Review Article

Too Much of a Good Thing: Updated Current Management and Perinatal Outcomes of Polyhydramnios

CME Credits

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Abstract

Amniotic fluid assessment is crucial in prenatal ultrasound to monitor fetal conditions, with polyhydramnios, characterized by excessive amniotic fluid, affecting 1%–2% of pregnancies. Polyhydramnios is linked to complications such as placental abruption, preterm labor, congenital anomalies, and postpartum hemorrhage, emphasizing the need for early detection and management. While idiopathic causes account for 60%–70% of cases, other causes include impaired fetal swallowing and increased urine production due to maternal, fetal, and placental conditions. Accurate amniotic fluid volume (AFV) assessment and surveying the underlying cause are important, with ultrasound methods such as deep vertical pocket (DVP) and amniotic fluid index (AFI) preferred. Polyhydramnios is defined by an AFV exceeding 2000 ml, an AFI over 24 cm, or DVP more than 8 cm. Management typically targets underlying causes, with treatments such as amnioreduction and indomethacin for severe cases. Antepartum monitoring includes detailed fetal ultrasound, genomic and genetic examinations, and tests for maternal diabetes and infections. Intrapartum management addresses complications such as malpresentation and shoulder dystocia, whereas postpartum care involves monitoring for uterine atony and hemorrhage. Perinatal outcomes in idiopathic polyhydramnios are generally poorer, with increased risks of fetal demise, preterm delivery, and neonatal complications, but these results may need further stratification and verification.

Keywords: Amniotic fluid, hydramnios, polyhydramnios

Introduction

Amniotic fluid is an important parameter for monitoring fetal conditions during prenatal ultrasound. Excessive fluid accumulation in the amniotic sac during pregnancy is typically defined as polyhydramnios or hydramnios.[1] The prevalence of polyhydramnios or hydramnios is approximately 1%–2% of pregnancies.[2] Polyhydramnios is associated with multiple perinatal complications, including placental abruption, preterm labor, preterm prelabor rupture of membrane, congenital anomalies, cord prolapse, abnormal fetal presentation, and postpartum hemorrhage.[3] Therefore, it is essential for physicians to detect polyhydramnios and arrange further diagnostic workup.

Various causes contribute to polyhydramnios, with idiopathic origins accounting for approximately 60%–70% of cases.[4] Other mechanisms, such as impaired fetal swallowing or overproduction of urine output, are documented in the literature.[4] This review aims to explore the diagnosis of polyhydramnios, associated underlying diseases, future updates, perinatal outcomes, and management strategies for antenatal counseling.

Physiology of Amniotic Fluid

Amniotic fluid provides a crucial environment for fetal development, facilitating the growth of extremities and lungs, acting as a bacteriostatic area, and serving as a buffer against trauma.[5] In addition, It protects against fetal umbilical cord

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compression.[6] In early gestation, amniotic fluid is considered a transudate of plasma derived from the mother's side through the uterine decidua and placenta surface. Later, the amniotic fluid originated from the fetal urine production, lung secretions, and fetal oral–nasal secretions. The circulation of this fluid is completed after fetal swallowing, transmembranous, and intramembranous flow.[7]

Assessment of Amniotic Fluid

Quantitative measurement of actual amniotic fluid volume (AFV) can be determined through dye dilution techniques or at the time of cesarean section.[8] However, these methods are impractical due to their invasive nature, time consumption, and inability to detect AFV before delivery if measuring the AFV during cesarean section.[9] Sonographic measurement is more approachable for clinicians. Although experienced physicians can qualitatively estimate AFV, semiquantitative methods are usually preferred due to reproducibility, ease of communication, and ability to follow-up.[10]

The AFV measurement by ultrasound can be divided into several methods: deep vertical pocket (DVP), amniotic fluid index (AFI), and two-diameter pocket.[11-14] These measurements require the probe to be perpendicular to the floor and parallel to the long axis of the maternal body.[4] In addition, the width of the pocket must be more than 1 cm without an umbilical cord or fetal parts inside the pocket.[15]

The DVP is the single largest pocket of amniotic fluid. The AFI is determined by summing the DVP of 4 quadrants divided by the umbilicus into upper and lower quadrants and by the linea nigra into right and left halves.[16] The two-diameter pocket method, defined as vertical multiplied by horizontal of the largest vertical pocket, is seldom used nowadays.[13] Although color Doppler has been employed to identify the umbilical cord in the pocket, it often leads to the overdiagnosis of oligohydramnios and is not recommended for routine use.[17]

Normal amniotic fluid levels usually range between 500 ml and 2000 ml.[12] However, the correlation between DVP or AFI to the actual amniotic fluid amount is very poor.[15] Definitions of polyhydramnios included a total AFV of more than 2000 ml,[18] amniotic fluid level >95th or 97th percentile[19,20] an AFI >24 cm or 25 cm[19,20] a DVP >8 cm,[21] or increased amniotic fluid level detected subjectively.

For twin pregnancies, the evaluation of AFV is extremely important as a clinical indicator for twin-twin transfusion syndrome (TTTS).[9] Although qualitative and semiquantitative sonographic techniques tend to underestimate the abnormality of AFV compared to dye-dilution technique, these sonographic techniques are still more widely used in clinical practice.[9] The standard procedure is similar to that used in singleton with a 4–8 MHz probe.[22] DVP is measured in both gestational sacs, excluding the umbilical cord or fetal limbs.[22] The criteria for oligohydramnios and polyhydramnios are the same as for singleton with cutoffs of 2 cm and 8 cm, respectively. This is reasonable because the 2.5th percentile and 97.5th percentile of maximum for twins are about 2.2 cm and 7.5 cm, respectively.[15,23]

Using artificial intelligence (AI) for measuring amniotic fluid holds promise for the future. However, current limitations included difficulties in differentiating between actual AF and reflected waves, nonautomated labeling of images, and ignoring uncertain aspects such as the angle and direction of the transducer.[24] Thus, it is still a long way from being applied to fetal health in clinical practice.

Idiopathic Polyhydramnios

About 40%–70% of polyhydramnios cases can be classified as idiopathic prenatally.[4,25] However, about 10% of these features are found to have anomalies after birth, primarily gastrointestinal atresia.[25] It is crucial to clarify that although idiopathic polyhydramnios is the most common cause of polyhydramnios, this diagnosis should only be made after excluding other underlying diseases. Monitoring sequential fetal biometry is recommended due to the potential for developing fetal growth restriction and macrosomia.[4] Those conditions also provide clues of high-risk fetal abnormalities such as trisomy 18 and 13 or genetic disorders like Costello syndrome.[26]

Maternal Condition-related Polyhydramnios

Maternal diabetes stands as the primary cause of maternal-related polyhydramnios. Other factors included alloimmunization and fetal–maternal hemorrhage.

Diabetes-related polyhydramnios

The hypothesis behind the diabetes-related polyhydramnios is because the maternal hyperglycemia leads to fetal hyperglycemia, triggering osmotic diuresis, and resulting in polyhydramnios. Evidence supports this hypothesis as AFI correlated with amniotic fluid glucose levels, making AFI a potential marker for glycemic control.[27]

Maternal alloimmunization (isoimmunization)

Maternal alloimmunization, also known as isoimmunization, typically arises from maternal exposure to alloantigens such as foreign erythrocyte surface antigens, which stimulate immunoglobulins production.[28] The consequence can evolve into hemolytic disease of the fetus and neonate (HDFN) and fetal hydrops once the immunoglobulins cross the placenta. RhD alloimmunization is the most common cause, though the prevalence of non-RhD alloimmunization is also increasing.[28] In Eastern populations, the prevalence of anti-RhD alloantibodies tends to be lower, but the severity of HDFN is higher.[29]

Among 23886 full-term babies, there were 15 cases of HDFN, including 6 cases of anti-RhE (40%), 3 cases of anti-RhE combined with anti-RhC (20%), 3 cases of anti-RhD (20%),

2 cases of anti-Mi (13.3%), and 1 case of anti-RhC (6.6%).[29] Polyhydramnios was identified as an isolated finding, possibly due to increased fetal cardiac output in anemic fetuses with maternal anti-C alloimmunization.[30] Fetal anemia enhances extramedullary hematopoiesis, leading to fetal hepatosplenomegaly, liver congestion, and impaired synthetic function. Consequently, the accumulation of extracellular fluid manifests as effusions, ascites, edema, and polyhydramnios.[31]

Fetal Condition-related Polyhydramnios

Polyhydramnios is associated with various fetal conditions. These include fetal structural abnormalities, macrosomia, fetal congenital infections (such as toxoplasmosis, syphilis, parvovirus B19 infection, rubella, cytomegalovirus (CMV), and herpes simplex virus), fetal genomic/genetic disorders, and multiple gestations.[32]

Reasons for impaired amniotic fluid swallowing

Many fetal structural anomalies that can impede amniotic fluid swallowing include central nervous system abnormalities, cleft palate, micrognathia, lesions with compression of esophagus and trachea (neck, mediastinal, lung masses, diaphragmatic hernia, and congenital high airway obstruction), gastrointestinal tract obstruction, and neurological or muscular disorders.[4]

Reasons for increased urine output

Macrosomic fetuses often exhibit increased urine output, which may physiologically lead to mild polyhydramnios.[4] Other causes of increased urinary outputs included (1) high-output cardiac states, (2) renal abnormalities, and (3) osmotic fetal diuresis. Certain fetal abnormalities can result in high-output cardiac states or heart failure, often overlapping with non-immune hydrofetalis. A particular condition known as "paradoxical polyhydramnios" is due to the ureteropelvic junction. The etiology is possibly related to impaired renal concentrating abilities.[4]

Fetal congenital infections

The prevalence of toxoplasma, rubella, CMV, herpes simplex, and other infections (TORCH) are lower in polyhydramnios compared to oligohydramnios.[32] Studies have reported infection rates ranging from 0.3% to 2.9% in polyhydramnios, whereas 25% of cases with oligohydramnios were confirmed to have CMV infection.[32,33] Parvovirus B19 infection, however, may lead to high output cardiac states and increased urine output,[34] prompting ongoing debate regarding routine screening for TORCH and parvovirus B19 serology among pregnant women.

Genomic and genetic conditions

Polyhydramnios has been associated with genetic syndromes and abnormal karyotypes such as Down syndrome.[35] A previous study disclosed chromosome abnormality or pathogenic copy number variants (CNVs) in 3.1% of patients with isolated polyhydramnios.[36] A more recent study survey of 600 patients with either isolated polyhydramnios or nonpolyhydramnios reported an overall prevalence of chromosomal aberrations at 5.8%.[37] Specifically, for isolated polyhydramnios, pathologic findings were noted in about 2.3% of cases (1.5% aneuploidy and 0.8% pathogenic CNVs) compared to 8.6% for nonisolated polyhydramnios (5.0% aneuploidy and 2.4% pathogenic CNVs).[37] Stratifying patients by polyhydramnios severity, the incidence of aneuploidy and pathogenic CNVs was 5.4%, 8.9%, and 10% for mild, moderate, and severe cases, respectively.[37] Although the latter study indicates that isolated polyhydramnios does not inherently increase the risk of chromosome abnormalities compared to the baseline population, caution is warranted due to inconsistent study findings and the limitations of prenatal ultrasound. In cases of nonisolated polyhydramnios associated with other fetal anomalies and a normal karyotype without pathologic CNVs, genetic testing such as gene sequencing may be considered for diagnosing genetic disorders such as Noonan syndrome, Bartter syndrome, and Greig cephalopolysyndactyly syndrome.[1,38] A study on postnatal follow-up of neonates prenatally diagnosed with polyhydramnios noted chromosomal or genetic disorders in 13% of cases, particularly those with severe polyhydramnios and reduced fetal movements.[38]

Placental-Related Polyhydramnios

Placental chorioangioma is the most common nontrophoblastic tumor of the placenta.[39,40] In cases of large placental chorioangioma (≥2.2 cm in diameter), the incidence of polyhydramnios is about 16.1%.[41] This may be the result of excessive transudation of fluid from the surface of the large.[42]

Multiple gestations, particularly monochorionic twin gestation, are known as high-risk pregnancies with numerous complications. In such cases, one twin may present with polyhydramnios, whereas the other experiences oligohydramnios, a manifestation of TTTS.[43] TTTS can also be regarded as a placental cause of polyhydramnios. The pathophysiology of this condition involves shared placental vascular anastomoses, leading to hypervolemia in the recipient twin, resulting in polyuria and polyhydramnios.[44]

Treatment and Further Management of Polyhydramnios

Treatments are usually based on the underlying cause and the severity of polyhydramnios. Symptomatic treatments include amnioreduction and the use of indomethacin.

The use of amnioreduction or expectant management for moderate-to-severe polyhydramnios is controversial. Amnioreduction is often reserved for pregnant women experiencing respiratory complaints and restrictive diaphragmatic movement related to polyhydramnios.[4,45] A retrospective study of 218 singleton pregnancies with 110 patients receiving amnioreduction, revealed that the rates of spontaneous preterm delivery were similar between groups. However, there was a higher percentage of vaginal delivery

in women who underwent amnioreduction compared with those managed by expectant management. (49.4% vs. 30.5% $P = 0.01$) and a lower rate of uterine atony (2.4% vs. 13.7%, $P = 0.006$).[46]

Indomethacin is used for the treatment of preterm labor or idiopathic polyhydramnios.[47] This prostaglandin synthetase inhibitor stimulates the fetal secretion of arginine vasopressin and reduces fetal renal blood flow, resulting in decreased urine output.[45] However, the usage of indomethacin for preterm labor has been associated with several perinatal complications such as intraventricular hemorrhage, periventricular leukomalacia, and necrotizing enterocolitis in meta-analyses.[48,49] Therefore, the Society of Maternal-Fetal Medicine does not recommend indomethacin therapy solely for the purpose of decreasing amniotic fluid in cases of polyhydramnios.[4]

Antepartum management

Surveying the underlying disease is paramount in the antepartum management of polyhydramnios. Detailed fetal ultrasound should be conducted to detect any structure abnormalities. The focus of sonography includes oral, esophageal, tracheal, or intestinal deformities; central nervous system; cardiac septal defects; pulmonary masses; diaphragmatic hernia; fetal stomach anomalies; fetal kidneys issues such as ureteropelvic junction obstruction; evidence of sacrococcygeal teratoma over the lower spine and pelvis; signs of fetal anemia (e.g., middle cerebral artery peak systolic velocity measurement exceeding 1.5 multiples of median); fetal hydrops; fetal movement; placental masses; and characteristics of trisomies.[4,45,50] Fetal biometry should be monitored before delivery due to the high potential for macrosomia. Conversely, idiopathic polyhydramnios with growth restriction should raise suspicion of chromosomal abnormalities such as trisomy 13 or 18.[4]

Laboratory tests are essential to diagnose gestational diabetes or pregestational diabetes. Maternal blood type and antibody screening should be conducted to check for alloimmunization, with further rosettes test diagnosing maternal–fetal hemorrhage by fetal D+cells in maternal Rh-negative cells. Congenital Infections such as syphilis, parvovirus B19, toxoplasma, rubella, CMV, and herpes simplex may be considered, especially with additional sonographic findings such as nonimmune hydrops, hepatomegaly, splenomegaly, or placentomegaly.[4,45]

There are no data supporting the benefit of diagnostic amniocentesis for isolated polyhydramnios.[51] However, these tests should be available to all pregnant women. Patients with additional ultrasound findings should also be offered genetic counseling.[4,52]

Antenatal fetal surveillance is not mandatory in pregnancy with mild idiopathic polyhydramnios.[4] Nonetheless, nonstress tests and serial ultrasounds to evaluate AFV and fetal development are feasible for mild and severe polyhydramnios due to the relatively higher perinatal morbidity and mortality.[53] The American College of Obstetricians and Gynecologists suggests weekly antenatal surveillance at 32–34 weeks of gestation for patients with moderate or severe polyhydramnios.[54] For mild idiopathic polyhydramnios, induction of labor before 39 weeks is usually not indicated.[4] However, the timing of delivery should be individualized based on the underlying disease or obstetrical indications such as gestational diabetes.[55]

Intrapartum management

Polyhydramnios is often complicated with malpresentation.[56] Therefore, an ultrasound should be performed to assess fetal presentation. If malpresentation is diagnosed, following local guidelines for cesarean section or external cephalic version is recommended.

Continuous cardiotocography monitoring is advised for pregnant women with polyhydramnios.[57] A higher rate of dysfunctional labor, cesarean section due to failure of labor progression, and operative vaginal delivery has been reported.[58,59] Close monitoring of the labor course and associated complications, such as cord prolapse, placenta abruption, and prolonged labor is prudent.[55]

Shoulder dystocia is another potential complication during intrapartum due to the risk of macrosomia. A meta-analysis of five studies involving 75,047 participants demonstrated an increased risk of shoulder dystocia in women with idiopathic polyhydramnios (Risk Ration: 3.52; 95% confidence interval [CI]: 2.08–5.96: $I^2 = 0\%$, $\tau^2 = 0$).[53] Giving these conditions, the Society for Maternal–Fetal Medicine recommends pediatrician support during labor for women even with mild idiopathic polyhydramnios and delivery at tertiary center for women with severe polyhydramnios are also recommended.[4]

Postpartum management

Obstetricians and medical team members should be aware of the risk of postpartum hemorrhage, as uterine overdistention may cause uterine atony.

A recent prospective cohort study revealed that the incidence of postpartum hemorrhage was significantly higher in women with idiopathic polyhydramnios (7.8% vs. 2.2; odds ratio [OR]: 1.60; 95% CI: 1.09–2.34).[60] However, a previous multicenter study showed no statistically significant difference in postpartum hemorrhage rates.[61] An updated meta-analysis demonstrated that patients with idiopathic polyhydramnios have an increased risk of postpartum hemorrhage (RR 1.98, 95% CI: 1.22–3.22; $I^2 = 84\%$).[53] Despite the conflicting results of current studies, it is advisable to prepare uterotonic agents in advance.[4]

Perinatal Outcomes of Polyhydramnios

Idiopathic polyhydramnios

Pagan *et al*. in a systemic review and meta-analysis showed that the risk of intrauterine fetal demise is higher in the idiopathic polyhydramnios group compared to control groups (OR: 7.64 [95% CI: 2.50–23.38]).[62] Secondary outcomes were also elevated, including neonatal death (OR: 8.68 [95% CI: 2.91–25.87]), neonatal intensive care unit admission rates (OR: 1.94 [95% CI: 1.45–2.59]), 5-min Apgar score <7

(OR: 2.21 (95% CI: 1.34–3.62]), and higher rate of cesarean delivery (OR: 2.31 [95% CI: 1.79–2.99]).[62]

One year later, Kechagias *et al*. suggested that idiopathic polyhydramnios may be associated with an increased risk of perinatal complications, such as preterm delivery (RR: 1.96, 95% CI: 1.35–2.86. I^2 = 92%), placental abruption (RR: 3.20,95% CI: 2.20–4.65; I^2 = 2%), labor induction (RR: 1.53, 95% CI: 1.18–2.00; I^2 = 95%), shoulder dystocia (RR: 3.52, 95% CI: 2.08–5.96; I^2 = 0%), delivery through cesarean section (RR: 1.60, 95% CI: 1.39–1.84; I^2 = 95%), and postpartum hemorrhage (RR 1.98, 95% CI: 1.22–3.22; I^2 = 84%) in a meta-analysis with 38 studies included. This systemic review and meta-analysis also demonstrated increased risk of adverse perinatal outcomes such as low APGAR score (RR = 3.0, 95% CI: 1.23–7.35; I^2 = 95%), stillbirth (RR: 4.75, 95% CI: 2.54–8.86; I^2 = 9%), and perinatal mortality (RR: 4.75, 95% CI: 2.67–8.48; I^2 = 37%).[53]

One of the hypotheses for the poor perinatal outcomes is that uterine overdistension and increased intrauterine pressure may lead to a degree of "placental insufficiency."[53]

However, these two studies did not stratify the data according to the severity of polyhydramnios and most of the included studies are observational studies with high diversity and different criteria. Therefore, interpreting the results of these two meta-analyses should be done with caution, and further studies are needed. For nonidiopathic polyhydramnios, perinatal outcomes may vary due to different underlying causes.

Nonidiopathic polyhydramnios

Nonidiopathic polyhydramnios encompasses a spectrum of conditions including fetal anomalies, maternal conditions, and placental abnormalities. Understanding these specific etiologies is essential for accurately predicting and managing outcomes. Currently, there is a notable absence of research examining perinatal outcomes associated with nonidiopathic polyhydramnios. Further studies comparing outcomes across various underlying causes could yield valuable insights for clinical practice.

Conclusion

Amniotic fluid assessment is essential in prenatal ultrasound for monitoring fetal conditions, particularly in cases of polyhydramnios, which affects 1%–2% of pregnancies and is associated with significant perinatal complications. Early detection and accurate AFV measurement, using methods such as DVP and AFI, are crucial. Management strategies should focus on identifying and addressing underlying causes, with specific treatments for severe cases. Comprehensive antepartum, intrapartum, and postpartum monitoring is vital to mitigate risks and improve outcomes, although idiopathic polyhydramnios remain linked to poorer perinatal outcomes. Further randomized studies may be needed to verify the results and stratify polyhydramnios into mild, moderate, and severe categories. Ongoing advancements in technology and AI offer promise for enhancing future care.

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