Polyhydramnios Oligohydramnios Placental Abnormalities



JU/5th Year Medical Students Dr Amal Barakat

AMNIOTIC FLUID

Amniotic fluid: A clear, slightly yellowish liquid that surround the fetus during pregnancy and it is contained in the amniotic sac.



AMNIOTIC FLUID COMPONENTS

•Amniotic fluid is 98% water and electrolytes.

•Signaling molecules, peptides, carbohydrates, lipids, and hormones make up the other 2%

-Amniotic fluid normally has a pH of 7.0 to 7.5.



AMNIOTIC FLUID





AMNIOTIC FLUID VOLUME

- ■10 WEEKS ==== 30 ml
- **-**20 WEEKS === 250 ml
- ■28 WEEKS === 800 ml

By 28 weeks, amniotic fluid reaches a volume of about 800 ml, where it plateaus until near term and then begins to decrease.



AMNIOTIC FLUID CIRCULATION

•AFV reflects the balance between AF flow pathways that increase AFV (urination, outflow of lung secretions) and those that decrease AFV (swallowing, intramembranous absorption).



AMNIOTIC FLUID FORMATION

- Active secretion by amniotic epithelium.
- Passive transudation from maternal circulation.
- Passive transudation from fetal circulation.
- •Fetal urine, Lung secretions and Gastrointestinal secretions.



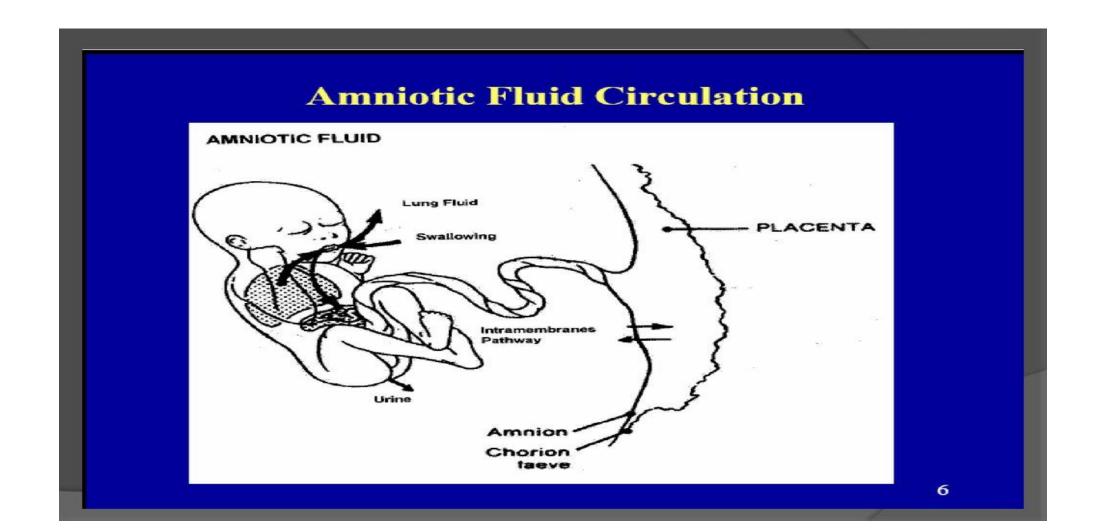
AMNIOTIC FLUID ELIMINATION

•Though there are many mechanisms for eliminating amniotic fluid, the two largest contributors to elimination are *fetal swallowing* and the *intramembranous pathway* [the transport of amniotic water and solutes across the amniotic membrane into the fetal circulation].

•The greatest contributor to amniotic fluid elimination is through fetal swallowing, seen as early as 11 weeks.



AMNIOTIC FLUID CIRCULATION



FUNCTIONS OF AMNIOTIC FLUID

- Fetal protection
- Umbilical cord protection providing a cushion between the fetus and the umbilical cord.
- Barrier against infection
- It serves as a reservoir of fluid and nutrients for the fetus containing: proteins, electrolytes, immunoglobulins, and vitamins from the mother.
- •It provides the necessary *fluid*, *space*, and *growth factors* to allow normal development and growth of fetal organs such as the musculoskeletal system, gastrointestinal system, and pulmonary system.

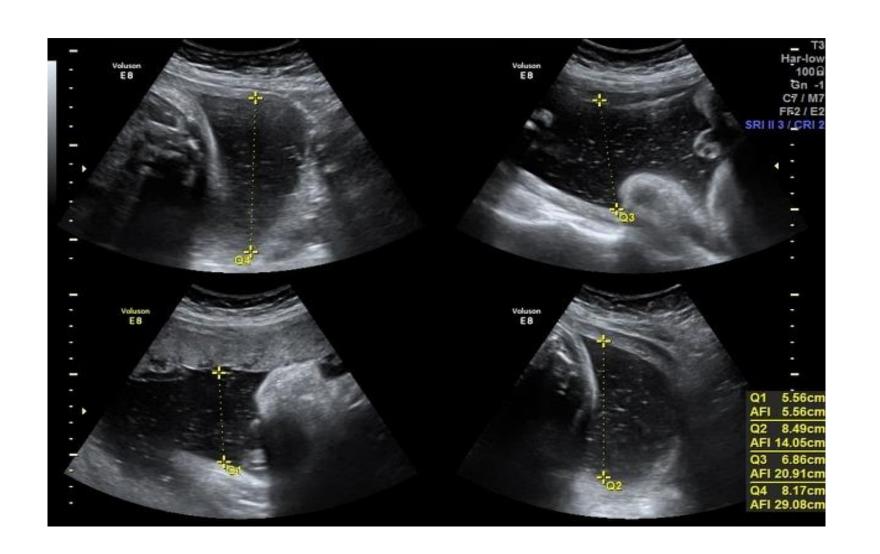
POLYHDRAMNIOS

 Polyhydramnios (also known as hydramnios) refers to an excessive volume of amniotic fluid.

- Prenatal diagnosis is based upon sonographic documentation of excessive amniotic fluid volume (AFV) by a quantitative technique:
- Amniotic fluid index (AFI) ≥24 cm
- Measured after 20 weeks by dividing the amniotic cavity into four quadrants using maternal linea nigra as the midline.
- Single deepest pocket (SDP) ≥8 cm
- Measured vertically in the AP plane at any location in the amniotic cavity.

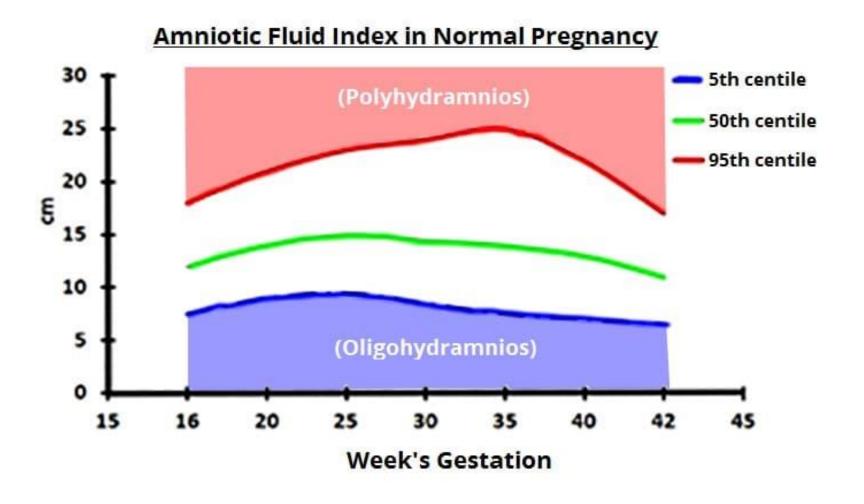


AMNIOTIC FLUID INDEX





AMNIOTIC FLUID INDEX IN NORMAL PREGNANCY





POLYHADRAMNIOS

- Excessive amount of liquor
- •MILD === AFI 25-30 cm
- •MODERATE === AFI 31-35 cm
- •SEVERE === AFI More than 35 cm



INCIDENCE

•The incidence of polyhydramnios in a general obstetric population generally ranges from 1 to 2 percent.



CAUSES

- **IDIOPATHIC** (65%)
- MATERNAL (15%)
- Rh isoimmunization
- DIABETES (GDM)
- FETAL (18-20 %)
- Multiple pregnancy (Twin-Twin Transfusion Syndrome)
- Fetal anomalies
- Chromosomal abnormalities (Trisomies 21,18,13)
- PLACENTAL (Less 1%)
- Placental chorioangioma
- Circumvallate Placental Syndrome



FETAL ANOMALIES ASSOCIATED WITH POLYHDRAMNIOS

- Any condition that prevents the fetus from swallowing e.g. oesophageal atresia, CNS abnormalities (Anencephaly, Opened spina bifid), muscular dystrophies, congenital diaphragmatic hernia.
- Esophageal or Duodenal atresia
- Anaemia alloimmune disorders, viral infections
- Fetal hydrops
- Twin-to-twin transfusion syndrome
- Increased lung secretions cystic adenomatoid malformation of lung
- Genetic or chromosomal abnormalities



CLINICAL TYPES

Depending on the rapidity of onset of Hydramnios

-ACUTE(rare)

- Sudden onset, appears in few days

CHRONIC

Insidious onset, appears in few weeks



CLINICAL FEATURES

Depending on the severity of polyhydramnios

- Asymptomatic
- General weakness
- Dyspnea, palpitation
- •Pressure symptoms: Heart burn, Indigestion, lower limb edema, Abdominal discomfort, leg and vulval varicosities and hemorrhoids



CLINICAL FEATURES

- It should be suspected clinically when uterine size is large for gestational age.
- The skin is tense and shiny
- The umbilicus is flat or everted
- The fetal parts are difficult to be palpated
- Difficult to hear fetal heart
- Abnormal lie and presentation
- High presenting part



DIAGNOSIS

- Ultrasound
- To confirm the diagnosis
- Single/Multiple
- Lie/Presentation
- Screen for congenital abnormalities
- Fetal Doppler
- Blood grouping = Rh, Serological testing for alloimmunization
- Bood sugar, Maternal Glucose Tolerance Test
- **TORCH** (Toxoplasmosis, Parvovirus, Rubella, Cytomegalovirus, Hepatitis)
- -Karyotyping (if appropriate)



COMPLICATIONS & POTENTIAL CONSEQUENCES

During pregnancy:

- Malpresentation
- PROM
- Preterm Labor
- Placental Abruption and Accidental hemorrhage

During Labor:

- Early ROM
- Cord Proplapse
- Increased operative delivery
- Uterine atony, PPH & Shock



COMPLICATIONS & POTENTIAL CONSEQUENCES

FETAL

•Increase prenatal morbidity & mortality mainly due to prematurity and the presence of an underlying abnormality or congenital malformation.

-After delivery:

the baby must be examined before its first feed by a pediatrician. A nasogastric tube should be passed to ensure there is not a tracheoesophageal fistula or esophageal atresia.



AIMS OF MANAGMENT

- Relieve the symptoms
- •Find a cause
- To avoid and to deal with complications



MANAGEMENT

- Most of the time the management is supportive and no medical intervention is required in the majority of women with polyhydramnios.
- If maternal symptoms are severe (e.g breathlessness), an **aminoreduction** can be considered.
- Aminoreduction is associated with PROM, infection and placental abruption (due to a sudden decrease in intrauterine pressure),.
- RE-accumulation may develop rapidly, and is therefore not performed routinely.
- Indomethacin can be used to reduces fetal urine production.
- 25 mg every 6 hrs/ Limit use to the lowest effective dose for the shortest duration possible.
- Indomethacin intake durin pregnancy is associated with premature closure of the ductus arteriosus, intraventricular hemorrhage and fetal renal impairment or dysfunction leading to oligohydramnios, therefore it should not be used beyond 32 weeks.

•WHEN and HOW TO DELIVER THOSE PATIENTS?



OLIGOHYDRAMNIOS

- Oligohydramnios refers to amniotic fluid volume (AFV) that is less than the minimum expected for gestational age.
- It is diagnosed by ultrasound examination, preferably based on an objective measurement:
- Amniotic fluid index (AFI) ≤5 cm
- Single deepest pocket (SDP) <2 cm</p>



INCIDENCE

•In the overall obstetric population, oligohydramnios occurs in <1 percent of preterm pregnancies and in 2 to 10 percent of pregnancies at 40 to 42 weeks



CLASSIFICATION

- Some clinicians classify oligohydramnios as:
- mild (AFI 4.1 to 5.0 cm)
- moderate (AFI 2.1 to 4.0 cm)
- severe (AFI 0 to 2.0 cm)
- Anhydramnios is the extreme end of the oligohydramnios spectrum



CAUSES

- Rupture of membranes
- Maternal causes: HTN, PET, SLE, CHRONIC KIDNEY DISEASE, CHRONIC ABRUPTION, POST TERM
- **Fetal causes**: IUGR, Intrinsic renal disorders (eg, cystic renal disease), Renal agenesis, Obstructive lesions of the lower urinary tract (eg, posterior urethral valves, urethral atresia).
- Drugs: Indomethacin, ACE Inhibitors
- Idiopathic



CAUSES

•The two most common disturbances leading to oligohydramnios are:

AF loss due to rupture of membranes

 Reduced urination due to fetal kidney disease or lower urinary tract obstruction



CLINICAL MANIFESTATIONS

- •Oligohydramnios may be suspected because the uterine size is less than expected for gestational age
- •As an incidental finding on an ultrasound examination or performed for another reason
- •The patient presents with prelabor rupture of membranes.



COMPLICATIONS

- Pregnancies complicated by oligohydramnios from any cause are at risk for:
- Pulmonary hypoplasia (if second-trimester oligohydramnios),
- Fetal deformation (if prolonged oligohydramnios)
- Umbilical cord compression
- Increased risk for fetal or neonatal death, which may be related to the underlying cause of the reduced AFV, the sequalae of reduced AFV, or both.



PROGNOSIS

The fetal prognosis depends on several factors:

- The underlying cause
- Severity (reduced versus no amniotic fluid)
- Gestational age at occurrence



PROGNOSIS

-Idiopathic oligohydramnios has a better prognosis than renal oligohydramnios, but the risk for adverse outcome is still increased, in part because some of these cases represent placental pathology, such as maternal vascular malperfusion



PROGNOSIS

- Oligohydramnios after amniocentesis
- •An exception to the poor prognosis is oligohydramnios related to second-trimester amniocentesis. In these cases, the membranes often "reseal," amniotic fluid reaccumulates, and pregnancy outcome is normal.



APPROACH

• Targeted History and Physical Examination looking for maternal and familial conditions and maternal medications that may be associated with oligohydramnios.

Rule out prelabor rupture of membranes (PROM)

•ULTRASOUND: Perform a detailed sonographic evaluation for findings that may account for reduced AFV.



ULTRASOUND

The evaluation should include:

- Assessment for fetal anomalies, particularly assessment of the:
- -Kidneys (presence, size, location, appearance [echogenicity, cysts, urinary tract dilation])
- Bladder (size and shape)
- -Umbilical cord fetal insertion site and vessel number
- Fetal sex (males are prone to renal dysplasia and agenesis, posterior urethral valves)
- Second-trimester markers suggestive of aneuploidy
- Fetal Growth Restriction
- Placental abnormalities (eg, chronic abruption)



MRI

•Fetal magnetic resonance imaging (MRI) can be helpful to better define complex fetal anomalies when this information will alter patient care, because it is less limited by lack of amniotic fluid than ultrasound. It may also detect anomalies missed on ultrasound.

GENETIC TESTING

• If no fetal anomalies are detected (ie, isolated oligohydramnios), the risk of genetic abnormalities does not appear to be increased above the baseline risk.

- If fetal anomalies are identified:
- AMNIOCENTESIS for microarray on amniocytes
- Noninvasive screening test (cell-free DNA) and discuss its limitations
- Trisomy 13 and triploidy are the most common chromosomal abnormalities associated with early oligohydramnios



DIAGNOSTIC AMINOINFUSION

- If oligohydramnios prevents adequate ultrasound assessment of the fetus, transabdominal amnioinfusion of approximately 200 mL of <u>saline</u> under ultrasound guidance to provide better visualization of fetal anatomy and thus improve diagnostic precision may be done.
- During the amnioinfusion, fluid may be collected for genetic studies.
- In addition, in patients with second-trimester oligohydramnios, intraamniotic dye injection can be of value for diagnosis of prelabor rupture of membranes (PROM) if physical and laboratory examinations do not support the diagnosis.



DIAGNOSTIC MATERNAL HYDRATION

- Oral hydration with one to two liters of water may be an alternative to amnioinfusion to transiently increase AFV for up to 48 hours, particularly in hypovolemic patients.
- Hydration with water appears to reduce maternal plasma osmolality and sodium concentration, resulting in osmotically driven maternal to fetal water flux; it also improves uteroplacental perfusion.
- The combined use of oral water ingestion and <u>desmopressin</u> (DDAVP) markedly and transiently increases AFV; however, use of DDAVP for this indication should be considered experimental, and used only under approved research protocols.

SERIAL AMNIOINFUSION OR AMNIOPORT

- Transabdominal amnioinfusion performed serially percutaneously or serially or continuously through a port has been used with some success in *research studies* to improve fetal outcome in pregnancies with idiopathic oligohydramnios, early oligohydramnios due to PPROM, and oligohydramnios due to lower urinary tract obstruction or fetal renal disease.
- Significant postnatal medical, emotional, and financial implications exist.
- Normal <u>saline</u> is infused to achieve a single deepest pocket (SDP) >2 cm.



VESICOAMNIOTIC SHUNT

• In fetuses with oligohydramnios due to lower urinary tract obstruction, fetal cystoscopy for diagnostic confirmation of posterior urethral valves followed by vesicoamniotic shunting has been used with some success to increase AFV and prevent adverse pulmonary, orthopedic, and renal sequelae.



TISSUE SEALANTS

- •A variety of tissue sealants (eg, fibrin glue, gelatin sponge, amniopatch) have shown some success in stopping leakage from ruptured membranes in case reports.
- Neither the safety nor the efficacy of these sealants has been established.



THERAPEUTIC MATERNAL HYDRATION

• Maternal hydration may be a useful long-term strategy to improve AFV in cases of idiopathic oligohydramnios. however, the effect on perinatal outcomes could not be assessed.

• It is recommended an oral intake of about 1500 mLs of hypotonic solutions daily, ideally for two weeks.

• There were many limitations to the available data, including inability to evaluate pregnancy, delivery, and neonatal outcome after the interventions; large differences in gestational age at diagnosis of oligohydramnios; different cut-offs for diagnosis; different hydration protocols (IV, oral, combination of IV and oral; different hydration volumes, hydration solutions, and durations of hydration); and different outcome measures and time intervals for their assessment.

PLACENTAL VARIANTS & ABNORMALITIES

•A normal placenta is round or oval-shaped and about 22 cm in diameter. It is 2 cm to 2.5 cm thick and weighs about a pound (470 gm).

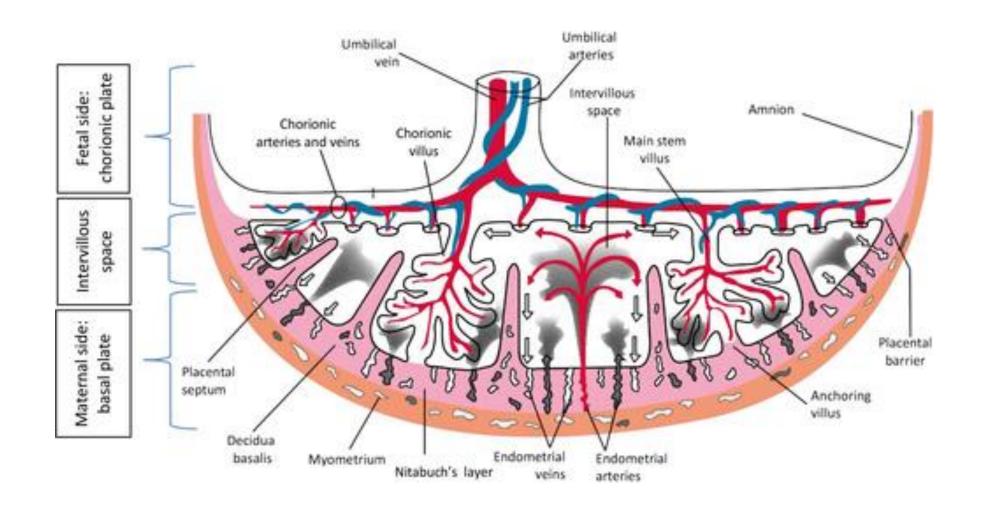


PLACENTAL STRUCTURE

- The placenta is composed of the chorionic plate on the fetal side and the basal plate on the maternal side.
- The fetal side and maternal side are separated by the intervillous space.
- The chorionic plate is a thick mass of connective tissue and contains the amnion, main stem villi and the chorionic arteries and veins, which are ramifications of the umbilical arteries and umbilical vein.
- The chorionic arteries and veins ramify into the arterioles and venules of the main stem villi.
- The main stem villi project into the intervillous space and are connected to the maternal basal plate by anchoring villi.



NORMAL PLACENTA





PLACENTAL VARIANTS

- Bilobed placenta (placenta bilobate, bipartite placenta, placenta duplex
- The succenturiate placenta
- Circumvallate placenta
- Circummarginate placenta
- The ring-shaped placenta
- Placenta fenestrate
- Battledore placenta (Marginal cord insertion)
- Placenta membranacea



PLACENTAL ABNORMALITIES

- Anatomical Abnormalities
- Low-lying placentas, placenta previa and abnormally invasive placentas are the most frequently occurring placental abnormalities in location and anatomy
- Abnormalities associated with structure and placental function
- Abnormalities associated with placental changes due placentamaternal effects such as pre-eclampsia and fetal erythroblastosis
- Mechanical abnormalities associated with the umbilical cord



PLACENTA PREVIA

- Complete Previa: Complete coverage of the cervical os by the placenta
- Marginal previa: If the leading egde of the placenta is less than 2 cm from the internal os
- Presentation: Painless Vaginal Bleeding started in the second half of pregnancy
- Soft, non tender uterus
- Malpresentation is found in 35%
- Vaginal examination is avoided
- Diagnosis by Ultrasound
- Corticosteroids are indicated at 24-34 weeks gestation
- Complications: APH, PPH, Shock, Hysterectomy, Death, Premature Delivery
- DELIVERY BY C/S IF MAJOR DEGREES



ABNORMALLY INVASIVE PLACENTAS

- PLACENTA INCRETA
- PLACENTA PERCRETA
- PLACENTA ACCRETA

 The defect of endometrial-myometrial interface leads to a failure of normal decidulization

- High morbidity and mortality because of severe and life threatening hemorrhage
- The most common risk factor is previous cesarian delivery
- Multidisciplinary care team
- RX: Cesarian HYSTERECTOMY



VASA PREVIA

•Vasa previa remains a hidden, rare complication that occurs approximately in 1/2500 pregnancies.

•Vasa previa is defined as the crossing of fetal vessels, unsupported by the placenta or the umbilical cord, between internal cervical os and the presenting part of the fetus.



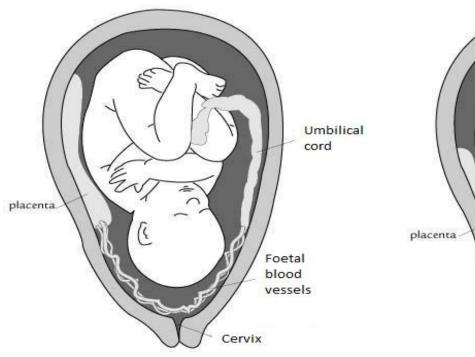
WHAT CAUSES VASA PREVIA?

•1- Velamentous insertions (where the cord inserts directly into the membranes, leaving unprotected vessels running to the placenta)

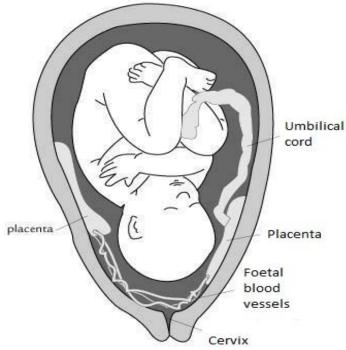
•2- Vessels crossing between lobes of the placenta such as in succenturiate or bilobate placent



WHAT CAUSES VASA PREVIA?



Vasa praevia with velamentous umbilical cord insertion



Vasa praevia with multi-lobed placenta



RISK FACTORS FOR VASA PREVIA

- 1- Pregnancy after ART
- 2- Low lying placenta & Placenta Previa
- 3-Bilobate placenta or Succenturiate placenta
- 4- Multiple gestation
- 5- Velamentous cord insertion



PRENATAL DIAGNOSIS OF VASA PREVIA

 Diagnosis by Vaginal Ultrasound and Colour Doppler

If diagnosed prenatally: Plan for C/S at 36 W



PRESENTATION OF VASA PREVIA

 The classic triad of the vasa previa is: membrane rupture >>> Rapid fetal bleeding >>> painless vaginal bleeding >>> fetal heart rate abnormalities – typically bradycardia, late decelerations, sinusoidal fetal heart rate pattern or >>> serious fetal injury & fetal death.

