Abstract
Amniotic fluid is the product of complex and dynamic fetal and placental physiologic processes. Disruption of the fine balance may result in overproduction or underproduction of fluid. Therefore, alterations in amniotic fluid volume serve as important markers of both in utero developmental defects as well as physiological responses to fetal hypoxemia and other metabolic disturbances such as maternal/fetal hyperglycemia. Both polyhydramnios and oligohydramnios may be associated with either major congenital anomalies or adverse perinatal outcomes. Although the ultrasonographic diagnostic criteria have yet to be firmly established, it is apparent that both subjective and objective criteria have been used successfully to identify these conditions. Polyhydramnios, particularly when severe and detected early in gestation, can be treated antenatally with serial amniocenteses. Oligohydramnios with intact membranes, especially when severe and in the absence of anomalies, is usually managed by delivery; however, further research is indicated to delineate management guidelines.

key words: Normal Amniotic Fluid Index – Pregnancies.

NORMAL AMNIOTIC FLUID INDEX
Amniotic fluid is a clear, slightly yellowish liquid that surrounds the unborn baby (fetus) during pregnancy. It is contained in the amniotic sac. While in the womb, the baby floats in the amniotic fluid. The amount of amniotic fluid is greatest at about 34 weeks into the pregnancy, when it averages 800 mL. About 600 mL of amniotic fluid surrounds the baby at full term (40 weeks gestation). The amniotic fluid constantly moves (circulates) as the baby swallows and "inhales" the fluid, and then releases it (1).

The amniotic fluid helps:
- The developing baby to move in the womb, which allows for proper bone growth
- The lungs to develop properly
- Prevents pressure on the umbilical cord
- Keep a constant temperature around the baby, protecting from heat loss
- Protect the baby from outside injury by cushioning sudden blows or movements (2).
- Amniotic fluid index more than 24 cm is called polyhydramnios. This condition can occur with multiple pregnancies (twins or triplets), congenital anomalies (problems that exist when the baby is born), or gestational diabetes (2).
- Amniotic fluid index less than 5 cm is known as oligohydramnios. This condition may occur with late pregnancies, ruptured membranes, placental dysfunction, or fetal abnormalities (2).
To estimate the volume of amniotic fluid within the uterus, the amniotic fluid index is used, which is measured against the amniotic fluid index chart (4).

**Amniotic Fluid Index Chart:**

When a woman is pregnant, she often undergoes a series of ultrasound examinations to attain a fetal biophysical profile. The amniotic fluid index (or AFI) is used to estimate the well-being of the fetus by doing an ultrasound of the uterus (5).

There are several approaches to test; the most commonly used methods are the four-quadrant technique and the 'single deepest pocket (5).

The amniotic fluid index chart shows the average volume of amniotic fluid in a pregnant woman based on gestational age in millimeter

**Table (1): Amniotic fluid index chart percentile values (4).**

<table>
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<th>50th</th>
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<td>82</td>
<td>90</td>
<td>145</td>
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<td>258</td>
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</tbody>
</table>
Techniques and Values of Amniotic Fluid Index:

**Technique**
Using the umbilicus and the linea nigra as horizontal and vertical axis, an imaginary divide is created inside the uterus, which is split into four quadrants. The deepest pocket without fetal parts or umbilical cord is then measured in millimeters or centimeters vertically. The sum of the four quadrants, in millimeters or centimeters, is amniotic fluid index. Usually, the normal value of amniotic fluid index varies from 50 mm to 250 mm (or 5 cm to 25 cm) (6).

![Fig (2): Measurement of amniotic fluid index (7).](image)

**Values**
Normal AFI varies from 80 mm to 180mm. An average AFI level is 80 mm to 140 mm when you are in your 20 weeks to 35 weeks of pregnancy. After 35 week, AFI levels usually begin to reduce (8).
The AFI < 50-60 mm is assumed as oligohydramnios, while AFI >250mm is assumed as polyhydramnios. With the pregnancy process going on, the AFI values are changing, and the percentile for fetus age is usually referred as the cutoff value (9).

**Low Amniotic Fluid Levels**
Having low levels of amniotic fluid inside the uterus during pregnancy can lead to numerous problems, which include miscarriage, premature birth, and stillbirth. There are often no symptoms experienced by the mother, except for their belly not growing in alignment with their duration of pregnancy, being smaller than it should (10).

**High Amniotic Fluid Levels**
The symptoms of polyhydramnios for this include edema, difficulty breathing, and excessive...
weight gain. Polyhydramnios can lead to many of the same complications as oligohydramnios, and the extra fluid can leak through the vagina in rare instances. Polyhydramnios can also lead to certain congenital complications, including Down’s syndrome (11).

Amniotic Fluid Abnormalities
Alterations in amniotic fluid volume, especially decreased amniotic fluid volume (oligohydramnios) have classically been considered as an indicator of poor perinatal outcome. The semiquantitative method of calculating an amniotic fluid index (AFI) by using ultrasound to measure the sum of the deepest pockets of amniotic fluid in the 4 quadrants of the maternal abdomen is the most common method of quantifying amniotic fluid volume.

Defining oligohydramnios as an AFI less than 5cm or less than 8cm (alternate definition) and polyhydramnios as greater than 25 cm (12), or greater than 18 cm alternate definition) are 2 frequently used classifications. Studies of AFI for gestational age have shown an increase in mean AFI until the early third trimester, with a decrease in mean AFI thereafter (13).

Although the risks associated with a low AFI are well established, less information is available regarding the clinical significance of a low-normal or borderline AFI. Recent studies suggest that up to 16% of patients with a low AFI (5-8 cm) will develop oligohydramnios within 4 days (13).

Recent studies, however, have challenged the relationship between amniotic fluid volume and poor prenatal outcome, especially the relationship between oligohydramnios and poor outcome near term (14).

Although little information is available on the perinatal risks associated with borderline AFI, these recent observations have lead to increased use of antepartum testing in women with a borderline AFI. Our objective was to determine the risk of adverse perinatal outcome associated with a borderline AFI, between 5 and 10 cm. This study was undertaken because it was felt that there was an alarming trend of inducing patients who were otherwise normal but only had reduced amniotic fluid volume after 37 weeks of gestation.

Polyhydramnios
Incidence and Origin
Polyhydramnios, or hydramnios, is defined as an excessive volume of amniotic fluid relative to the gestational age. Polyhydramnios may be acute or chronic. Acute polyhydramnios is usually a fulminant second-trimester process, with fluid accumulating rapidly over a period of a few days (15).

Chronic polyhydramnios has a more gradual onset and course, often presenting in the third trimester. The incidence varies, depending on whether the diagnosis is clinical or sonographic. Overall, polyhydramnios complicates approximately 0.3–1.6% of all pregnancies (16). Chronic polyhydramnios is more frequent, exceeding the incidence of acute polyhydramnios by a 50: 1 ratio.

Risk factors for polyhydramnios may be broadly divided into maternal, fetal, placental and idiopathic origins.

<p>| Table (2): Risk factors for hydramnios(16). |
|-----------------|-----------------|
| Maternal conditions | Isoimmunization |
| Placental conditions | Chorioangioma |</p>
<table>
<thead>
<tr>
<th>Fetal conditions</th>
<th>Circumvallate placenta</th>
</tr>
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<tbody>
<tr>
<td>Multiple gestations</td>
<td>Twin-to-twin transfusion syndrome</td>
</tr>
<tr>
<td>Gastrointestinal</td>
<td>Esophageal atresia, duodenal or jejunal atresia, annular pancreas, midgut volvulus, diaphragmatic hernia, omphalocele, gastroschisis</td>
</tr>
<tr>
<td>CNS lesions</td>
<td>Anencephaly, hydrocephalus, encephalocele, spina bifida, microcephaly, hydranencephaly</td>
</tr>
<tr>
<td>Skeletal malformations</td>
<td>Arthrogryposis multiplex, osteogenesis imperfecta, thanatophoric dysplasia</td>
</tr>
<tr>
<td>Fetal tumors</td>
<td>Cystic adenomatoid malformation of the lung, sacrococcygeal teratoma, cervical teratoma</td>
</tr>
<tr>
<td>Cardiac disease</td>
<td>Severe congenital heart disease, fetal arrhythmias</td>
</tr>
<tr>
<td>Genetic disorders</td>
<td>Down syndrome, trisomy 13 and 18, Pena-Shokeir syndrome, multiple congenital anomalies, myotonia dystrophica</td>
</tr>
<tr>
<td>Fetal renal and endocrine disorders</td>
<td>Vasopressin insufficiency</td>
</tr>
<tr>
<td>Hematologic disorders</td>
<td>Homozygous α-thalassemia, fetomaternal hemorrhage</td>
</tr>
<tr>
<td>Intrauterine infections</td>
<td>Rubella, syphilis, toxoplasmosis, parvovirus</td>
</tr>
<tr>
<td>Miscellaneous</td>
<td>Nonimmune hydrops fetalis, fetal retroperitoneal fibrosis</td>
</tr>
<tr>
<td>Idiopathic</td>
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</table>

Diabetes mellitus is the most common maternal factor, occurring in approximately 25% of cases (15). The exact mechanism for polyhydramnios with diabetes is unclear. It may represent fetal polyuria secondary to fetal hyperglycemia. However, van Otterlo and colleagues, measuring fetal urinary output by ultrasonography, found no increase in urine output in 12 of 13 diabetic pregnancies complicated by polyhydramnios (17).

Alternatively, fetal glycosuria may lead to an increase in amniotic fluid osmolality, resulting in water transfer from the fetal compartment to maintain osmolar equilibrium. Pedersen, however, found no association between amniotic fluid glucose concentration and volume (17).

Isoimunization is another, albeit decreasing, cause of polyhydramnios. The proposed inciting mechanism is extramedullary hematopoiesis in response to fetal anemia, which results in portal hypertension and hypoalbuminemia. The decrease in colloid oncotic pressure, as well as hydrostatic venous engorgement, leads to extravasation of fluid into the interstitium of the placenta (18).

How this extravascular fluid results in hydramnios is unclear. The extracellular fluid could possibly be transferred across the placenta and membranes into the amniotic cavity. Alternatively, the interstitial
fluid in the placenta could perhaps interfere with water transfer between the fetal and maternal compartments, resulting in fetal volume overload, polyuria, and ultimately polyhydramnios. Fetal conditions have been observed in approximately 20% of polyhydramnios cases. Fetal malformations of the central nervous system (CNS) comprise almost 50% of fetal anomalies, with anencephaly being the most common. The postulated mechanisms for polyhydramnios due to CNS malformations include centrally-mediated reduction in fetal swallowing, fetal polyuria resulting from insufficient production of vasopressin from the fetal pituitary, and transudation of fluid across the uncovered meninges. Gastrointestinal anomalies constitute the second leading structural fetal cause. Any gastrointestinal obstruction proximal to the ligament of Treitz, such as duodenal or esophageal atresia, may interfere with the effective removal of amniotic fluid by the alimentary tract (18).

Fetal circulatory disturbances account for approximately 7% of fetal anomalies responsible for hydramnios. Structural cardiac malformations and persistent fetal arrhythmias may result in right and left heart failure. Presumably, the resulting increase in venous pressure causes an elevation in hydrostatic pressure in the fetal capillaries, with transudation of fluid into the interstitial space. This mechanism would occur systemically in the fetus, leading to the characteristic appearance of non-immune hydrops (subcutaneous edema, ascites, pleural and pericardial effusions), as well as in the placenta, resulting in polyhydramnios (18).

Other circulatory disturbances can also result in polyhydramnios. In twin-to-twin transfusion syndrome, the recipient twin becomes plethoric and may develop hydramnios, either through volume overload, increased renal blood flow, and polyuria, or through a hydropic placenta. The donor twin becomes anemic, often leading to oligohydramnios and the “stuck twin” syndrome. Placental chorioangiomas and sacrococcygeal teratomas are other abnormalities in which large arteriovenous shunts may lead to high-output cardiac failure and ultimately polyhydramnios (19).

Inadequate fetal respiratory activity secondary to anomalies may prevent fluid absorption at the alveolar/capillary interface, leading to polyhydramnios. Examples include compressing tumors, such as cystic adenomatoid malformations, displaced abdominal contents, such as congenital diaphragmatic hernia, and thoracic wall abnormalities, such as thanatophoric dysplasia.

Polyhydramnios not associated with an identifiable cause is labeled “idiopathic” and accounts for 30–60% of cases. Further research is necessary to identify other as yet undetermined causes. One such possibility is a disorder of intra-amniotic prolactin regulation by the chorion and decidua. Under normal circumstances, prolactin may be partially responsible for control of water homeostasis in the intra-amniotic environment. In vitro studies on human amnion have shown reduced diffusion of water in response to ovine prolactin administered on the fetal side of the membrane. Hence, an overproduction of decidual prolactin may impair diffusional flow of water away from the amniotic compartment, leading to polyhydramnios (20).

**Clinical Presentation**

The maternal signs and symptoms of polyhydramnios are usually caused by the over-distended uterus and its compressing effect on intra-thoracic and intra-abdominal organs. Elevation of the diaphragm can result in dyspnea and occasionally respiratory distress. Back and abdominal discomfort are also frequent complaints, as are nausea and vomiting. Edema of the lower extremities may result from
compression of the inferior vena cava (15).

**Diagnosis of Polyhydramnios**

The diagnosis of polyhydramnios had formerly been a clinical one, retrospectively based on the presence of more than 2000 ml of amniotic fluid at the time of delivery or membrane rupture. Antenatal suspicion was raised by difficulty in palpating fetal parts, distant fetal heart sounds by unamplified auscultation, a tense uterine wall, and disproportionate growth of the fundal height. Historically, amniography was used to qualitatively assess amniotic fluid volume. This method was subsequently supplanted by static ultrasonographic imaging, which was used to calculate total intrauterine volume (TIUV). However, inaccuracies in measurement as well as the advent of real-time ultrasonography led to the abandonment of TIUV. Real-time ultrasonography is now the primary means of amniotic fluid volume assessment; however, strict ultrasonographic criteria have never been uniformly adopted. Chamberlain and colleagues arbitrarily defined polyhydramnios as a fluid pocket of at least 8 cm in vertical and transverse diameters (21).

Using this criterion, the incidence of polyhydramnios in a select high-risk referral population was 3.2%. Those patients with polyhydramnios had a higher incidence of major congenital anomalies (4%), macrosomia (33%), and perinatal mortality (3.3%) compared to a control group with normal amniotic fluid volume. More recently, the amniotic fluid index (AFI) has replaced the largest vertical pocket in many ultrasound units. An AFI of greater than 20 cm was arbitrarily defined as excessive amniotic fluid volume (22).

**Perinatal Complications**

The increased perinatal morbidity and mortality associated with polyhydramnios are due to both an increase in congenital/genetic anomalies and preterm births. Perinatal mortality used to approach 100% with acute polyhydramnios; however, with aggressive repetitive amniocentesis, survivors have been reported.

Chronic polyhydramnios tends to have a better prognosis, especially if idiopathic in origin. Perinatal mortality has ranged from 34% to 69% in older studies. However, Chamberlain and colleagues quoted a 3.3% mortality when the diagnosis was made sonographically. Some of the variation in survival may be a function of diagnostic criteria differences and prenatal therapy, as well as improved survival of both preterm and anomalous infants (23).

Polyhydramnios may be complicated by preterm labor in up to 26% and premature rupture of membranes in up to 19% of cases. Both may occur as a result of overdistention of the uterus. Malpresentations are also encountered more frequently, as a result of both the abundance of amniotic fluid in which the fetus may maneuver and the earlier gestational age at the time of delivery. Other intrapartum complications may include placental abruption due to rapid decompression of the uterus at the time of rupture of membranes, dysfunctional labor patterns, and postpartum hemorrhage as a result of uterine atony (16).

**Clinical Management**

Treatment of polyhydramnios may be medical or surgical or both. The method chosen will depend on the etiology, severity, clinical symptoms, and gestational age at diagnosis, as well as the presence and type of associated anomalies.

If the diagnosis is made on the basis of ultrasonographic findings, an attempt should be made to
establish the cause. In cases that are not acute or severe and are not associated with a fetal malformation, patients should be rescanned periodically to assess the progression or improvement of the fluid volume. Some reports have documented gradual resolution of polyhydramnios, either spontaneously or as a result of treating the underlying cause (e.g., control of hyperglycemia, intrauterine transfusion of the anemic fetus). These pregnancies progressed uneventfully after resolution of the polyhydramnios, with no adverse sequelae observed (24).

In the absence of rapidly progressive polyhydramnios or maternal symptoms, management is expectant. If a patient experiences increasing dyspnea, back pain, or preterm labor, hospitalization for possible tocolysis and amniocentesis should be considered. Medical management, including salt restriction, diuretics, and intra-amniotic vasopressin has not proved beneficial. Indomethacin has been suggested as a therapeutic modality to reduce the amniotic fluid volume, because it has been observed to decrease urinary output in neonates being treated for patent ductus arteriosus. A reduction in amniotic fluid has been observed in one series of eight patients with hydramnios treated with indomethacin, as documented by decreasing fundal height measurements and largest vertical fluid pocket by ultrasonography. This observation further confirms the important contribution of fetal urination in overall amniotic fluid dynamics. Although case reports and early studies suggested the therapeutic benefit of indomethacin in the treatment of polyhydramnios, it is not typically used in the third trimester, due to its recognized effects of in-utero narrowing of the fetal ductus arteriosus, which can result in pulmonary hypertension postnatally (25). Therapeutic amniocentesis, or amnioreduction, is an effective modality for acute decompression of the tense and distended uterine cavity. It is typically performed for relief of maternal symptoms or preterm labor. It should be performed under ultrasonic guidance to avoid fetal contact, using a long 20-gauge amniocentesis needle which is often connected via plastic tubing to a suction bottle. Amnioreduction is usually accomplished over 30–45 minutes, although no ideal time period for drainage has been established. During this time, uterine contractions may occur, which can be uncomfortable for the patient. Typically, these contractions will abate spontaneously within 24 hours after the procedure has been completed. The quantity of amniotic fluid that removed has also not been established and may be dependent on gestational age, severity, and rapidity of reaccumulation. Volumes aspirated in various reports have ranged from 200 to 4000 ml. There has been concern that too rapid or too extensive a decompression could result in placental separation. Amniocentesis may need to be repeated initially 2–3 times in the first week, followed by weekly amnioreduction or as clinically indicated. Periodic evaluation of maternal electrolytes and serum protein may need to be assessed if frequent amniocenteses are required although no studies have demonstrated the efficacy of such surveillance (18).

**Perinatal outcomes of Pregnancies with Borderline versus Normal Amniotic Fluid Index**

Many studies have been done to show the association of a borderline amniotic fluid index with some adverse perinatal outcomes and, in most findings, the occurrence of maternal and fetal complications was reported more often in pregnancies with borderline AFI than in those with normal AFI. However, there were no specific perinatal cares or other care protocols for these patients and that could be because of different reasons such as the variations in the study designs, the likelihood of a borderline index varied from 6-44% and 25-35% and the absence of receiver-operating characteristic curve to determine the thresholds of adverse outcomes, and therefore, more research will be required to find out the effect of AFI on adverse pregnancy outcome (26).
Amniotic fluid cavity filled with liquor amnii is a natural floating bed for fetus required for its existence and growth in sterile environment, regulation of temperature, a voidance of external injury and reduction of impact of uterine contractions. Amniotic fluid has high influence on the fetal outcome and is directly related to the perinatal morbidity and mortality. This study aims to compare the pregnancy outcomes in patients with borderline Amniotic Fluid Index (AFI) between 5-10 cm and normal AFI = 10-24 cm (27).

Nature has made floating bed in a form of amniotic fluid cavity filled with liquor amnii for the requirement of fetus, for its existence and growth in sterile environment, regulation of temperature, avoidance of external injury and reduction of impact of uterine contractions. It is necessary for baby's proper growth and development. It cushions the fetus from physical trauma, permits fetal lung growth and provides barrier against infections (28).

This study aims to compare the pregnancy outcomes in patients with borderline AFI between 5-<10 cm and normal AFI >/= 10-24 cm in terms of maternal and fetal complications so that appropriate intervention if needed can be taken at appropriate time (27).

Inclusion criteria were pregnant women at more than 37 weeks gestation, singleton pregnancy, those with intact membranes and AFI between 5 and 24 cm. Exclusion criteria were women with history of congenital uterine anomalies, history of genital tract malignancies. For all the selected cases, thorough history was taken, and complete examination was done. The previous obstetric records and ultrasound reports were reviewed. Period of gestation was calculated by last menstrual period, those who didn’t remember last menstrual period or having irregular cycle period of gestation was calculated by the patient’s early scan (29).

Ultrasonography (USG) was taken as the medium of assessing AFI. USG was done at the Department of Radiology, IOM-TUTH by an experienced consultant radiologist. Amniotic fluid index was measured using technique as described by Phelan, Patient was kept in supine position; a linear, curvilinear or sector transducer was used, uterus was divided into 4 quadrants using the maternal sagittal midline vertically and an arbitrary transverse line approximately halfway between symphysis pubis and upper edge of uterine fundus. Transducer was kept parallel to the maternal sagittal plane and perpendicular to the maternal coronal plane throughout. The deepest unobstructed and clear pocket of amniotic fluid was visualized and the image frozen. Ultrasound calipers were manipulated to measure the pocket in a strictly vertical direction. The process was repeated in each four quadrants and pocket measurement summed, which gives AFI. If AFI is less than 9, four quadrant evaluation needs to be performed three times and average the values. Grading of AFI was done as described by Phelan et al. (22), AFI between 5.1-9 was termed borderline oligohydramnios, AFI between 10-24 was termed normal (30).

After amniotic fluid index (AFI) assessment, they were categorized into two groups: those with AFI 5-9 cm (Group 1) and with AFI 10-24 cm (Group 2) accordingly. Pregnancy outcomes was noted in terms of mode of delivery, amniotic fluid colour & fetal birth weight (31).

Considering induction of labour, in this study, Group 1 had higher rate of induction of labour when compared with Group 2. When mode of termination was looked upon, in Group 1 Cesarean section rate was 51.8% compared to 26% in case of Group 2. There were various reasons associated with it. Cases with AFI of 5 cm or less were directly taken for Cesarean section assuming increasing intrapartum
complications if induction was conducted (32).

Thus, overall Cesarean section rate was higher in Group 1. Most common indication for Cesarean sections were fetal distress followed by failed induction. Coming to instrumental delivery, vacuum was the only used instrument and both the groups had equal number of instrumental deliveries. All these results were comparable to several studies. Found that intrapartum oligohydramnios was associated with an increased risk of Cesarean section, indication mostly being fetal distress (32).

References


