The Egyptian Journal of Hospital Medicine (January 2023) Vol. 90 (2), Page 3327-3331

Association between Oligohydramnios and Placental Lesions and Their Effect on Fetal Growth

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ABSTRACT

Background: The amniotic sac, which houses the amniotic fluid that is a clear, slightly yellowish liquid that surrounds the foetus throughout pregnancy. This environment supports the foetus' natural growth and development.

Objectives: This study aimed to investigate the association between oligohydramnios and different placental pathological lesions and its effect on fetal growth during pregnancy.

Patients and method: This single cohort descriptive study was conducted in Department of Obstetrics & Gynecology, Faculty of Medicine, Zagazig University through the period from September 2020 to September 2022. A total of 48 pregnant women presented by oligohydramnios.

Results: Neonates with isolated oligohydramnios had lower birth weights as a defining feature and a higher rate of neonatal morbidity with low Apgar score. They were admitted more often to neonatal intensive care unit (NICU). Oligohydramnios is linked to a higher rate of surgical delivery and, consequently, a higher rate of maternal morbidity.

Conclusion: Oligohydramnios without fetal structural and chromosomal abnormalities and in the absence of known maternal disease is associated with unfavourable neonatal outcomes as well as lesions caused by placental vascular malperfusion. We believe that it belongs to a spectrum of "placental insufficiency" (probably in a milder form than preeclampsia and fetal growth restriction).

Keywords: Oligohydramnios, Placental lesions, Fetal growth.

INTRODUCTION

The amniotic sac, which houses the amniotic fluid that is a clear, slightly yellowish liquid that surrounds the foetus throughout pregnancy. This environment supports the foetus' natural growth and development. Amniotic fluid provides for ongoing foetal growth in an unrestricted, sterile, and thermally controlled environment, while enabling proper development of the foetal respiratory, gastrointestinal, urinary, and musculoskeletal systems. By its strong bacteriostatic characteristics, it shields the foetus against injury and infection. It shields the developing embryo from vascular and nutritional impairment and prevents compression of the umbilical cord placenta [(1)].

Decreased amniotic fluid volume in relation to gestational age is referred to as oligohydramnios. The amniotic fluid index (AFI), which is derived by summing the centimeter depth of the greatest vertical pocket in each of four equal quadrants, is used to define it semi-quantitatively [(2)]. Oligohydramnios can have a substantial effect on the mother's and the fetus's outcomes. It may result in cord compression, foetal pulmonary hypoplasia, foetal growth restriction, a low Apgar score, admission to the neonatal intensive care unit (NICU), and foetal mortality. Obstetric problems can include labour that is protracted because of inertia and a higher frequency of surgical interventions [(3)]. The objective of the present study was to describe the association between oligohydramnios and placental pathology and analyse the effect of oligohydramnios with placental pathology on fetal growth during pregnancy.

PATIENT AND METHODS

This single cohort descriptive research was done in the Department of Obstetrics and Gynecology, Zagazig University's College of Medicine through the period from September 2020 to September 2022. 48 pregnant women presented by oligohydramnios were admitted at Zagazig University Hospital.

Inclusion criteria: Singleton pregnancy. Presence of oligohydramnios measured by ultrasound when the deepest vertical pool or the amniotic fluid index is 5 cm or less. Absence of major fetal malformations. Intact amniotic membrane.

Exclusion criteria: Multiple pregnancies. Pregnancy after term. Membranes prematurely rupturing before term. Fetal death. Renal agenesis (Potter's syndrome). Polycystic kidney. Urethral obstruction (atresia/posterior urethral valve obstruction). Women who had any medical disorders as gestational hypertension, preeclampsia, heart failure and chronic liver diseases.

All women in this study were subjected to full detailed medical and clinical examinations. Investigations included ultrasound for measurement of fetal growth, for measuring the amount of amniotic fluid and the placenta, documentation of fetal viability and presence of gross fetal anomalies. Standard US was obtained at 28 weeks of pregnancy, weekly measurement of amniotic fluid volume and biweekly foetal biometry were part of the US examination was done, fetal growth restriction (FGR) was diagnosed when estimated fetal weight and/or AC < the tenth percentile, confirmed on at least two measurements conducted back-to-back, two weeks after the US standard was established.

Ultrasonography with Doppler depending on the initial degree of blood flow anomalies, Doppler examinations of foetal circulation comprised weekly or

Received: 16/10/2022
Accepted: 19/12/2022

biweekly monitoring of the umbilical artery and middle cerebral artery blood flow velocity waveforms.

After delivery, all placentas were immediately fixed in buffered formalin for placental biopsy. Following a physical examination, the following standard samples were taken for routine diagnosis: three samples in one block from the umbilical cord, a membrane roll, and three blocks from the chorionic disc, two from the disk's centre and one from its edge. Each placenta yielded five chunks. Sections were frequently stained with H & E after samples were routinely prepared for histology.

Evaluation of neonates:

Pediatricians checked each newborn as soon as they were born. The neonatal records yielded the following information:

Charts were used to determine birth weight percentiles for various gestational ages. APGAR ratings, admission to the neonatal intensive care unit (NICU) and newborn mortality.

Ethical approval: Before starting of the study, permission was obtained from Institution Review Board IRB (#6518-7-12-2020), Faculty of Medicine, Zagazig University. A written informed consents were taken from the admitted pregnant women included in the study after being informed about the objectives and hazards of the study. The study's protocol complied with the Helsinki Declaration, which is the World Medical Association's code of ethics for research on humans.

Statistical analysis

Microsoft Excel software was used to code, enter, and analyse historical data, basic clinical examinations, laboratory investigations, and outcome measurements. The Statistical Package for the Social Sciences (SPSS version 20.0) program was used to import the data and perform analysis.

The following tests were used to determine whether differences were significant: Difference and association of qualitative variable by Chi square test (X^2), differences between quantitative independent groups by t test. Depending on the type of data, qualitative were represented as number and percentage, while quantitative continues group data were represented by mean ± SD. P value was chosen at 0.001 for very significant results and 0.05 for outcomes that were significant.

RESULT

Table (1**)** showed that the mean age of the studied cases was 24.60 ± 4.17 years and the mean BMI was 27.05 ± 3.32 kg/m^2. 14% of the studied group were workers, only 4% were smokers, 60% of the studied group had previous C.S and the other 40% had normal delivery.

Table (1): Descriptive data of the studied group (n = 48)

	N	%
Age/years		
< 25	29	60%
≥ 25	19	40%
Work		
Yes	6	14%
No	42	96%
Smoking		
Yes	2	4%
No	46	96%
Gravidity		
1	22	46%
2	6	12%
More than 2	20	42%
Parity		
0	23	48%
1	13	28%
2	8	16%
More than 2	4	8%
Mode of delivery		
Cs	29	60%
Vaginal	19	40%
Bodey Mass Index (BMI)		
< 30	29	60%
≥ 30	19	40%

Number of cases was 48 cases

Table (2) showed that the mean neonatal birth weight was 2950 ± 4.17 gm and the mean gestational age was 38.60 ± 2.74 weeks. Only 4% of the neonates died, 23% had NICU admission, 6% had Apgar score < 7 at 5 m and 6% were small for gestational age (SGA).

Table (2): Descriptive date of the studied neonates and Neonatal outcome after delivery

	Range	Mean ± SD
Neonatal birth weight	1600 – 3700	2950 ± 4.17
Gestational age at delivery	37.0 – 40.0	38.60 ± 3.74
Neonatal outcome	**N**	**%**
Neonatal death		
Yes	2	4.0
No	46	96.0
NICU admission		
Yes	10	23.0
No	38	79.0
APGAR Score < 7 at 5m		
Yes	3	6.0
No	45	94.0
Small for gestational age		
Yes	3	6.0
No	45	94.0

Table (3) showed that the mean amniotic fluid index was 3.18 ± 1.11, while there was 6% of studied eases had abnormal umbilical artery Doppler.

Table (3): Ultrasound (U.S) findings in the studied group

U.S Findings		
Amniotic fluid index (AFI)	**Mean ± SD**	3.18 ± **1.11**
	Range	1.9 – 4.4
Umbilical artery Doppler	**Normal**	47 (94%)
	Abnormal	3 (6%)

Table (4) showed that the mean placental weight was 433 ± 6.54 g and 4% of the studied females had retro placental hemorrhage.

Table (4): Placental characteristics in the studied group

Placental characteristics		
Placental weight (g)	**Mean ± SD**	433 ± **6.54**
	Range	311 – 577
Retro placental hemorrhage	**Yes**	2 (4%)
	No	48 (96%)

Table (5) showed that 19% had abnormal cord insertion, 25% had hypercoiled cord and 10% had hypocoiled cord. Umbilical coiling index (UCI) is defined as the total number of coils divided by the total length of the cord in centimeters.

UCI < 10 percentile—hypocoiled, 10^{th}-90^{th} percentile—normocoiled, > 90th percentile—hypercoiled.

Table (5): Umbilical Cord characteristics in the studied group

Umbilical Cord characteristics	N	%
Abnormal cord insertion		
Yes	9	19.0
No	39	81.0
Hyper coild cord		
Yes	12	25.0
No	36	75.0
Hypocoild cord		
Yes	5	10.0
No	43	90.0

Table (6) showed that 4% had fetal thrombotic vasculopathy and avascular villi while 6% had thrombosis.

Table (6): Vascular and villous lesions consistent with FTOD (fetal thrombo-occlusive disease) characteristics in the studied group

Vascular and villous lesions consistent with FTOD	N	%
Fetal thrombotic vasculopathy		
Yes	2	4.0
No	46	96.0
Avascular villi		
Yes	2	4.0
No	46	96.0
Thrombosis		
Yes	3	6.0
No	45	94.0

Table (7) showed that 27% had intervillous fibrin deposition, 4% had villous agglutination, 27% had villous infarction and 6% had increase syncytial knots.

Table (7): Villous changes related to maternal malperfusion in the studied group

Villous changes related to maternal	N	%
Intervillous fibrin deposition		
Yes	13	27.0
No	35	73.0
Villous agglutination		
Yes	2	4.0
No	46	96.0
Villous infarction		
Yes	13	27.0
No	35	73.0
Increase syncytial knots		
Yes	3	6.0
No	45	94.0

Table (8) showed that there was a significant correlation between estimated fetal weight and amniotic fluid index.

Table (8): Correlation between fetal biometric indices and amniotic fluid index in study group

		Amniotic fluid index
Biparital diameter (BPD)	Rs	0.948
	P	0.021
Abdominal circumference	Rs	0.156
	P	0.041
Estimated fetal weight	Rs	0.787
	P	0.001*
Femur length	Rs	0.174
	P	0.035

Rs: Spearm correlation co-efficient. P-value > 0.05 to be statistically insignificant. P-value < 0.05 to be statistically significant. P-value < 0.001 to be highly statistically significant.

DISCUSSION

In our study the mean maternal age was distributed as 24.60 ± 4.09 with minimum 18 and maximum 35, which coincide with the study done by **Casey *et al.*** [4] where the mean maternal age was 23.9 years but in the study of **Hadas *et al.*** [5] the mean maternal age was 30.6 ± 5.4 years. **Spinillo *et al.*** [6] found that the mean maternal age was 31.5 years.

In the present study, the mean BMI was 27.05 ± 3.32 kg/m^2, which is near to the results by **Hadas *et al.*** [5] where the mean BMI was 24.4 ± 4.4 Kg/m^2. **Spinillo *et al***. [6] stated that the Mean BMI was 29.1 ± 4.6 Kg/m^2.

In our study the incidence of oligohydramnios was 46%, in primi- and 54% in multi-gravida, which is in agreement with the study done by **Hadas *et al*.** [5] where the incidence of oligohydramnios was 46.3% in primigravida. The mean amniotic fluid index was 3.18 ± 1.11 in our study, but in the study done by **Wang *et al.*** [7] it was 3.39 ± 6.39. It is believed that the redistribution of blood flow favouring the foetal heart and brain over the lungs, digestive tract, and kidney is what causes the progressive decline of amniotic fluid volume associated with FGR [8]. There is proof that the amniotic fluid volume decrease in FGR pregnancies is not just a foetal circulatory response to hypoxia. In reality, the placenta of humans can facilitate the transfer of water from the mother to the foetus by either hydrostatic or osmotic mechanisms, which are likely controlled by water channel proteins [9, 10]. Experiments on animals and in vitro by **Adriano *et al.*** [11] and **Bos *et al*.** [12] assert that preeclampsia and hypoxia can affect the amount of amniotic fluid by obstructing both transplacental water transport and transmembranous water reabsorption. Venoconstriction brought on by a mismatch between the intervillous space and villus, as occurs in placental underperfusion, can change the transvillous pressure and impact the transport of liquids through the placenta [13].

As regards smoking, only 4% were smokers, which agrees with **Hadas *et al.*** [5] as there was 4.6% of the studied group were smokers**. Saleemuddin *et al.*** [14] found that 3.5% of their studied group were smokers.

In the current study, 60% of the studied group had previous C.S and the other 40% had normal delivery but in the study done by **Krishna *et al.*** [15] there were 42% of the studied group had previous C.S and the other 58% had normal delivery.

In the present study, the mean neonatal birth weight was 2950 ± 4.17 gm, which is near to the results by **Hadas *et al.*** [5] as the mean neonatal birth weight was 3180 ± 435.7gm.

As regards to the mean gestational age at delivery, it was 38.60 ± 2.74 weeks, which coincide with the results by **Hadas *et al.*** [5] where the mean gestational age at delivery was 39.5 ± 1.3 weeks. **Spinillo *et al.*** [6] stated in their study group that the average gestational age at delivery was 39.1 weeks.

In the current study, the umbilical artery Doppler was normal in 94% and abnormal in 6% of pregnant females with oligohydramnios, which agrees with the study of **Krishna *et al.*** [15] where the umbilical artery Doppler was normal in 93% and abnormal in 7% of pregnant female with oligohydramnios.

Regarding to the mean placental weight, it was 433 ± 6.54 gm and there was 4% of the studied group had retroplacental hemorrhage. This is near to the results by **Hadas *et a1*.** [5] as the mean placental weight was 477 ± 100 gm and there was 2.8% of the studied females had retro-placental hemorrhage. Also, **Hendrix *et a1*.** [16] stated that the mean placental weight was 441.8 ± 128.9 gm in their study group. In the study done by **Spinillo *et al*.** [6] it was 469.6 ± 184.9 gm.

In the present study, 23% of the study group had NICU admission, which is the same as that of **Krishna *et al.*** [15] and near to the results by **Hadas *et a1*.** [5] as 23.1% of the study group had NICU admission in their study. **Spinillo *et al.*** [6] stated that 21.8% of their study group had NICU admission.

As regard to Apgar score, there was 6% had Apgar score < 7 at 5 m but in the study done by **Krishna *et a1*.** [15] there was 15% had Apgar score < 7 at 5m. **Hadas *et al.*** [5] in their study stated that there was no neonates had Apgar score < 7 at 5m.

In our study, 6% of the neonates were small for gestational age (SGA) but in the study by **Krishna *et al.*** [15] 18% of the neonates were small for gestational age (SGA). **Hadas *et al.*** [5] stated that between the research group and the control group, there was a substantial difference concerning lower birth weight (3180 ± 435.7 vs. 3395 ± 551.4 g). In the present study, there was 19% had abnormal cord insertion, which is near to the results by **Hadas *et al.*** [5] as 22.2% had abnormal cord insertion. In the current study, 25% of the study group had hypercoiled cord but in the study done by **Hadas *et al.*** [5] 25.9% had hypercoiled cord. In the study done by **Hadas *et al*.** [5] there was 12.9% of their study group had hypocoiled cord but in the present study, 10% had hypocoiled cord. **Spinillo *et al.*** [6] found that 11.3% of their study group had hypocoiled cord.

As regards vascular and villous foetal thrombo-occlusive disease-related lesions (FTOD), there was 4% had fetal thrombotic vasculopathy, 4% had avascular villi and 6% had thrombosis of placental lesions in the study group. In the study done by **Hadas *et al.*** [5] there was vascular lesions consistent with FTOD represent 5.5% and villous lesions consistent with FTOD represent 3.7% of the placental lesions in their study group. Previous research by **Taylor *et al.*** [17] has mainly described the impact of placental vascular pathology on birth weight and adverse outcome in the SGA population, as the main risk group for placental insufficiency.

In the present study, 27% had intervillous fibrin deposition, 4% had villous agglutination, 27% had villous infarction and 6% had increase syncytial knots.

The complex systems that underlie healthy placental function are important for foetal growth because they make sure that the foetus is continuously supplied with the nutrients and oxygen it requires, that waste is removed, and that it is protected from pathogens.

These vital functions are disrupted by placental dysfunction or damage, which can lead to stunted growth and even stillbirth [18]. Hypoxia-reoxygenation insults to the developing placental villi early in pregnancy are regarded to be the most frequent mechanism of placenta harm. Failure of the spiral arteries' physiological transition, which results in constrained maternal blood flow into the intervillous area, triggers this process. A MVM-like histology will emerge from variations in oxygen tension that cause oxidative stress and free radical damage to the placental villi. These pathologic lesions represent a severe form of early placental malfunction [19].

A recent study by **Akolekar *et al.*** [20] Including more than 6000 babies showed that the bulk of unfavourable perinatal outcomes—such as stillbirth, low cord pH, emergency Caesarean sections for foetal distress in labour, and admission to the newborn intensive care unit—occurred among foetuses of the right gestational age.In our investigation, there was a strong relationship between amniotic fluid index and estimated foetal weight. In a prior study by **Hendrix *et al.*** [16], they demonstrated that prenatal growth velocity, as determined by the relative change in foetal abdominal circumference over time, can be used to more accurately predict the outcomes of newborns. The first step in concentrating resources for antenatal surveillance that incorporates measurements of foetal hemodynamic adaptations to diminished placental function is to identify these foetuses with the highest risks for perinatal morbidity. Hence, women with suspected foetal development restriction should receive advice regarding the best time to deliver, which is generally seen as being between 37 and 38 weeks [21].

CONCLUSION

Oligohydramnios is linked to placental vascular malperfusion lesions and poor neonatal outcomes even in the absence of known maternal illness and foetal anatomical and chromosomal abnormalities. It ought to be viewed as a component of the "placental insufficiency" spectrum, in our opinion (probably in a milder form than preeclampsia and fetal growth restriction).

Sources of funding: This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Conflicts of interest: There are no conflicts of interest, according to the authors.

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