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 Objectives

1. To know how to diagnosis fetal death
2. To be able to discuss and manage fetal death
3. To have an idea about possible causes
4. How to prevent recurrence

 Fetal death: etiology and pathological findings

 Intrauterine fetal death (IUFD)

Fetal death at any time after 20 weeks of gestation and/or weight of > 500 grams and before the start of labor.It complicate 1% of pregnancies.

**Causes of Fetal Death**

The aetiology of fetal demise is unknown in 25-60% of all cases. In cases where a cause is clearly identified, the cause of fetal death can be attributable to fetal, maternal, or placental pathology.

 **Maternal**

* Prolonged pregnancy (>42 wk)
* [Diabetes](http://emedicine.medscape.com/article/127547-overview) (poorly controlled)
* [Systemic lupus erythematosus](http://emedicine.medscape.com/article/1146456-overview)
* [Antiphospholipid syndrome](http://emedicine.medscape.com/article/261691-overview)
* Infection
* Hypertension
* [Preeclampsia](http://emedicine.medscape.com/article/221777-overview)
* [Eclampsia](http://emedicine.medscape.com/article/253960-overview)
* Hemoglobinopathy
* Advanced maternal age
* Rh disease
* Uterine rupture
* Maternal trauma or death
* Inherited thrombophilias

**Fetal**

* Multiple gestations
* Intrauterine growth restriction
* Congenital abnormality
* Genetic abnormality
* Infection (ie, parvovirus B19, CMV, listeria)
* Hydrops

**Placental**

* Cord accident
* Abruption
* Premature rupture of membranes
* Vasa previa
* Fetomaternal hemorrhage
* Placental insufficiency

**Risk factors (weak predictive value)**

* African American race
* Advanced maternal age
* History of fetal demise
* Maternal infertility
* History of small for gestational age infant
* Small for gestational age infant
* Obesity
* Paternal age

**Diagnosis of Fetal Death**

History and physical examination are of limited value in the diagnosis of fetal death. In most patients, the only symptom is decreased fetal movement. An inability to obtain fetal heart tones upon examination suggests fetal demise; however**, this is not diagnostic and death must be confirmed by ultrasonographic examination.** Fetal death cannot be confirmed by auscultation and CTG may be misleading as maternal heart rate may be recorded. Suspected IUD should be confirmed by US imaging of the FH by a Practitioner experienced in Ultrasonography and who is able to discuss the findings, with minimum delay. Other relevant findings may include overlapping skull bones, hydrops or oligohydramnios.Fetal demise is diagnosed by visualization of the fetal heart and the absence of cardiac activity.

Straight- X-ray abdomen (Spalding sign): collapse of skull bones it usually appears 7 days after I.U.F.D. Hyper flexion of the spine crowding of the ribs shadow (Robert’s sign) Appearance of gas shadow in great vessels: 12 hours

Complications

 Psychological upset

Infection: Once the membranes rupture, infection, especially by gas forming organism like CI. Welchi.

Blood coagulation disorders

During labor . Uterine inertia and PPH

Management depends on

1-Single or multiple gestation

2-Gestational age at death

3-The parents wish

**Management of Fetal Death**

Once the diagnosis of fetal demise has been confirmed, the patient should be informed of her condition. Often, allowing the mother to see the lack of cardiac activity helps her to accept the diagnosis.

If expectant management is planned: Await spontaneous onset of labour during the next four weeks Reassure the woman that in 90% of cases the fetus is spontaneously expelled during the waiting period with no complicatons.

Labor induction should be offered after diagnosis. Patient responses vary in regard to this recommendation; some wish to begin induction immediately, while others wish to delay induction for a period of hours or days until they are emotionally prepared.

When a dead fetus has been in utero for 3-4 weeks, fibrinogen levels may drop, leading to a coagulopathy. This is rarely a problem because of earlier recognition and induction. In some cases of twin pregnancies, induction after the death of a twin may be delayed to allow the viable twin to mature.

Induction may be accomplished with preinduction cervical ripening followed by intravenous oxytocin . Patients with a history of a prior cesarean delivery should be treated cautiously because of the risk of uterine rupture, just as in any birth following cesarean delivery .

Early fetal demise may be managed with laminaria insertion followed by dilatation and evacuation. In women with fetal death before 28 weeks' gestation, induction may be accomplished using prostaglandin E2 vaginal suppositories (10-20 mg q4-6h), misoprostol (ie, prostaglandin E1) vaginally or orally (400 mcg q4-6h), and/or oxytocin (preferred in women with prior uterine surgery). In women with fetal death after 28 weeks' gestation, lower doses should be used.

. Pain management in patients undergoing induction of labor for fetal demise is usually easier to manage than in patients with live fetuses. Higher doses of narcotics are available to the patient and often a morphine is sufficient for successful pain control. Should a patient desire superior pain control to intravenous narcotics, epidural anesthesia should be offered.

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**Evaluation of Fetal Demise**

The time following a fetal death is extremely difficult for both the family and the health care providers. In this stressful time, use of a checklist is helpful to prevent oversights in the evaluation of the mother, fetus, and placenta as well as to ensure that the emotional needs of the family are met.

Up to 60% of stillbirths have no identifiable etiology. Attempting to determine the cause of fetal death remains important because it may influence estimates of recurrence and future preconceptional counseling, pregnancy management, prenatal diagnostic procedures, and neonatal management.

Many institutions use a selective workup based on clinical findings. For example, when clinical findings strongly suggest a cause for the fetal demise either no further testing or limited testing is performed. Causes deemed fairly obvious include cord accident (ie, prolapse, entanglement, true knot, tight nuchal cord), anencephaly, or previously known lethal karyotype. In such cases, no further workup is necessary.

If severe clinical abruption is present, testing can be limited to toxicology screening and possibly a thrombophilia workup.

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|  | A systematic approach to fetal death is valuable in determining the etiology 1-History A-Family history Recurrent abortions Congenital anomalies Abnormal karyotype Hereditary conditions Developmental delay B-Maternal History I-Maternal medical conditions DM HPT Thrombophilia Autoimmune disease Severe Anemia Epilepsy Consanguinity Heart disease II-Past OB Hx Baby with congenital anomaly / hereditary condition IUGR Gestational HPT with adverse sequel Placental abruption IUFD Recurrent abortions  |

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|  | Current Pregnancy Hx Maternal age Gestational age at fetal death HPT DM/ Gestational DM Smooking , alcohol, or drug abuse Abdominal trauma Placental abruption PROM or prelabor SROM Specific fetal conditions Nonimmune hydrops IUGR Infections Congenital anomalies Chromosomal abnormalities Complications of multiple gestation Work up  |

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The most important part of the work-up of a fetal demise is the autopsy of the fetus. The decision to proceed with an autopsy must be made by the parents and informed consent is necessary. With parents who are resistant to the idea of a complete autopsy, a limited fetal evaluation should be discussed with the family. Although uncommon, postmortem MRIs can provide valuable information in the evaluation of a fetus when an autopsy cannot be performed.

The placenta and the membranes should be carefully examined, including cultures. Placenta Weight Staining Adherent clots Structural abnormality Velamentous insertion Edema/ hydropic changes Membranes Stained Thickening

 [ CLOSE WINDOW ]

**This is an example of a checklist to be used following fetal death. Courtesy of Santa Clara Valley Medical Center.**

Fetal karyotype can be obtained from a sample of amniotic fluid (preferred), fetal blood, or fetal tissue (skin or fascia lata). Fetal karyotype should be considered in all cases. It is especially important if the fetus is dysmorphic, has growth retardation, is hydropic, or has anomalies or other signs of chromosomal abnormality. Chromosomal analysis should also be considered in patients with multiple pregnancy losses, especially with a history of second- and third-trimester losses or when a parent has a balanced translocation or mosaic chromosomal pattern

A summary of the protocol for the fetus and placenta is as follows:

* Careful inspection
* Placental cultures for suspected listeria infection
* Radiographs, if indicated
* Autopsy
* MRI, if no autopsy
* Fetal karyotype

**Maternal Studies**

Maternal studies that should also be considered during the workup of a fetal demise include the following:

* Diabetes testing using hemoglobin A1C and a fasting blood glucose
* Syphilis screening using the VDRL or rapid plasma reagent test
* Thyroid function testing (ie, TSH, FT4)
* Urine toxicology screening

The above tests have traditionally been a part of an evaluation for the etiology of fetal demise. If diabetes screening has been performed during the prenatal period, repeat testing for diabetes is probably not necessary. Similarly, if the patient has no signs or symptoms of thyroid disease, thyroid dysfunction is unlikely to be the cause of the demise. However, these tests are inexpensive

**Commonly accepted tests**

* Thorough maternal history
* Fetal autopsy
* Placental evaluation
* Karyotype
* Indirect Coombs test
* Serologic test for syphilis
* Testing for fetal-maternal hemorrhage (Kliehauer-Betke or other)
* Urine toxicology screen
* Parvovirus serology

**Useful in some circumstances**

Thrombophilia evaluation to include the following:

* Lupus anticoagulant
* Anticardiolipin antibodies
* Factor V Leiden

Prothrombin mutation

* Protein C, protein S, and antithrombin III deficiency

**Uncertain use**

* TSH
* Hemoglobin A1C
* TORCH titers
* Placental cultures
* Testing for other thrombophilias

**Management of Future Pregnancy**

If a particular medical problem is identified in the mother, it should be addressed prior to conception. For example, tight control of blood glucose prior to conception can substantially reduce the risk of congenital anomalies in the fetus. Preconceptional counseling is helpful if congenital anomalies or genetic abnormalities are found. Genetic screening and detailed ultrasound can evaluate future pregnancies. In some cases, such as cord occlusion, the patient can be assured that recurrence is very unlikely.

Fetal death of unknown cause is a special problem. Because a large number of etiologies of fetal demise exist, a provider has difficulty determining risk of stillbirth for any particular pregnancy. Although recurrent fetal loss is uncommon, patients are naturally anxious. Most patients find increased fetal surveillance with the next pregnancy reassuring, even though such testing is not clearly beneficial. We recommends antepartum testing starting at 32-34 weeks' gestation in an otherwise healthy mother with history of stillbirth.Weekly biophysical profile or fetal heart rate testing can be combined with maternal kick counts in the third trimester. For patients who have experienced earlier loss, frequent ultrasound is reassuring.

Optimal management of chronic medical conditions is important prior to the next pregnancy.

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