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Polyhydramnios or Excessive Fetal Growth Are Markers for Abnormal Perinatal Outcome in Euglycemic Pregnancies

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Abstract

Objective—This study aims to investigate the perinatal outcome of fetuses with polyhydramnios and/or accelerated growth among women with a normal oral glucose challenge test (oGCT).

Methods—Singleton, nonanomalous pregnancies with an oGCT(< 130 mg/dL) at 24 to 28 weeks, who subsequently demonstrate polyhydramnios (amniotic fluid index > 24 cm or maximum vertical pocket > 8 cm) and/or accelerated growth (abdominal circumference > 95th percentile) on two-third trimester examinations were studied. Maternal demographics, delivery, and neonatal information were recorded. Cases were compared with a reference group (normal oGCT with neither abnormal third-trimester growth nor polyhydramnios).

Results—A total of 282 pregnancies were in the study group, and 663 were in the reference group. Deliveries in the study group were at a higher risk for birth weight (BW)% > 90%, standard deviation, and postpartum hemorrhage when compared with the reference group (adjusted odds ratio: 2.3–5.6). Pregnancies complicated by both polyhydramnios and accelerated fetal growth were significantly more likely result in a BW% > 90% (odds ratio [OR]: 18.5; 95% confidence interval [CI]: 8.9–38.6) and PPH (OR: 4.2; 95% CI: 2.4–7.6).

Conclusion—Pregnancies with normal oGCT that develop polyhydramnios and accelerated growth are at higher risk for maternal and neonatal complications. Isolated polyhydramnios without accelerated growth increases the risk for delivery complications but not neonatal morbidity.

Keywords

polyhydramnios; accelerated growth; normal glycemic screening; perinatal outcome

Amniotic fluid is fundamental for proper fetal development and growth. The perinatal implications of polyhydramnios, excessive amniotic fluid, on near-term pregnancies without

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Conflict of Interest
None.

evidence of diabetes are not defined. Polyhydramnios (amniotic fluid index [AFI] ≥ 24 or maximum vertical pocket ≥ 8 cm)^{1,2} is seen in 1 to 2% of all pregnancies. Both fetal and maternal conditions can lead to an accumulation of excessive amniotic fluid. Fetal anomalies associated with polyhydramnios include aneuploidy, structural abnormalities, central nervous system anomalies, and hydrops.^{3,4} The most common maternal etiology for polyhydramnios is poorly controlled diabetes.⁵ Additional maternal risk factors include infections and alloimmunization. Finally, 50% of polyhydramnios remains idiopathic.⁶

In those pregnancies with a known etiology for polyhydramnios, adverse perinatal events are frequent and include an increased risk of preterm delivery as well as perinatal mortality.⁷ Multiple studies have evaluated the risk adverse perinatal outcome in the presence of idiopathic polyhydramnios. These studies also demonstrate increased maternal and neonatal morbidity, specifically cesarean delivery and postpartum hemorrhage for the mother and macrosomia, neonatal intensive care unit (NICU) admission and depressed APGAR scores for the fetus. These studies, however, do not exclude pregnancies with any of the potential risk factors for polyhydramnios, such as maternal diabetes.⁸⁻¹² Finally, we could not find a prior study that evaluated the relationship between accelerated fetal growth (AFG) and polyhydramnios in pregnancies with normal glucose screening. Thus, we sought to elucidate the relationship between polyhydramnios, AFG, and perinatal outcome in nonanomalous pregnancies with normal glycemic screening. We hypothesized that pregnancies with normal glycemic control had increased perinatal risks when they complicated with polyhydramnios and/or AFG.

Methods

This is a retrospective; institutional review board approved study (HP-00059684) performed at a single tertiary referral center from 2009 through 2015. Ultrasound evidence of AFG was defined as abdominal circumference greater than the 95% or overall estimated fetal weight (EFW) at greater than the 90th percentile. The EFW determination was by Hadlock et al.¹³ Polyhydramnios was defined as an AFI of ≥ 25 cm or greater or maximum vertical pocket of ≥ 8 cm or greater. All measurements occurred after 28^{0/7} weeks of gestation and were confirmed on a subsequent scan at least 7 days after initial ultrasound evidence of polyhydramnios and/or AFG. Glucose screening was performed using a two-step approach with an initial 50 g glucose challenge test (oGCT) which if abnormal was followed by 3-hour 100 g glucose tolerance test (oGTT).^{14,15} An abnormal oGCT was defined as a 1-hour value after a 50 g oGCT is ≥ 130 mg/dL or greater.¹⁶ Inclusion criteria were singleton nonanomalous fetuses born at ≥ 34 weeks of gestation. Exclusion criteria were (1) chromosomal or structural abnormalities; (2) multiple gestations; (3) abnormal glucose screening or diagnosis of diabetes; (4) incomplete glucose screening during pregnancy; (5) alloimmunization; (6) fetal infection. Cases of accelerated growth or polyhydramnios were identified from an ultrasound-based database (Viewpoint 5.6.21.12). The control group was created by randomly selected two to three individuals from the same ultrasound day with a similar gestational age. This was done to reduce selection bias.

Data on maternal characteristics and pregnancy outcomes were collected from hospital obstetrical and neonatal records. Patient characteristics included maternal age, body mass

index, race, parity, maternal comorbidities (history of chronic hypertension, pregestational diabetes, preeclampsia, infections [HIV, hepatitis C], autoimmune, and respiratory disease), and maternal weight gain. Delivery outcomes were: gestational age at delivery, mode of delivery, vaginal lacerations, shoulder dystocia, estimated blood loss at delivery, and placental weight at delivery. Neonatal outcome variables included stillbirth, birth weight percentile, arterial pH, APGAR score, NICU admission, NICU length of stay, neonatal respiratory complications (respiratory distress syndrome or intubation more than 6 hours), neonatal hypoglycemia (defined as lowest blood sugar within 24 hours of delivery), and neonatal blood sugar within 6 hours of life. Fetal macrosomia was defined as the birth weight more than 4,000 g. An adverse composite neonatal outcome as previously defined by the Vermont Oxford Network was generated if a neonate had any of the following: intraventricular hemorrhage, hypoxic–ischemic encephalopathy, necrotizing enterocolitis, sepsis, or death.

For analysis, gravidas were divided into two groups. One group was composed of individuals with no ultrasound evidence of polyhydramnios and/or AFG (control group). Individuals with either polyhydramnios or AFG comprised the study group. A subanalysis was also performed to individually examine ultrasound factors (polyhydramnios alone, AFG alone, and polyhydramnios/AFG) in comparison to the control group. Categorical data were analyzed using chi-square and Fisher's exact test. Adjusted odds ratios (aORs) were calculated where appropriate, controlling for maternal factors. Continuous variables were analyzed according to their distribution. The probability of < 0.001 was considered significant after correcting for multiple comparisons with the Bonferroni correction. All statistical analyses were performed using SPSS version 21.0 (SPSS Inc., Chicago, IL).

Results

A total of 945 patients met inclusion criteria. Within this population, 282 patients had isolated polyhydramnios and/or AFG, and 663 had normal AFI and growth parameters. Maternal demographics of study and control groups are given in ►Table 1. The median gestational age at delivery was not significantly different between the control group and the study group. In the study group, both birth weight (3,352 vs. 3,038 g) and birth weight centile (56.2 vs. 29.1%) were significantly larger for the study group ($p < 0.001$ for each). In addition, delivery was more likely to be complicated by shoulder dystocia (odds ratio [OR]: 3.4; 95% confidence interval [CI]: 1.3–8.5), primary C-section (aOR: 2.1, 95% CI: 1.2–3.8), and postpartum hemorrhage (aOR: 2.3, 95% CI: 1.3–3.8). None of the neonatal outcomes differed significantly between the two groups (►Table 2).

Each ultrasound factor was analyzed separately as well. First, a comparison was made between individuals with polyhydramnios only and control group, and a significant increase was found in the rate of shoulder dystocia (OR: 4.5, 95% CI: 1.6–12.9). Next, in comparing AFG alone to control, a significant increase was seen in the rate of birth weight greater than the 90th percentile (OR: 7.7, 95% CI: 3.4–17.8) and macrosomia (OR: 5.0, 95% CI: 1.9–13.0). Next, in the presence of both polyhydramnios and AFG, there was a significant increase in risk postpartum hemorrhage (OR: 4.2, 95% CI: 2.4–7.6), primary C-section for arrest disorder (OR: 6.1, 95% CI: 1.7–22.4), birth weight greater than the 90th

percentile(OR: 18.5, 95% CI:8.9–38.6), and macrosomia(OR: 11.5, 95% CI: 5.2–25.6) (►Table 3). No correlation was seen between AFI and birth weight percentile in both those with only polyhydramnios and those with both polyhydramnios and fetal abdominal circumference (AC) > 95th (►Fig. 1).

Discussion

This study demonstrates that polyhydramnios with and without AFG increase the risk for adverse perinatal outcomes in patients with normal glycemic screening. For the mother, there is an increased risk of shoulder dystocia, cesarean delivery, and postpartum hemorrhage. For the neonate, there is an increased risk of macrosomia. However, in patients with normal glycemic screening, polyhydramnios without evidence of fetal macrosomia (normal AC) is not associated with large for gestational age or any other adverse neonatal outcome.

The significance of our study is that it is first to evaluate polyhydramnios with and without AFG. Prior studies demonstrate a threefold increased risk for macrosomia with polyhydramnios.¹⁰ This finding, however, was not quantified by the presence of AFG. In the absence of AFG, the risk of macrosomia is not increased. But, the addition of polyhydramnios to AFG significantly enhances the risk of macrosomia. This study demonstrates the presence of AFG without polyhydramnios has a fivefold increased risk of macrosomia. This risk jumps to 11-fold when polyhydramnios is seen as well. In diabetic pregnancies, the etiology of polyhydramnios is thought to be related to maternal hyperglycemia which leads to fetal hyperglycemia which results in osmotic diuresis.^{17–19} Unfortunately, in the absence of cord blood samples to correlate fetal metabolic status and amniotic fluid volume, the relationship remains unknown. Also, our study evaluated the accelerating fetal growth. The AC is an efficient tool for the prediction of neonatal birth weight.²⁰ The presence of AFG associated with maternal diabetes has rarely been associated with congenital anomalies. A large AC, however, has been associated with macrosomia, shoulder dystocia, postpartum hemorrhage, and other perinatal complications.^{21–24} After excluding anyone with impaired glycemic control, the risks of a large AC are still demonstrated.

The risks of intrapartum complications with polyhydramnios with and without AFG were also evaluated. The risk of shoulder dystocia in our study was significant with polyhydramnios. This risk remained significant even when individuals with accelerated growth, a known risk factor for shoulder dystocia, were excluded. Also, cesarean delivery was increased. Also, 41% of primary cesarean deliveries were done for arrest disorders, a rate much higher than prior published studies.²⁵ Also, the risk of primary cesarean delivery for arrest disorder was sixfold higher in those with both accelerated growth and polyhydramnios. This finding provides critical information to the obstetrical team managing a labor course at risk for arrest disorders and fetal malposition.

In contrast to prior studies, we found no increase in the rate of NICU admission or 5-minute APGAR < 7 in our study population.^{9,10} Likewise while others have found an association between hypoglycemia and a large AC, we found no such correlation nor did we find that

neonatal hypoglycemia was associated with any of the ultrasound findings.²¹ Infants were found to be larger for delivery in the presence of hypoglycemia with ultrasound findings; however, this did not correlate with hypoglycemia.

The strengths of this study are the well-characterized patient population with information about the prenatal, intrapartum, and postnatal course. Also, we have a relatedly large sample size of pregnancies with only isolated polyhydramnios. The limitations of this study are its retrospective nature, and we do not have information from umbilical cord samples needed to correlate ultrasound findings with neonatal metabolic effects. Also, only 69% of our neonates had an evaluation of their blood sugar after delivery. Thus, the nonsignificance found in our population may be related to the limited amount of data.

In conclusion, we have demonstrated that in a euglycemic population, the presence of polyhydramnios, both with and without accelerated fetal abdominal growth is a risk factor for intrapartum complications. In individuals with both findings, the risk of arrest disorders, postpartum hemorrhage, and fetal macrosomia is significantly increased. Thus, polyhydramnios in a near-term pregnancy should alert individuals to intrapartum risks and precautions should be taken in the prevention of postpartum hemorrhage and shoulder dystocia. These findings are critical in the safe management of labor and delivery.

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Fig. 1.

Correlation between amniotic fluid index and birth weight percentile for each ultrasound finding. Polyhydramnios only (green), linear $y = 0.2434x + 35.819$, $R^2 = 0.0014$; accelerated fetal growth (red), $y = 0.5829x + 63.593$, $R^2 = 0.0182$; polyhydramnios and fetal abdominal circumference (blue), $y = 0.1223x + 73.847$, $R^2 = 0.0001$.

Table 1

Maternal characteristics between individuals in reference population in comparison to individuals with either isolated polyhydramnios or accelerated fetal growth

Characteristics	Reference (<i>n</i> = 663)	Polyhydramnios and fetal abdominal circumference (<i>n</i> = 282)	<i>p</i> Value
Maternal age, y	24.8 (14–46)	28.3 (15–45)	0.001
Parity	1 (0–9)	1 (0–9)	0.584
Body mass index	26.9 (16.2–64.6)	27.5 (16.1–63.8)	0.248
Pregnancy weight gain (kg)	10.83(–9.1 to 41.3)	14.5(–6.9 to 35.1)	0.001
1-hour GST	96 (48–134)	102 (53–134)	0.0168
Asthma	13.3% (88)	12.4% (35)	0.707
Chronic hypertension	13.7% (91)	14.9% (42)	0.637
Preeclampsia	4.1% (27)	0.4% (2)	0.001
Smoking	13.1% (87)	16.3% (73)	0.153

Abbreviation: GST, glucose screening test.

Table 2

Comparison of perinatal outcomes for individuals in reference group in comparison to individuals with either polyhydramnios or accelerated fetal growth

	Reference (663)	Polyhydramnios and fetal abdominal circumference (282)	
	<i>n</i> (%)	<i>n</i> (%)	aOR (95% CI)
Induction ^a	22.2% (147)	24.8% (70)	1.33 (0.9, 2.1)
Shoulder dystocia	1.2% (8)	4.0% (11)	3.4 (1.3, 8.5) ^b
3/4 th laceration	1.1% (7)	1.4% (4)	0.7 (0.2, 2.1) ^b
Cesarean delivery	26.4% (175)	34.4% (97)	1.4 (0.9–2.1)
Primary C-section ^a	12.2% (81)	17.3% (49)	2.1 (1.2, 3.8)
Indication for C-section arrest disorder ^a	25% (20/80)	41.7% (20/48)	1.9 (0.7–5.1)
Postpartum hemorrhage ^a	9.0% (57)	17.5% (47)	2.3 (1.3, 3.8)
Macrosomia	2.0% (13)	9.6% (27)	6.4 (2.5–16.1)
Greater than 90 th percentile for GA at delivery ^a	2.1% (14)	11.7% (33)	5.6 (2.5, 12.9)
NICU admission ^c	15.2% (101)	12.5% (35)	1.0 (0.6, 1.3)
Respiratory complications ^c	4.5% (30)	5.8% (26)	1.6 (0.9, 2.9)
Hypoglycemia ^c	6.0% (29/506)	6.8% (27/243)	1.3 (0.7, 2.2)
NICU composite outcome ^c	2.6% (18)	1.7% (8)	1.3 (0.4, 4.1)

Abbreviations: aOR, adjusted odds ratio; CI, confidence interval; GA, gestational age; NICU, neonatal intensive care unit. Note: Bold values indicate $p < 0.001$.

^aAdjusted for total weight gain and maternal age.

^bNot adjusted odds ratios secondary to low number of outcomes.

^cAdjusted for gestational age at delivery, race, and maternal age.

Table 3

Comparison of individual ultrasound factors to reference group

	Reference group (663)	POLY only (131)	OR (95% CI)	AFG (76)	OR (95% CI)	POLY and AFG (74)	OR (95% CI)
GA delivery	39.0 (34.0–43.0)	39.4(35.0–42.0)	1.03 (0.9, 1.23)	38.5 (36.0–41.3)	1.03 (0.9, 1.23)	39.0 (33.0–41.0)	1.03 (0.9, 1.23)
Induction	22.2% (147)	32.1% (42)	1.6 (1.1, 2.5)	11.8% (9)	0.5 (0.2, 0.9)	24.3% (18)	1.1 (0.6, 2.0)
Shoulder dystocia	1.2% (8)	5.3% (7)	4.5 (1.6, 12.9)	2.7% (2)	2.3 (0.5, 10.9)	2.8% (2)	2.4 (0.5, 11.5)
Cesarean delivery	26.4% (175)	32.1% (942)	1.3 (0.9–2.0)	35.5% (27)	1.6 (0.9–2.6)	37.8% (28)	1.7 (1.1, 2.8)
Primary C-section	12.2% (81)	20.9% (26)	1.9 (0.9, 3.8)	14.1% (11)	0.8 (0.4, 1.9)	20.4% (12)	0.7 (0.3, 1.4)
Primary C-section for arrest disorder	25.0% (20/81)	34.6% (9/26)	1.6 (0.6, 4.2)	30.0% (3/11)	1.1 (0.3, 4.7)	66.7% (8/12)	6.1 (1.7, 22.4)
PPH	8.7% (60)	12.5% (33)	1.4 (0.9, 2.2)	16.0% (12)	1.9 (0.9, 3.7)	26.0% (20)	4.2 (2.4, 7.6)
Gt 90%	2.0% (13)	3.0% (17)	1.3 (0.6, 3.2)	14.5% (11)	7.7 (3.4, 17.8)	28.8% (21)	18.5 (8.9, 38.6)
Macrosomia	1.7% (11)	3.2% (10)	1.9 (0.8, 4.5)	9.2% (7)	5.0 (1.9, 13.0)	24.7% (186)	11.5 (5.2, 25.6)
Hypoglycemia	6.0% (29)	6.8% (17)	1.1 (0.6, 1.9)	4.8% (3)	0.7 (0.2, 2.4)	6.1% (3)	0.9 (0.3, 3.1)
NICU admission	14.8% (94)	16.1% (44)	1.1 (0.7,1.6)	10.3% (8)	0.6 (0.3, 1.4)	13.1% (7)	0.8 (0.4, 1.9)
Respiratory complication	4.6% (29)	7.3% (20)	1.7 (0.9, 3.1)	2.6% (2)	0.6 (0.1, 2.4)	5.6% (3)	1.2 (0.4, 4.1)

Abbreviations: AFG, individuals with accelerated fetal growth and no evidence of polyhydramnios; CI, confidence interval; GA, gestational age; Gt, greater than 90% percentile at delivery; NICU, neonatal intensive care unit; OR, odds ratio; POLY and AFG, individuals with both polyhydramnios and evidence of accelerated growth; POLY only, individuals with polyhydramnios and no evidence of accelerated growth; PPH, postpartum hemorrhage. Note: values in bold denotes $p < 0.001$.