

3. Management of Intra-Uterine Death

Contributed by

Prof. Malik Gunawardana
 Dr. Gamini Perera
 Dr. P.D. Liyanagama
 Dr. Gyan de Silva
 Dr. Sarath Karunarathna
 Dr. P. Amaradivwakara
 Dr. H. D. Kalansuriya
 Dr. T.V.K. Vitananchchi
 Dr. S.K. Gunasinghe

Management of Intra-Uterine Death

Contents	Page
3.1 Scope of the guideline	59
3.1.1 Definition	59
3.2 Aetiology	60
3.2.1 Maternal	60
3.2.2 Fetal	60
3.2.3 Placental	60
3.3 Prevention /Prediction	61
3.4 Diagnosis	62
3.5 Complications	63
3.6 Management	64
3.6.1. Initial counselling	64
3.6.2. Maternal investigations	64
3.6.3. Accomplishing the delivery of the fetus	67
3.6.4 Management after delivery of the fetus and placenta	69
3.7 References	70
3.8 Further reading	70

Introduction

The guidelines are to provide recommendations to aid General Practitioners and Obstetricians in the management of pregnancy with an **Intra-Uterine Death (IUD)**. This treatment could be initiated in a primary care setting or in centres with advanced facilities. The objectives of the guidelines are prevention, early diagnosis, investigating, treatment and counseling of the patient.

3.1 Scope of the guideline

The death of a fetus at any stage of pregnancy is a tragic and a very sensitive event and the obstetricians' aim is to;

- Establish the diagnosis
- Investigate the cause
- Delivery
- Subsequent counseling of the parents.

3.1.1 Definition

- Intrauterine Death may be defined as the retention of a demised fetus after a period of viability.

3.2 Aetiology

Unexplained causes account for 25-60% of all fetal demise; the incidence increases with increasing gestational age. In cases where a cause is clearly identified, the cause of fetal death can be attributable to fetal, maternal or placental pathology.

3.7.1 Maternal

- Prolonged pregnancy (>42 wk)
- Diabetes mellitus (poorly controlled)
- Systemic lupus erythematosus
- Infection
- Hypertension
- Pre-eclampsia
- Eclampsia
- Haemoglobinopathy
- Extremities of maternal age
- Rh disease
- Uterine rupture
- Antiphospholipid syndrome
- Acute, severe maternal hypotension
- Maternal death

3.7.2 Fetal

- Multiple gestation
- Intrauterine growth restriction
- Congenital abnormality
- Genetic abnormality
- Infection (i.e., parvovirus B19, CMV, Listeria)

3.7.3 Placental

- Cord accident
- Abruption
- Premature rupture of membranes
- Vasa praevia

3.3 Prevention /Prediction

- Prenatal screening for Blood group, viral infections.
- Prevent consanguineous marriages.
- Prevent early marriage and pregnancy (Showghy¹⁰ has stated that pregnancy at the age of 16 years and less can increase the IUD risk factor by 4 times.)
- Better education can directly influence the implementation of reproductive health.
- Several causes like chromosomal abnormalities, are not preventable even with modern medical knowledge, whereas others like post-maturity are completely preventable.
- Intrauterine fetal death secondary to Rh isoimmunization can be prevented with correct administration of anti-D Ig.
- Proper prenatal care.¹⁵
- Proper Diabetic and other diseases control. (Hypertension, SLE, Antiphospholipid syndrome, etc.)
- Fetal monitoring for cases with the high risk can lead to prevention of IUD.
- Ultrasound can be used to diagnose placental situation and in prediction of IUD.

3.4 Diagnosis

The absence of fetal heart sounds remains the mainstay of clinical diagnosis supported by lack of fetal movements and regression of uterine size.

Auscultation of the fetal heart by Pinnard stethoscope is dependent upon the experience of the operator, and reported lack of fetal movement by the mother may be unreliable.

The ultrasonic examination confirms the diagnosis. USS diagnosis of death is recognized by an absent fetal heart pulsation, skull collapse and retraction of brain tissue.

An attempt should always be made to determine the cause of the fetal death because this may dictate further management.

Sometimes there are situations where advice should be given for delivery without delay. The presence of ruptured membranes with retained dead fetus is a recipe for intrauterine sepsis. A major placental abruption is associated with a risk of rapid onset coagulopathy.

3.5 Complications

Problems associated with retained dead fetus;

- Infection,
- Maternal distress- Psychological manifestations- Depression, rejection of pregnancy, withdrawal,
- Coagulopathy,
- Rhesus isoimmunization in rhesus negative mother,
- Necessity for surgical intervention and its concomitant complications.

Coagulopathy only seems to occur after 16 weeks of gestation, and in general only when the dead fetus has retained in utero for more than 4 weeks.

DIC due to retained dead fetus dictates a necessity to empty the uterus.

Spontaneous abortion or labour will occur in 80% of women within 2 weeks. Only 10% remain undelivered for more than 3 weeks.

Patient presenting with an established coagulopathy and if she is not in labour, the use of heparin to arrest consumption of coagulation factors and allow time for spontaneous recovery is well described. But if the patient present with coagulopathy and in labour or if there is active hemorrhage, heparin is contraindicated. Treatment should then be directed towards replacement of deficient factors using fresh blood, cryoprecipitate or fresh frozen plasma (FFP).

3.6 Management

3.6.1 Initial Counseling

(Grade X)

Confirmation of diagnosis,
Breaking the bad news,
Sympathetic hearing,
Discuss the options of early induction or conservative management for 1-2 weeks.

3.6.2 Maternal Investigations

3.6.2.1 Basic investigations

(Grade X)

- Urine full report,
- Urine culture and anti-bacterial sensitivity test (ABST),
- Full blood count,
- Random blood sugar levels,
- High vaginal swab and endocervical canal swab for culture and anti-bacterial sensitivity test (ABST).
- Coagulation screen;
 - Bleeding time,
 - Clotting time,
 - Serum fibrinogen levels,
 - Serum fibrinogen degradation products,
 - Prothrombin time,
 - Activated partial thromboplastin time.
- Ultrasound scan;
 - Confirmation of IUD,
 - Fetal presentation and lie,
 - Placental localization,
 - Detection of placental abruption and the volume of the haematoma,

To detect uterine pathology (Detection of fibroids and their location, to detect uterine abnormalities,

To detect any other pelvic/abdominal pathology.

3.6.2.2 Special investigations

(Grade Y)

- o Diabetes mellitus detection;
 - Fasting blood sugar,
 - Post-prandial blood sugar,
 - Glucose tolerance test,
 - Haemoglobin A1c levels,
 - Fructoseamine.
- o Syphilis -screening;
 - Veneral disease research laboratory test (VDRL).
 - confirmatory
 - Treponima Pallidum haemagglutination test (TPHA).
 - ELISA Antibody.
 - Fluorescent Treponim anti-immunoglobulin test (FTAIG).
- o Thyroid function test;
 - Thyroid stimulating hormone (TSH),
 - Free thyroxine levels (FT4, FT3).
- o Urine toxicology screening,
- o Blood culture and anti-bacterial sensitivity test (ABST) (in cases of maternal pyrexia or presence of signs of septicaemia).

The above tests have traditionally been a part of an evaluation for the aetiology of fetal demise. If diabetes screening has been performed during the prenatal period, repeat testing for diabetes is 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 and normal results may be reassuring to the patient.

Additional tests that should be considered are as follows;

(Grade Y)

- o Antibody screening,
- o Kleihauer-Betke test,
- o Lupus anticoagulant and anticardiolipin antibody testing,
- o Thrombophilia panel: While some authorities recommend maternal testing in all cases of fetal demise, a more selective approach is to limit testing to patients who have a history of venous thrombosis, a positive family history, a placental infarction, severe pre-eclampsia occurring in the second or early third trimester, abruption, or intrauterine growth retardation. Testing is most accurate if performed several months postpartum,
- o Infection: Infection is a rare cause of fetal demise. Authorities' opinions vary as to which panel of tests is appropriate. If no obvious cause for the demise is established or if clinical signs or symptoms suggest infection, consider testing for;
 - i. Cytomegalovirus (acute and chronic titers),
 - ii. Rubella virus (acute and chronic titers, if not immune),
 - iii. Parvovirus (acute and chronic titers) and,
 - iv. Toxoplasma gondii (acute and chronic titers).

A more cost-effective approach is to limit testing for cytomegalovirus, rubella virus, and T. gondii to those patients in whom clinical findings suggest the possibility of intrauterine infection. (e.g., those with intrauterine growth restriction, microcephaly).

3.6.3. Accomplishing the Delivery of the Fetus

Vaginal delivery should be aimed unless there are specific indications for abdominal delivery. **(Grade X)**

Spontaneous labour could be awaited for up to two to four weeks or the labour could be induced. **(Grade Y)**

3.6.3.1 The induction of labour

The use of hypertonic solutions in the presence of dead fetus is obsolete.

The following facts need attention.

- i. Any agent which increase uterine tone has the potential capacity to rupture the uterus.
- ii. The myometrium is sensitive to prostaglandins at any gestation, but there is evidence that sensitivity increases with advancing gestation.
- iii. It does not matter which prostaglandin is administered or which route is used, as there will always be dose-effect relationship. Nevertheless, some analogues and some methods of administration are associated with a lower incidence of side effects.
- iv. The myometrium is relatively as insensitive to oxytocin as it is further away from term, and thus sensitivity increase with advancing gestation.
- v. Cervical 'ripeness' or favourability as defined by Bishop's score reflects oxytocin sensitivity.
- vi. The simultaneous administration of prostaglandin and oxytocin is commonly associated with iatrogenic damage to the uterus & cervix.

Surgical methods of induction i.e. artificial rupture of membranes (ARM), artificial separation of membranes (ASM) are **not** recommended because of the higher risk of infection.

A. Recommendations of induction

(Grade Y)

Oxytocin should be used for the induction of labour when the cervix is favorable.

The prostaglandin chosen should be one, which is available.

When prostaglandin is used the initial dose should be adjusted according to the gestation and further dose should be administered according to uterine response.

One should avoid the simultaneous administration of prostaglandin and oxytocin.

Adequate pain relief should be offered to make mother perfectly comfortable when required.

Consideration of the welfare of the fetus has no place.

3.6.3.2 Indication for Caesarean Section in the presence of a dead fetus

i. Absolute indications

(Grade X)

- Known placenta praevia of a major degree,
- Severe cephalo-pelvic disproportion,
- Previous classical Caesarian Section,
- Incipient uterine rupture- most commonly in neglected labour,
- In the presence of uterine rupture,
- Hand prolapse.

ii. Relative indications

(Grade Y)

- Previous LSCS,
- Transverse lie or shoulder presentation near term.

3.6.4 Management after delivery of the fetus and placenta

- o Careful inspection of the fetus and placenta. **(Grade X)**
- o Placental cultures for suspected listeria infection (To obtain placental cultures, separate the amnion and the chorion and submit a culture specimen using Stuart medium.)
- o Radiographs, if indicated, **(GradeY)**
- o Autopsy if indicated, **(GradeY)**
- o MRI, if no autopsy,
- o Chromosomal analysis of the fetus and placenta,
- o Counseling of parents and relations; **(Grade X)**
 - Explanation of probable or definite causes when available or events that led to the death in utero,
 - Arranging mementoes if parents wish (photographs of fetus, etc.),
 - Therapy with tranquilizers, hypnotics,
 - Psychological support by medical and ward staff,
 - Referral to a Psychologists/Psychiatrists when indicated,
- o Suppression of lactation; **(Grade X)**
 - Firm breast support and discourage expression of milk,
 - Mild analgesics for breast tenderness and pain,
 - Bromocriptine therapy.
- o Contraception as appropriate. **(GradeY)**

3.7 References

1. Fretts RC. Maternal age and fetal loss. Older women have increased risk of unexplained fetal death, Br Med J 2001; 322 (7283): 430.
2. Showghy S, Milaat W. Early teenage marriage and subsequent pregnancy outcome, East Med Healt J 2000; 6(1): 46-53.
3. Kiely J L. Fetal death during labor. Am J Obstet Gynecol 1985; 173:721-27.

3.8 Further reading

1. Zhang J, Cai W. Risk factors with antepartum fetal death. Early Hum Dev 1992; 28: 193–200.
2. Petitti, D. The epidemiology of fetal death. Clin Obstet Gynecol 1987; 30: 253–8.
3. Fretts, R, Usher R. Causes of fetal death in women of advanced maternal age. Obstetr Gynecol 1997; 89: 40–5.
4. Kochenour, N. Other causes of fetal death. Clin Obstet Gynecol 1987; 30: 312–9.
5. Rasmussen S, Albrechtsen S., Irgens L., Lorentz M., Dalaker K., Maartmann-Moe H., Vlatkovic L, Markestad T. Unexplained antepartum fetal death in Norway, 1985-97: diagnostic validation and some epidemiologic aspects. Acta Obstetricia et Gynecologica Scandinavica. 2003; 82(2):109-115.
6. Wen, S.W. Lei, Huizhong. K., Sauve M.S.. Determinants of Intrapartum fetal death in a remote and indigent population in China. J Perinatol. 2004, 24(2):77-8.
7. Incerpi M H. Stillbirth evaluation, what tests are needed? Am J Obstet Gynecol; 1998, 178: 1121-8.
8. Pajntar M. Maternal and neonatal outcome related to delivery time following PROM, Int Obstet and gynecol 1997; 58; 281-6.
9. Smith Get. Life table analysis of the risk of prenatal death at term and post term in singleton pregnancies. Am J Obstet Gynecol 2001; 184: 489-94.
10. Abu-Heija A. Grand multi parity. Is it risk? Int J Obstet Gynecol 1997; 59: 213-16.
11. Hyattsville R. Fetal mortality by maternal education and prenatal care.<http://www.yahoo/cdc.gov/nchs/products/pubds/series/st20/pre-1/st20-31htm> online. 2002.

12. Nathnagle M. Risk factors for late or no prenatal care following Medicaid expansions in California. *Matern Child Health J* 2000; 4(4): 251-59.
13. Froen J F. Risk factors for sudden intra uterine un-explained death. *J Obstet Gynecol*. 2001; 184: 694-702.
14. Al-Abudulkareem, A.A., Ballal SG. Consanguineous marriage in an Urban area of Saudia Arabia. *J Commun Health* 1998; 23(1): 75-83.
15. Rosham E.K. Seasonality of low birth weight in Indigenous Australias. *Aust Z J Pub Health* 1998; 22(6): 669-72.
16. Conde-Agudelo, A., Belizan, J. M., Diaz-Rossello, J. L. Epidemiology of fetal death in Latin America. *Acta Obstetricia et Gynecologica Scandinavica*. 79(5): 371-378, May 2000.
17. Gunton J E. Outcome of pregnancies complicated by pregastational diabetes mellitus. *J Obstet Gynecol* 2000; 40: 38-43.
18. Mondestin, MA.J., Ananth C. V., Smulian, J. C., Vintzileos A. M. Birth weight and fetal death in the United States: The effect of maternal diabetes during pregnancy. *Am J Obstet & Gynecol*. 2002; 187(4): 922-926.
19. Carrena A. Depression in women suffering prenatal loss. *Int J Obstet Gynecol* 1997; 62: 149-53.
20. Shiener E. Determining risk factors for intra partum fetal death. *J Reprod Med*. 2000; 45:419-24.
21. Rabson S. Subsequent birth outcomes after an unexplained still birth, *NZJ Obstet Gynecol* 2001; 41(1): 29-35.
22. Seppo H. Aoonotic and viral infection in fetal loss after 12 weeks. *Br J Obstet Gynecol* 2000; 104: 942-5.
23. Pajantar M. Maternal and neonatal outcome related to delivery time following PROM. *Int Obstet Gynecol* 1997; 58: 281-6.
24. Surkan P. J., Dickman S.O., Paul W. Cnattingius, S. Previous preterm and small-for-gestational-age Births and the subsequent risk of stillbirth. *N Eng J Med*. 2004; 350(8): 777-785.
25. Zhang J. Klebanoff M. A. Small-for-Gestational-Age Infants and Risk of fetal death in subsequent pregnancies. *N Eng J Med*. 2004, 350(8): 754-756.
26. Airass U. Chronic hypertension during pregnancy. <http://www.ranz.coy.edu.au/open/exam/motol/cases.htm> online 2002.
27. Divon M. Fetal and neonatal mortality in the post term pregnancy. *A.m J Obstet Gynecol*. 1998; 178: 726-31.

28. Cenantigus S. Difference in late fetal death rates in association with determinates of SGA fetus. *Br Med J* 1998; 316(3134): 1483-87.
29. Dikenson G. E. Obstetric and prenatal outcome from the Australian and New Zeland twin-twin transfusion syndrome. *Am J Obstet Gynecol*. 2000; 182: 706-12.
30. Paulli R.M. Outcome of twin pregnancies complicated by a single IUFD. <http://www/Mourningmultiplied/htm> on line 2002.