

Polyhydramnios-A literature review

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ABSTRACT :

Polyhydramnios is the term used to describe an excess accumulation of amniotic fluid. Polyhydramnios is defined as an increase in the amniotic fluid in pregnancy and is associated with increased maternal and neonatal morbidity and mortality. The severity of this disease process varies, but up to one in five of the neonates affected by this condition are born with a congenital anomaly. Prompt diagnosis by ultrasound and workup for the underlying cause of polyhydramnios can direct care, including the use of reductive amniocentesis or indomethacin for symptomatic severe polyhydramnios. To mitigate the high morbidity and mortality associated with this condition, delivery is recommended at a tertiary center. This activity reviews the evaluation and treatment of polyhydramnios and highlights the role of the interprofessional team in evaluating and managing pregnant women with this condition. This clinical condition is associated with a high risk of poor pregnancy outcomes . The reported prevalence of polyhydramnios ranges from 0.2 to 1.6 % of all pregnancies.

Keywords: amniotic fluid; high risk pregnancy; polyhydramnios.

INTRODUCTION :

Polyhydramnios is a pathologic excess of amniotic fluid volume (AFV) in pregnancy. It represents a high-risk obstetric condition with increased perinatal and maternal morbidity and mortality due to a higher incidence of intrauterine fetal demise, preterm labor, premature rupture of membranes, cord prolapse, fetal macrosomia, breech presentation, cesarean delivery, and postpartum hemorrhage. In pregnancies affected by polyhydramnios, approximately 20% are due to a congenital anomaly. However, 60% to 70% are idiopathic with no identified underlying cause.

Polyhydramnios is caused by a disruption of the AFV equilibrium, which under normal circumstances balances fluid production and absorption. The clinical presentation and severity of polyhydramnios depend on the extent of the amniotic fluid volume and the underlying cause. The diagnosis is made by ultrasound with a single deepest measure fluid pocket that exceeds eight centimeters or an amniotic fluid index that is 25 centimeters or more.

Antepartum evaluation and fetal surveillance are indicated in severe polyhydramnios to identify the underlying cause, direct care, and time the delivery. Reductive amniocentesis and indomethacin are indicated only for severe cases of polyhydramnios. Delivery is recommended at a tertiary care center.

ETIOLOGY :

An underlying disease is only found in 17% of cases in mild polyhydramnios. In contrast, an underlying disease is detected in 91% of cases in moderate to severe polyhydramnios . The literature lists the following potential etiologies

- fetal malformations and genetic anomalies (8–45%)
- maternal diabetes mellitus (5–26%)
- multiple pregnancies (8–10%)

- fetal anemia (1–11%)
- other causes, e.g. viral infections, Bartter syndrome, neuromuscular disorders, maternal hypercalcemia. Viral infections which can lead to polyhydramnios include parvovirus B19, rubella, and cytomegalovirus. Other infections, e.g. toxoplasmosis and syphilis, can also cause polyhydramnios.

Advances in detailed ultrasound scanning and the prevention of Rhesus isoimmunization in the last decades have changed the relative frequency of these etiologies and significantly reduced the number of idiopathic cases.

Well-known malformations which impair the swallowing reflex include esophageal atresia, duodenal atresia and neuromuscular disorders such as myotonic dystrophy. Increased urine production, as occurs with increased cardiac output associated with fetal anemia, can also result in increased production of amniotic fluid. These changes can also occur in the context of chromosomal disorders such as trisomy 21 and different syndromes. Duodenal atresia is the most important etiology in cases with trisomy 21.

Poorly managed gestational diabetes is associated with fetal macrosomia and polyhydramnios but the pathogenesis has not been elucidated yet. One possible explanation is fetal hyperglycemia resulting in increased osmotic diuresis which subsequently leads to polyuria. This theory is supported by evidence of a strong association with high glycosylated hemoglobin values (HBA_{1c}) in cases with polyhydramnios. According to the AWMF S3-guideline, polyhydramnios can be an indication of diabetogenic fetopathy. However, due to the wide range in amniotic fluid volumes, polyhydramnios does not play an important role in monitoring gestational diabetes. The prevalence of polyhydramnios in maternal cases with diabetes mellitus is 18.8%. As the cause could also be fetal metabolic syndrome, children born after pregnancy complicated by polyhydramnios should be followed up by a pediatrician.

COMPLICATIONS

The risk of the following obstetric complications is increased when polyhydramnios is present due to over-expansion of the uterus¹³:

- maternal dyspnea
- premature membrane rupture
- preterm labor
- abnormal fetal presentation
- umbilical cord prolapse
- postpartum hemorrhage: due to reduced uterine myometrial tone

EPIDEMIOLOGY

Polyhydramnios occurs in 1% to 2% of all pregnancies. It is often identified incidentally in the asymptomatic patient during sonographic evaluation for other conditions in the third trimester. Idiopathic polyhydramnios is often a self-limiting condition, but, rarely, a cause for increased AFV may be discovered after birth.

Idiopathic polyhydramnios is less likely to be associated with an underlying disease process. However, an underlying disease or congenital anomaly has been identified in 91% of cases with more severe polyhydramnios. These patients are more likely to be symptomatic due to significant amniotic fluid volume. Chamberlin used ultrasonography to obtain qualitative AFV to evaluate the perinatal mortality rate (PMR) in 7562 patients with high-risk pregnancies. The PMR of patients with normal fluid volumes was 1.97 deaths per 1000 patients. The PMR increased two-fold to 4.12 deaths per 1000 patients with polyhydramnios.

PATHOPHYSIOLOGY

The pathophysiology of polyhydramnios is not entirely understood. Under normal circumstances, an equilibrium exists balancing the production and absorption of amniotic fluid. Amniotic fluid is mainly derived from fetal urination and less from fetal lung fluid. Fluid absorption occurs mainly through fetal swallowing. It is estimated that the fetus produces 500 to 1200 mL of urine and swallows 210-760 mL of amniotic fluid daily. Any imbalance in the complex regulatory mechanisms that regulate the amniotic fluid volume can result in polyhydramnios.

In cases of maternal hyperglycemia, fetal urine excretion increase is thought to be due to the increase of osmotic diuresis contributing to increased AFV production. Neuromuscular disorders or infections may inhibit normal muscular activity, including swallowing, thereby decreasing amniotic fluid resorption and polyhydramnios. Bartter syndrome is a rare autosomal recessive condition that affects fetal renal tubular function and results in sodium loss and polyuria, causing severe polyhydramnios.

In twin-twin transfusion syndrome, there is a donor and recipient fetus. In this condition, the placenta causes a fluid imbalance between the twins such that the recipient twin is volume overloaded and produces more urine. This affects 8% to 10% of monochorionic diamniotic pregnancies and is diagnosed in the presence of polyhydramnios and oligohydramnios caused by placental vascular anastomoses and blood flow imbalance between twins.

High output cardiac states (i.e., hemolytic anemia and infection with parvovirus B19) can increase fetal urine output and polyhydramnios. Common aneuploidies such as trisomies 21 (Down syndrome), 18 (Edward syndrome), and 13 (Patau syndrome) can be associated with polyhydramnios.

TREATMENT / MANAGEMENT

The treatment varies based on the severity of polyhydramnios and its underlying cause. Idiopathic and mild polyhydramnios rarely require treatment. A maternal-fetal medicine consultation is recommended in severe and symptomatic polyhydramnios or a known fetal anomaly setting to initiate interval ultrasound scans for growth and fetal assessment to determine the timing and mode of delivery. It is recommended that delivery occurs at a tertiary facility due to potential maternal and neonatal morbidity and mortality associated with severe polyhydramnios. Additionally, the consultation of a maternal-fetal medicine specialist is indicated for symptomatic severe polyhydramnios for reductive amniocentesis and treatment for twin-twin transfusion syndrome (TTTS). Selective fetoscopic laser photocoagulation or laser therapy is indicated in severe cases of TTTS to ablate placental anastomosis and is performed at specialized tertiary fetal medicine centers.

Reductive amniocentesis is beneficial and recommended only as a symptomatic treatment for pregnant women with significant respiratory complaints associated with restricted diaphragmatic movement and for those with substantial discomfort associated with excessive amniotic fluid. The procedure timing varies based on maternal symptoms, and on average, 1.5 to 3 L of amniotic fluid may be withdrawn. Significant adverse events post amniocentesis are uncommon but include preterm labor, placental abruption, and premature rupture of membranes. Polyhydramnios usually recurs after amnioreduction making the efficacy of the amnioreduction procedure limited. Serial amniotic fluid monitoring is indicated every one to three weeks post-procedure.

Indomethacin, a prostaglandin synthetase inhibitor, has demonstrated the ability to decrease amniotic fluid volume and is useful as a tocolytic in preterm labor. The dose is 2.2 to 3 mg/kg/day. Moise postulated that a dose of 25 mg every six hours was adequate for the treatment of fluid reduction in polyhydramnios. Indomethacin use results in a transient decrease in fetal urine output but is also associated with neonatal complications, including premature closure of the ductus arteriosus, oligohydramnios, periventricular leukomalacia, and necrotizing enterocolitis. Its use in polyhydramnios should be restricted to severe cases of symptomatic polyhydramnios with preterm labor, and the society of Maternal-Fetal Medicine (SMFM) recommends against the use of indomethacin solely to decrease amniotic fluid.

The delivery timing depends on the severity of polyhydramnios, underlying congenital malformations, and presentation of preterm labor or premature rupture of membranes. Mild and idiopathic polyhydramnios are not indications for labor induction, and the mode of delivery should be based on the usual labor characteristics determined by maternal and fetal factors. When pregnant women with polyhydramnios present in labor, an ultrasound should determine fetal presentation. An external cephalic version (ECV) may be performed for breech presentation without contraindications. Continuous electronic fetal monitoring is recommended during labor. There is an associated risk

for delayed first stage of labor due to uterine overdistension and an increased rate of amniotomy. The risk of cord prolapse increases due to the lack of fetal head engagement, which warrants cesarean delivery.

The risk of shoulder dystocia increases due to fetal macrosomia risk, with a reported increased incidence of fetal weight exceeding 4000 grams. Neonatal support should be available to women with polyhydramnios as there is an increased incidence of transient tachypnea of the newborn (TTN), which results in increased neonatal intensive care (NICU) admissions. Postpartum hemorrhage is increased due to uterine atony from uterine overdistension. Preparation should be made to respond to postpartum hemorrhage promptly.

There is no consistent evidence to support bed rest to prevent complications associated with polyhydramnios, and bed rest is associated with increased risk for venous thromboembolic disease in pregnancy.

CONCLUSION

Polyhydramnios diagnosed on ultrasound requires further maternal and fetal diagnostic tests. Maternal gestational diabetes should be excluded and maternal ToRCH screening is recommended. Detailed morphological testing should be planned for the fetus. Polyhydramnios can significantly impact pregnancy outcomes due to its association with complications such as preterm labor and increased risks during delivery. Management involves addressing underlying causes like maternal conditions or fetal anomalies, with interventions ranging from close monitoring to therapeutic procedures like amnioreduction. The prognosis varies depending on the severity and underlying causes, but with proper management, many pregnancies affected by polyhydramnios can still result in favorable outcomes.

REFERENCES

1. Golan A, Wolman I, Sagi J. et al. Persistence of polyhydramnios during pregnancy – its significance and correlation with maternal and fetal complications. *Gynecol Obstet Invest.* 1994;37:18.
2. Many A, Hill L M, Lazebnik N. et al. The association between polyhydramnios and preterm delivery. *Obstet Gynecol.* 1995;86:389.
3. Smith C V, Plambeck R D, Rayburn W F. et al. Relation of mild idiopathic polyhydramnios to perinatal outcome. *Obstet Gynecol.* 1992;79:387.
4. Alexander E S, Spitz H B, Clark R A. Sonography of polyhydramnios. *AJR Am J Roentgenol.* 1982;138:343.
5. Hill L M, Breckle R, Thomas M L. et al. Polyhydramnios: ultrasonically detected prevalence and neonatal outcome. *Obstet Gynecol.* 1987;69:21.
6. Hobbins J C, Grannum P A, Berkowitz R L. et al. Ultrasound in the diagnosis of congenital anomalies. *Am J Obstet Gynecol.* 1979;134:331.
7. Dashe J S, McIntire D D, Ramus R M. et al. Hydramnios: anomaly prevalence and sonographic detection. *Obstet Gynecol.* 2002;100:134.
8. Brace R A. Physiology of amniotic fluid volume regulation. *Clin Obstet Gynecol.* 1997;40:280.
9. Harding R, Bocking A D, Sigger J N. et al. Composition and volume of fluid swallowed by fetal sheep. *Q J Exp Physiol.* 1984;69:487.
10. Pritchard J A. Deglutition by normal and anencephalic fetuses. *Obstet Gynecol.* 1965;25:289.
11. Alexander E, Spitz H, Clark R. Sonography of Polyhydramnios. *AJR Am J Roentgenol.* 1982;138(2):343-6. doi:10.2214/ajr.138.2.343
12. Sohaey R, Nyberg D, Sickler G, Williams M. Idiopathic Polyhydramnios: Association with Fetal Macrosomia. *Radiology.* 1994;190(2):393-6. doi:10.1148/radiology.190.2.8284386
13. Hashimoto B, Callen P, Filly R, Laros R. Ultrasound Evaluation of Polyhydr Amnios and Twin Pregnancy. *American Journal of Obstetrics and Gynecology.* 1986;154(5):1069-72. doi:10.1016/0002-9378(86)90752-0
14. Sivit C, Hill M, Larsen J, Lande I. Second-Trimester Polyhydramnios: Evaluation with US. *Radiology.* 1987;165(2):467-9. doi:10.1148/radiology.165.2.3310100
15. Barkin S, Pretorius D, Beckett M, Manchester D, Nelson T, Manco-Johnson M. Severe Polyhydramnios: Incidence of Anomalies. *AJR Am J Roentgenol.* 1987;148(1):155-9. doi:10.2214/ajr.148.1.155.