SUBJECT :::Midwifery

CHAPTER No 08

PREPARED BY: Uroosa qayoom

Management of complications during pregnancy

Qno 01 Antepartum hemorrhage or bleeding during pregnancy

Antepartum hemorrhage is defined as bleeding from genital tract after28th week of pregnancy and before the birth of the baby. According to WHO, bleeding from genital tract during pregnancy before the viability is called APH.

Types/causes

It has mainly two types:

1) placental bleeding : it includes placeta previa and Abruptio placenta.

2) Extra placental bleeding :it occurs due to local cervicovaginal lesions.

Placenta previa

Placenta previa is defined as implantation of the placenta in the lower segment lie alongside or in front of the presenting part. Placenta previa occurs approximately 1 of every 250 births. It is 1 in 200 in India. 1/3 of all antepartum hemorrhage occurs due to placenta previa.

Causes of placenta previa.

The exact cause of implantation of placenta in lower segment is not known but according to postulated theories or hypothesis there are few factors responsible for placenta previa.

1) Dropping down theory: according to this theory, the fertilized ovum drops down to lower iterine segment. Poor decidual reaction in the upper segment may be the cause of formation of Central placenta previa.

2) Multiple pregnancies: The large placental bed of the twin placenta is prone to low implantation of a least part of placenta.

3) Defective decidua: It causes spreading of the chorionic villi over a wide area in the uterine wall encroaching on the lower segment.

4) Big surface area of placenta: Big surface area of placenta can droops into the lower

segment which leads to placenta previa.

Other causes include multiple pregnancy, multiparty, maternal age more the >35years , race like Asian women, infertility treatment, presence of uterine scar, previous c section, prior curette, prior placenta previa, smoking, and any abnormalities of placenta.

Degrees of placenta previa: there are four types of placenta previa depending upon the degree of extension of placenta to lower segment.

Type 1(low lying) : The major part of placenta is attached to the upper segment and only the lower margin encroaches into the lower segment but not up to the os.

Type 2(marginal) : The palcenta reaches the margin os the internal os but not cover it.

Type 3(incomplete or partial central) : The placenta covers the internal os partially, covers the internal os when closed but does not entirely do so when fully dilated.

Type 4(central or complete) : The placenta completely covers the internal os even after it is fully dilated.

Note* For clinical purpose, the types are graded into mild degree (type 1 and 11 anterior) and major degree types 2 posterior, 3 and 4).

Signs and symptoms

Symptoms The only symptom of placenta previa is painless vagibal bleeding. Pain in abdomen is absent unless spontaneous labour ensures.

Signs : The bleeding is usually bright red color and the amount varies with the proportion of separation of placenta. Abdominal examination: the abdomen is soft, relax, elastic, normal consistency and without any localized area of tenderness, persistence of malpresentation commonly breech, head is floating, it can't be pushed down into the pelvis, slowing of the fetal heart rate on pressing the head into pelvis which soon recover promptly called as Stallworthy sign.

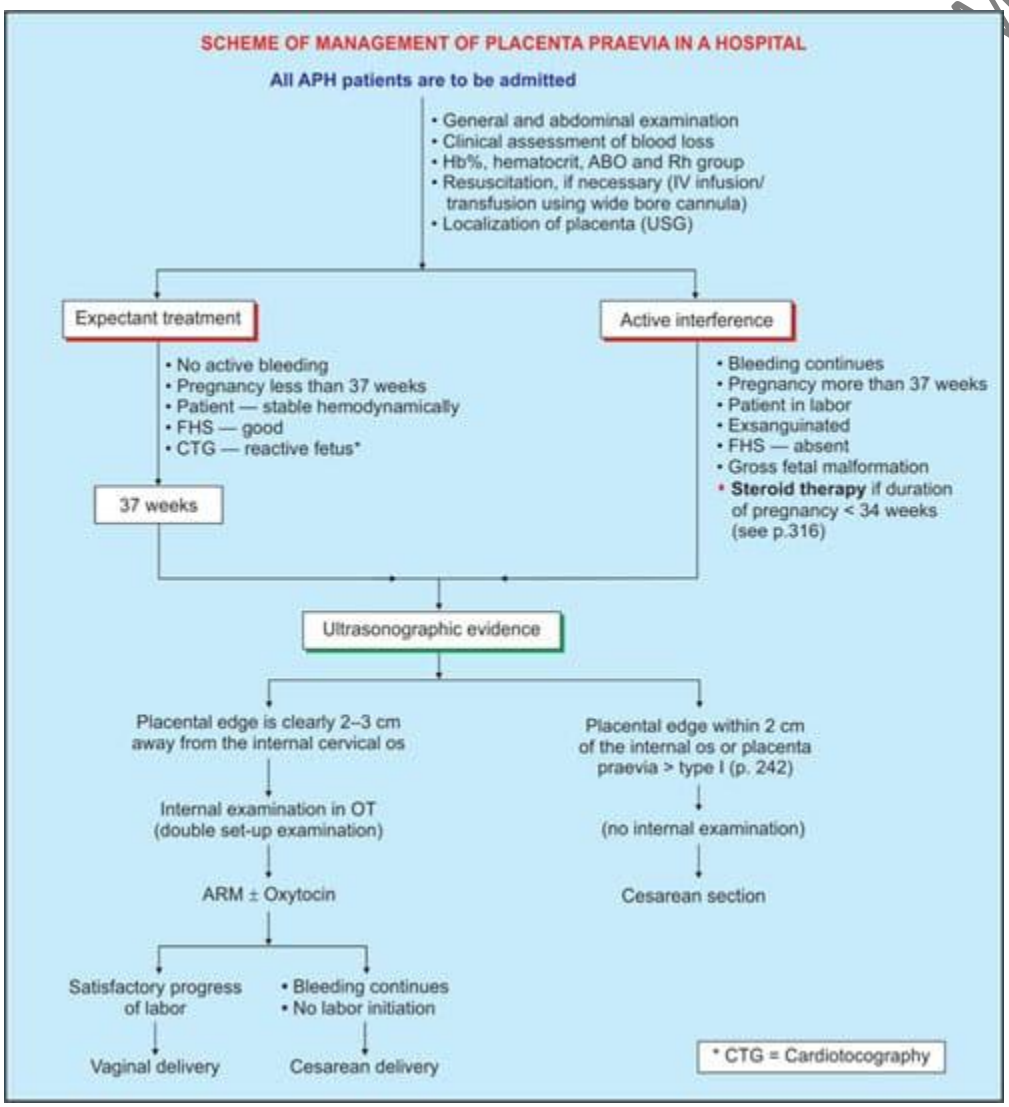
Diagnosis: 1) inaging studies-A) sonography (transabdominal ultrasound TAS and transvaginal ultrasound TVS, transperineal USG, color Doppler flow study, 3D power Doppler study. B) magnetic resonance imaging for better diagnosis of placenta previa and placenta accreta. C) clinical examination- By internal examination, direct visualization during cesarean section and examination of placenta following vaginal delivery.

Complications : Maternal -During pregnancy 1) Antepartum hemorrhage with varrying degree of shock is an inevitable complications. 2) Malpresentation, increased incidence of breech and transverse lie 3) premature labour either spontaneous or induced.

During labour : 1) Early repture of membranes 2) cord prolapse due to abnormal attachment of cord 3) slow dilatation of cervix 4) intrapartum hemorrhage 5) increased incidence of operative interference 6) PPH 7) retained placenta.

During Pureprium: sepsis is increased due to increased operative interference, placenta site near to vagina and anemia. Sibinvolution and embolism.

Scheme for management of placenta previa.



Abruptio placenta : it is the form of antepartum hemorrhage where the bleeding occurs due to premature separation of normally situation placental. Hospital records show incidence of 1 in 250 deliveries and 2/3 abruptions occurs before 36 weeks.

Varieties of abruptio placenta

Revealed : following separation of the placenta, the blood insinuates downwards between the membranes and the decidua. Ultimately, the blood comes out of the cervical canal to be visible externally and this is the commonest type.

Concealed : The collected blood is prevented from coming out of the cervix by the presenting part which presses on the lower segment. The blood collects behind the separated placenta or collected in between the membranes and decidua. Sometimes blood may percolate into amniotic sac after rupturing the membranes. In any of the circumstances blood is not visible outside. This is rare type.

Mixed : in this type, some part of the blood collects inside and a part is expelled out. Usually one variety predominates the other. This is quite common.

Etiology of abruptio placenta

The exact etiology of separation of normao situated placenta remains obscure in majority of cases, following are some of the risk factors

1) General factor : high birth order pregnancies with gravid more than>5 , three times more common than a first birth, advancing age of mother, poor socioeconomic condition and smoking.

2) Hypertension in pregnancy: it is the most important cause of abruption. Pre eclamsia, gestational hypertension and essential hypertension all are associated 10-50%.

3) Trauma : traumatic separation of placenta usually leads to it's marginal separation with escape of blood outside. Trauma may be due to external cephalic version, RTA, needle puncture at amniocentesis.

4) sudden uterine decompression: sudden uterine decompression of uterus leads to diminished surface area of uterus adjacent to placental attachment.

5) short cord: either relative or absolute can bring about placental separation during labour by mechanical pull.

6) supine hypotension syndrome: there is a passive engorgement of the uterine and placental vessels resulting in repture and extravasation of the blood.

7) sick placenta: poor placentation, as evidenced by abnormal uterine artery doppler waveform.

Other factors include placental anomaly, folic acid deficiency, torsion of the uterus, cocaine abuse, thrombophillias and prior abruption.

Clinical features: clinical features of abruptio placenta depends upon degree of separation, type of abruption and variety of abruption. Some of the C/M are small to moderate amount of bright or dark red vaginal bleeding, acute abdominal pain associated with vaginal bleeding, uterine tenderness and high uterine toxicity often described as " Board like abdomen"., incro in size of uterus particularly if the bleeding is concealed, failure of uterus between contractiona, fetal heart sound absent with concealed and mixed type, urine output usually diminished.

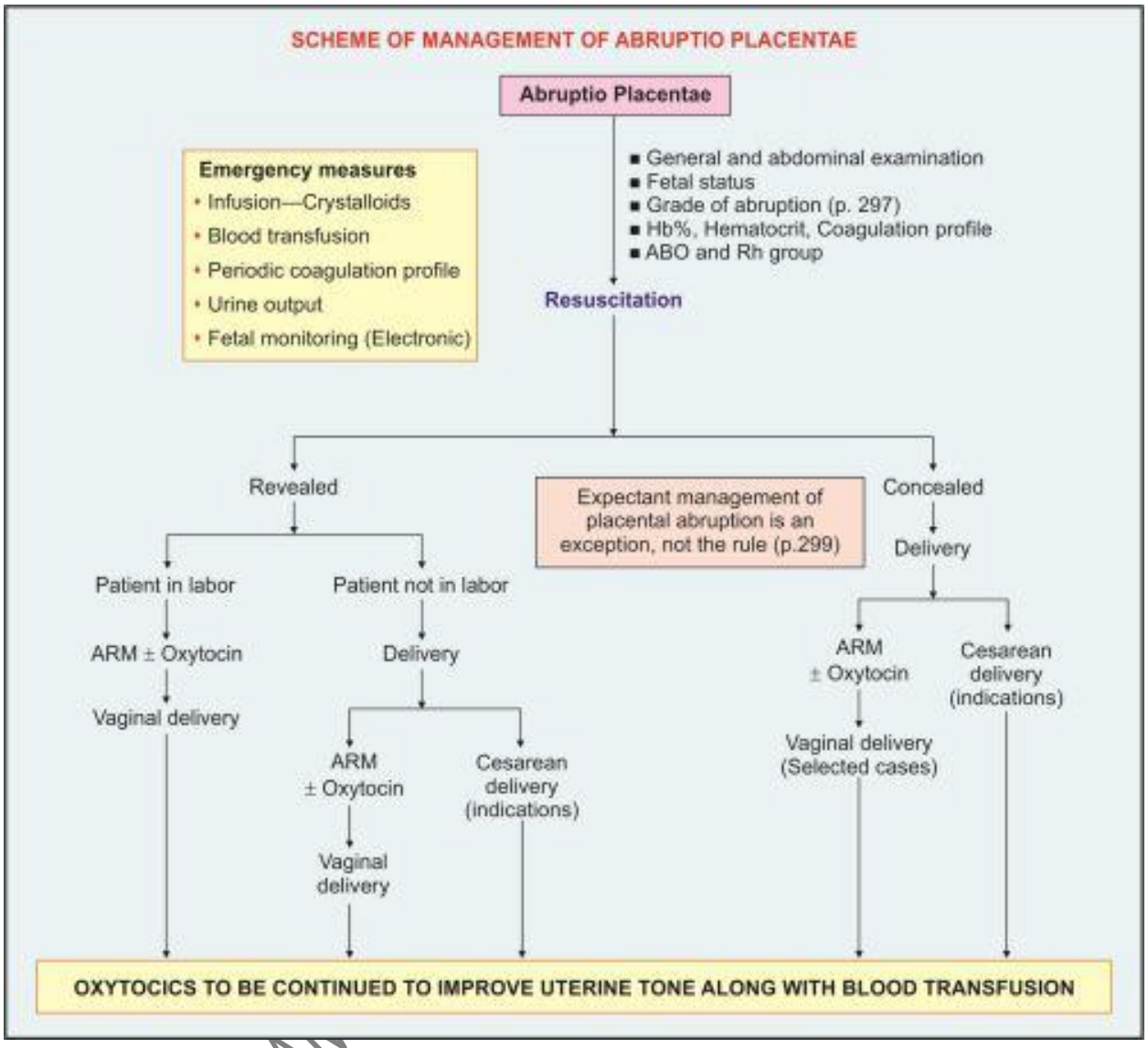
Diagnosis 1) ultrasound -- to visualize the placenta and presence of clot or hematoma.

2) coagulation profile: to rule out DIC (BT, CT, fibrinogen level, platelet count, PT, PTT, fibrin degradation products) 3) Renal function test.

Complications: In revealed type -- maternal risk is proportionate to visible blood loss and maternal death is rare.

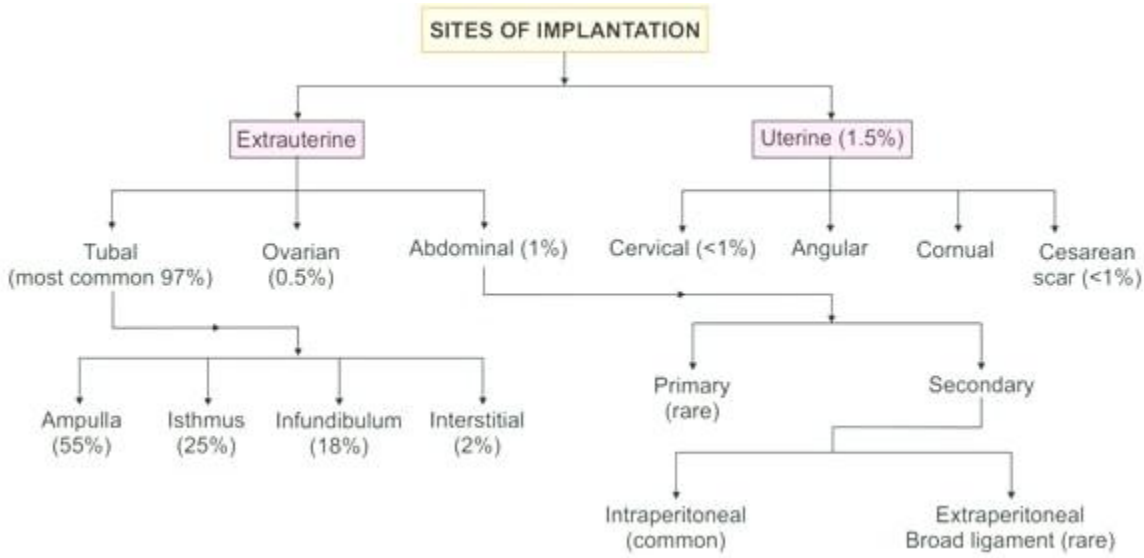
In concealed type : the following complications may occur--- hemorrhage, shock, blood coagulation disorder, Oligurea, anurea, pph and puerperal sepsis.

Scheme f management of abruptio placenta



Ectopic pregnancy : An ectopic pregnancy is one in which the fertilized ovum is implanted and develops outside the normal uterine cavity. Ectopic pregnancy still contributes significantly the cause of maternal mortality and morbidity.

Site of implantation



Risk factors : history of pelvic inflammatory disease, history of tubal ligation, contraceptive failure, previous ectopic pregnancy, tubal reconstructive surgery, history of infertility, IUD use, tubal endometrosis and salphingitis.

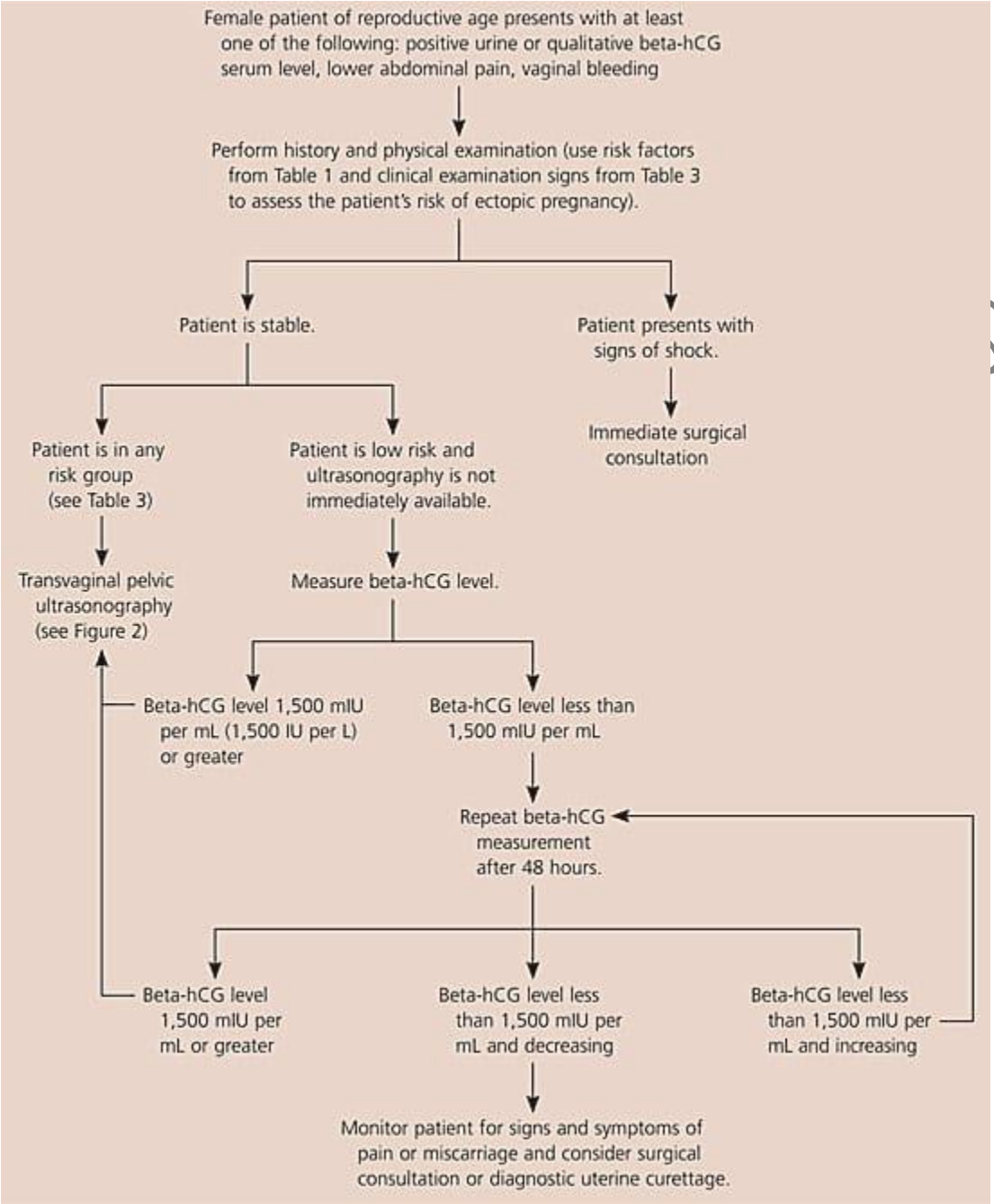
Clinical manifestation: in acute --- triad of symptoms are present I. e, abdominal pain 100%, vaginal bleeding, amenorrhea. Vomiting, fainting attack, pallor, features of shock like tachycardia, hypotension. Abdominal examination--- tense, tender, bowel may be distended. Pelvic examination-- vaginal mucosa and bulky uterus slightly present.

In unrepture: Amenorrhea, features of pregnancy, pain in abdomen generally flank pain, uterus usually soft showing evidence of early pregnancy, well circumscribed tender mass may be felt.

In chronic: bladder irritation, rectal tensmus, rise in temperature may be due to infection or due to absorption of products of degerated blood accumulated in the abdomen.

Investigation: blood examination, culdocentesis (aspirations of noncloting blood with Hematocrit greater than 15% signifies reptures ectopic pregnancy, estimation of HCG, and sonography.

Scheme for management of ectopic pregnancy.



Vesicular mole or hydratiform mole

Definition: it is the abnormal condition of the placenta where there are partly degeneration and partly proliferation changes in the young chorionic villi. This results in the formation of clusters of small cysts of varying sizes.

Incidence rate : it is highest in Philippians 1 in 80 prgenancies. Lowest in European countries 1 in 752 and in USA 1 in 2000.The incidence rate in India is about 1 in 400 pregnancies.

Cause : cause is not definitely known, but it appears to be related to the ovular defect as it sometimes affects one ovum of twin pregnancy. However, the following factors and hypothesis have been forwarded:

1) Highst prevalence among teenage pregnancies and those women's over 35 year of age. 2) prevelence appears to vary with race and ethnic origin 3) Faulty nutrition causes by inadequate intake of protein, carotene is associated with increased risk 4) Disturbed maternal immune mechanism 5) cytogenic abnormalities 6) and history of prior hydratiform mole.

Types of hydratiform mole / vasicular mole:

There are two types of abnormalities --- completely and incomplete mole

1) complete mole : The whole conceptus is transformed into a mass of vessicles. It is the result of fertilization of annucleated ovum (has no chromosome) with a sperm which duplicate giving rise to 46 chromosomes of paternal origin only.

2) incomplete mole : A part of trophoblastic tissue only shows molar changes. There is a fetus or at least an amniotic sac. It is the result of fertilization of an ovum by 2 sperms, so the chromosomal no is 69.

Clinical manifestation: pre eclamsia develops in 20% of cases, usually before 20w gestation., Hyperthyroidism develops in 10% of cases manifested by enlarged throid gland, breast signs of pregnancy, Abdominal examination:: uterus is larger than the period of amenorrhea in 50% of cases, 50% with inactive or dead mole., uterus is doughy in consistency, fetal parts and heart sound can't be detected except in partial mole, passage of vesicles (sure sign) , bilateral ovarian cysts (5 -20 cm) in 50%of cases.

Treatment As soon as the diagnosis of vasicular mole is established the uterus should be evacuated. The select method depends on the size of uterus. Cross matched blood should be available. Suction evacuation: carried under general anaesthetic but not that much which relax the uterus. An infusion of oxytocin 500 ml of 5%glucose should be maintained throughout the procedure. Hysteoromy : it may be needed for evacuation of large mole to minimize and facilitate control of bleedy. Hysterectomy should be considered in women over 40years who have completed their family for fear or risk of developing chariocarcinoma.

Hyperemesiss Gravidarum

Definition: Hyperemesiss Gravidarum or pernicious vomiting refers to excessive nausea and vomiting that results in fluid and electrolyte imbalance, marked weight loss, aetonuria and nutritional deficiencies. (According to REEDER MARTIN). The incidence of HG ranges from 0.3%to 3%depending on literature source.

Risk factors: multiple gastation, hydratiform mole, non use of multivitamin before 6 week of gestation or during the peri conceptional period, heartburn and acid reflux, nonpregnant women who experience nausea and vomiting related to oestrogen based medication, motion sickness, migraine, female fetus, family history of HG.

According to hormonal theory :excess of HCG and estrogen can trigger vomiting center that leads to HG. According to psychosomatic theory bodily changes and self concept the mother develops neurosis and psychological discomfort that may cause vomiting. Dietary deficiencies : Due to low deficiency of vitamin B1, B6 and protein may be the effect rather than cause. Allergic or immunological basis, decrease gastric motality, any pathology of liver, kidney, heart and brain disorder can leads to HG.

Types of HG there are two types -- Early HG and late HG

Early HG is defined as vomiting occur throughout the day but no evidence of dehydration and starvation. Late HG there is evidence of dehydration and starvation.

Clinical manifestation: pernicious vomiting (anything taken orally is rejected) , vomiting initially watery and bilious (weight loss is seen), oliguria, seldom mental symptoms, epigastric pain, constipation, Ptyslism, fatigue, anorexia. Dehydration, muscle wasting, ketosis, weigh loss >5% of pregnancy weight, tachycardia, postural hypotension, sunken eyes , acetone smell in breath

Investigation: Liver function test : elevated alanine aminotransferase ALT) and aspartate transaminase (AST) are four times more severe cases. Blood urea nitrogen and creatinine mag be slightly elevated, ketones is present in both blood and urine, serum electrolytes there may be hypokalemia, hyponatremia, and loss of hydrogen and chloride.

Complications: circulatory failure, jaundice due to liver involvement, retinal hemorrhage, Wernicke's encephalopathy (thiamine deficiency), Korsakoff 's syndrome (disorientation and loss of memory), renal insufficiency, Delerium, coma and in severe cases sometimes it leads to death.

Management for HG:

Management of nausea and vomiting symptoms:

Drink and eat little and often, meal high in carbohydrate and low in fat is better, meals should be cold because cold reduce smell related nausea, avoid caffeine and alcohol as these can enhance dehydration.

Medical management

Drugs Antiemetics 1) inj Promethazine 250 mg

2)Inj trifluopromazine 10 mg

3) Tab metachlopromide 10 mg

4(Tan Ondosterone

5)Nutritional supplements like vitamin B1, B6, vitamin B12 and vitamin C

6) Fluid theraply) 3 liter 50%dextrone and RL infusion in 24 hours and k+supplement fluid.

Gastational diabetes mallitus(GDM)

Definition: GDM is defined as carbohydrate intolerance of variable severity with onset or first recognition during present pregnancy.

Etiology:1) pregy pre diabetic state 2) pregnancy Leads to marked insulin resistance that will increase insulin requirement and cause GDM 3) 60-80%womens with obesity leads to GDM 4) family history of diabetes.

Effects of diabetes on pregnancy

Abortion, preterm labour, infection, increased incidence of pre eclamsia, maternal distress, diabetic retinopathy, pph, puerperal sepsis.

Effects of diabetes on fetus and neonate

A) fetal: fetal macrosomia, congenital malformation, birth injury and fetal death.

B) neonate: Hypoglycemia, RDS, polycythemai, hypocalcemai, Hypomagnesaemia.

C) During labour: prolongation of labour due to big baby, shoulder dystocia, perineal injury, operative interference.

D) puerperium: puerperal sepsis and lactation failure.

Gastational diabetes diet

30 kcal/kg for normal weigy women

24kcal/kg for overweight women

12kcal/kg for morbidy obese women.

Note :Diet should contain carbohydrate 50%, protein20% and fat 25-30%. Usually three meal regimen, with breakfast 25% of total intake, lunch 30, dinner 30%.

Management :

Preconceptional counselling : Fetal congenital malformation are significantly low (0.8-2%) in women's who receive preconceptional counselling. Patients glycemic control and vascular status are assessed. folic acid supplementation (0.4mg/day) . Woman are taught about self glucoss monitoring. Appropriate advice about diet and insulin is given.

Principles of management :1) careful antenatal supervision and glycemic control 2) To find out the optimum time and method of delivery. 3) Arrangement for the care of newborn.

Antenatal cars: Antenatal supervision should be at monthly interval upto 20th week, there after at 2 week interval. At times patient need stabilization of blood glucose and for a monitoring a fetus.

Sonographic examination: in GDM cases or normal prego (3-4week) interval is extremely helpful, not only to diagnose varieties of congenital malformation of fetus but also to detect fetal macrosomia or growth restricted rate.

Insulin therapy: when diabetes is first detected during pregnancy and can't be controlled by diet alone, it should be treated with insulin therapy.

Hospital management : GDM patients are admitted 34-36weeks prior. Early hospitalization facilitates 1) stabilization of diabetes 2) minimizes the incidence of pre eclampsia, polyhydramnios and preterm labour. 3) To select out appropriate time and method of delivery.

Induction of labour : Diabetes women controlled on insulin are considered for induction of labour after 38week of complete.

Cesarean section: if macrosomia, difficult to control diabetes, elderly primigravida , multigravidae with bad obstetric history, polyhydramnios, Malpresentation.

Care of baby : Asphyxia is anticipated and be treated effectively. Look for any congenit malformation, check blood glucose within 2 hours after birth to avoid problems of hypoglycemia, all babies should receive 1mg vitamin k IM and ensure early breast feeding.

Hypertensive disorders in pregnancy

Pre eclampsia (PE) : PE is a multisystem disorder of unknown etiology characterized by development of hypertension to the extent of 140/ 90mmhg or more with proteinurea after 20th week. According to new guidelines(ACOG) American Congress of obstetricians and gynecologist in 2013 , in India, the incidence of pre eclampsia is reported to be 8-10%among primigravida and in multigravidae 5%.

Etiology: Failure of trophoblastic invasion mediators (abnormal placentation), vascular endothelial damage, immunological intolerance between maternal and fetal tissues, coagulation abnormalities, genetic predisposition, dietary deficiency or excess.

Clinical types of pre eclampsia : There are two types of pre eclampsia I. e mild and severe

Mild pre eclampsia: This includes rise of bp of more than 140/90 mmhg but less than 160 mmhg systolic and 110 mmhg diastolic without significant proteinurea.

Severe pre eclampsia: this includes rise of systolic bo more than 160 mmhg or diastolic more than 110 mmhg.

Clinical manifestation : 1) slight swelling over the ankles 2) Tightness of the ring on the finger 3) swelling may extend to face, abdominal wall, valva and even the whole body.

Alarming signs :1) Headache (occipital or frontal region) 2) Disturbed sleep 3) Diminished urinary output 4) epigastric pain.

In severe pre eclampsia : Bp more than 160/110 , Oligurea (urine output less than 400 ml/ day) , proteinurea (more than 5gm/day), visual disturbance and retinal hemorrhage, HELLP syndrome (hemolysis, elevated liver enzymes level, and low platelet count less than 1,00,000 cells/mm3, weight gain more than 1 pond (500gm) per week or 4-5 pond (2500) per month.

Complications: 1) pre eclampsia changes in eclampsia 2) PPH 3) IUGR 4) abruptio placenta 5) Disseminated intra vascular coagulation (DIC), Olighydrominios.

Investigation : 24 hour urine collection to determine ratio of protein to creatinine and proteinurea.

Ophthalmoscopy (examination of interior of eye) - for retinal edema and hemorrhage

Blood pressure monitoring, LFT - to monitor raised level of liver enzymes eg Alanine Aminotransferase and aspartate Aminotransferase. Clotting studies - if there is severe pre eclampsia or thrombocytopenia, sonography - for assessment of fetal growth, volume of amnioti fluid, Doppler Velocimetry.

Tool over test :: This test is done between 28- 32 weeks. First take bp from one side then advice the patient to roll on her back and measure again bp, if diastolic bp increase greater than 20mmhg from rolling side to back position then test consider positive. There is a chances of develop hypertension later if test is positive.

Prevention: Regular antenatal check up, calcium supplementation (2 gm/ day), nutritional and anti oxidant supplement and give anti thrombin agents like low dose aspiring 60 mg daily.

Management : 1) provide bed rest in lateral position and adequate fluid

2) Advice protein diet (about 100 gm protein daily)

3) Give total calory of 1600 kcal/ day

4) monitor deep tendon reflex and presence of hyperreflexia because hyperreflexia indicate CNS irritability.

5) monitor renal function through BUN, serum creatinine and creatinine clearance.

6) Monitor intake and output to assess renal perfusion

7) corticosteroid may use to promote lung maturity

8) Administer antihypertensive drugs

9) In hypertensive crisis (if diastolic bp more than 110 mmhg) administer

A) Labetalol 200 mg in 200 ml NS at 20 mg / hour via IV and double every 30 minutes.

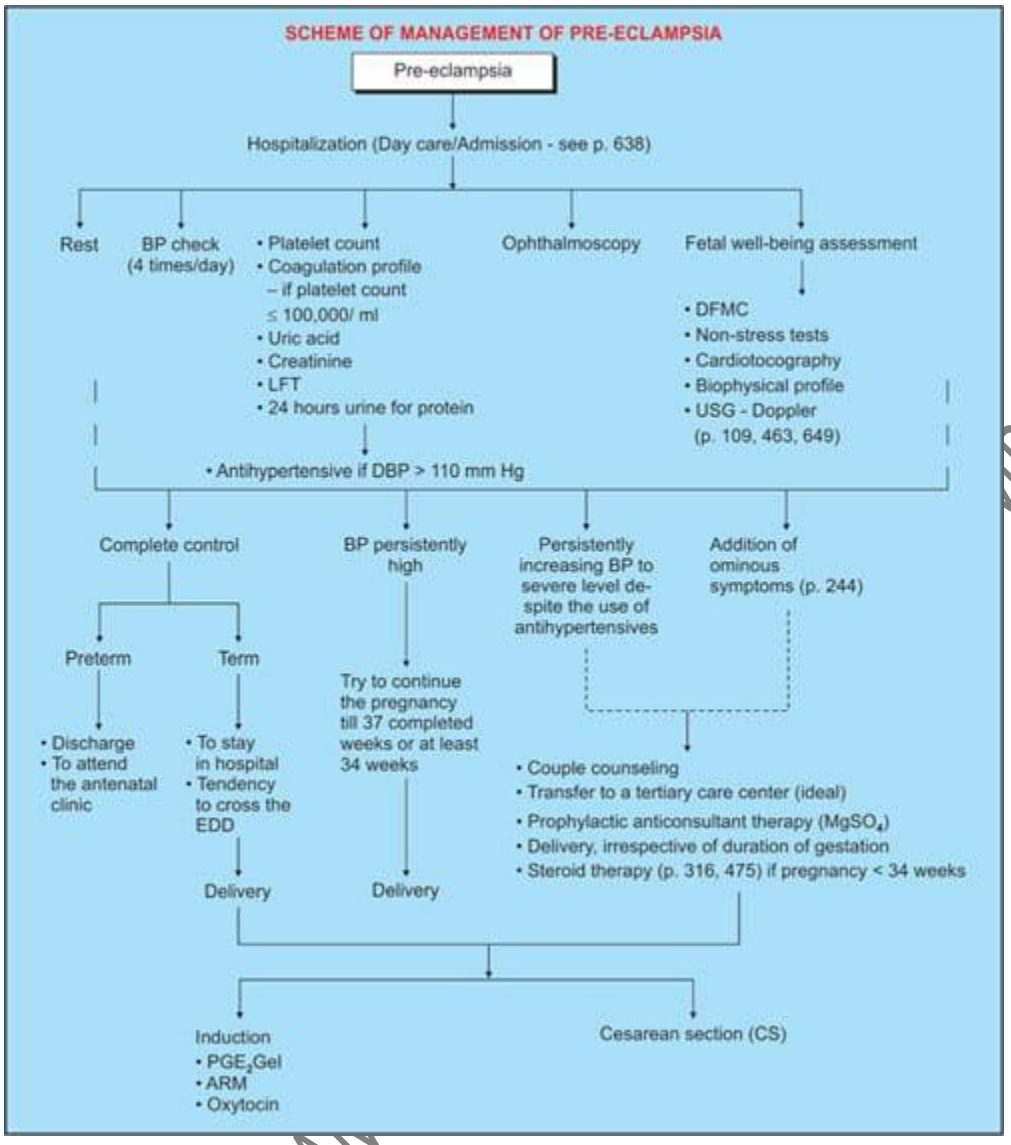
B) Labetalol use as a first line treatment of hypertension during pregnancy

C) Nitroglycerin 5 mg / min iv

D) sodium nitropruside 0.25 to 5 mg / kg / min iv

E) Magnesium sulphate to prevent seizures, it may continue for 24- 48 hours after delivery.

Scheme of management for pre eclampsia



Eclampsia (seizure)

Definition : pre eclampsia when complicated with grandmal seizures or coma is called eclampsia. The term eclampsia is derived from a Greek word meaning 'like a flesh of lightening' . It is mainly develops between 20 week of gestation to the end of first postpartum week.

Incidence: # The incidence varies widely from country to country and even between different zones of the same country

 # in developing countries it's prevalence is far and few but in developing ones, particularly in rural areas, it is still high and contributes significantly to maternal deaths

 # The hospital incidence range from 1 in 500 to 1 in 30 pregnancies.

More common in primigravida (75%) and 5 times more common in twins. It occurs between the 36th week and term in more than 50% cases.

Cause of eclampsia : cause of cerebral irritation leading to convulsions is not clear, the irritation may be proveked by ::

1) Anoxia : spasm of the cerebral vessels can increases cerebral vascular resistance and fall in cerebral O2 consumption.

2) cerebral edema may contribute to irritation

3) cerebral dysthymaia increases following anoxia and edema

4) Excessive release of excitatory neurotransmitters can cause convulsions.

Onset of seizures :Ante partum (before onset of labour) -50% , intra partum (during labour) -30%, post partum (within 48hours of dely) -20%.

Clinical manifestation

1) promontory stage : The patient becomes unconscious, there is twitching of the muscles of face, tongue and limbs. Eye balls roll or are turned to one side and become fixed. This stage lasts for about 30 seconds.

2) Tonic stage : The whole body goes into a tonic spasm. The trunk, limbs are flexed and hands clenched. Respiration ceases and the tongue protudes the teeth. Cynosis appear and eyeballs become fixed. This stage lasts for about 30seconds

3) clonic stage : All the voluntary muscles undergo alternate contraction and relaxation. The twitching start in the face then involve one side of the extremeties and ultimately the whole body. Biting of tongue occur. This stage lasts for 1-4 minutes.

4) stage of coma: following the fits, the patient passes to stage of coma. It may last for a brief period or in other deep coma persists till another convulsions.

Management immediate management

1) To prevent serious maternal injury from fall 2) prevent aspiration 3) To maintain airways 4) Ensure oxygenation.

Medical management

Magnesium sulphate therapy is the therapy of choice to control seizures. It depresses CNS and relax smooth muscles including uterus.

Diazepam therpy (40 mg in 500 ml of 5% dextrone at 30 drops /minute) for sedation and muscle relaxation.

Phenytoin sodium therapy for control of convulsions.

Administer diuretics e. g frusemide

Administer thiopentone sodium (0.5gm in 20ml 5%dextrone) slow iv in status epilepticis.

Note * if labour not present (if fits are not controlling, terminate the pregnancy with in 6-8 hrs by induction of labour or c section.)

If labour present (Artificial repture of membrane and vagibal delivery or c section).

Polyhydramnios

Definition : polyhydramnios is defined as a state where liquor amnii exceeds 2000 ml that can leads to discomfort to the patient. 1- 2 % or 1 in 1000 pregnancies suffer from polyhydramnios and is more common in Multiparae than primigravida.

Causes : A) Fetal anomalies (20%)

i) Twin to twin transfusion syndrome: A complication of monochorionic multiple pregnancies in which one fetus receives more blood flow than the other from the placenta. One twin (recipient) has polyhydramnios, Polycythemia with Circulatory overload, while the other twins (donor) has olighydrominios, anemia and IUGR.

ii) Anencephaly (in 50%cases) : Deficient development of vault of skull and brain tissues called anencephaly. Excess amniotic fluid secretion due to transudation (oozing of a fluid through pores of a membrane) from exposed meninges, absence of fetal swallowing reflex and suppression of fetal anti diuretic hormone secretion which cause exceesive urination and ultimately leads to polyhydramnios.

iii) open spina bifida : Excess amniotic fluid due to transudation from exposed meninges.

iv) Esophageal or Duodenal Atresia: it causes decrease amniotic fluid swallowing.

V) Facial cleft and neck masses : it also decreases amniotic fluid swallowing that leads to polyhydramnios.

Vi) Hydrops fetalis : The clinical condition generalized edema in infants due to Erythroblastosis fetalis.

V) Aneuploidy : A condition of having an abnormal number of chromosomes.

B) placental anomalies: chorioangioma: A vascular tumor of the chorioan.

C) Maternal causes: Diabetes: It is presumed that a raised maternal blood sugar raises fetal blood sugar that can result fetal diuresis and ultimately polyhydramnios occur. Cardiac and renal disease: may lead to edema of the placenta leading to increase in transudation.

D) multiple pregnancy: multiple pregnancy is about 10 times more common than it's overall incidence.

E) Idiopathic 50-60%

Types of polyhydramnios : There are two types of polyhydramnios acute and chronic

1) Acute polyhydramnios: (rare) it develops suddenly to a rapid increase in volume, between 20-24 week's gestation.

Chronic polyhydramnios (commenest) : Gradual in onset usually starting from 30 th week of pregnancy.

Clinical manifestation: # Dyspnea and palpitation when fluid volume exceeds 3000 ml.

Edema at legs and vulva and hemorrhoids.

Taut (markedly enlarged with tense, shiny and strike) abdomen. # Fundal height more than period of gestation t. # Fluid thrill can be elicited in all direction over the uterus. # Fetal part and fetal heart sound not well defined. # consistent malpresentation # Evidence of pre eclampsia. # abdominal girth more than normal # uterus is globular in shape.

Complications: Maternal (During pregnancy) : * pre eclampsia (25%) , malpresentation, premature rapture of membrane and preterm.

During labour: Early repture of membrane, cord prolapsed due to sudden escape of amniotic fluid, uterine inertia, increased chance of operative delivery due to malpresentation, postpartum hemorrhage due to atonic uterine.

During Puerperium : subinvolution

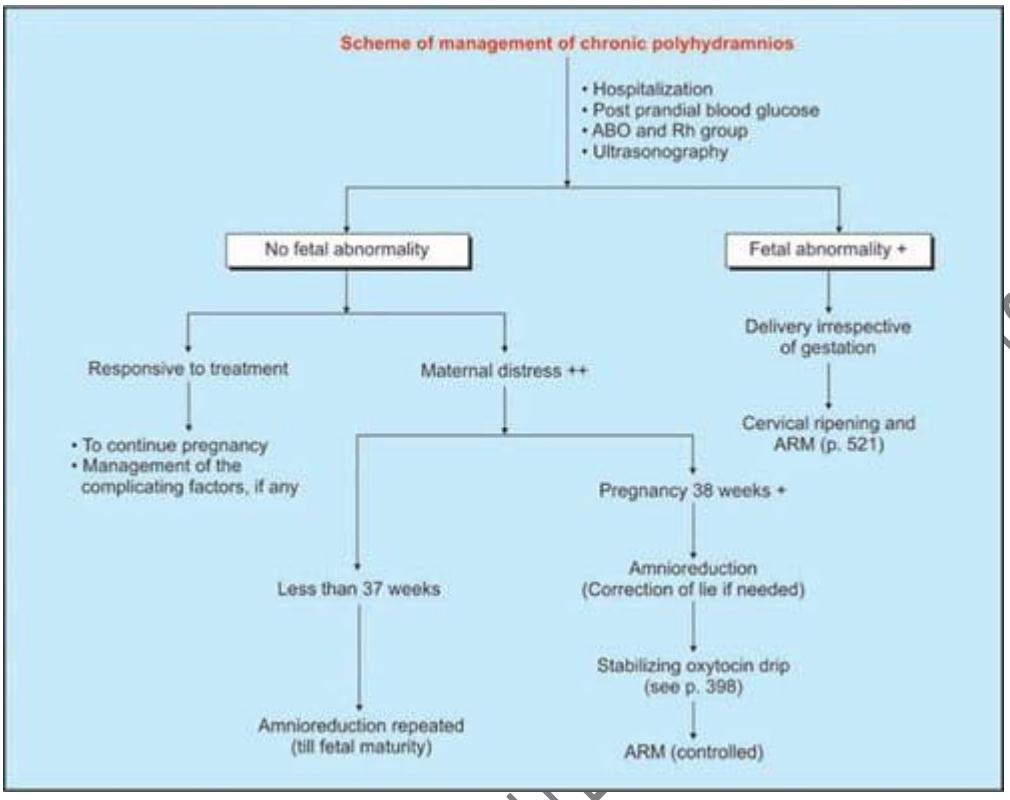
Fetal complications : May be death to prematurity and congenital abnormalities.

Investigation: ultrasound: used to detect of congenital anomalies, AFI, vertical pocket, lie, multiple pregnancy and malpresentation.

Amniotic fluid test: increase alpha fetoprotein AFP in open neural tube defect like anencephaly, spina bifida etc.

Management (as per cause) : # provide bed rest in the left lateral position to encourage placental perfusion and diureses. # Give up right position to releive dyspnea# performan amniocentesis to reduce the amniotic fluid volume in women who are experiencing severe discomfort and respiratory embarrassment. # Give orally Indomethacin (25mg) which decrease amniotic fluid by reducing fetal urine output. # Administer diuretic drugs as prescribed. # if pregnancy is more than 37 weeks, perform delivery by induction of labour through oxytocin infusion. # if congenital fetal abnormality present, terminate pregnancy.

Scheme for management of polyhydramnios



Olighydrominios

Definition: it is extremely rare condition where the liquor amnii is deficient in amount to extend of less than 200 ml. Clinically amniotic fluid index less than 5 cm (< 10th percentile) and vertical pocket less than 2 cm.

Causes : Fetal causes - 1) Fetal chromosomal or structural anomalies of kidney or lungs e. g renal agenesis, obstructed uropathy etc. 2) spontaneous repture of membrane, intrauterine infection, postmaturity, and IUGR. 3) Amnion nodosum - rounded or ovul opaque elevations in the placenta (1-6 mm in diameter) . 4) Amniotic fluid leakage.

Maternal causes: Hypertension, Uteroplacental insufficiency and dehydration.

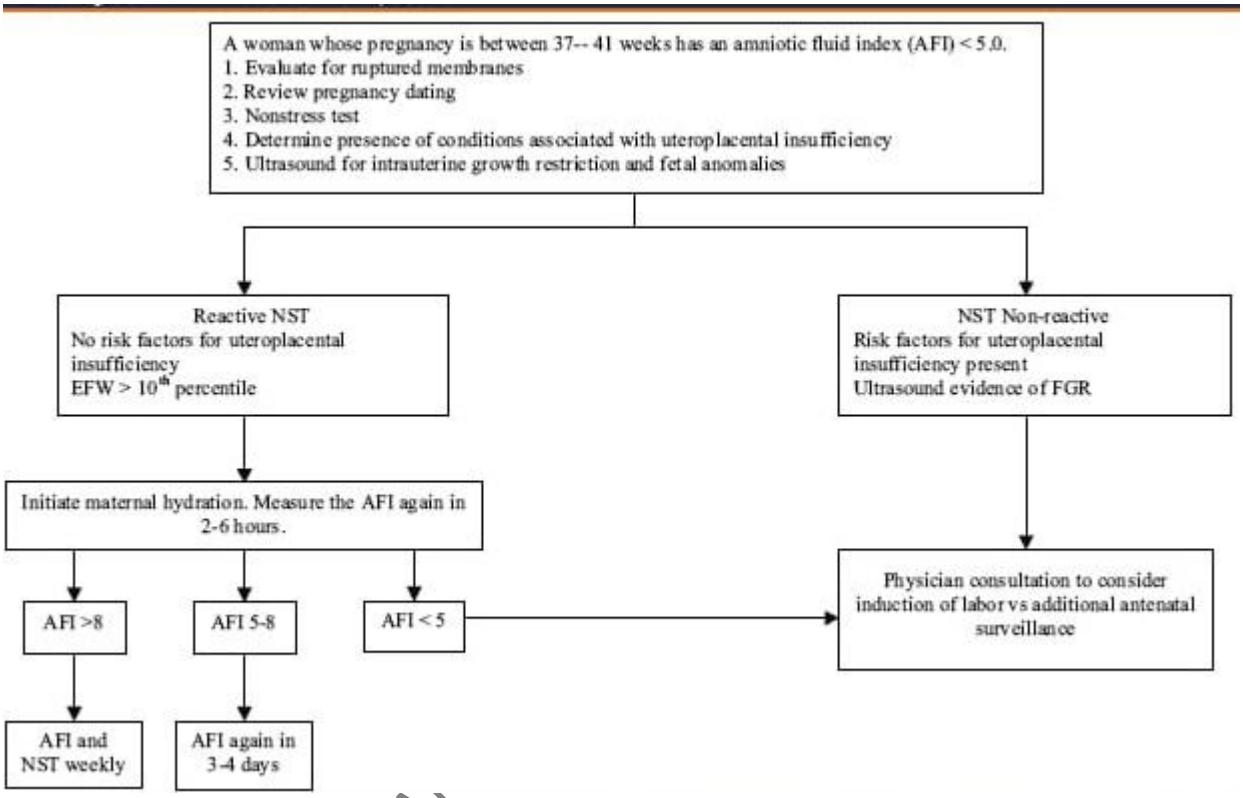
Clinical manifestation: 1) uterine size is smaller than the period of amenorrhea 2) mother feels less fetal movement 3) uterus is small and compact (feels full of fetus) 4) malpresentation (mostly breech) 5) Fetal parts easily felt.

Complications: Fetal complications: • compression malformation like baby has a squashed looking face, flattening of nose, micrognathia, and talipus. • olighydrominios associated fetal defects like renal agenesis or potter's syndrome with pulmonary hypoplasia.

Maternal complications: increased chance of operative delivery and prolonged labour.

Treatment : iv administration of fluid (fructose) and essential amniotic acid. **Amnioinfusion** The instillation of fluid usually normal saline into amniotic sac to increase the amniotic fluid volume.

Scheme for management of Oligohydramnios



IUGR (intrauterine Growth restriction or fetal growth restriction)

Definition : IUGR is said to be present in those babies whose birth weight is below the 10th percentit of average for the gastational age. in developed countries, it's incidence rate is about 2-8%, among the term babies about 5% and post term babies is about 15% .

Types : Based on clinical evaluation and ultrasound examination, the small fetuses are divided into:

1) Fetus who are small and healthy. The birth weight is less than 10th centile for these gestation age. They have normal ponderal index, normal subcutaneous fat and usually have uneventful neonatal course.

2) Fetuses whose growth is restricted by pathological process (true IUGR) , the fetuses are divided into Symmetrical and Asymmetrical IUGD.

Symmetrical (early onset 20%) : The fetus is affected from the noxious effect very early in the phase of cellular hyperplasia, the total cell no is less. This type of growth restriction is most often causes by structutal or chromosomal abnormalities or congenital infection (TORCH).

Asymmetrical (late inset 80%) : Fetus is affected by in lateral months during the phase of cellular hypertrophy. The total cell no remains the same but size is smaller than normal. The pathological processes that too often results in asymmetrical growth retardation, are maternal disease extrinsic to the fetus. These diseases alter the fetal size by reducing uteroplacental blood flow or by restricting the O2 and nutrient transfer or by reducing the fetal size.

Etiology : Maternal causes: constitutional: small women, slim, low BMI, maternal genetic and racial background are associated with small babies.

Maternal nutrition before and during pregnancy : critical substrate requirement for fetal growth such as glucose, Ami no acids and O2 are deficient during pregnancy. This is an imp causes of IUGR with undernutrition.

Toxins : like alcohol, smoking, cocaine, heroin and drugs can also cause IUGR.

Fetal causes: 1) Structural anomaly either circulatory, renal and other diaorder.

2) chromosomal abnormalities: Triploidy and Anencephly, trisomies (13,18,21 and Turner's syndrome).

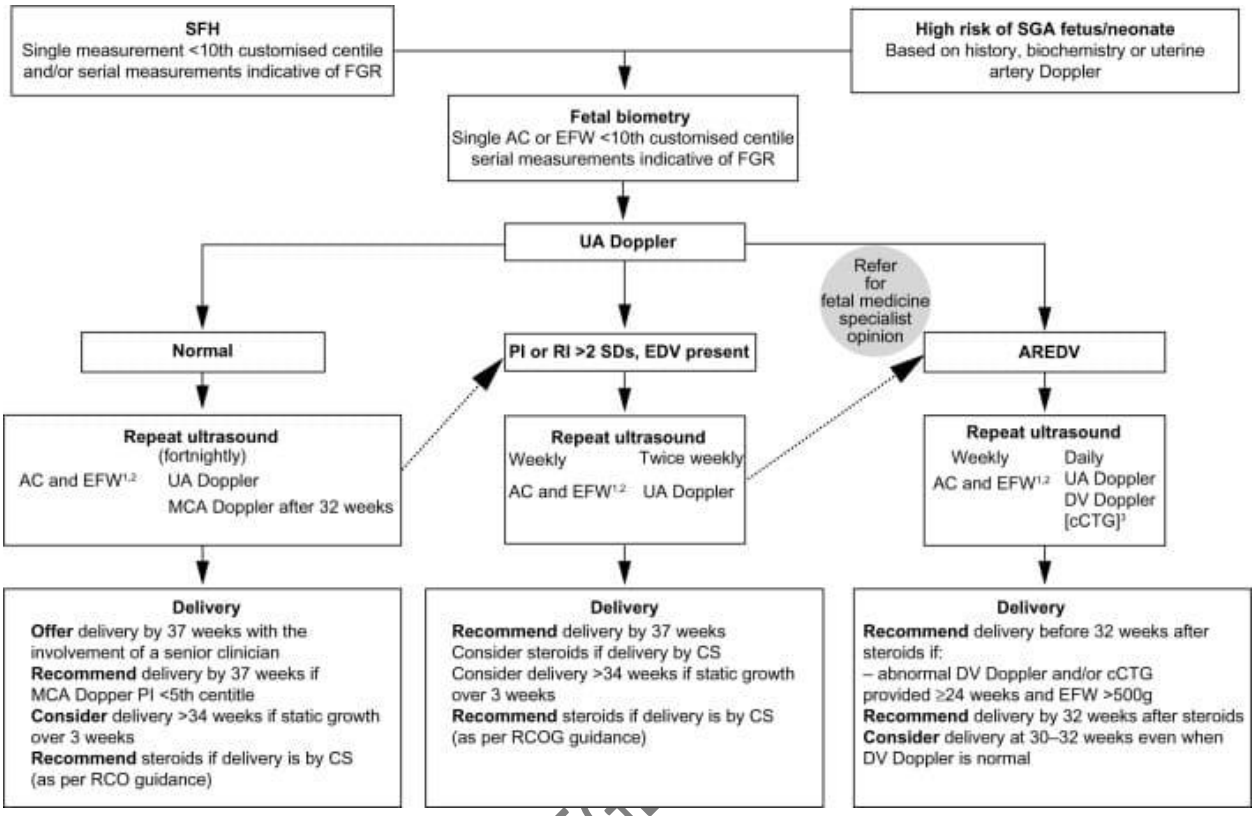
3) infection like TOTCH 4) multiple pregnancy.

Placental causes:it includes causes of poor uterine blood flow to the placental site for a long time

Signs and symptoms :1) Weight deficit at birth is about 600g below the minimal in centile standard. 2) Length is unaffected 3) Head circumference is relatively larger than the body in asymmetrical variety. 4) Dry and wrinkled skin because of less subcutaneous fat, scaphoid andomen 5) Thin meconium stained, vernix caseosa and thin umbilical cord and old man look. 6) planter creases are well defined 7) Baby is alert, active and having normal cry 8) Reflexes are normal.

Diagnosis:History collection, physical examination, clinical palpation of the uterus for fundal height and SFH symphisia pubis height.

Management for IUGR



Post maturity and post term pregnancy

Defintion: pregnancy continuing beyond 2 weeks of the expected date of delivery (>294days) is called post maturity or post term pregnancy. This occurs in an estimated 3- 12%of pregnancies.

Etiology: 1) wrong dates - due to inaccurate LMP (most common) .

2) Biological variability (hereditary) may be seen in the family.

3) Maternal factors: primiparity, previous prolonged pregnancy, sedentary habit, eldery multiparae.

4) Fetal factors : congenital anomalies like Anencephly, abnormal fetal HPA axis and adrenal hypoplasia that leads to diminished fetal cortisol response.

5) Placental factors : sulfatase deficiency that leads to low estrogen.

Clinical manifestation : Baby - General appearance 1) Baby looks thin and old

2) skin is wrinkled

3) absence of vernix casosa

4) Body and the cord are stained with greenish yellow color.

5) Head is hard without much evidence of molding.

6) nails are protruding beyond the nail beds.

7) weight often more than 3kg and length is about 54cm.

Liquor amnii: scanty and may be stained with meconium.

Placenta : This is evidence of aging of the placenta manifested by excessive infarctions and calcification.

Cord : There is diminished quantity of Wharton's jelly which may precipitate cord compression.

Diagnosis : 1) Hardness of fetal skull bones by x ray

2) Decrease amniotic fluid amoun less than 200 ml.

3) USG shows increase biparietal diameter, abdominal circumference and femur length.

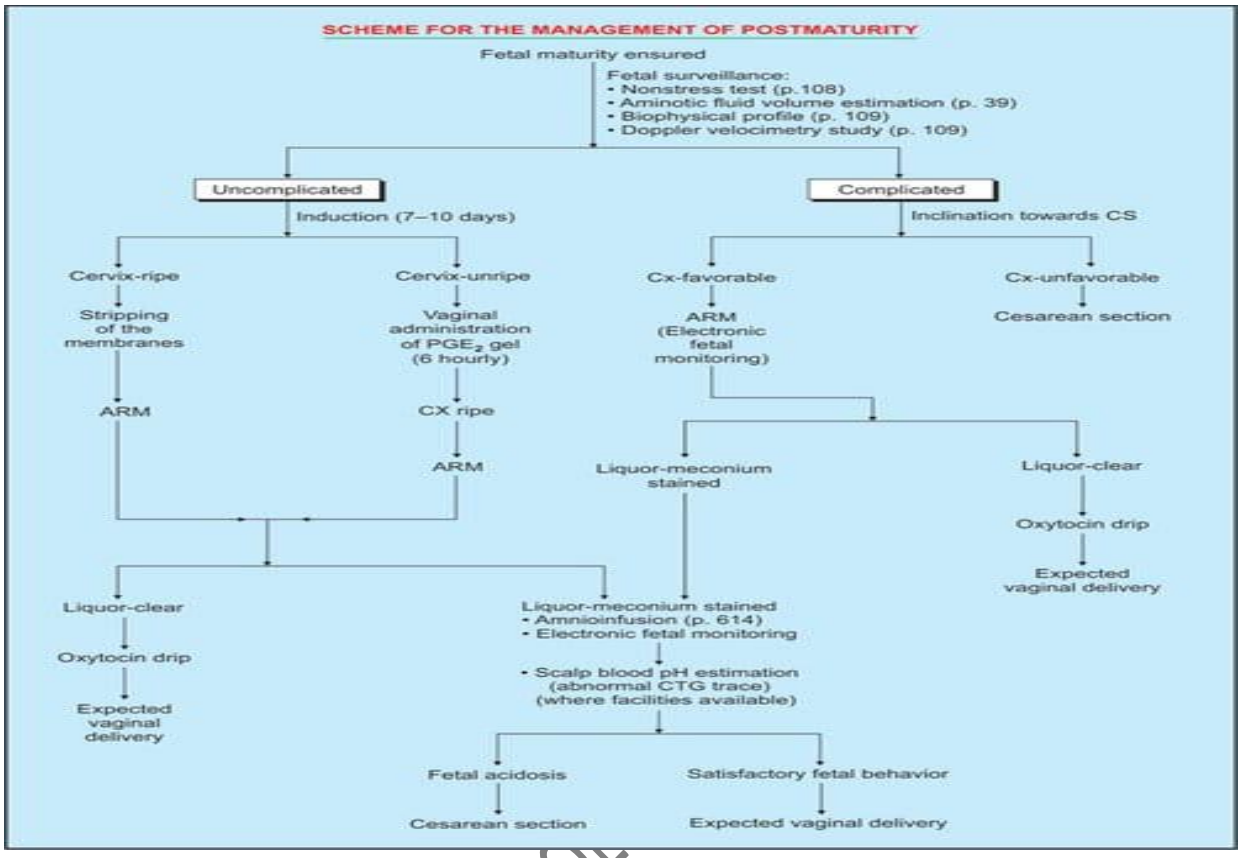
4) orange color of cells, presence of phosphatidyl glycerol.

Complications : Fetal hypoxia and acidosis, meconium aspiration, operative delivery, shoulder dystocia, macrosomia and Dysmaturity and Oligohydramnios.

Management : if uncomplicated - cervix ripening done by PGE2 gel vaginally, artificial repture of membrane and vaginal delivery by oxytocin drip.

If complicated : AROM if favorable cervix , caesarean if cervix unfavorable and meconium present in liquor.

Scheme for management of post maturity.



Intrauterine death

Definition : Death of a fetus in uterine cavity after 20 weeks of pregnancy or fetal weight more than 500 gm to before birth is termed as IUD.

Causes : causes of fetal death are complications of maternal (5-10%), placental (20-35%) or fetal (25-40%) like:::

Hypertensive disorder, severe anemia, Hyperpyrexia e. g malarial, Rh-incompatibility, maternal or fetal infection (herpes, TORCH) , APH, fetal chromosomal abnormalities and true knotting of umbilical card.

Diagnosis: Reduce fundal height, absence of fetal heart sound and fetal movement, hyperflexion of fetal spine, maternal weight loss, macerations (dissolution of dead fetus reyai ed in uterus by aseptic degenetative process), Spalding sign (overlapping of cranial bones due to liquefaction of brain matter).

Management : - spontaneous expulsion occurs within 2 weeks of death (80%) cases.

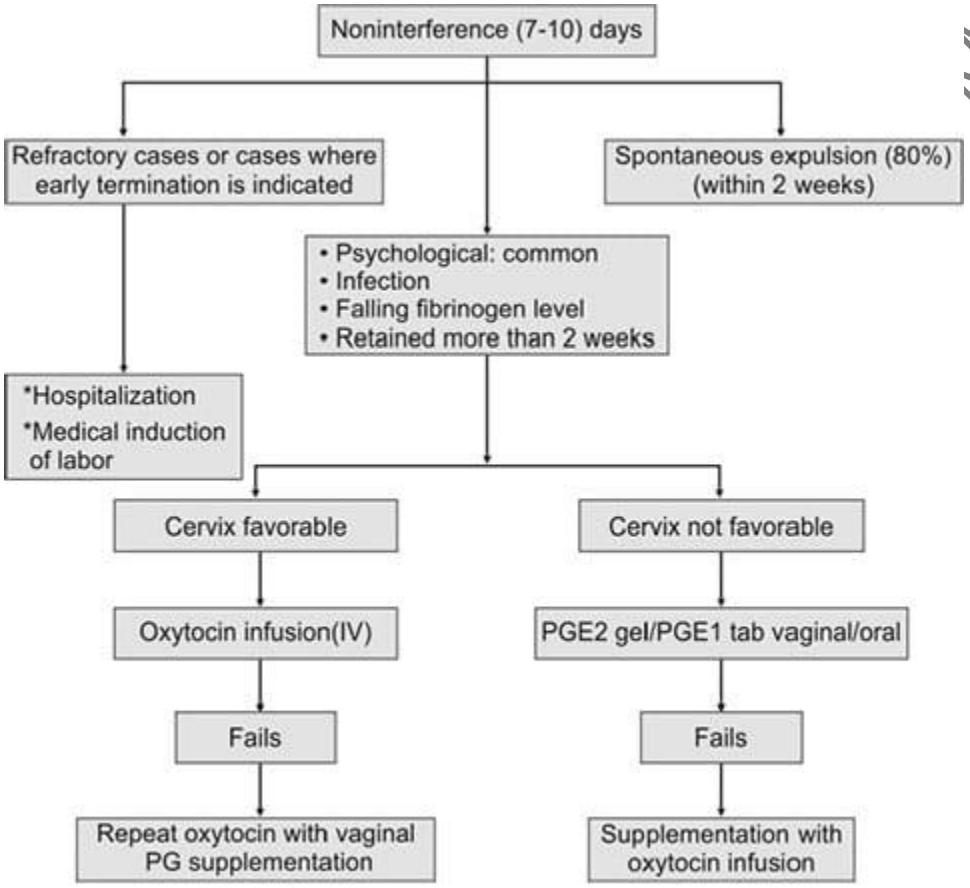
Medical induction of labour (if remains more than 2 weeks after death)

Oxytocin infusion (5-10 unit of oxytocin in 500ml RL) ifcervix is favorable.

Exogenous vaginal prostaglandin (PGE2) gel may be used to soften the cervix before induction of lanour.

Oral misoprostol (PGE1) 25-50u hm.

Scheme of management



IV, intravenous; PG, prostaglandin.

Multiple pregnancy (Twin pregnancy)

Definition : The presence of two or more embryos in the uterus is called multiple pregnancy. The incidence of natural twin pregnancy is 1:94. 20%of women, who have undergone treatment with fertility drugs, develop multiple pregnancies and it is the most common cause.

Varieties : Twins (2 fetuses) , Triplets (3 fetuses), Quadruplets (4fetuses), Quintuplets (5fetuses), sextuplets (6 fetuses).

Tyes of twins

A) Dizygotic twins or biovular twins or fraternal twins (80%) -Twins from two separate fertilized ova.

B) Monozygotic twin or identical or Uniovular twins 20%- Twins develop from single fertilized ova.

C) Siamese twin : or conjoined twins(< 1%) : A Culturally insensitive term for congenitally United twins. It occurs in case of division occur after 12 days of development of embryonic disc of single fertilized ovum.

Etiology : The cause of twinning is not known. The frequency of monozygotic twins remains constant throughout the globe and is probably related to maternal environment. Here are some of the prevelence for twin pregnancy :

1) Race : The frequency is highest among Negroes, lowest among Mongols and intermediate amongst Caucasians.

2) Hereditary : There is a hereditary predisposition likely to be more transmitted through the female (maternal side).

3) Advancing age : Advancing age of the mother increased incidence of twinning between the age of 30 -35 years.

4) Influnce of pariety : The incidence is increased with increasing parity, especially from fifth gravida onward.

5) iatrogenic : Drugs used for induction of Ovulation may produce multiple fetuses to the extent of 20-40% following gonadotropin therapy, although to a lesser extent (5-6%) following clomiphene citrate.

6) superfecundation ; Is the fertilization of two different ova released in the same cycle, by separate acts of coitus within a short period of time.

7) superfetation : is the fertilization of two ova releases in different menstrual cycles. The nidation and development of one fetus over another fetus is theoretically possible untill the decidua space is obliterated by 12 week of pregy.

Signs and symptoms : Excessive fetal activity, uterus large for gestation, palpation of 3-4 large parts in the uterus, auscultation of more than one FHR, excessive weight gain.

Complications: Twin to twin transfusion syndrome, polyhydramnios, malpresentation, cord prolapse, malpresentation and prolonged labour.

Management : 1) induction of labour by 38 weeks for dochorionic twin and between 36-37 weeks for monochorionic twins. 2) prepare for cesarean delivery for abnormal presentation. 3) Administer Oxytocin medication after delivery to prevent PPH from uterine overdistention.

Teenage pregnancy :

Definition : Teeanage pregnancy is pregnancy in human female under the age of 20years.pregnancy can take place in pubartal females known as adolescent pregnancy.

Risk factors: 1) low socio economic group2) sexual abuse 3) single parenthood 4) neglected child.

Causes : 1) curiosity and experimentation 2) peer pressure 3) family related problems 4) lack of information.

Signs and symptoms : 1) Abdominal distension 2) breast change / enlargement 3) fatigue 4) missed period 5) nausea 6) light headache 7 frequent urination.

Diagnosis : weight gain, abdominal examination (palpation - feel fundis), pelvic exay(softening of cervix, enlightenment of uterus) , USG.

Effects (Maternal effect) 1) Ane. Anemia 2) pregnancy induced hypertension 3) UTI 4) malnutrition 5) effect of STD

Fetal effect : 1) Low birth weight 2) mental retardation 3) fetal death 4 psychological disability

Social effet) 1) isolation 2) guilt 3) stress 4) social interaction loss 5) low self esteem 6) lack of support group

Treatment : all option should be available like abortion, pregnancy, family support.

* asses for smoking , alc and other drugs

* educate nutrition, * educate sleep * educate rest ., * information regarding contraception. * encourage to continue further schooling.

Prevention :1) knowledge based awareness regarding contraception and prevent STD.

2) Assistance (postponed sexual activity untill marriage)

3) peer counselling program.

Elderly primigravida :

Definition women having there first pregnancy at or above the age of 30 year's (according to FIGO - 35 years) are called elderly primigravida.

Types : There are two types of elderly primigravida :

1) women married late but conceives soon after.

2) women married early but conceives long after marriage.

Complications : During pregnancy : 1) abortions, pre eclampsia , abruptio placenta, uterine fibroids, tendency ofpostmaturity, IUGR.

During labour : 1) prelabour, 2) prolonged labour due to uterine inertia 3) impaired joint mobility 4) inelasity of soft tissues 5) maternal and fetal distress 6) increased chances of c section 7) retained placental.

Fetal complications : 1) pretermbirth 2) IUGR 3) fetal congenital malformation .

Puerperium : 1) increased morbidity due to operative interference 2) failing lactation.

Management : preconception counselling.

Grandmultipa

Definition : Grand multiparae relates to a pregnant mother who has got previous 4 or more viable births. The incidence has been gradualally declining over the couple of decades due to acceptance of small family norms

Complications : During pregnancy : abortion, placenta previa, anaemia, hemorrhoids, varicose vein hiatal hernia and malpresentation.

During labour : cord prolapse, CPD, Obstructed labour, repaired uterus, PPH, increased operative interference.

During puerperium : increased morbidity, Subinvoltion and lactation failure.

Management : these cases are considered as high risk as such they require adequate antenatal care and should have a mandatory hospital delivery.