-1 % of pregnancies.
- 40-60 % of abruptions occurred before 37 weeks of gestation and 14% occurred before 32 weeks.→ The perinatal death rate is approximately 12 % (versus 0.6 % in non- abruption births). The majority of perinatal deaths (up to 77 %) occur in utero due to intra uterine asphyxia ; deaths in the postnatal period areprimarily related to preterm deliver

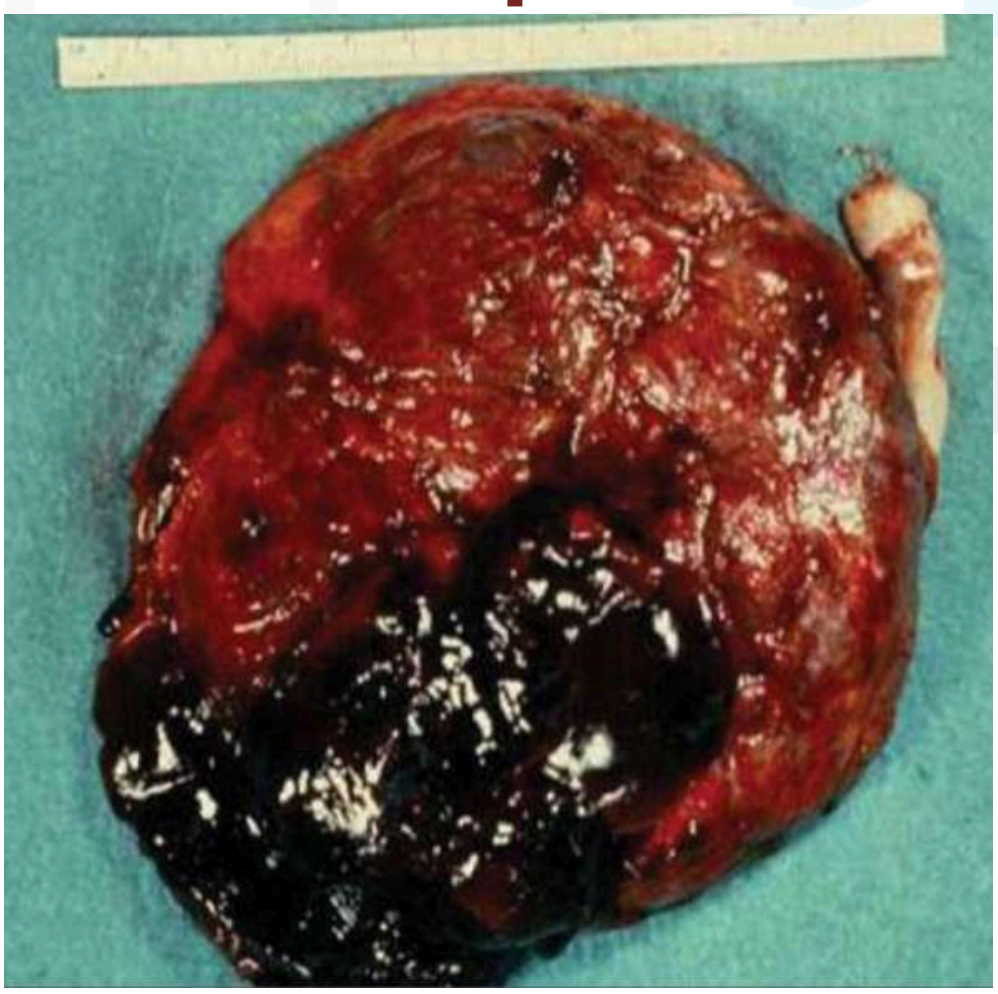
Normal placenta

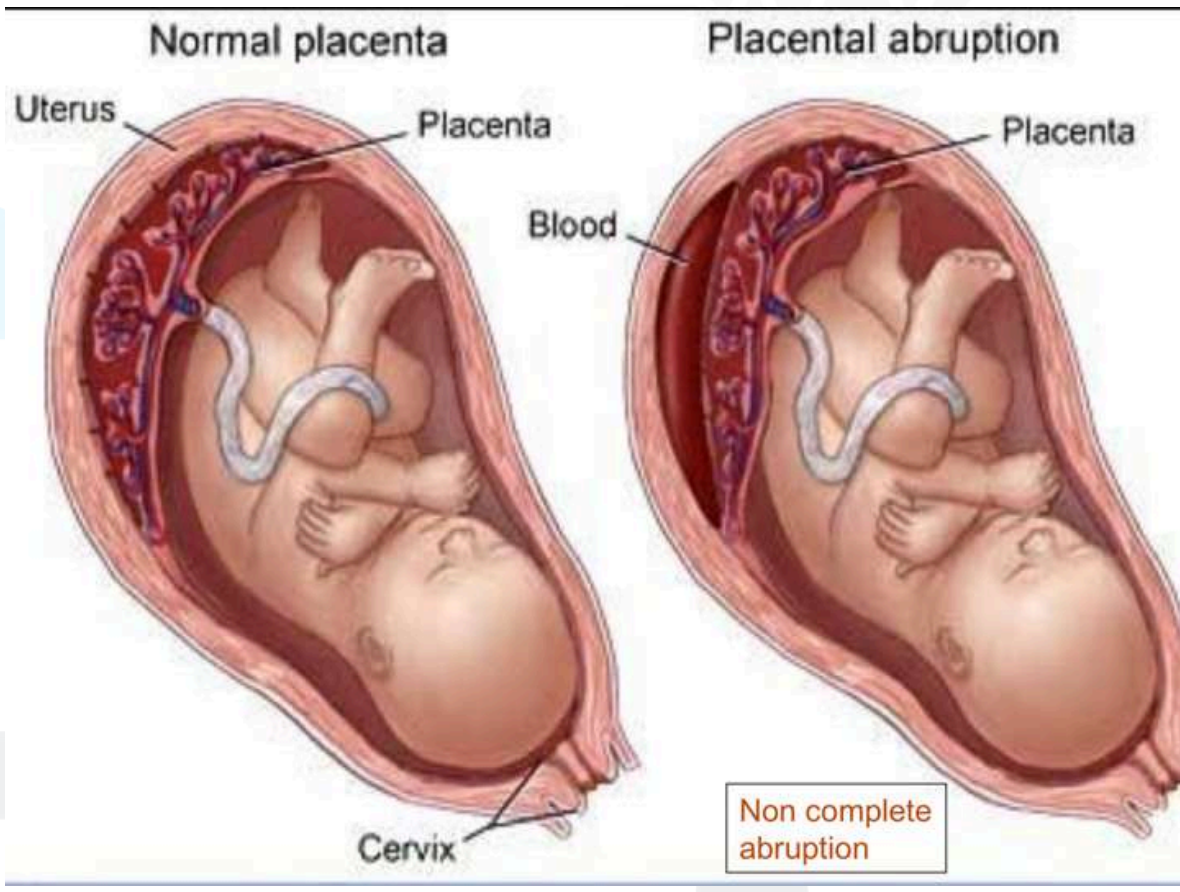


this is the Amniotic membrane

(chorionic + amniotic) fuse during the first trimester

Abrubtion plcenta





Premature Placental Separation (Abruption Placentae)
Under normal physiological conditions, placental separation occurs during the third stage of labor. Therefore, any separation before this stage is considered premature and termed placental abruption.

Placenta: Structure and Function
The placenta is the critical interface between maternal and fetal circulations, functioning as the site of gas exchange, nutrient delivery, and waste elimination. It is considered a fetal organ, despite being attached to the maternal uterus.

When abruption occurs, the separated portion of the placenta becomes non-functional, compromising oxygen and nutrient transfer to the fetus. The clinical impact on the fetus is directly proportional to the degree of placental separation.

Less than 50% Separation

- The fetus initiates compensatory mechanisms to preserve vital organ perfusion:
  - Blood is preferentially distributed to the brain and heart.
  - Renal perfusion is reduced, leading to decreased urine output.
-
- Since fetal urine is the primary source of amniotic fluid in the second half of pregnancy, reduced urination results in oligohydramnios.
- Fetal movement decreases gradually, which the mother may perceive as reduced activity.

More than 50% Separation

- This degree of separation often leads to acute fetal distress.
- Approximately 20% of such cases result in intrauterine fetal demise.
- Fetal heart rate monitoring (e.g., via CTG) is essential and may reveal:
  - Bradycardia
  - Late decelerations
  - Absent variability
  - Or intrauterine fetal death, depending on the extent and duration of separation.
-

In all cases of placental abruption, fetal movement is reduced:
- In mild separation, the decrease is gradual.
- In severe separation, the reduction is often sudden and pronounced, which mandates urgent fetal heart rate evaluation.

Decidua Basalis and Placental Anatomy

Decidua Basalis (Maternal Component of the Placenta):
- The decidua basalis is a specialized layer of the endometrium formed through a process called decidualization. This transformation occurs under hormonal influence during early pregnancy.
- It functions as a protective interface, ensuring that abruption or cellular sloughing affects this layer rather than damaging the underlying endometrial tissue.
- Preservation of the endometrium is essential for the potential of future pregnancies.

Placental Septa and Cotyledons:
- Septa originating from the decidua basalis project into the intervillous space, where maternal and fetal blood interact without mixing.
- These septa divide the maternal surface of the placenta into approximately 40 cotyledons, which are the functional units of exchange.

Fetal Circulation within the Placenta:
- The umbilical cord contains:
  - One umbilical vein: carries oxygenated blood from the placenta to the fetus.
  - Two umbilical arteries: carry deoxygenated blood from the fetus to the placenta.
-
- These vessels branch into chorionic arteries and veins, which further divide into spiral-shaped arterioles and venules. This spiral configuration increases the surface area available for exchange with maternal blood, enhancing the efficiency of nutrient and gas transfer.

Mechanism of Placental Abruption

1. Initial Trigger: Rupture of Maternal Blood Vessel

- Abruption begins with rupture of a maternal blood vessel, either a vein (low-pressure system) or an artery (high-pressure system).
- This endothelial injury activates the coagulation cascade, leading to clot formation.

A. Venous Rupture (Low-Pressure System)

What Happens:

- Slow bleeding occurs due to rupture of maternal veins.
- The body may stop the bleeding by forming a clot.
- However, the accumulated blood between the endometrium and placenta creates shearing forces, leading to partial placental separation.

◆ **Clinical Picture:**

Chronic Abruption

1. Gradual decrease in fetal movements.
2. Minimal intermittent spotting.
3. Ultrasound findings:
   - Smaller abdominal size due to intrauterine growth restriction (IUGR).
   - Oligohydramnios due to reduced fetal renal perfusion and urine output.
4.
5. Often associated with preeclampsia, especially in preterm cases, as both conditions share a common pathophysiology.
6. This presentation is known as chronic abruption.
7. Retrospective diagnosis: after delivery, the placenta is sent to pathology. The pathologist may find organized clots, confirming previous abruption.

B. Arterial Rupture (High-Pressure System)

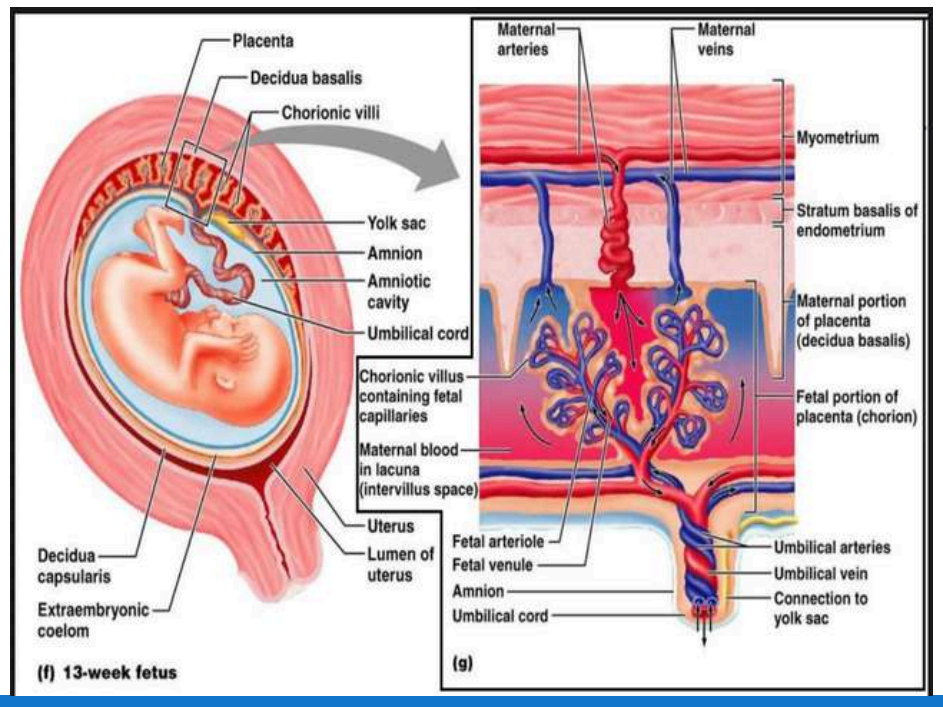
What Happens:

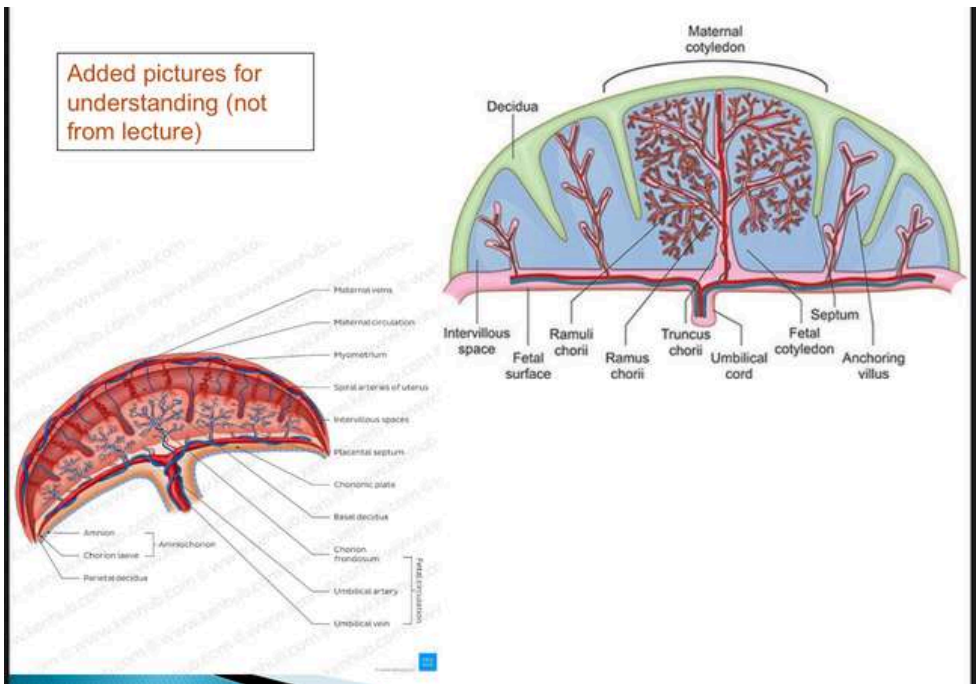
- Bleeding is usually severe and ongoing due to arterial rupture.
- The body repeatedly tries to activate the coagulation cascade, but the bleeding does not stop.
- Continuous consumption of clotting factors leads to Disseminated Intravascular Coagulation (DIC).

◆ **Clinical Picture:**

Acute Severe Abruption

1. Antepartum hemorrhage (visible vaginal bleeding).
2. Fetal distress or intrauterine fetal death is common.
3. DIC is most frequently seen when placental separation exceeds 50%.
4. Note: DIC is specific to placental abruption, and does not occur with placenta previa.
5. The bleeding is maternal, leading to hypovolemia, which further reduces blood supply to the fetus.





What Causes Vessel Rupture in Placental Abruption?
Placental abruption is initiated by the rupture of maternal vessels (veins or arteries). But what causes these vessels to rupture? Below are the primary mechanisms and risk factors:

1. Preeclampsia (Main Risk Factor in >50% of Abruptions)

- **Preeclampsia is the most significant risk factor for placental abruption.**
- **It results from abnormal placental implantation, leading to abnormal development of maternal and fetal arteries.**
- **These vessels are typically small and high-resistance, making them prone to ischemia, necrosis, and eventually rupture.**

2. Direct Abdominal Trauma
Examples:

- **Road Traffic Accidents (RTA)**
- **Domestic violence (e.g., falling, physical assault)**

Key Notes:

- **In RTA, especially with acceleration-deceleration forces:**
  - **The uterine muscle stretches during acceleration and contracts during deceleration.**
  - **The placenta cannot stretch or contract like the uterus, which leads to mechanical shearing and separation.**
-
- **History taking is crucial: Always ask about vaginal bleeding and fetal movements.**
- **Hospital observation is essential:**
  - **Ideally: 24 hours**
  - **At minimum: 4 hours if patient refuses longer stay**

3. Smoking and Cocaine Abuse

- **Up to 10% of cocaine users may experience placental abruption, typically in the third trimester.**
- **Both nicotine and cocaine have strong vasoconstrictive effects:**
  - **This leads to fragile blood vessels that rupture easily.**

- **Counseling on smoking and drug cessation is an essential part of abruption prevention.**

4. Rapid Uterine Decompression

- **As pregnancy advances, there is an increase in:**
  - **Fetal weight**
  - **Amniotic fluid**
  - **Intrauterine pressure**

Scenarios that increase intrauterine pressure:

- **Twin pregnancy**
- **Polyhydramnios**

Mechanism:

- **Sudden decompression (e.g., rapid rupture of membranes) leads to rapid shrinking of the uterine cavity.**
- **The uterine muscle contracts quickly, but the placenta cannot shrink due to its lack of elasticity or muscle fibers.**
- **This mismatch results in placental separation.**

Examples:

- **Twin pregnancy → after delivery of the first twin, placenta may separate.**
- **Polyhydramnios → especially after sudden rupture of membranes; controlled and gradual fluid release is safer.**

Note: This mechanism is the same as that seen in acceleration-deceleration injuries (see point 2).

Additional Placental and Circulatory Anatomy Notes

Fetoplacental Unit:

- **Composed of both:**
  - **Fetal part of the placenta**
  - **Maternal part (decidua basalis)**

Umbilical Cord:

- **Contains 3 vessels:**
  - **2 arteries (carry deoxygenated blood from fetus)**
  - **1 vein (carries oxygenated blood to fetus)**

Vessel Branching:

- **Vessels divide as follows:**
  - **Main stem vessels**
  - **→ Secondary divisions**
  - **→ Tertiary divisions**
  - **→ Spiral vessels in the chorionic villi**

Bleeding Characteristics:

- **Maternal venous bleeding:**
  - **Low-pressure system**
  - **Often stops spontaneously**
- **Maternal arterial bleeding:**
  - **High-pressure system**
  - **More severe**
  - **Can lead to massive bleeding and abruption**

Chorionic Villi Shape:

- **The spiral structure of fetal chorionic villi increases surface area, enhancing maternal-fetal exchange.**

Pathogenesis and Pathophysiology :

● **The immediate cause is rupture of maternal vessels in the decidua basalis, where it interfaces with the anchoring villi of the placenta. The accumulating blood splits the decidua, separating a thin layer of decidua with its placental attachment from the uterus. The bleed may be small and self-limited, or may continue to dissect through the placental-decidual interface, leading to complete or near complete placental separation. The detached portion of the placenta is unable to exchange gases and nutrients; when the remaining feto-placental unit is unable to compensate for this loss of function, the fetus becomes compromised.**

● **A small proportion of all abruptions are related to sudden mechanical events, such as blunt abdominal trauma or rapid uterine decompression like in (multiple pregnancy , twins , polyhydramnios) , which cause shearing of the inelastic placenta due to sudden stretching or contraction of the underlying uterine wall**

Part of trophoblastic cells make chorionic villi (which contains fetal blood vessels), and other part invade decidua basalis, so if there are no sufficient endometrium implantation, cause abnormality in vessels make it easily ruptured.

Suboptimal trophoblastic implantation may also explain the increased risk of abruption among women with a prior cesarean, uterine anomalies (bicornuate uterus), uterine synechiae, and leiomyoma
only first gand smoker

The mechanism underlying the relationship between smoking cigarettes or cocaine abuse and abruption is unclear. One hypothesis is that the vasoconstrictive effects of smoking (or cocaine) cause placental hypoperfusion(vasoconstriction), which could result in decidual ischemia, necrosis, and hemorrhage leading to placental separation.

About 5-10% of cocaine abuser will have abruption

Thrombin plays a key role in the clinical consequences of placental abruption, and may be important in its pathogenesis, as well.

- It is formed via two pathways: Either decidual bleeding leads to release of tissue factor (thromboplastin) from decidual cells, which generates thrombin.

OR decidual hypoxia induces production of vascular endothelial growth factor (VEGF), which acts directly on decidual endothelial cells to induce aberrant expression of tissue factor which then generates thrombin

The production of thrombin can lead to the following clinical sequelae:

1) Uterine hypertonus and contractions, as thrombin is a potent, direct uterotonic agent. So preterm labor and preterm delivery, if it occur in labor it cause rapid progress of labor (delivery less than 3hrs)
2) Triggering of coagulation e.g DIC

The major risk factors of placental abruption :

1) Previous abruption : the most important risk factor. The risk of recurrence has been reported to be 5-15 %,. After two consecutive abruptions, the risk of a third rises to 20-25 %.
2) Hypertension : 5 fold increased risk of severe abruption
3) Premature rupture of membranes (before 37w, it lead to chorioamnionitis)
4) Chorioamnionitis (uterine, placental and fetal membranes sealed from bacteria in vagina, so rupture of membranes lead to ascending infection from vagina, lead to chorioamnionitis .... Inflammatory process and cytokine release make BV more friable and easily ruptured
5) Abdominal trauma/accidents
6) Cocaine abuse
7) Polyhydramnios
8) Smoking during pregnancy : it is associated with a 2.5-fold increased risk of abruption
9) Maternal age advanced maternal age 39 ...
10) Parity
11) Multi-fetal gestation.
12) Thrombophilias.
13)Short cord (< 30 cm) normally 30-80 cm

If duration between rupture of membranes and labor (known as latency period) increases, risk of abruption increases... and duration increases if rupture occurs more distant from term, but bout 90% have rupture of membranes go in labor after one day

In maternal age and parity, there is no defined cause but it found to have larger placental size, so it at more risk for abruption and previa also

Thrombophilia :

1- hereditary : usually with past medical hx of thromboembolism
2- acquired : antiphospholipid syndrome (but this need the presence of (obstetric &non-obstetric) criteria to do work up in AP

- It is mostly with acquired thrombophilia (mostly anti-phospholipid syndrome), in obstetric criteria these patient ate more liable for abruption.
- Also in hereditary, mainly factor 5 Leiden mutation, prothrombin gene mutation.
- Not all patients with abruption need to diagnose thrombophilia, because thrombophilia mainly caused DVT.
- In APT (anti-phospholipid thrombophilia), thrombosis occur at atypical sites (as subclavian, hepatic), not in calf

Acute placental abruption : Abrupt onset of :

1) Vaginal bleeding. Painful
2) Abdominal pain (and/or back pain (according to site of placenta)).
3) Uterine contractions : usually high frequency and low amplitude, but a contraction pattern typical of labor is also possible and labor may proceed rapidly.
4) The uterus is often firm, and may be rigid and tender. Woody feel
5) Absent or low fetal movement.

- Vaginal bleeding ranges from mild and clinically insignificant to severe and life-threatening. Blood loss may be underestimated because bleeding may be retained behind the placenta and thus difficult to quantify (Concealed). The amount of vaginal bleeding correlates poorly with the degree of placental separation and does not serve as a useful marker of impending fetal or maternal risk. Maternal hypotension and fetal heart rate (FHR) abnormalities, however, suggest clinically significant separation that could result in fetal death and severe maternal morbidity.
- When placental separation exceeds 50 %, acute DIC and fetal death are common

In examination of abruption:
1. Vital signs (pressure, pulse)
2. Amount of vaginal bleeding and severity of abruption.
3. In abdominal examination:
- Put your hand on abdomen at uterine fundus, you feel uterine contractions, tense rigid uterus without relaxation.
- Normally in labor, contractions occur at (3-4 in 10 minutes which last about 40 sec), between contractions uterine tone completely relaxed, in thesepatients there are tetanic contractions (more than 5 contractions in 10 minutes last about 15sec or 20sec = mild contractions, without relaxation between contractions).
- Examine concealed abruption (patient has no bleeding or minimal bleeding, with abdominal pain.. And it will be hypovolemic, and mainly preterm labor

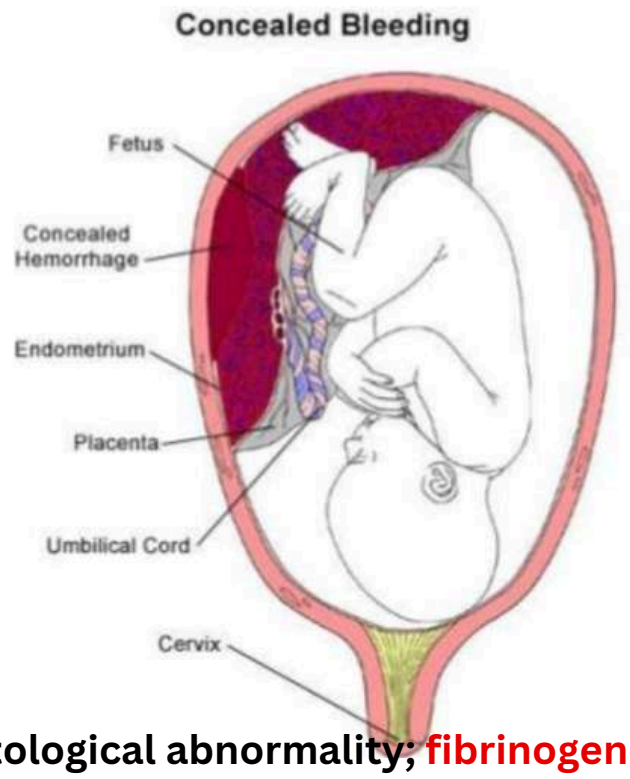
Table 1. Definition of different grades of placental abruption

Grade	Definition
0	Asymptomatic with a small retroplacental clot
1	Vaginal bleeding with no signs of maternal or fetal compromise
2	Vaginal bleeding with signs of fetal compromise
3	Vaginal bleeding accompanied by uterine tetany, abdominal pain, and signs of fetal and maternal compromise Coagulopathy in 1/3 of cases

- **Occasionally, the signs and symptoms of abruption develop after rapid uterinedecompression, such as after uncontrolled rupture of membranes in the setting of polyhydramnios or after delivery of a first twin. Signs and symptoms of abruption also may occur after maternal abdominal trauma or a motor vehicle crash. In these cases, placental abruption generally presents within 24 hours of the precipitating event and tends to be severe.**

Amount of bleeding doesn't indicate the severity of abrupto except in very heavy vaginal bleedin

In 10 -20 % of placental abruptions, patients present with only preterm labor, and no or scant vaginal bleeding. In these cases, termed 'concealed abruption,' all or most of the blood is trapped between the fetal membranes and decidua, rather than escaping through the cervix and vagina. Therefore, in pregnant women with abdominal pain and uterine contractions, even a small amount of vaginal bleeding should prompt close maternal and fetal evaluation for placental abruption. In other cases, a small concealed abruption may be asymptomatic and only recognized as an incidentalfinding on an ultrasound. Large for date uterus



Laboratory findings

The degree of maternal hemorrhage correlates with the degree of hematological abnormality; fibrinogen levels have the best correlation with severity of bleeding**.**
Initial fibrinogen values of ≤200 mg/dL are reported to have 100% positive predictive value for severe postpartum hemorrhage, while levels of ≥400 mg/dL have a negative predictive value of 79%. (coagulopathy)
- Mild separation/hemorrhage may not be associated with any abnormalities of commonly used tests of hemostasis.
- Severe abruption can lead to DIC, which occurs in 10-20 % of severe abruptions with death of the fetus.
CBC : Hb , pletlet count
Coagulation profile (pt , ptt , inr) >> change in cases of sever bleeding
Fibrinogen factor 1
Repeat the investigation again if the initial ones was normal
Coagulation profile (mainly fibrinogen and platelets) and Hb (because of hemorrhage).
Normal labs not ensure that no bleeding and coagulation cascade failure occur (stop of abruption), so you should to repeat labs after some hours to ensure

Why postpartum hemorrhage occur?

- After delivery of baby, delivery of placenta occur (stage 3 in labor), so you have to cut placenta and umbilical cord from maternal uterine arteries.

- Maternal artery is tortious to increase amount of blood supply to the baby, so in 10 weeks pregnant blood flow to baby is about 60ml / minute ... but at term it reaches 1000ml minute, if blood volume 5L or 6L it takes 5 minutes consumed and die occur, so hyper coagulo state during labor occur to constrict these vessels and prevent bleeding, so if these amount of blood consumed by abruption bleeding occur.

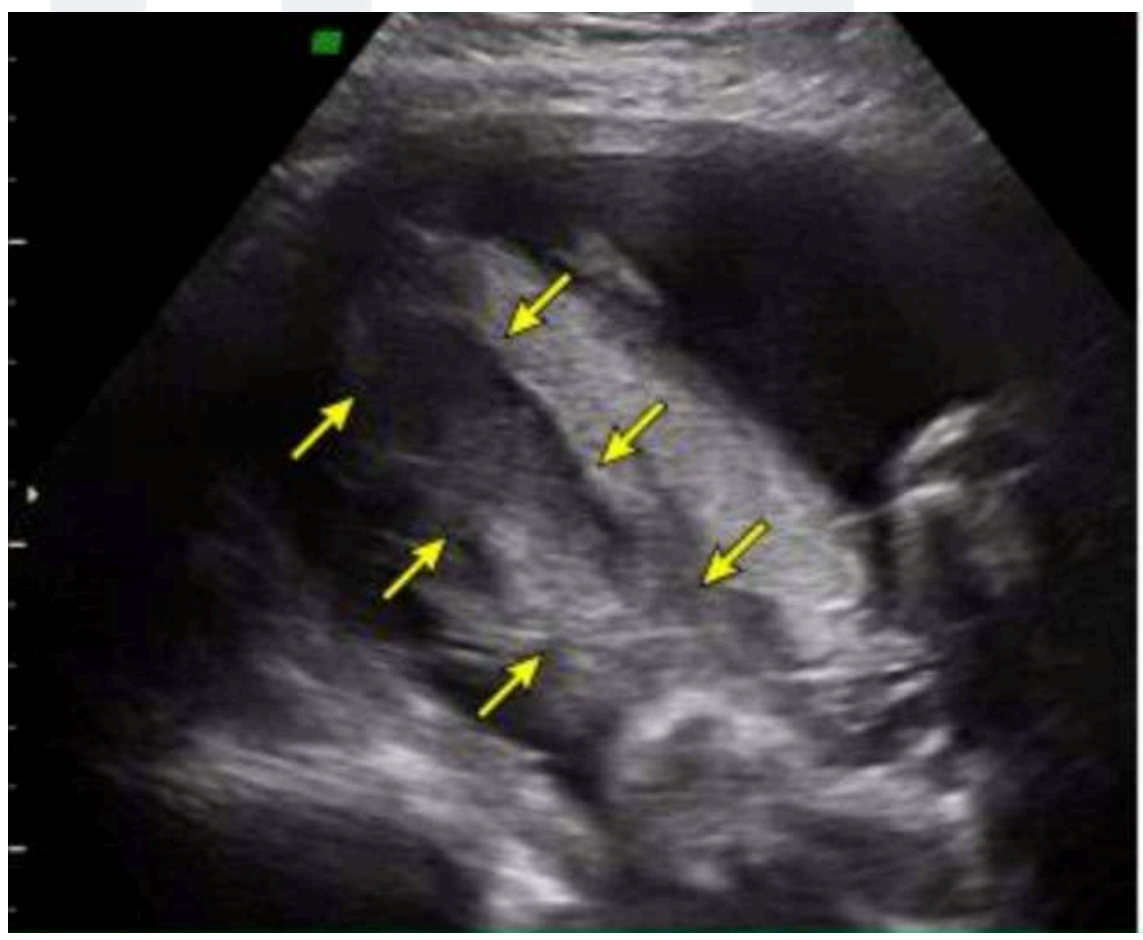
- Contraction of myometrium also will constrict these vessels, but if these uterine muscles fatigued because of tetanic contractions caused by abruption, postpartum hemorrhage occur

Imaging:

- **Identification of a retro-placental hematoma is the classic ultrasound finding of placental abruption. Retro-placental hematomas have a variable appearance; they can appear solid, complex, and hypo-, hyper-, or iso-echoic compared to the placenta.**
- **The sensitivity of ultrasound findings for diagnosis of abruption is only 25-50 %, but the positive predictive value is high (88 %) when ultrasound findings suggestive of abruption are present.**
- **The absence of retro-placental hematoma does not exclude the possibility of severe abruption because blood may not collect behind the placenta.**
- **MRI can detect abruptions missed by ultrasound examination, but increased diagnostic certainty is unlikely to change management or be cost-effectiv**

Diagnosis of abruption mainlyclinical diagnosis

US image retroplacental hematoma



Posterior placenta w/ large retroplacental hematoma outlined by arrows

Hematoma my be hypo, iso (as placenta) or hyper... known some times as jello-sign (moves when you move abdomen)

Consequences :

For the mother, the potential consequences of abruption are primarily related to the severity of the placental separation , while the risks to the fetus are related to both the severity of the separation and the gestational age at which delivery occurs.

With mild placental separation, there may be no significant adverse effects. As the degree of placental separation increases, the maternal and perinatal risks also increase.

Maternal:

- **Excessive blood loss and DIC generally necessitate blood transfusion and can lead to hypovolemic shock, renal failure, adult respiratory distress syndrome, multi-organ failure, peri-partum hysterectomy and, rarely, death .**
- **Emergency cesarean delivery for fetal or maternal indications.**

Fetal and neonatal:

- **Perinatal morbidity and mortality related to hypoxemia, asphyxia, low birth weight, and/or preterm delivery.**
- **Fetal growth restriction FGR (with chronic abruption).**

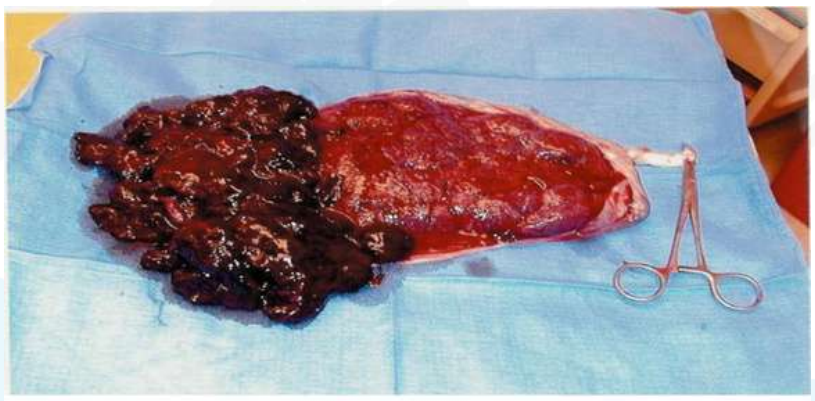
-Most commonly abruption occurs at term.

- Most common cause of fetal death is asphyxia, not prematurity.

-If abruption at preterm, there are two morbitis, which related to abruption and which related to preterma (prematurity

Placental pathology :

- **Gross examination of placenta after delivery may reveal the abruption.**



- **Histopathological examination of the placenta shortly after an acute abruption may not reveal any abnormality. In less acute cases, an organizing retroplacental hematoma indenting the parenchyma may be noted.**

In addition to features of recent infarct (needs 4-6 hours to develop

Chronic abruption:

- **Women with chronic abruption experience relatively light, chronic, intermittent bleeding and clinical manifestations of ischemic placental disease that develop over time, such as oligohydramnios (termed chronic abruption– oligohydramnios sequence), FGR, and preeclampsia.**

They are also at risk of preterm premature rupture of membranes.

- **Coagulation studies are usually normal.**
- **Ultrasound examination may identify a placental hematoma (retromembranous, marginal, or central), and serial examination may reveal FGR and/or oligohydramnios.**

- **Histological examination of the placenta may show chronic lesions, such as chronic deciduitis (lymphocytes with or without plasma cells), maternal floor decidual necrosis, villitis, decidual vasculopathy (specifically, in the vessels of the extraplacental membrane roll), placental Infarction, intervillous thrombosis, villous mal-development, and hemosiderin deposition**

Diagnosis :

The diagnosis of abruptio placentae is primarily clinical, but findings from imaging, laboratory, and postpartum pathologic studies can be used to support the clinical diagnosis

Chronic abrupto NOT THE ACUTE ONE associated with
(IUGR , oligohydramnios , preeclampsia , PROM)
Villiminous cord insertion also associated with (IUGR , oligohydramniosseverity of AP :
The main source of bleeding is from the mother , so the mother will be affected first:
1- maternal stability : vital signs (tachycardia , hypotension , shock >> sever AP)
2- fetal condition : (distress ,death>> sever AP)
3- amount of bleeding ; if
• Heavy amount = sever AP
• Mild amount ; you need to assess the above 2 factors because the bleeding amount isn't a good indicator for severity , unless ongoing heavy bleeding then it is sever
4- coagulopathy : DIC>> sever AP
To assess the presence of DIC : prolonged PT,PTT , thrombocytopenia , high D-dimer and fibrin degradation products , low fibrinogen (<100 mg\dl)

Managment:

Initial intervention for woman w/ potentially sever acute abruption : (admission to labor room)

A) Stabilization of the mother:

1) I.V fluid : Secure intravenous access with at least one, and preferably two, wide-bore intravenous lines. (1st step, immediately but cannulas)

2) Closely monitor the mother's hemodynamic status (heart rate, blood pressure, urine output). Urine output should be maintained at above 30 cc/hour and monitored with a Foley catheter.

3) Keep maternal oxygen saturation >95 percent and keep the patient warm.

4) Draw blood for a complete blood count, blood type and Rh (preparation of 4 units PRBCs), and coagulation studies. Repeat coagulation tests in patients with clinical signs of severe abruption as coagulopathy may develop or worsen over time.

5) Call for help.

6) Notify the anesthesia team. Anesthesia-related issues in these patients include management of hemodynamic instability, technical issues related to bleeding diathesis, and the potential need for emergency cesarean delivery.

7) Notify the blood bank so blood replacement products (red blood cells, FFP, cryoprecipitate, platelets) will be readily available, if needed.

B) Immediately initiate continuous fetal monitoring.

Always related to other:
Abruption– DIC– fetal death

In women with DIC, transfuse blood and blood products to achieve the following minimum levels:

- **Platelet count ≥50,000/microL**
- **Fibrinogen ≥100 mg/dL**
- **Prothrombin (PT) and partial thromboplastin time (PTT) less than 1.5 times control**
- **Hematocrit 25-30 %**

Normal values of DIC profile:
1- Fibrinogen 150-600 mg/dl
2- PT 11-16 se
3- PTT 22-37 sec
4- Fibrin degredation product (FDP) < 10µg/dl

If fetus is death, focus on resuscitation for mother (it will has DIC)

After initial interventions, the management depends on :

1- Severity of abruption (mild or severe)

2- Gestational age (mature or immature)

- **Severe abruption: characterized by one or more of the following:**

A) Ongoing major blood loss (external vaginal bleeding or concealed dentified by ultrasound).

B) Clinically unstable maternal status tachycardia/hypotension/tachypnia/oligourea)

C) Coagulopathy.

D) Dead fetus or compromized (Non reassuring FHR

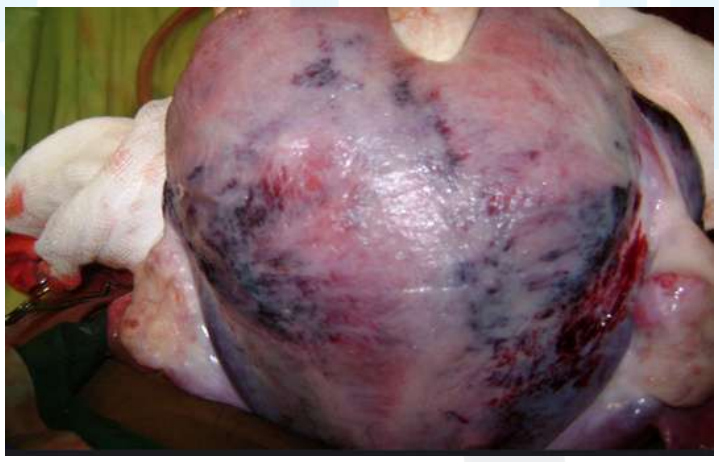
1) In pregnancies complicated by severe abruption → Expeditious delivery (vaginal or cesarean) (At any gestational age)
→ Vaginal delivery is reasonable if the mother is stable and the fetal heart tracing is reassuring. With a clinically significant abruption, the patient is often contracting vigorously, but if she is not in active labor, then amniotomy and administration of oxytocin frequently result in rapid delivery.
- The fetus should be continuously monitored.
- An attempt at vaginal birth should only be undertaken if there is access to immediate cesarean delivery, if necessary.

General anesthesia or spinal?
Depends on:
1. Spinal make hypotension.
2. Easier and faster for anesthesiologist.
3. Ccoagulopathy = no spina

Prompt cesarean delivery is indicated if the mother is unstable or the fetal heart tracing is nonreassuring and vaginal delivery is not imminent, or when vaginal delivery is contraindicated (malpresentation, prior classical cesarean delivery) or unsuccessful (failure to progress).
● Postpartum: Postpartum haemorrhage (PPH) should be anticipated in women who have experienced APH, so active management of third stage of labour is strongly recommended.

After delivery of anterior shoulder > give oxytocin + controlled cord traction

At cesarean delivery, blood extravasated into the myometrium (called a Couvelaire uterus) may be observed. (seen only with abruption, not other causes of antepartum hemorrhage).. (may not respond to uterotonic agents as oxytocin and uterus preserved lax, so bleeding occur



2) In non severe abruption : depends on gestational age
1- If immature fetus : Expectant management
2- If mature fetus : Delivery
- The optimum timing of delivery is not established, A senior obstetrician should be involved in determining the timing and mode of birth of these women

Expectant:
Admitte to hospital, observations, give steroids (to enhance lung maturity of the baby), give investigations, listen to fetal heart, if patient is negative blood group give anti D, keep patient in hospital to 37 or 38 weeks, then delivery

Expectant management :
1) Hospital stay: There are no compelling data to guide the length of a hospital stay in these pregnancies. A reasonable approach is to monitor the patient in the hospital until the bleeding has subsided for at least 48 hours, fetal heart rate tracings and ultrasound
examinations are reassuring, and the patient is asymptomatic. At that point, discharge may be considered. Importantly, the patient should be counseled to return immediately should she experience further bleeding, contractions, reduced fetal movement, or abdominal pain.
2) Single course of antenatal corticosteroid (26-35 weeks of gestation).
3) Serial assessment of fetal well being tests : NST / biophysical profie / doppler studies for fetal blood vessels / sonographic estimation of fetal weight to assess growth.
4) Anti-D immune globulin for Rh(D)-negative women.
5) Schedule delivery at 37-38 weeks because of the increased risk of stillbirth.
- Delivery before 36-37 weeks is indicated if additional complications arise (FGR, preeclampsia, PROM, non-reassuring fetal assessment, recurrent abruption with maternal instability

Placenta previa

Previa = going before

The presence of placental tissue that extends over or lies proximate to the internal cervical os (in the lower uterine segment). Sequelae include the potential for severe bleeding and preterm birth, as well as the need for cesarean delivery.

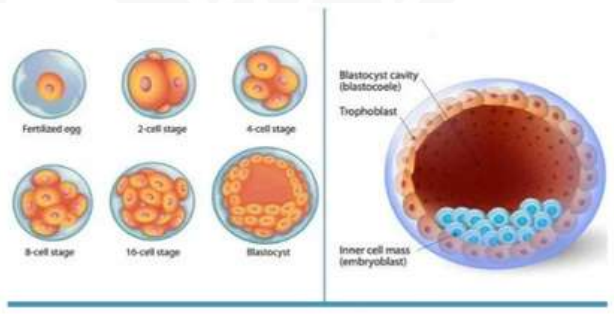
- Incidence: 3.5-4.6 per 1000 births.

- Normally placenta located away from lower uterine segment or surface.
- Proximal part to cervix is fetal part.
- If placenta are preceded by to the cervix it is placenta previa

- Most common risk factor for placenta previa is previous CS, so because of increased cases of CS, placenta previa increased recently because of maternal request of CS or IVF which increase incidence of placenta previa

- Differentiation of site of placenta depends on implantation of blastocyst.
- After fertilization, cells begin to divide to reach about 15 cell at day 5, this known as blastocyst, during fertilization it is in tube, it go to uterine cavity at day 7 to day 12.
- Blastocyst have cavity and trophoblastic cells (which later on form placenta), and inner cell mass (embryo blast, form baby later on).
- Blastocyst search about best environment.
- Best environment contains good endometrium, good blood supply, good surface area for implantation.... These factors found at fundus (normally).
- About 11% of blastocysts implanted at site where it enter the uterus.
- About 11% or 12% descend toward the cervix or lower uterine segment.

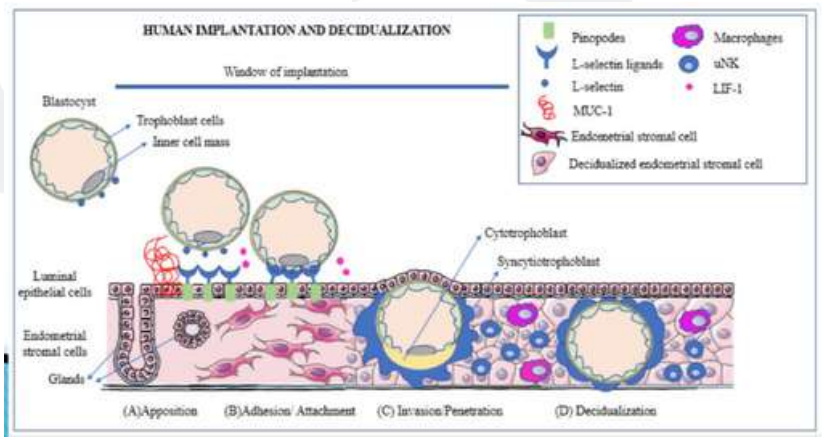
Added picture



What guide blastocyst to implanted at the cervix??

CS scar:

- because it is reach in collagen, and blastocyst need environment that reach in collagen.
- Have some types of proteins found in endometrium that increase the receptivity of blastocyst, this type of protein found in high amount at CS scar.
- CS scar is fibrous tissue and hypoxia occur, so induce angiogenesis, so blastocyst go toward cervix.



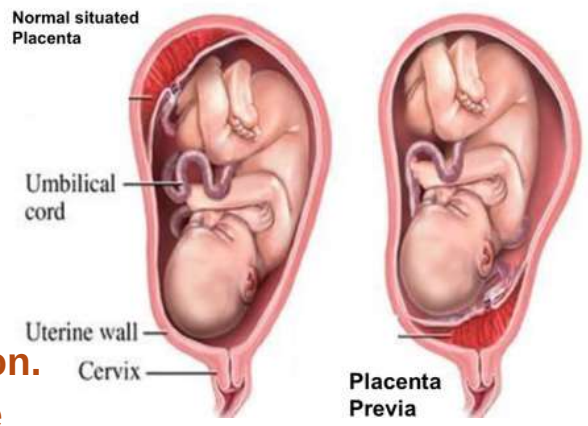
Steps of implantation:

1. Apposition: proximate to proper site for implantation
2. adhesion: direct contact with endometrium
3. invasion.

- If endometrium is not found (this occur at CS scar), because there are recurrent injury of all uterine layers (serosa, myometrium, endometrium), so if endometrium not found blastocyst try to implant to second layer which is myometrium, this known as MORBIDLLY ADHERANT PLACENTA or PLACENTA ACUETA SPECTRUM.
- Decidua basalis are not found in non- pregnant women, sloughing occur for it.
- Dysfunction of INVASION gives morbidly adherent placenta.
- Dysfunction of APPOSITION and ADHEASION.

- During pregnancy isthmus elongate to form lower uterine segment.
- When compare isthmus with uterine fundus or body (according to contents), muscles in isthmus lower than that in uterine body or fundus, so you find elastin and collagen and smooth muscle fibers.
- So placenta previa patient has also postpartum hemorrhage, because muscle content of isthmus lower than that in body or fundus = lower uterine muscle contractions = more postpartum hemorrhage

-Placenta Previa : presence of placenta in the lower uterine segment wither reaching the cervix or not
-Upper uterine segment : smooth muscle fiber (myometrium).
-Lower uterine segment : collagen, elastic, smooth muscle
-So the contractility of the lower segment is lesser than that of the upper one



Why bleeding from placenta occur in previa?
- Because of cervical changes (dilation and effacement) of cervix, interruption of blood vessel occur, so digital vaginal examination should to avoided to avoid interruption of placental vessels.
- We have two types of examination: digital vaginal examination & speculum examination.
- Also sexual activity should be avoided, because it increase PG release, whichincrease uterine contractions, causes changes on surface, this known as PELVIC REST

Pathogenesis :

The pathogenesis of placenta previa is unknown. One hypothesis is that the presence of areas of suboptimal endometrium in the upper uterine cavity due to previous surgery or pregnancies promotes implantation of trophoblast in, or unidirectional growth of trophoblast toward, the lower uterine cavity. Another hypothesis is that a particularly large placental surface area, as in multiple gestation or in response to reduced uteroplacental perfusion, increases the likelihood that the placenta will cover or encroach upon the cervical os.

Pathophysiology :

Placental bleeding is thought to occur when gradual changes in the cervix and lower uterine segment apply shearing forces to the inelastic placental attachment site, resulting in partial detachment. Vaginal examination or coitus can also disrupt the intervillous space and cause bleeding. Bleeding is primarily maternal, but fetal bleeding can occur if a fetal vessel is disrupted

Major risk factors for placenta previa:

1. **Previous placenta previa : Recurrence rate is 4-8%**
2. **Previous cesarean delivery.**
3. **Multiple gestation.**
4. **Multi-parity.**
5. **Advanced maternal age.**
6. **Infertility treatment.**
7. **Previous abortion.**
8. **Previous intrauterine surgical procedure.**
9. **Maternal smoking.**
10. **Maternal cocaine use.**

Prelabor CS may increase risk more than intrapartum CS, because changes that occur during labor not found in prelabor CS, so liability to injury of myometrium more in prelabor.

Multiple pregnancies may cause previa because mother may have on placenta that have to be sufficient for 2 fetal cells, or 2 placenta, so it try to take widest part of uterus which may reach cervix or lower uterine segment.

Endometriosis and assist reproductive teqniologist (as IVF or intrauterine insemination) may increase risk of placenta previa.
- This is because the frequency and amplitude of uterine muscle contraction which is the cause of movement of blastocyst and go toward fundus or cervix, they found the frequency and amplitude in them more than spontaneous pregnancy, so this increase in amplitude and frequency of movement may lead to move blastocyst toward cervi

Smoking and cocaine abuse, as same as CS scar, it cause hypoxia and ischemia.

Clinical features:

There are two presentations :

1. **Antepartum hemorrhage.**
2. **Ultrasound presentation and course.**

Antepartum hemorrhage :

- In the second half of pregnancy, the characteristic clinical presentation is unprovoked painless vaginal bleeding, the blood is usually bright red and ranges in volume from scant to heavy, which occurs in 70-80 % of cases.

- An additional 10 -20 % of women present with both uterine contractions and bleeding. Which is similar to the presentation of abruptio placenta.

- In approximately one-third of affected pregnancies, the initial bleeding episode occurs prior to 30 weeks of gestation; this group is more likely to require blood transfusions and is at greater risk of preterm delivery and perinatal mortality than women whose bleeding begins later in gestation . An additional one-third of patients becomes symptomatic between 30 and 36 weeks, while most of the remaining patients have their first bleed after 36 weeks. About 10 % of women reach term without bleeding.

→ Digital vaginal examination is contraindicated in any woman beyond 20 weeks of gestation who presents with vaginal bleeding, until rule out of placenta previa by ultrasound.

In placenta previa blood not collect behind placenta, so mild = mild, heavy =heavy, no silent bleeding.

Because about 2 third of patients of previa occur preterm, so most common cause of fetal death in it is prematurity (not asphyxia)

Ultrasound presentation and course:

- 1-6 % of pregnant women display sonographic evidence of a placenta previa between 10 and 20 weeks of gestation when they undergo obstetrical ultrasound examination. The majority of these women are asymptomatic and 90 % of these early cases resolve.

→ Two theories have been put forth to account for resolution of the previa:

1)The lower uterine segment lengthens from 0.5 cm at 20 weeks of gestation to more than 5 cm at term. Development of the lower uterine segment relocates the stationary lower edge of the placenta away from the internal os.

2) Progressive unidirectional growth of trophoblastic tissue toward the fundus within the relatively stationary uterus results in upward migration of the placenta. This phenomenon has been termed trophotropism.

- If the previa persists with advancing gestational age, it is less likely to resolve

- Patients with previa comes to hospital at 1st

, 2nd & 3rd trimesters, up to 6% percent

of theme between 10th week and 20th week when you do US find placenta at lower uterine segment or may covering the cervix, in this case you don’t diagnose it as placenta previa, you describe the placenta (ex: found about 2cm from cervix).

In this case you have to follow the patient for placental migration, follow up to 28 weeks up to 32 weeks, you follow up to these weeks because at which the lower uterine segment start to elongate (lower uterine segment for 20 weeks pregnant women is 0.5cm, at term it approximately 10cm.

If location of placenta after 28 weeks differ, this known as PLACENTAL MIGRATION, actually it didn’t migrate, but the lower uterine segment elongate, this known as DYNAMIC CLASSIFICATION.

The 2nd theory known as TROPHOTROPISM, when the baby developed, demands will be more, and because of the lower blood supply at lower uterine segment than upper uterine segment so placenta try to cover more space toward upper uterine segment to take more blood supply, with progression of labor, baby depends more on part of placenta which has more blood supply nor on lower part, so lower part of placenta will be atrophied, on US it will be smaller, so you may describe it as migrated placenta.

So 90% of patient with low lying placenta or placenta covering the cervix between 10-20 weeks the will be not previa after 20 weeks, so follow up is very important

Associated conditions : Placenta previa has been associated with an increased risk of several other pregnancy complications:

1. **Placenta accreta: Placenta accreta complicates 1-5% of pregnancies with placenta previa and an unscarred uterus.**
2. **Malpresentation: The large volume of placenta in the lower portion of the uterine cavity predisposes the fetus to assume a non-cephalic presentation.**
3. **Preterm labor and rupture of the membranes : Ante-partum bleeding from any cause is a risk factor for preterm labor and premature rupture of membranes.**
4. **Vasa previa and velamentous umbilical cord : they are uncommon, but when present they are often associated with placenta previa.**
5. **Congenital anomalies : Population-based cohort studies have reported an increase in the overall rate of neonatal congenital anomalies in pregnancies complicated by placenta previa, but no single anomaly or syndrome was associated with the disorder.**

Diagnosis of placenta previa :

- **Is based on identification of placental tissue covering or proximate to the internal cervical os on an imaging study, typically ultrasound. (A distance greater than 2 cm from the os excludes the diagnosis of previa).**
- **Transabdominal U/S examination is performed as the initial examination; if it shows placenta previa or the findings are uncertain, transvaginal U/S (TVS) should be performed to better define placental position.**
- **The overall false positive rate of transabdominal U/S for diagnosis of placenta previa is high (up to 25 %), so the diagnosis should be confirmed by TVS unless the previa is clearly central.**
- **Superior performance of TVS over transabdominal U/S for diagnosis of placenta previa. (provides a clearer image of the relationship of the edge of the placenta to the internal cervical os)**

Why digital vaginal examination contraindicated while transvaginal US recommended?
Because in US there are angle present between probe and cervix so not slugged inside the cervix

Classifications of placenta previa :

The traditional classification of placenta previa describes the degree to which the placenta encroaches upon the cervix in labour (opened cervix) and is divided into :

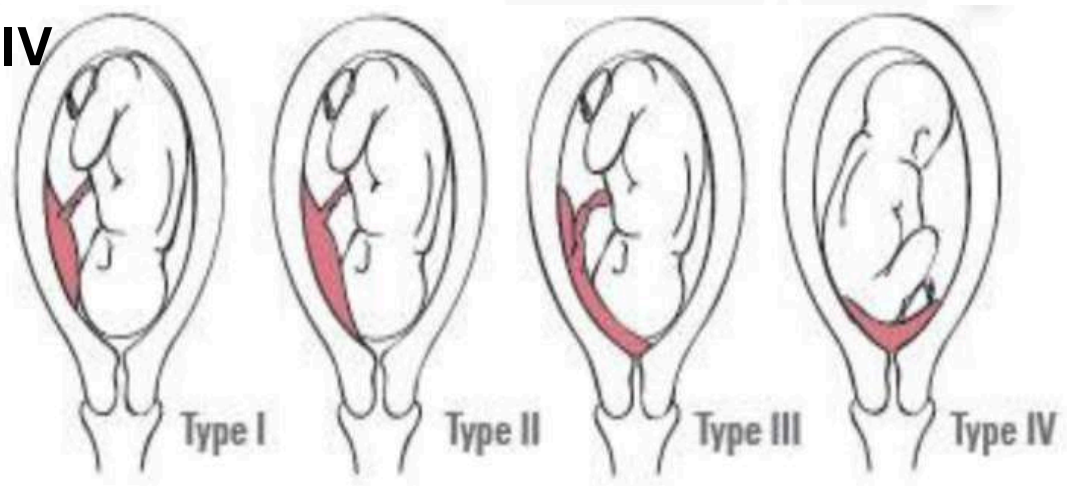
- Grade I (low lying)(lateral): Placenta is in lower segment, but the lower edge does not reach internal os

- Grade II (marginal): Lower edge of placenta reaches internal os, but does not cover it.

- Grade III (partial)(incomplete centralis): Placenta covers internal os partially.

- Grade IV (complete) (complete centralis) (total): Placenta covers internal os completely

Minor placenta previa: Grade I and Grade II
Major placenta previa: Grade Ill and Grade IV



If major, patient cant deliver patient vaginally..
But after dilation of cervix, minor developed to major, so any placenta previa (2cm within internal os, it is CS

Breech (malpresentation) associated with previa

In previa patient, you should to rule out:

1. Malpresentation (breech)

2. Congenital anomalies (no specific anomaly associated, but incidence of anomaly found to be higher)

3. Completely adherent placenta.

4. Vasa previa & filamentous cord

5. Preterm labor, which caused by bleeding

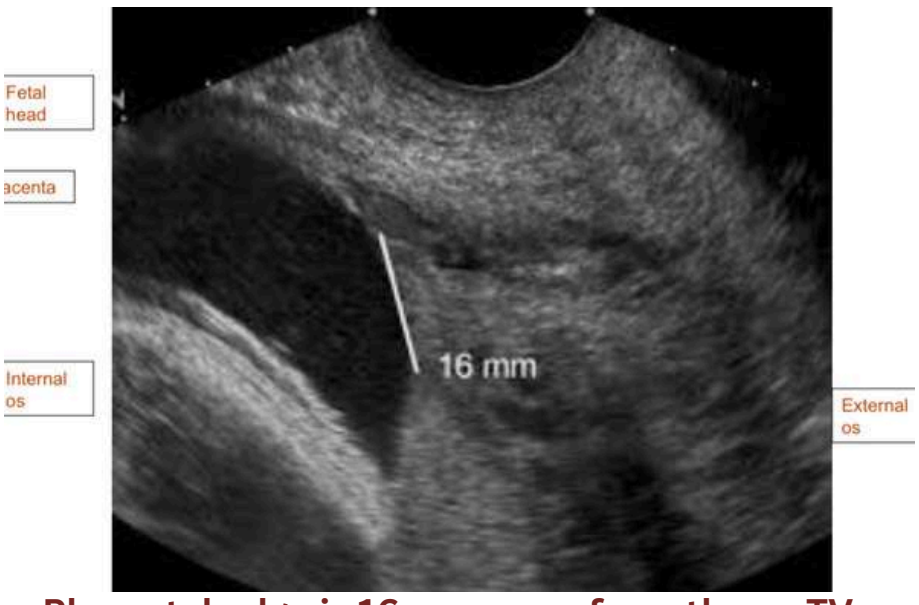
- The previous system for classifying Placenta Previa is imprecise.

- Nowadays, the diagnosis and classification of Placenta Previa depends on the actual distance from the placental edge to the internal cervical OS at TVS, using standard terminology of millimeters away from the OS or millimeters of overlap.

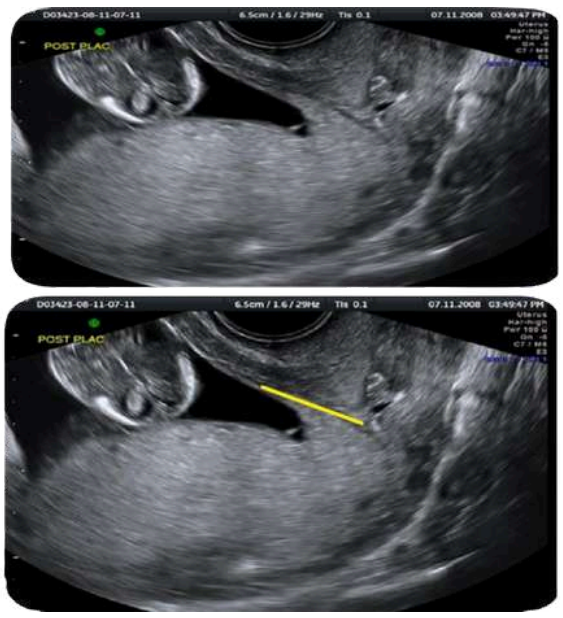
A placental edge exactly reaching the internal os is described as 0 mm.

- If the placental edge is more than 20 mm away from the internal os :

No Placenta Previa



Placental edge is 16 mm away from the os TV ultrasoun



MRI is well-suited to the assessment of placental-cervical relationships because of the differing magnetic resonance characteristics of the two tissues. However, it is not used for diagnosis of placenta previa because of its high cost, limited availability, and the well-established safety and accuracy of transvaginal sonography . MRI is most useful for diagnosis of complicated placenta previa, such as previa-accreta and suspected posterior placenta previa.

- posterior: away from anterior uterine wall

- Anterior: proximal from anterior uterine wall

- Post diagnostic evaluation :

(1)Exclusion of placenta accreta : Using transvaginal ultrasound ± MRI.

(2)Follow-up ultrasound examination : When the placenta previa was diagnosed in the second trimester, a follow up ultrasound examination for placental location in the third trimester is recommended (between 32-36 weeks).

- At anterior uterine wall the bladder located, so by US bladder with urine inside gives shadowing, so it easier to define placenta previa, But if it posterior, there are no shadowing, so it is more possible to define previa, so you go for MRI.

- Also if there are dysfunction in invasion, this give morbidly adherent placenta, so you use MRI to define diagnosis.

Morbidity and mortality :

- Maternal:Placenta previa increases the risk of antepartum (RR 9.8), intrapartum (RR 2.5), and postpartum hemorrhage (RR 1.9). For this reason, women with placenta previa are more likely to receive blood transfusions (12 versus 0.8 percent without previa) and undergo postpartum hysterectomy, uterine/iliac artery ligation, or embolization of pelvic vessels to control bleeding (2.5 versus 0 percent without previa). The risk is particularly high in those with previa-accreta.

- Neonatal:The principal causes of neonatal morbidity and mortality are related to preterm delivery, rather than anemia, hypoxia, or growth restriction

Management of placenta previa:

The management of pregnancies complicated by placenta previa is best addressed in terms of the clinical setting:
1) Asymptomatic placenta previa
2) Bleeding placenta previa

Asymptomatic previa:

If diagnosed before 28 weeks, follow up to 28-32 weeks, if previa persist, admit to hospital up to delivery = 37 weeks (some say no need for admission)
- While patient in the hospital for follow up, you insert to large bore cannulas for to check Hb and correction for anemia
- Also you give steroids to enhance the life maturity of the baby

Asymptomatic placenta previa :

- **Follow-up transvaginal ultrasound examination :**

- For pregnancies >16 weeks :

- If the placental edge is ≥ 2 cm from the internal os, the placental location is reported as normal and follow-up ultrasound for placental location is not indicated.

- If the placental edge is < 2 cm from, or covering, the internal os : follow-up ultrasonography for placental location is performed at 32 weeks of gestation.

- At 32 weeks follow up ultrasound :

- If the placental edge is ≥2 cm from the internal os, the placental location is reported as normal and follow-up ultrasound for placental location is not indicated.
And these patients can be delivered vaginally safely.

- If the placental edge is still <2 cm from the internal os or covering the cervical os,

1- Admission to hospital for observation till delivery. (? Outpatient)
2- Avoid sexual intercourse.
3- Single course of antenatal corticosteroid should be administered to pregnancies at 26 to 35 weeks of gestation.
4- Follow-up TVS is performed at 36 weeks. if placenta previa persists, schedule cesarean section at 37 weeks of gestation

Bleeding placenta previa :

initial interventions for women with bleeding placenta previa : (admission to labour room)

A) Stabilization of the mother :

1. I.V fluid - Secure intravenous access with at least one, and preferably two, wide-bore intravenous lines

2. Closely monitor the mother's hemodynamic status (heart rate, blood pressure, urine output). Urine output should be maintained at above 30 mL/hour and monitored with a Foley catheter

3. Keep maternal oxygen saturation >95 percent and keep the patient warm.

4. Draw blood for a complete blood count, blood type and Rh (preparation of 4 units PRBCs), and coagulation studies.

5. Call for help.

6. Notify the anesthesia team. Anesthesia-related issues in these patients include management of hemodynamic instability, technical issues related to bleeding diathesis, and the potential need for emergency cesarean delivery.

7. Notify the blood bank so blood replacement products (red blood cells, fresh frozen plasma, cryoprecipitate, platelets) will be readily available, if needed.

B) Immediately initiate continuous fetal monitoring.

- **Severe bleeding and /or non reassuring FHR → Emergency cesarean section**

- Anesthesia : General anesthesia is typically administered for emergency cesarean delivery, especially in hemodynamically unstable women or if the fetal status is nonreassuring. However, regional anesthesia is an acceptable choice in hemodynamically stable women with reassuring fetal heart rate tracings.

- **Mild bleeding + Reassuring FHR + G.A < 37 weeks → conservative management**

Conservative management :

1- Symptomatic women often remain hospitalized from their initial bleeding episode until delivery.

2- Correction of anemia.

3- 4 unites of PRBCs should be available.

4- Anti-D immune globulin for Rh(D)-negative women.

5- Schedule cesarean section at 37 weeks.

6- Delivery is indicated emergently if any of the following occur:

- **Any vaginal bleeding with a non-reassuring fetal heart rate tracing unresponsive to resuscitative measures.**
- **Life-threatening refractory maternal hemorrhage.**
- **Labor.**

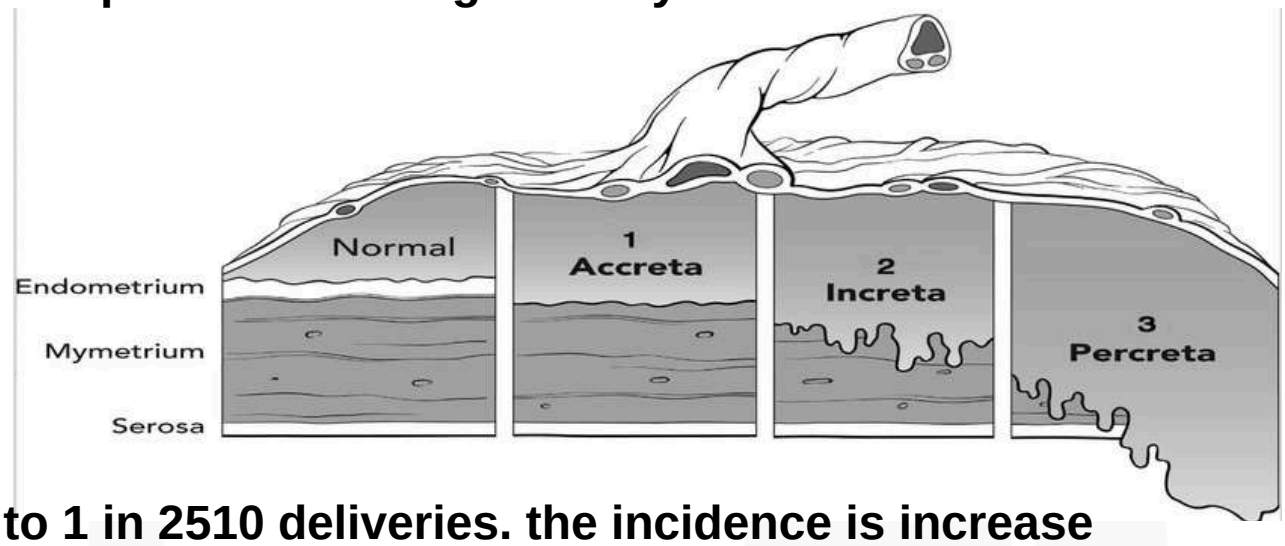
Morbidly adherent placenta

Refers to abnormal deep implantation of the placenta. There are three types according to the depth of invasion :

1) Placenta accreta (79%) : Chorionic villi attach to myometrium rather than decidua.

2) Placenta increta (14%) : Chorionic villi penetrate into the myometrium.

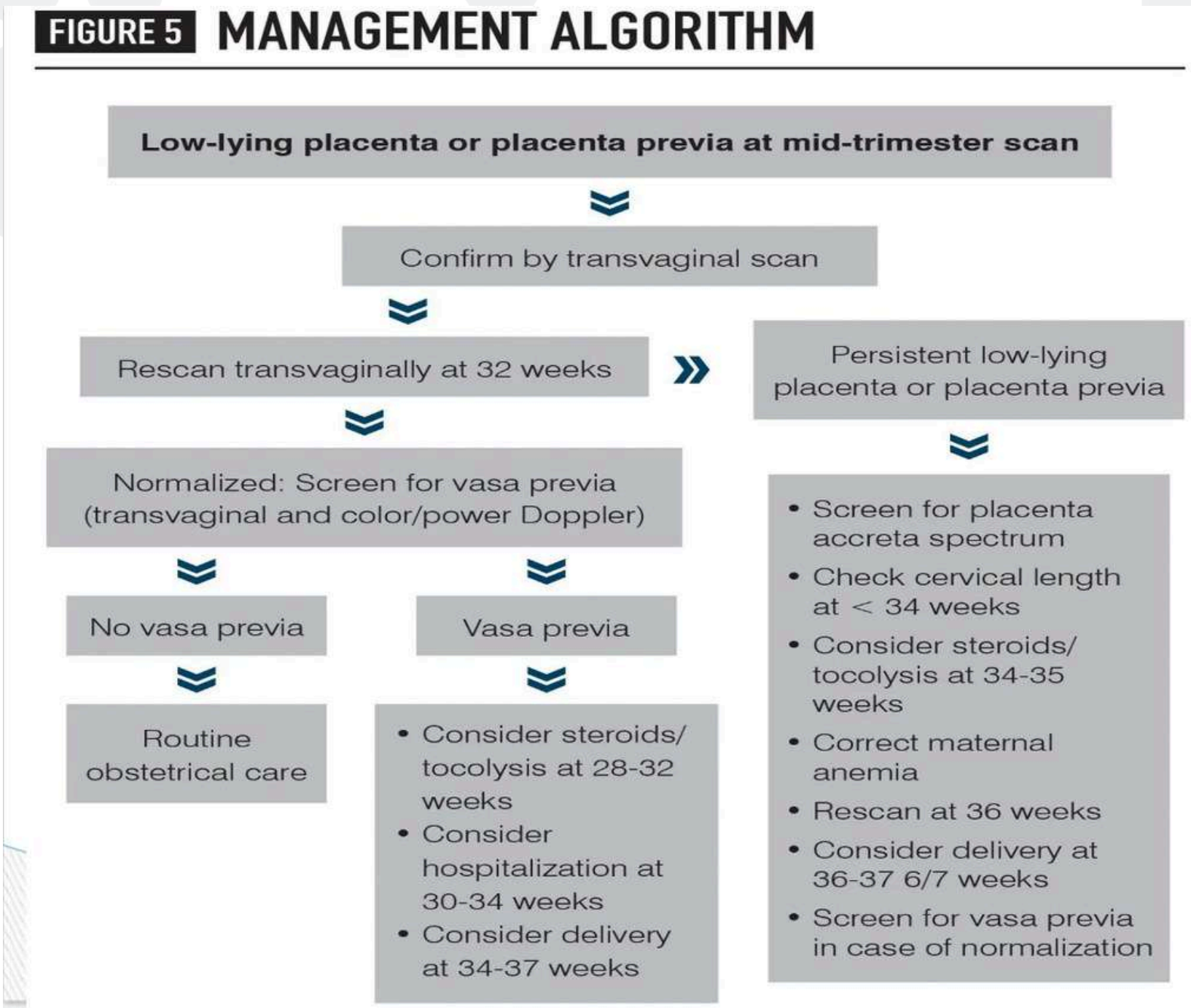
3) Placenta percreta (7%) : Chorionic villi penetrate through the myometrium to the uterine serosa or adjacent organs



- **The incidence ranging from 1 in 533 to 1 in 2510 deliveries. the incidence is increase due to the increasing prevalence of cesarean delivery.**

- **Pathogenesis : The pathogenesis of placenta accreta is not known with certainty.**

The most common theory is that defective decidualization (thin, poorly formed, or absent decidua) related to previous surgery or to anatomical factors (endocervix, lower uterine segment, endosalpinx, uterine anomaly) allows the placenta to attach directly to the myometrium. This theory is supported by the observation that 80 % of these cases are associated with a history of previous cesarean delivery, curettage, and/or myomectomy.



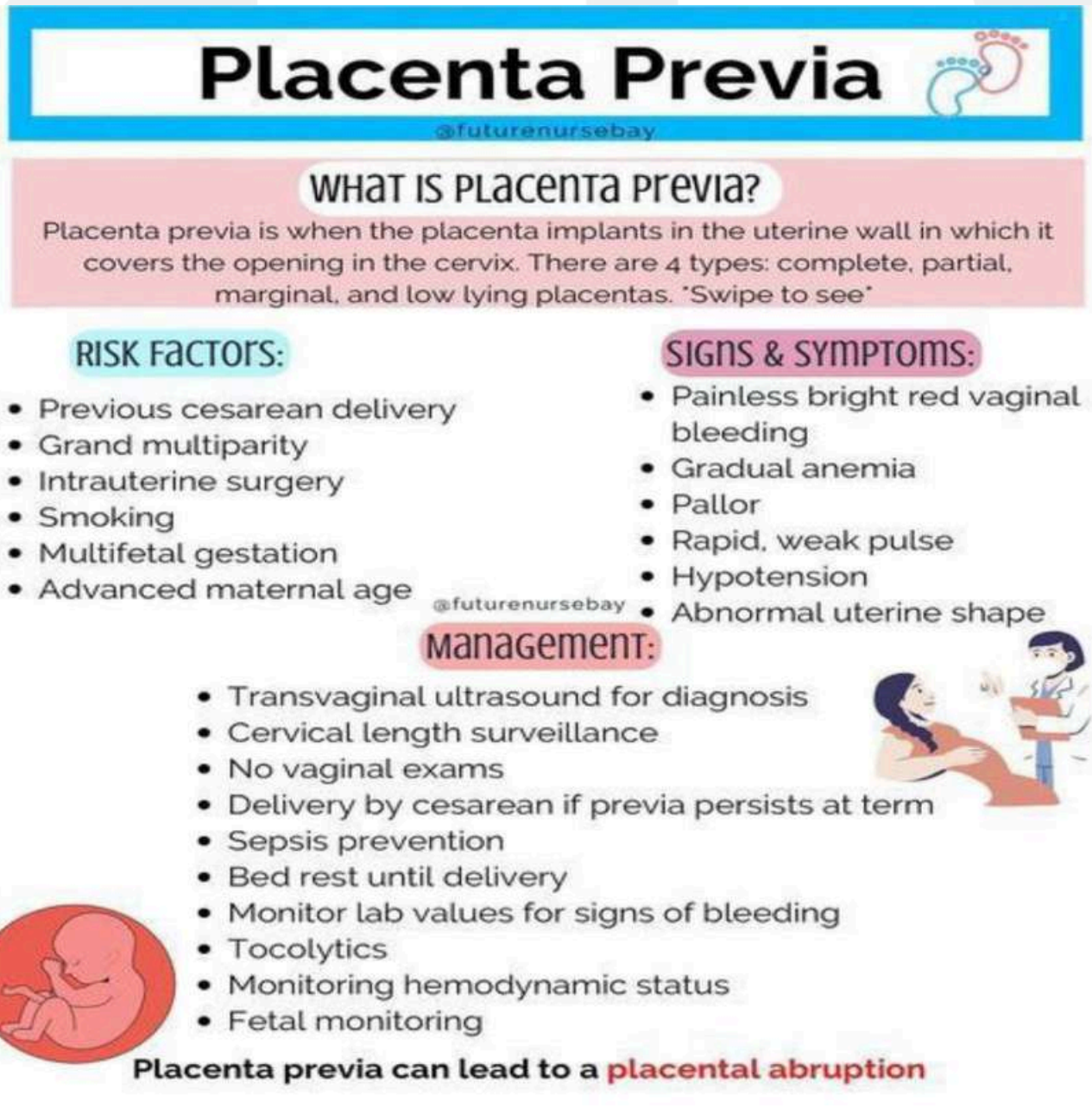
Risk factors : The most important risk factor for placenta accreta is
1) placenta previa after a prior cesarean delivery. In women with placenta previa, the frequency of placenta accreta increases with an increasing number of cesarean deliveries as follows:

- **No previous cesarean birth, 1-5 %.**
- **One previous cesarean birth, 11-25 %.**
- **Two previous cesarean births, 35-47 %.**
- **Three previous cesarean births, 40 %.**
- **Four or more previous cesarean births, 50-67 %.**

In the absence of placenta previa, the frequency of placenta accreta still increases with an increasing number of cesarean deliveries, but the incidence is much lower. In women without placenta previa, the frequency of placenta accreta is :

- **One previous cesarean birth, 0.3 %.**
- **Two previous cesarean births, 0.6 %.**
- **Three previous cesarean births, 2.4 %.**

2- History of uterine surgery.
3- Maternal age >35years.
4-History of pelvic irradiation.
5- Infertility procedures (IVF).
6- Asherman syndrome: intrauterine adhesion, these patient because of recurrent evacuation (mostly because of illegal abortion), they have recurrent injury of endometrium, this make it deficient or cause reactions which leads to adhesion formation.



Clinical presentation: there are two presentations :

1) The first clinical manifestation of placenta accreta is usually profuse, life-threatening hemorrhage that occurs at the time of attempted manual placental separation. Part, or all, of the placenta remains strongly attached to the uterine cavity, and no plane of separation can be developed.

- **Sequelae : Poorly controlled hemorrhage related to placenta accreta/increta/percreta is the indication for one to two thirds of peripartum hysterectomies. Additional potential sequelae of massive hemorrhage include DIC, adult respiratory distress syndrome, renal failure, unplanned surgery, and death.**

2) Diagnosed on prenatal sonographic evaluation of the placenta in a woman with risk factors for accreta (previa, previous cesarean delivery).
→ Placenta percreta with bladder invasion can cause hematuria.

Diagnosis :

- **Prenatal diagnosis of placenta accreta is typically based upon the presence of characteristic findings on ultrasound examination.**
  - **MRI can be more useful than ultrasound in two clinical scenarios:**

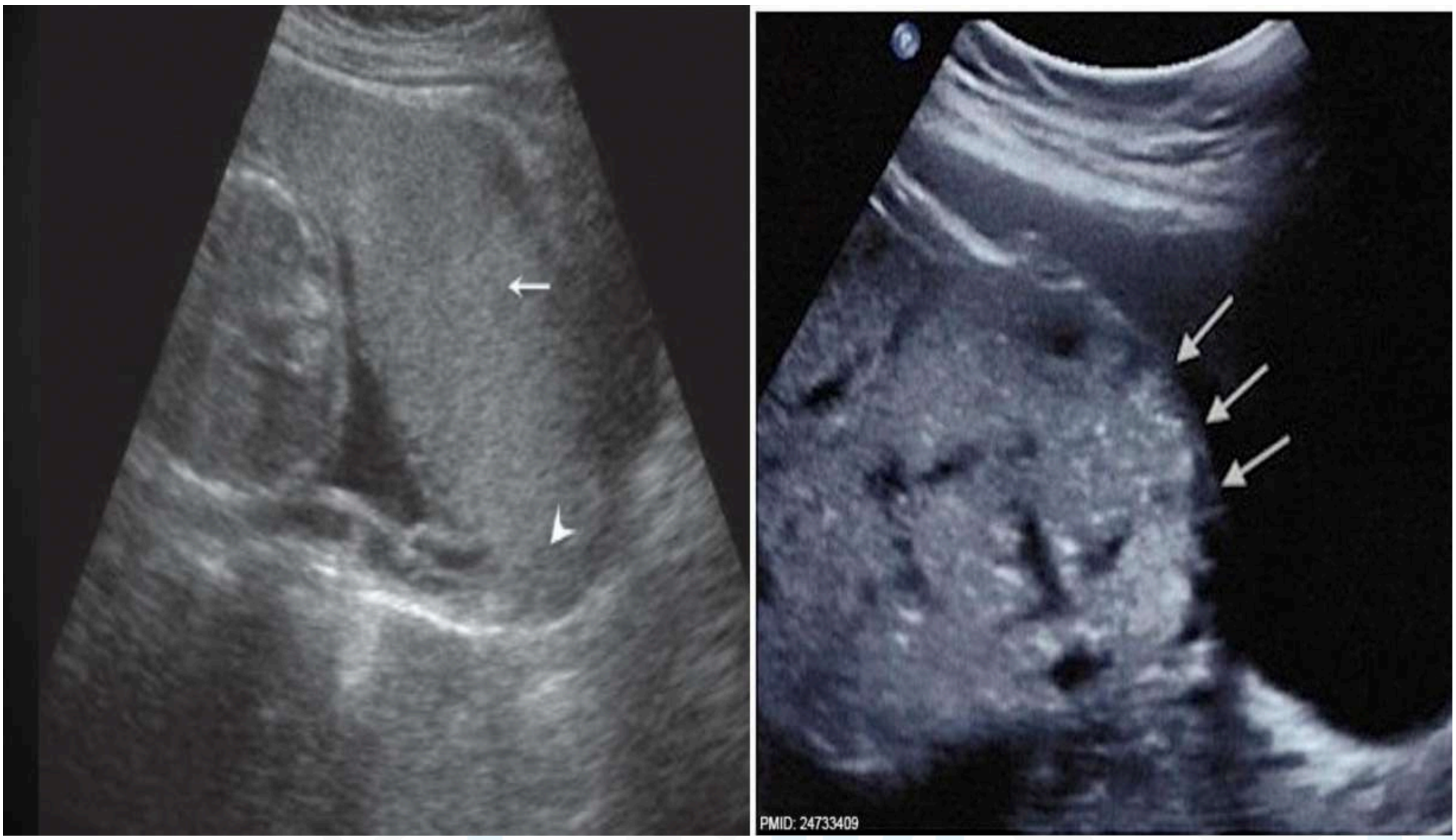
(1) Evaluation of a possible posterior placenta accreta because the bladder cannot be used to help clarify the placental-myometrial interface.

(2) Assessment of the depth of myometrial and parametrial involvement, and, if the placenta is anterior, bladder involvement

- **Postnatal diagnosis is based on histological examination of the placenta or the placenta and uterus.**

→ Women with a placenta previa or a low anterior placenta and prior uterine surgery should have thorough sonographic evaluation of the interface between the placenta and myometrium between about 18 and 24 weeks of gestation. At this gestational age, the diagnosis is suspected or excluded in virtually all cases. MRI can be useful when the ultrasound findings are uncertain.

After 3rd stage of labor (delivery of placenta), you do active management = give oxytocin & controlled contraction).
-Controlled contraction: check if uterus is contracted by your non dominant hand, hen put it suprapubic, do upward downward pressure, then by your other hand do down contraction to the cord, trying to make separation for placenta.
-If it not separated also after 30 minutes, this known as RETAIN PLACENTA (stuck in the uterus), in this case, patient go to theater, under anesthesia we do manual removal of placenta by trying to put your hand in the uterus at the plane found between placenta & uterine wall and separate them.
-If you try to do manual removal for placenta implanted into myometrium, this plane not found, so if you try to separate, active bleeding = massive bleeding.
-So manual removal for retain placenta never to done in the labor room, only in theater, to assist laparotomy and hysterectomy.



Management of placenta accreta :
There are two options:

1. **Cesarean hysterectomy.**
2. **uterine conservation with the placenta left in situ. has high risk of bleeding and sepsis.**

- **All patients with placenta accreta should be counseled about the suspected diagnosis and potential sequelae (Hemorrhage, blood transfusion, cesarean hysterectomy, maternal ICU admission). Consultation with a maternal-fetal medicine specialist is desirable.**

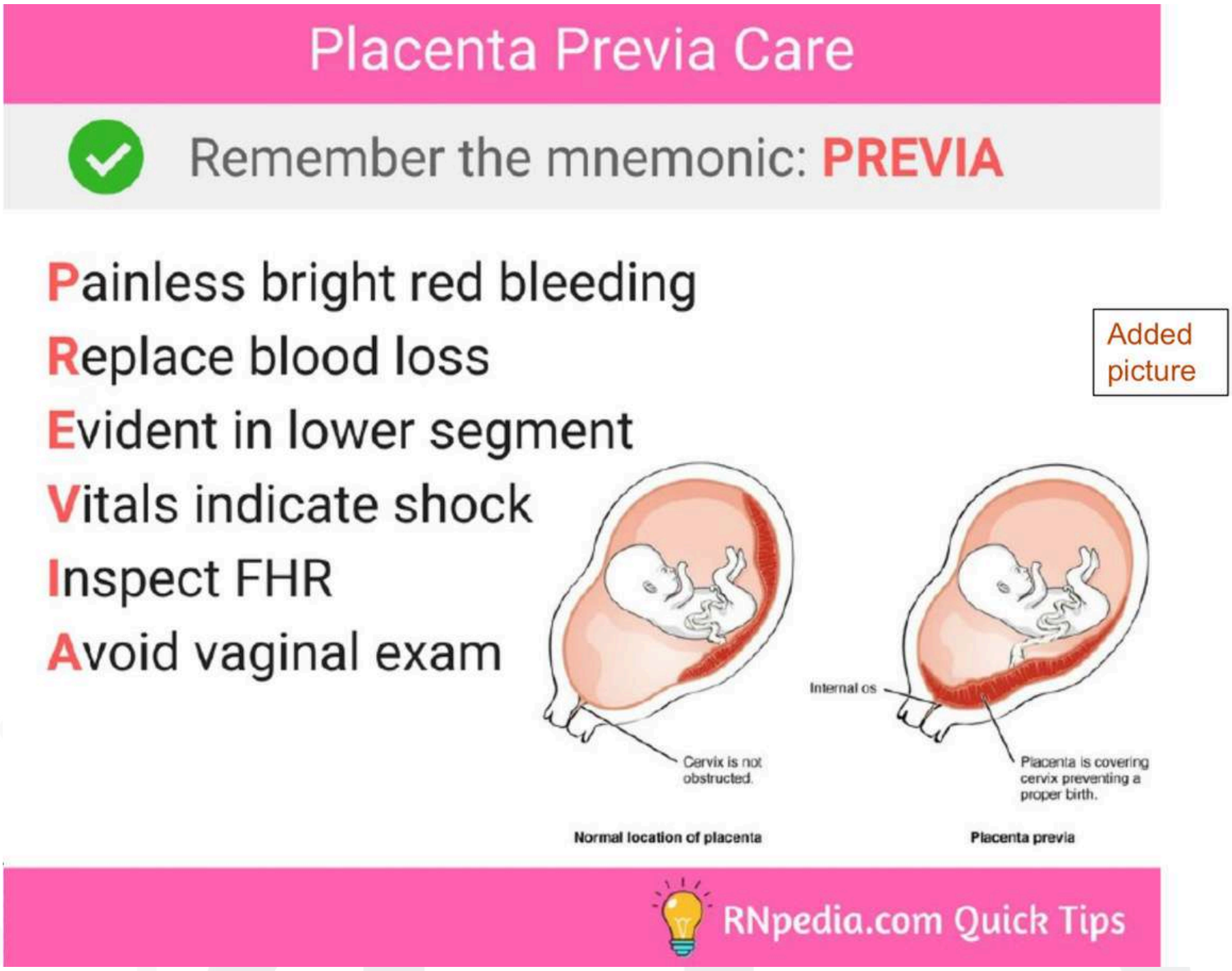
- **Management by a multidisciplinary team and delivery in a tertiary care facility improve outcomes and lower complication rates. The team includes maternal-fetal medicine specialists, anesthesiologists, neonatologists, blood bank and nursing personnel. It is desirable to have a surgeon present who has extensive experience with wide dissection of the lower uterine segment and parametrial areas in the event such dissection or bladder resection is required.**

- **Delivery should be scheduled electively for optimal availability of necessary personnel and facilities. Planned delivery is associated with less intraoperative blood loss than emergency delivery.**

- **The optimum gestational age for scheduled delivery is controversial. Some experts have recommended delivery of previa-accreta at 34 to 36 weeks of gestation.**

- **Adequate blood and clotting factors should be available at the time of delivery. The magnitude of blood loss is difficult to predict antepartum.**

• **Preoperative placement of balloon catheters into the internal iliac arteries. The catheters may be inflated intermittently during hysterectomy, thus potentially decreasing blood loss and providing optimum exposure of the operative field. They may also be used for embolization of persistent bleeders.**



Uterine rupture and uterine scar dehiscence

● **Complete disruption of all uterine layers, including the serosa. It is a life- threatening pregnancy complication for both mother and fetus.**

Other adverse outcomes include complications related to severe hemorrhage, bladder laceration, hysterectomy, and neonatal morbidity related to intrauterine hypoxia. Most uterine ruptures in resource-rich countries are associated with a trial of labor after cesarean delivery (TOLAC). In resource-limited countries, many uterine ruptures are related to obstructed labor and lack of access to operative delivery.

● **Uterine dehiscence generally refers to an incomplete, and frequently clinically occult, uterine scar separation where the serosa remains intact and is not usually associated with hemorrhage or adverse maternal or perinatal outcomes.**

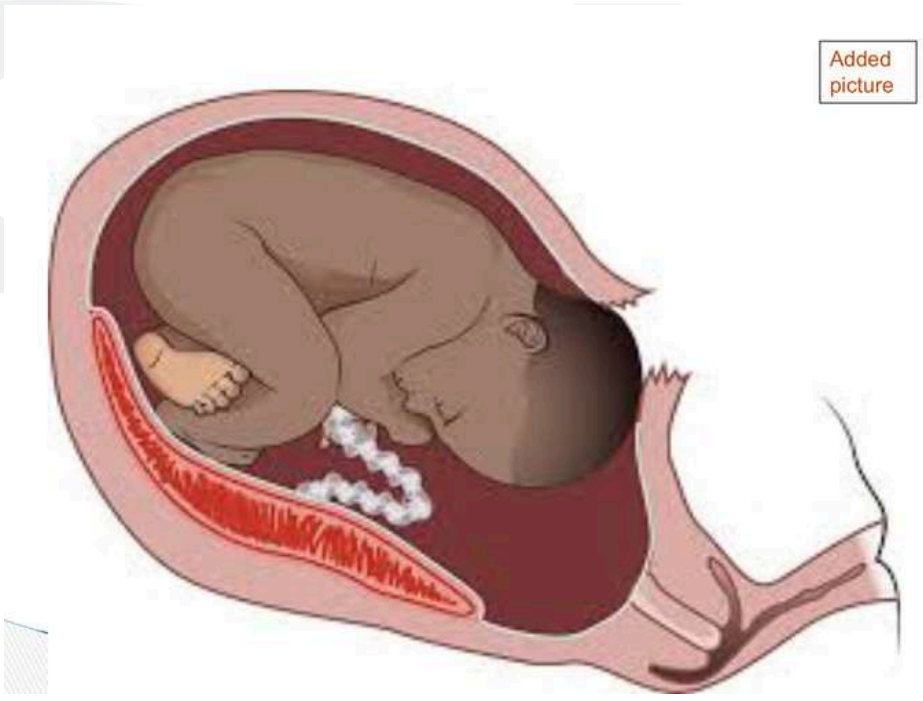
Uterine rupture occurs mainly with:

1. **Increased frequency of CS, where CS scar considered as the weakest area in the uterus because it is fibrous tissue, so it cant stretch as smooth muscle fiber, so it easily to rupture because of increase in the intrauterine pressure, especially with contraction.**
   - **If it try to deliver vaginally after CS, this know Trial Of Labor After Caesarean Delivery (TOLAC), but under specific criteria.**
2. **Patient has obstructed labor and don't go for CS, uterine rupture occur after some days of trying to deliver**

— After uterine rupture for laboring patient, station of baby not felt.
— If patient is post-partum, also there are no contractions, so bleeding occur.
— Bleeding + Atonic uterus (soft uterus) + not responding with oxytocin = possible uterine rupture.

- **Are postpartum rupture occurs after delivery?**

No, this seen mainly with multiparity or gram-multipara (para 5 or more), these deliver more quick than primigravida, so uterus is very sensitive and given oxytocin at lower degree, if increases will cause tetanic contraction.
The uterus of multipara because of pregnancy and delivery every time uterus under go hypertrophy and hyperplasia, so there are fibrous tissue also with smooth muscle in uterus, so it is week compared to primigravida, and more risk for rupture.
Patient deliver at term of delivery, and rupture take place, so it diagnosed after delivery.



- **The overall incidence of uterine rupture in women with a prior cesarean delivery (at lower uterine segment, by section of bladder, the lowest frequency of rupture, so it is the best site to do CS) varies between 0.3-1 %.**

- If CS done on lower uterine segment, but in transverse section, frequency increase to 2%
- Highest risk if prior CS at upper uterine segment, this known as CLASSICAL CS, so vaginal delivery contraindicated for them

Patient presentation :

The premonitory signs of uterine rupture include :

1- Fetal heart rate (FHR) abnormalities, there is no FHR pattern pathognomonic of rupture. The most common FHR abnormality is fetal bradycardia, which may be sudden or preceded by decelerations. Also to monitor uterine contractions.

2- Vaginal bleeding, is not a cardinal symptom, as it may be modest, despite major intra-abdominal hemorrhage which cause shock. and patient may become hemodynamically unstable.

3- Sudden or worsening abdominal pain, continuous.

4- Uterine contraction abnormalities, a gradual decrease in the amplitude of consecutive contractions, the so-called "staircase sign", then cessation of contractions.

5- Loss of station of the fetal presenting part.

CLASSICAL CS: any CS in upper uterine segment, wither transverse or vertical

→ In postpartum women, occult uterine rupture that occurred during delivery is characterized by pain and persistent vaginal bleeding despite use of uterotonic agents.

Management:
Most common cases managed by hysterectomy, but some times we try to preserve the uterus, especially if rupture occurs at site of scar and easily repaired.

VASA PREVIA

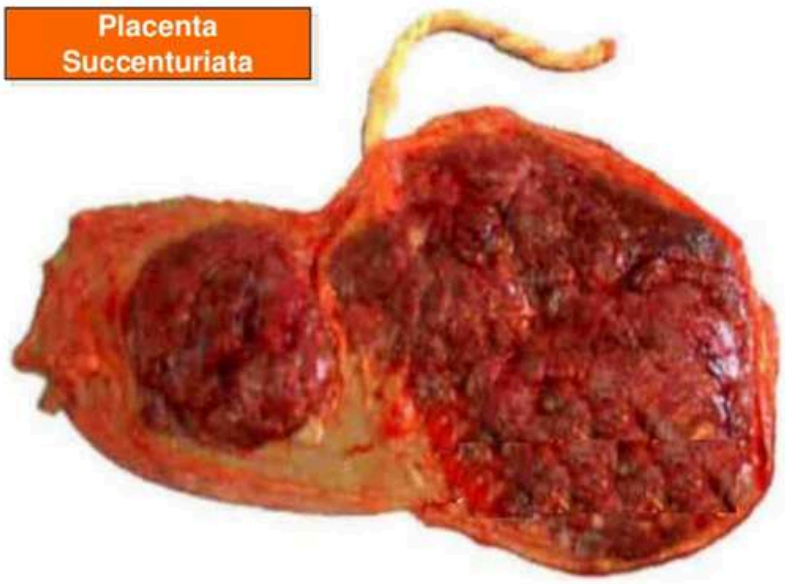
Fetal blood vessels are present in the membranes covering the internal cervical os. The membranous vessels may be associated with a velamentous umbilical cord (type 1 vasa previa) or they may connect the lobes of a bilobed placenta or the placenta and a succenturiate lobe (type 2 vasa previa).

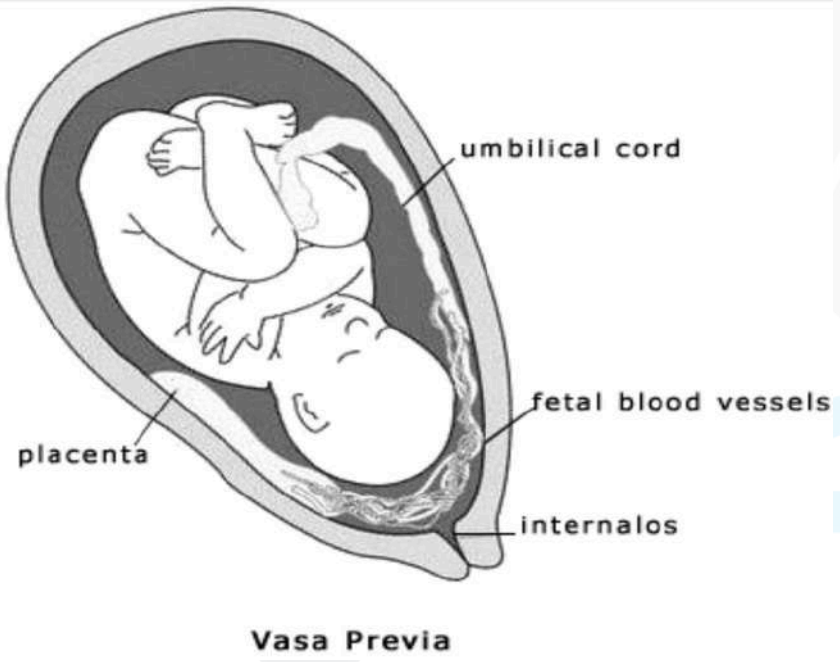
Prevalence: The prevalence of vasa previa is approximately 1 in 2500 deliveries, but is much higher in pregnancies conceived following use of assisted reproductive technologies (prevalence as high as 1 in 202). The prevalence is also increased in second-trimester low-lying placentas or placenta previa (even if resolved), bilobed or succenturiate lobe placentas in the lower uterine segment, and multiple gestations.

- Multiple gestations commonly come from assessed reproductive technology (ART).
- ART risk for multiple pregnancy, placenta previa, so it is risk factor for vasa previa.

Velamentous cord insertion: when placenta inserted marginal (normally it center of placenta, also marginal considerd normal), while cord and its arterial and venous divisions are in the amniotic membranes not in the placenta.

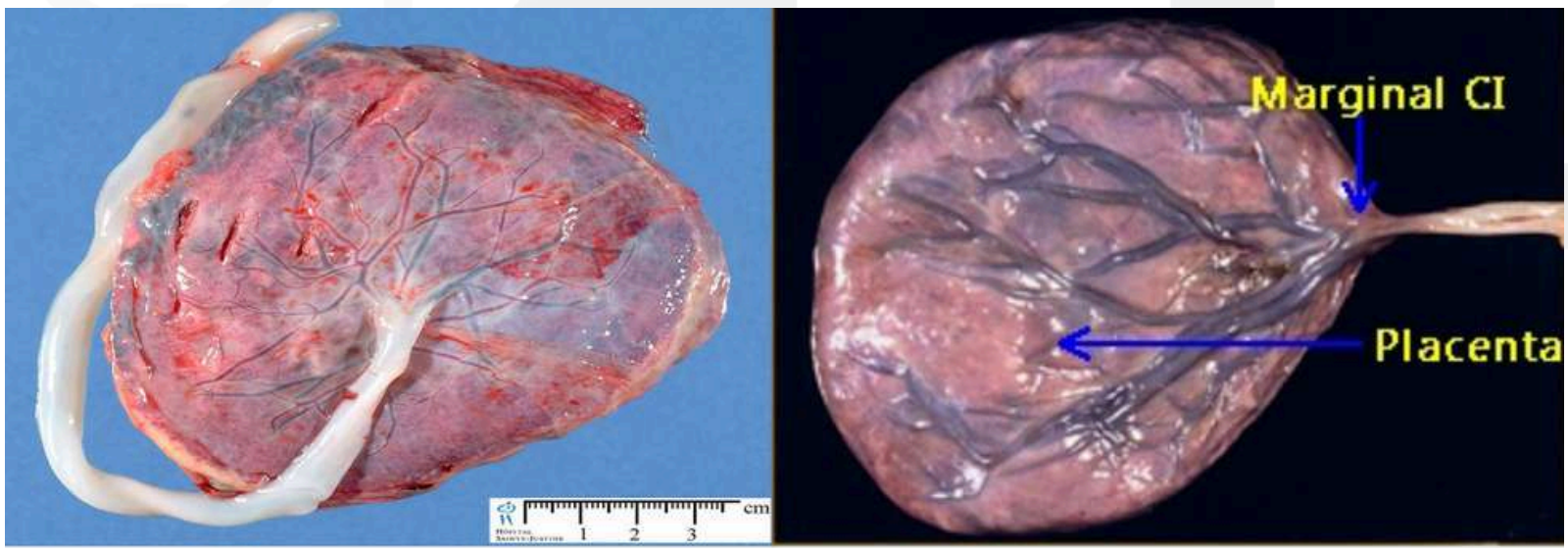
- This case seen more with placental previa (low lying placenta)
- This known as TYPE I vasa previa.
- 2nd type of placenta previa occur with patients have immature lobe of placenta, or major lobe and accessory / succenturiate lobe
- What occur in 2nd type is because there are connection between two membranes, so cord insertion between two lobes, if one lobe at anterior uterine wall, the second at posterior uterine wall, membranes above the cervix and fetal BV inserted within.



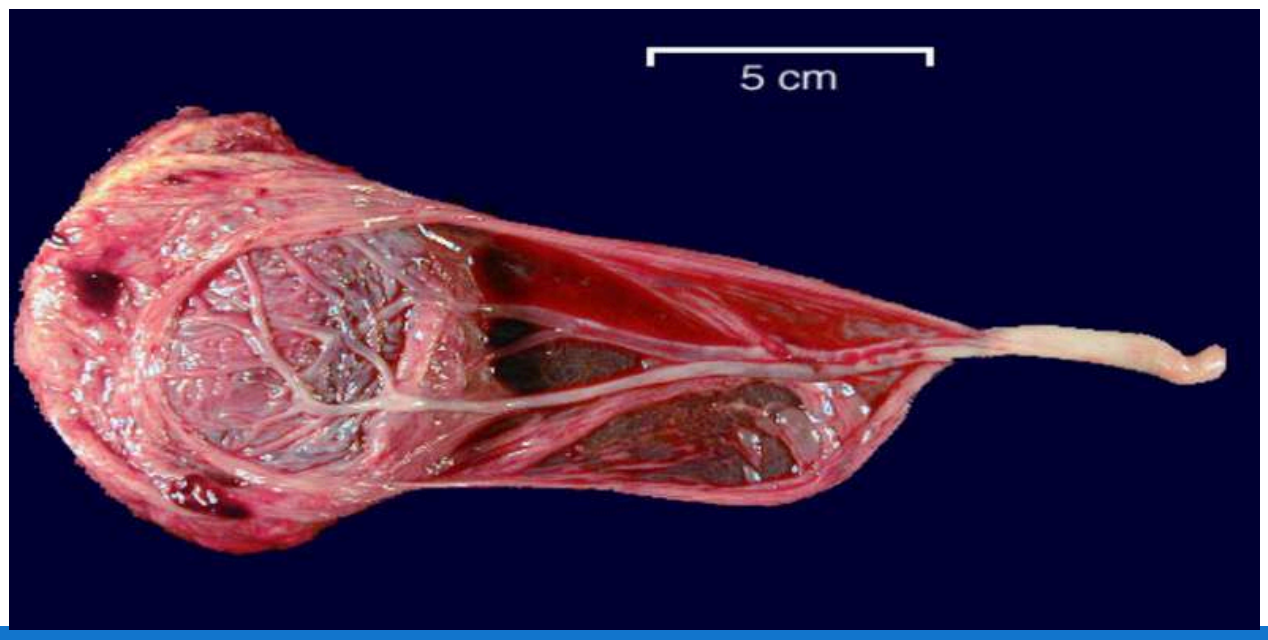


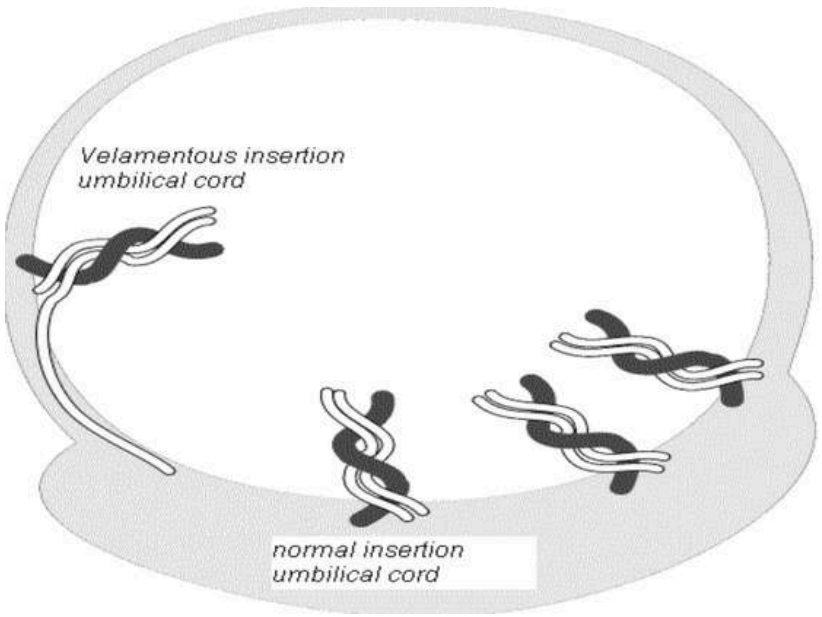
Vasa Previa

Normal ~~placenta~~ (marginal, central)



Velamenatous cord insertion





Clinical significance :

1.Vaginal bleeding after rupture of membranes (spontaneously or artificially). This is fetal bleeding , results in fetal anemia and hypotension, leading to fetal heart rate abnormalities, such as a sinusoidal pattern; fetal death due to exsanguination can occur within minutes.

2. The membranous vessels are at risk of compression from the fetal presenting part since they are not protected by the structure of a normal umbilical cord.

You can know if there is maternal or fetal blood also by other method.

- **Fetal Hb is resistace for acidic and alkaline media (doesn't denature), so you can use acidic or alkaline media to test.**
- **Disadvantage for this test that the shortest test time about 4 minutes, during these 4 minutes you may deliver patient, and you gave to know that fetal blood is low (about 200cc)**

● Antenatal diagnosis is based primarily on identification of membranous fetal vessels passing across the internal cervical os by real-time and color Doppler ultrasound, and it can be detected as early as 16 weeks GA.

*****In the absence of prenatal diagnosis, a clinical diagnosis of vasa previa should be suspected in the setting of vaginal bleeding that occurs upon rupture of the membranes and is accompanied by fetal heart rate abnormalities, particularly a sinusoidal pattern or bradycardia.**

• Confirmation that the blood is fetal via Apt, Kleihauer-Betke tests, or other tests (Ogita, Londersloot) supports the diagnosis;
However, there is usually no time to wait for test results before performing an emergency cesarean delivery for fetal distress.

Management :

1. **Admission in the third trimester till delivery.**
2. **Single course of corticosteroids.**
3. **Serial fetal assessment : NST two to three times daily.**
4. **Scheduled delivery early, between 35-36 weeks.**
5. **Emergency cesarean section should be done if any of the following occur : labor/premature rupture of membranes/fetal distress by NST.**

Patient with APH →
1. History.
2. Examination : general including vital signs, abdominal examination then vaginal examination (inspection and speculum examination)
- Digital vaginal examination is contraindicated , until placenta previa rule out by ultrasound.
3. Investigations :
- Ultrasound : fetal assessment , placental localization , retroplacental clots,.....
- CTG : to assess fetal heart rate and uterine contractions.
- Blood : CBC, KFT, LFT, coagulation profile, blood group, cross match.

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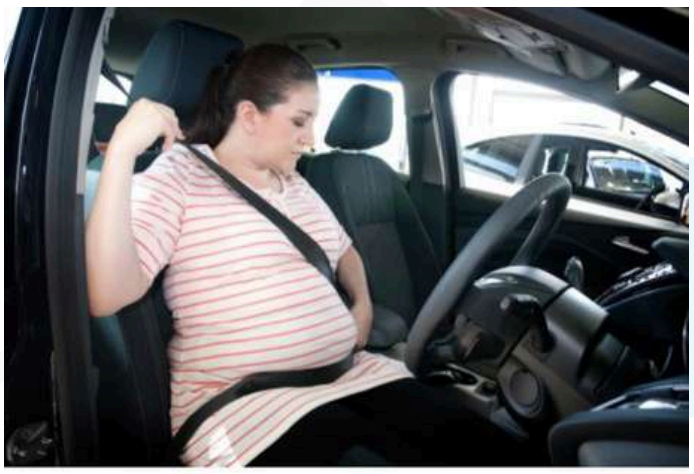

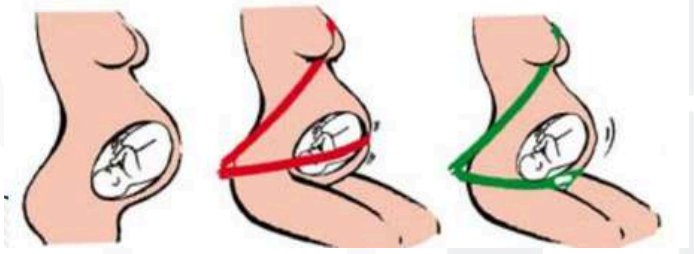

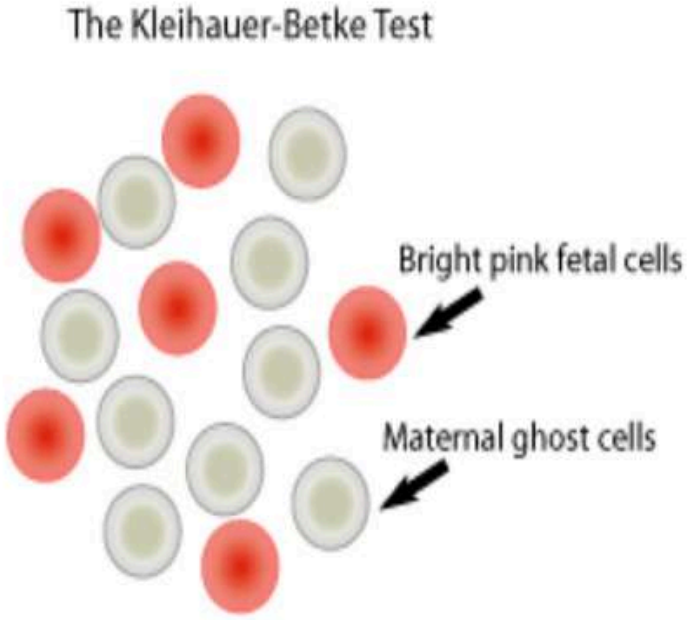

يا ربّ،
إن ضاقت بنا السُبل، وتهنا في دروب لا تؤدي إليك،
فأمسك بأيدينا بلطفك، واهدِ قلوبنا بنورك
ولا تجعل سعينا هباءً، ولا جهدنا ضائعًا في سرابٍ لا يُثمر

اللهم
إنّا نستجير بك من طريق بلا هدى
ومن عمل بلا بركة
ومن قلبٍ لا يرى، وعينٍ لا تبصر الحق

هب لنا من لدنك توفيقًا يسكن أرواحنا
وبصيرةً تضيء لنا عتمات الطريق
واجعلنا ممّن إذا عملوا أخلصوا، وإذا دعوا أُجيبوا، وإذا ساروا إليك، وصلوا

Thank You !