# Polyhydramnios and Oligohydramnios

# The amniotic fluid (A,F) ;

**Is initially secreted by the amnion, then by the 10th week gestation it comes from a transudate of the fetal serum through the fetal skin& the umbilical cord ,from 16th weeks on word the fetal skin become impermeable to water &the A.F is produced mainly by the fetal kidneys as fetal urine &from lung fluid & it is removed by the fetal swallowing .A.F production is increased progressively until term at a rate of 30 ml each week ;**

**At 10 week =30 ml.**

**At 20 week 300 ml.**

**At 30 week=600 ml.**

**At 38 week 1000 ml.**

**At 40 week 800 ml & at 42 week around 350 ml .**

# FUNCTIONS OF AMNIOTIC FLUID;

**Shock absorber – protects from external trauma.**

**Protects cord from compression.**

**Permits fetal movements – development of musculoskeletal system, prevents adhesions.**

**Swallowing of AF enhances growth & development of GIT.**

**AF volume maintains AF pressure – reduces loss of lung liquid – pulmonary development.**

**Maintenance of fetal body temperature.**

**Some fetal nutrition, water supply.**

**Bacteriostatic properties – decreases potential for infection.**

# Polyhydramnios;

**Is defined as excess of amniotic fluid greater than 2000 ml ,but since quantitative evaluation of A.F is impractical the most commonly used definition is by ultrasound by using;**

**1-*amniotic fluid index (AFI)* which is =adding together the measurements of the largest pool of fluid found in each of the 4 quadrant of the uterus &**
**Polyhydramnios is defined as AFI of greater than 25cm Or by**

**2-founding an amniotic fluid pool or pocket free from limbs or cord greater than 8 cm .**

## Types of Polyhydramnios;

**1- Acute Polyhydramnios; when amniotic fluid accumulates rapidly & it occur before 24 weeks of gestation.**

**2-Chronic polyhydramnios; when amniotic fluid accumulates gradually usually diagnosed at the third trimester.**

## Causes of polyhydramnios;

**1-Maternal causes(15%);**

**a- Rh isoimmunization.**

**B-Diabetes mellitus .**

**2-Fetal causes (18);**

**a-multiple pregnancy.**

**B-fetal anomalies ;**

- ***CNS anomalies ;anencephaly, encephalocele.**
- ***GIT anomalies ; esophageal atresia ,gastroschisis.**
- ***skeletal abnormality ;osteogenesis imperfecta.**
- ***cardiac abnormality.**
- ***chromosomal abnormalities ;Down's syndrome, trisomies 13 &18.**
- **congenital infections ;syphilis ,rubella,toxoplasma**

**3-Placental causes(1%) ;placental chorioangioma .**

**4- Idiopathic causes (65%).**

## Diagnosis;

***Clinically*;**

**Pt complains of abdominal discomfort &swelling & in acute type there is difficulty in breathing (dyspnea), abdominal pain & even vomiting .**

**By examination ;Uterine size is larger than date ,it may be tense tender ( in acute cases ) & in severe cases there is oedema of the anterior abdominal wall even the vulva there is also difficulty in define the fetal parts & in detecting fetal heart & fetal presentation .**

**Mal presentation & unstable lie is common .**

***Investigations*; by ultrasound AFI more than 25 cm or founding an amniotic fluid pool or pocket free from limbs or cord greater than 8 cm .**

## Complications;

## 1- maternal;

**1- pregnancy induced hypertension .**

**2- preterm labor**

**3-premature rupture of membranes.**

**4-respiratory &abdominal discomfort .**

**5- intrapartum complications;**

***abruption.**

***cord prolapse .**

**6- malpresentation & unstable lie with increase incident of emergency C/S**

**7- Increase incidence of post partum hemorrhage .**

## 2- Fetal complications;

There is increase in mortality and morbidity due to prematurity &other complications such as abruption &cord prolapse & infection due to rupture of the membranes.

## Treatment;

**Polyhydramnios without symptoms & with no fetal abnormalities require no treatment.**

**In other cases the aim of treatment is establishing the cause and relieving maternal discomfort .**

***finding the cause ;by**

**1-U/S examination to detect fetal & placental abnormalities. lethal fetal abnormalities is treated by termination of pregnancy as in anencephaly .**

**2- maternal investigations ;**

***blood group & Rh.**

*** screening for diabetes.**

*** screening for congenital infections (TORCHS) by serelogical tests.**

*** fetal karyotype by amniocentesis .**

**Each cause is managed accordingly .**

**2- relieving maternal discomfort; by repeating decompression by amniocentesis in acute cases .In special situations indomethacin used to decrease fetal A.F production**

# Oligohydramnios;

**Is deficiency of amniotic fluid & is defined as AFI less than 5 cm 0r by measuring the deepest pool of amniotic fluid or which is less than 2 cm .**

**Diagnosis ;decrease fetal movement & abdominal size .on examination the uterus is smaller than date uterus. U/S examinations will confirm the diagnosis** .

## Causes;

1-post-term pregnancy .

2-preterm premature rupture of membranes.

3-IUGR .

4-fetal anomalies ;renal (renal agenesis &urethral obstruction ),non renal (triploidy ,congenital heart block ).

5- Leaking of amniotic fluid after amniocentesis.

6-Preclampsia and chronic hypertension.

## Complications;

***Mainly fetal;***

***1- pulmonary hypoplasia .***

***2-pressure deformities .***

***3-amniotic adhesions and bands .***

***4-intrapartum complications ;cord compression and fetal distress .***

## Management ;

**DEPENDS UPON**

- **AETIOLOGY**
- **GESTATIONAL AGE**
- **SEVERITY**
- **FETAL STATUS & WELL BEING**

**References;**

**1-DEWHUREST TEXTBOOK OF GYNAECOLOGY AND OBSTETRIC.**

**2-OBSTETRICS BY TEEN TEACHERS.**