ORIGINAL PROF-1611

POLYHYDRAMNIOS; STUDY OF CAUSES AND FETAL OUTCOME

DR. SAADIA TARIQ
Senior Registrar
Gynae Unit-1,
Lahore General Hospital
Lahore

DR. SADIA CHEEMA DR. ADEELA AHMAD Dr. Naela Tarique

ABSTRACT... Polyhydramnios is though an uncommon problem but very distressing for patient. **Objectives:** To locate the causative factors and neonatal outcome in polyhydramnios. **Design:** Case series. **Setting:** Department of Obstetrics and Gynaecology unit 1, Lahore General Hospital, Lahore. **Period:** From January 2004- December 2005. **Subjects and Methods:** Total 82 diagnosed cases of polyhydramnios in 3rd trimester were included in this study. **Results:** According to the results of this study polyhydramnios can occur in primigravida as well as multigravida. Causative factor are mainly idiopathic after which the most important is fetal defects. Diabetes is also associated finding with polyhydramnios in 26.8% cases. The impact of polyhydramnios on neonatal outcome is that most of the babies were born without any significant effect. There were only 26 babies (31.5%) in which anomalies were present and neural tube defects were common. **Conclusions:** Idiopathic polyhydramnio being the most common type. Improved prenatal and antenatal screening and early detection of congenital anomalies may help to minimize the morbidity of the patient.

Key words: Polyhydramnios, Multigravida, Neural Tube Defects.

INTRODUCTION

In polyhydramnios amniotic fluid's largest vertical pool is more than 8 cm or amniotic fluid index (AFI) above the 95th centile for gestational age¹. Normal value is up to 6 cm. In older studies the incidence of polyhydramnios was 3.5% but more recent studies give an incidence of 0.2% due to earlier diagnosis and better management of pregnancies with fetal congenital abnormalities². In majority of cases the fetus is normal and there is no causative factor in the mother as well, prognosis for such cases is good³.

Causes of polyhydramnios are multifactorial, maternal cause is diabetes mellitus and fetal causes are duodenal atresia, sacrococcygeal teratoma, chorioangiooma, twin to twin transfusion syndrome, and chromosomal anomaly.

Some are compatible with life and some are not. One is duodenal atresia with increased risk of prenatal asphyxia and death, even when the karyotype is normal and no associated anomalies are present. Death could be caused by vagal over activity due to distention of the upper GIT⁴.

The anomaly detection rate in pregnancies with the help of ultrasound in Hydramnios was nearly 80% irrespective of the degree of amniotic fluid increase⁵. Residual anomaly risk after normal sonographic evaluation was mild or moderate and 11% if sever. Three sectional views of neck and upper chest are useful for in utero detection of esophageal pouch that may enhance the prenatal diagnosis of esophageal atresia. The positive predictive value for prenatal ultrasound for detecting EA is 100% with a sensitivity of 80%⁶.

Polyhydramnios may occur with gestational diabetes but there was no significant difference in Apgar score (1 and 5 min), newborn hypoglycemia metabolic acidosis and hyperbillrubinaemia. Hydramnios in women with GDM was associated with increased risk of prenatal morbidity and mortality⁷.

There is increase risk of preterm labor in polyhydramnios. Preterm delivery related to multiple gestation polyhydramnios was associated with enhanced amniotic expression and activity of cyclooxygenase type-2⁸. The babies being delivered near term have better prognosis than the babies of less gestation. These patients require hospital admission.

POLYHYDROMNIOS

There is increased rate of perinatal morbidity and mortality.

Mohsin in 2000 concluded that fetal BPP appears to be an effective technique for the assessment of fetal condition⁹.

MATERIAL AND METHODS

This study was conducted in Gynae unit 1 of Lahore General hospital, Lahore, over the period of From January 2004- December 2005. Method used was to do a thorough physical examination after a detailed history of the patients on clinical diagnosis of polyhydramnios were sent for ultrasonic confirmation after which if confirmed were included in the study and Proforma was filled. Routine Lab investigation was requested. Complete labor record was made along with mode of delivery and duration. Complete physical examination of baby by obstetrician and pediatrician with recording of Apgar score and any anomalies found, and any resuscitation carried out on the baby. Data thus collected was analyzed for results and compared with international as well as local studies.

RESULTS

During the study period of one year 3740 patients came for antenatal checkup, out of them 82(2.19%) patients had polyhydramnios. The results showed that 53 patients (64.6%) women belonged to age 30-39 years with next majority between 20-29 years i.e. 25 (30.4%). Only 4 patients (5%) were more than 40 years of age as show in table-I. It was also seen that majority of the women having polyhydramnios, were multigravida.

Patients presented with polyhydramnios were having fetal congenital anomalies in 26 cases (31.7%) cases while no congenital anomaly was detected in 56 (68.5%) cases. Details of fetal congenital anomalies were shown in table-II.

Regarding severity of polyhydramnios 45 patients (54.8%) presented with mild degree of polyhydramnios, 26 (31.7%) with moderate and 11 (13.3%) with sever degree of polyhydramnios, shown in table-III. In most of

Table-I. Age distribution and with poyhydramnios (82 cases)			
Age (years)	No. of patients	%age	
20-29	25	30.4%	
30-39	53	64.6%	
> 40	04	5.0	

Table-II. Associated fetal anomalies (82 cases)			
Anomalies	No. of Patients	%age	
No anomaly	56	68.3%	
Neural tube defects	11	13.4%	
Hydrocephaly	08	9.7%	
Exomphalos	02	2.4%	
Ventricular septal defects	02	2.4%	
Esophageal Artesia	01	1.2%	
Duodenal Artesia	02	2.4%	

severe degree of polyhydramnios fetal congenital anomalies were common and majority were detected at early gestation, so termination of pregnancy was done in these cases.

Table-III. Degree of polyhydramnios (82 cases)			
Severity	No. of patients	%age	
Mild (AFI 25-30cm)	45	54.8%	
Moderate (AFI 31-35cm)	26	31.7%	
Sever (AFI >35cm)	11	13.5%	

In 58 (70.7%) cases no associated maternal disease was detected, while impaired glucose tolerance was present in 22 (26.8%) cases. Out of these 57% were on insulin therapy and the rest i.e. 43% were having family history of diabetes mellitus. In my study 2 cases were of Rh- iso immunization, viral infection and smoking could not be implicated as a causative factor in polyhydramnios as shown in table-IV.

POLYHYDROMNIOS

Table-IV. Associated maternal condition with polyhydramnios			
Maternal condition	No. of patients	%age	
No disease	58	70.7%	
Diabetes mellitus	22	26.8%	
Rh-ISO immunization	02	2.4%	
Viral infections	-	-	
Smoking	-	-	

Regarding fetal outcome 56(68.2%) babies delivered alive, still birth in 20(24.3%) babies and early neonatal death seen in 6 babies, which mainly caused by prematurity. As the risk of pre term delivery is common in polyhydramnios. Detail of fetal outcome shown in table-V.

Table-V. Fetal outcome			
Outcome	No. of patients	%age	
Alive births	56	68.2%	
Still births	20	24.3%	

DISCUSSION

Polyhydramnios is an uncommon complication associated with pregnancy. Such pregnancies are high risk pregnancies and need to be thoroughly investigated. The clinical problems associated with polyhydramnios, apart from fetal anomaly, are maternal discomfort, difficult clinical examination of fetus and premature labor; it is diagnosed accurately by clinical examination and confirmed by ultrasonography. In cases where polyhydramnios is of mild to moderate degree and no cause is found in the mother as well as in fetus perinatal outcome is good¹⁰.

In our study the incidence of polyhydramnios was found to be 02.1%. Bryan M. Hibbard (1998) gave an incidence of 1%11 Hill et al² provide incidence of 0.9% after an ultrasonic assessment of more than 9000 prenatal patients in a study spread over span of 10 years. Desmedt et al (1990)¹² observed the incidence of major congenital anomalies in acute polyhydramnios to be 63%, in sub acute, 65%, and in chronic polyhydramnios, 14%. In our study congenital anomaly seen in 31.7% cases, which were mostly seen in sever polyhydramnios and most of the anomalies were detected earlier due to USG. Hotta et al concluded that sever polyhydramnios does not always result in lethal abnormalities¹³. Neural tube defects are easily detectable by ultrasound examination in first and second trimester, similarly serious structural abnormalities like septal defects and anterior abdominal wall defects can be easily diagnosed by mid trimester scan¹⁴. If early diagnosis is made maternal morbidity can be reduced by offering termination of pregnancy at an early gestation when it is psychologically and physically less traumatic to the mother.

Cases of polyhydramnios in 3rd trimester where no fetal congenital anomalies are detected on ultrasound maternal morbidity and fetal morbidity & mortality can occur by excessive abdominal distention, sudden premature rupture of membrane, placental abruption, and cord prolapse, fetal malpresentation, premature labor, postpartum hemorrhage and high risk of operative deliveries.

In 1995 Many et al¹⁵ concluded in his study that it is the underlying cause of polyhydramnios rather than the relative excess of amniotic fluid which is responsible for premature labor. He found the incidence of preterm labor to be 22.2% in diabetes mellitus, 39% with congenitally malformed baby and 12.6% in unexplained polyhydramnios. In our study 6 babies underwent early neonatal death mainly due to pre maturity and fore term delivery.

Impaired glucose tolerance was an etiological factor in 26.8% cases for polyhydramnios in our study. In contrast to our study Smith et al observed higher incidence of large for gestation age fetuses in mild idiopathic polyhydramnios, with no other adverse effect on fetus¹⁶. Sometime uncontrolled diabetes in first trimester leads to congenital anomaly in the fetus which causes polyhydramnios in mothers, so ultrasound examination

at 18-22 wks is mandatory to exclude major congenital abnormalities and structural defects at this stage^{17,18}.

Lazehnik et at¹⁹ also concluded by his study, that prevalence of large for gestational age fetuses is 2.7times greater with coexistent polyhydramnios. But in our study neither the severity of polyhydramnios nor the presence of maternal diabetes mellitus strengthens the relationship between polyhydramnios and large for gestational age new born infants.

In this series antiprostaglandin treatment with indomethacin was not attempted because of the fetal risk of oligohydramnios²⁰ premature constriction of the ductus arteriosus²¹.

In a study conducted by Phelan Et al, An increased incidence of fetal macrosomia, premature births, non-reactive non stress tests, perinatal morbidity, and fetal anomalies was observed. These data suggest that if polyhydramnios is encountered during an ultrasound evaluation, consideration should be given to the possibility of latent or uncontrolled diabetes mellitus or fetal macrosomia or anomaly. Fetal surveillance and genetic evaluation also should be consideration²².

The incidences of major congenital anomaly and fetal macrosomia were significantly related to qualitative amniotic fluid volume.

CONCLUSION

Polyhydramnios though an uncommon problem associated with pregnancy, can be very distressing for the patient.

The study proved idiopathic being the most common causative factor of polyhydramnios so with improved prenatal and antenatal screening and early detection of congenital anomalies and causative factors might help to minimize the morbidity of the patient.

Awareness regarding contraception and effective contraception measures may be helpful in reducing parity and thus associated risk of polyhydramnios and increased fetal anomalies.

The study also gives us an understanding of the impact of this condition on the fetus, which can be effectively managed if early detection and regular follow ups are carried out.

Copyright© 08 July, 2010.

REFERENCES

- Moor TR,Cayle JE. The amniotic fluid index in normal human pregnancy. Am J Obstet Gynecol 1990;162:1168-73.
- Hill LM, Breckle R, Thomas ML, Fries JK; Polyhydramnios. Ultrasonographically detected prevalence and neonatal outcome. Obstet Gynecol 1987;69:21-25.
- Chamber lain PF, Manning FA, Morrison L et al. Ultrasound evaluation of amniotic fluid volume 11. The relationship of increased amniotic fluid volume to perinatal outcome. Am J Obstet Gynecol 1994;150:250-54.
- Brantberg A. Blaas HG. Salvesen KA. Haugen SE. Mollerlokken G. Eik-Nes SH. Fetal duodenal obstruction: increased risk of prenatal sudden death. Ultrasound Obstet Gynecol 2002; 20;439-46.
- Dashe JS, Macintire DD. Ramus RM. Samtos-Ramos R. Twicker DM. Hydramnios: anamaly prevalence and sonographic detection. Obstet Gynecol 2002;100:134-9.
- Shulman A, Mazkereth R, zalel Y, Lipitz S, Avigad I et al. Prenatal identification of esophageal atresia: the role of ultrasonography for evaluation of functional anatomy. Prenat Diagn 2002;22; 669-74.
- Shoham I, Wiznitzer A, Silberstein T, Fraser D, Holcberg G, Gestational diabetes complicated by hydramnios was not associated with increased risk of perinatal morbidity and mortality. Eur J Obstet Gynecol Reprod Biol 2001;100:46-9.
- leguizamon G,Smith ,younis H,Nelson DM,Sadovsky Y. Enhancementof amniotic cyclooxygenase preterm type 2 activity in woman with preterm delivery associated with twins or polyhydramnios. Am J Obstet Gynecol 2001;184:117-22.

POLYHYDROMNIOS

- Mohsin H. Neonatal outcome as predicted by Biophysical profile. J Coll Physicians surg pak 2000;7:149-51.
- 10. Plating-kemp A, Ngu Yen T, Chang E, et al. **Idiopathic polyhydramnios and perinatal outcome.** Am J Obstet Gynecol 1999;181:1079-82.
- 11. Bryan M. **The fetal membranes and amniotic fluid.** In: Principles of Obstetrics, London Butterworth & Co; 1988:94-8.
- 12. Golan A, Wolman I. Langer R, David MP. Fetal malformation associated with chronic polyhydramnios in singleton pregnancies. Eur J Obstet Gynecol Report Biol 1992;47:185-8.
- 13. Desmedt E, Henry OA, Steinberg Lh. Beischer NA. Acute and subacute polyhydramnios in singleton pregnancies. Aust N Z J Obstet Gynaecol 1990;30;191-5.
- Hotta M. Ishimatsu J. Manabe A, Hamada T, Yakushiji m. Polyhydramnios; ultrasonic detection of fetal and maternal condition. Kurume Med J 1994;41;31-6.
- Biggiro JR Jr, Wenstrom KD, Dubard MB, Cliver sp. Hydramnios, prediction of adverse perinatal outcome. Obstet Gynecol 1999;94:773-7.
- 16. Many A, Hill LM, Lazebnik N, Martin JG. **The association between polyhydramnios and preterm delivery.** Obstet Gynecol 1995;86;389-91.

Article received on: 03/02/2010

Correspondence address: Dr. Saadia Tariq Senior Rgistrar, Gynae Unit 1, Lahore General Hospital, Lahore saadiatariq@ymail.com

- 17. Phelan JP, Park YM, Ahn MO, Rutherford SE. **Polyhydramnios and perinatal** 1990;10;347-50.
- Smith CV, Plambeck RD. Rayburn WF, Albaugh KJ. Relation of mild idiopathic polyhydramnios to perinatal outcome. Obstet Gynecol 1992;79;387-9.
- 19. Thompson O, Brown R, Gunnarson G, Harrington K. **Prevalence of polyhydramnios in population screened by first and second trimester ultrasonography.** J Perinat Med 1998;26:375-77.
- Glantz JC, Abramovicz Js, Sherer Dm. Significance of idiopathic mid trimester, polyhydramnios. Am J Perinatal 1994;11:305-8.
- 21. Lazehink N, Hill IM, Guzick D, Martin JG, Many A. Severity of polyhydramnios does not affect the prevalence of large for gestational age newborn infants, J Ultrasound Med 1996;15;385-8.
- 22. Nordstrom L. Westgren M. Indomethacin treatment for polyhydramnios. Effective but potentially dangerous. Acta Obstet Gynecol Scand 1992;71;239-41.
- Mohen D. Newnham JP, D'Orsogna I. Indomethacin for the treatment of polyhydramnios. A case of constriction of the ductus arteriosus. Aust N Z J Obstet Gynaecol 1992;32;243-6.
- Phelan JP. Park YM. Ahn MO. Rutherford SE. Polyhydramnios and perinatal outcome. J Perinatol 1990;10;347-50.

Tariq S, Tarique N, Ahmad A, Malik M, Aleem S.

Polyhydramnios; Study of causes and fetal outcome.

Professional Med J Dec 2010;17(4):660-664.

Received after proof reading: 22/10/2010

Wars are not won by evacuations.

Article Citation:

Accepted for Publication: 08/07/2010

Winston Churchill

Professional Med J Dec 2010;17(4): 660-664.