

Perinatal Outcome in Idiopathic Polyhydramnios

Naser Omar Mustafa Malas, JBOG* Tameem Mazen Mohammed Jayousi, JBOG*
Mohammed Fehmi Miqdadi, JBOG* Wa'el Odeh Mahmood Ma'ani, JBOG*

Objective: To determine if idiopathic polyhydramnios is associated with adverse perinatal outcome.

Design: A retrospective study.

Method: Sixty-nine women with singleton pregnancies who were discovered to have idiopathic polyhydramnios and who were delivered in a period of sixteen months (July 2002-October 2003). These were compared with 150 pregnant women with normal amount of liquor. Analytic study of preterm delivery (<37 weeks gestation), low birth weight (<2.5 kg), macrosomia (>4.0 kg), malpresentation, Apgar score at 5 minutes <7, rate of C/S delivery, neonatal hospitalization and death was considered. Analysis was done using X² test.

Results: This study showed an increase in malpresentation, C/S and Macrosomia.

Conclusion: Antenatal diagnosis of polyhydramnios requires careful search for associated underlying maternal and fetal conditions. Adverse perinatal outcomes are less in idiopathic polyhydramnios than in polyhydramnios due to a known cause.

Bahrain Med Bull 2005;27(1):

Polyhydramnios may be defined as an amniotic fluid index above the 95th centile for gestational age¹. It complicates approximately 0.4-3.5 % of pregnancies and it can be divided into three groups: mild (amniotic fluid index 25-30), moderate (AFI 30.1-35) and severe (AFI >35)^{2,3}.

Polyhydramnios may occur as a result of a variety of fetal, maternal and placental abnormalities⁴ These include major congenital abnormalities, chromosomal aberrations, multiple gestations, maternal diabetes and Rh. isoimmunisation. In about 65% of cases none of these can be identified and a diagnosis of idiopathic polyhydramnios can be made⁵.

Abnormal increase in amniotic fluid volume has been associated with increased frequency of perinatal morbidity and mortality rates such as pre-maturity, low birth-weight and perinatal death. Idiopathic polyhydramnios is not necessarily associated with higher rates of poor outcome⁶.

* Fetal medicine subspecialty from Queen Charlotte's Hospital
Princess Haya Hospital
Royal Medical Services
Jordan

The purpose of this study is to determine if idiopathic polyhydramnios is associated with an increased hazard to the fetus.

METHODS

During a period of sixteen months from 20 July 2002 to 20 October 2003 total of 2142 deliveries took place at our hospital. Amniotic fluid volume was assessed ultrasonographically using the 4-quadrant method of Phelan et al³. Patients whose AFI was greater than 24 cm were diagnosed as having polyhydramnios. These were followed up weekly to identify and exclude known cause of polyhydramnios. Exclusion criteria consisted of any of the following: 1) congenital anomalies; 2) multiple gestation; 3) placental abnormalities; 4) diabetes mellitus; and 5) Rh.isoimmunisation. The same ultrasonographic database was also used to select 150 matched control subjects with normal AFI and no evidence of any of the exclusion criteria. The two groups were then compared.

Clinical endpoints studied were macrosomia (>4.0 kg), low birth-weight (<2.5kg), pre-term delivery (<37 weeks), malpresentation at delivery, C/S rate, neonatal death, Apgar score at 5 minutes <7 and admission to neonatal ICU. The rate of each outcome was calculated and the two groups compared with the χ^2 test. $P<0.05$ was considered significant.

RESULTS

Hundred and three (4.8%) were found to have polyhydramnios. Among these there were 69 cases of idiopathic polyhydramnios representing 67%. Other causes were diabetes mellitus occurring in 24.4%, congenital abnormalities and multiple gestation each accounting for 3.9% and Rh. isoimmunisation accounting for about 1%. Idiopathic polyhydramnios was further classified into mild polyhydramnios (AFI 25-30 cm), which accounted for 84% and moderate polyhydramnios (AFI 30.1-35 cm) in 16%. There were no cases with severe polyhydramnios (AFI >35 cm).

Outcome measures were summarized in the table.

Table 1. Outcome measures

Outcome measure	Study Group (No. =69)		Control Group (No.=150)	
	No.	%	No.	%
Macrosomia(>4.0Kg)	14	20.3	11	7.3
Malpresentation	8	11.6	4	2.7
C/S	17	24.6	13	8.7
Pre-term delivery (< 37 Wk)	6	8.7	12	8
Low Birth Wt.<2.5 Kg)	5	7.2	12	8
5 Min. A/S <7	2	2.9	4	2.7
Neonatal ICU admission	4	5.8	8	5.3
Neonatal death	0	0.0	0	0.0

The most striking difference was the high incidence of malpresentation in the study group. There were 8 (11.6%) cases of malpresentation, which is more than 4 times than the 2.7% rate in the control group ($p<0.01$). There was also an increase in the incidence of macrosomia and caesarian section, which were around three times more common in the idiopathic

polyhydramnios group than in the control group ($p < 0.01\%$). However, the rate of both pre-term delivery and low birth-weight were similar in the two groups. Similarly, there was no significant difference in the incidence of low apgar score at 5 minutes or the admission to neonatal ICU. There was no perinatal death in either group.

DISCUSSION

This study shows that polyhydramnios is more common in our community than what was shown in other studies^{2,7}. However, similar to other previous studies, idiopathic polyhydramnios accounted for 67% of cases and 84% of these were mild^{5,6}. Although many authors found an increased frequency of both maternal and fetal complications in cases of polyhydramnios, this does not necessarily hold true for idiopathic polyhydramnios^{8,9}. In the study of Chamberlain et al polyhydramnios was found to be associated with higher rate of both maternal and perinatal complications¹⁰. Similarly, Phelan et al found increased incidence of pre-term delivery rate in patients with polyhydramnios¹¹. However, neither of these studies was limited to idiopathic polyhydramnios. Many et al found an increased rate of pre-term delivery in patients with known cause of polyhydramnios but not in idiopathic cases^{2,8}. Panting-kemp et al studied the effect of idiopathic polyhydramnios on perinatal outcome and did not find an increased incidence of preterm delivery, low birthweight or perinatal death⁶. They stated that the lack of poor outcome may be related to the fact that most cases in their study group were in the mild polyhydramnios range. The results of our study are consistent with those of Panting-kemp et al⁶. Most of the cases were in the mild range and the outcome might have been different if there had been more moderate and severe cases of idiopathic polyhydramnios.

This study was similar to several previous studies in showing a high incidence of macrosomic infants^{6,9-14}. The reason for this association is not clear, since all patients in our study group were screened for gestational diabetes. Smith et al suggested that this could be due to subclinical glucose intolerance causing both the polyhydramnios and macrosomia or an increased fetal urine production due to greater fetal size⁹.

The rate of malpresentation in our study was higher than that in previous studies for both the study group and the control group⁶. Both macrosomia and malpresentation contributed to the increased incidence of C/S in our study group. However, Gonen et al found that early induction of labor in macrosomic infants was not effective in preventing C/S for cephalopelvic disproportion¹⁵.

CONCLUSION

This study showed that apart from the increased incidence of macrosomia, malpresentation and C/S, idiopathic polyhydramnios does not seem to have adverse perinatal outcome. The optimum management for these pregnancies is yet to be settled. Meanwhile, they should probably be managed as any other normal pregnancy.

REFERENCES

1. Moore TR, Cayle JE: The amniotic fluid index in normal human pregnancy. *Am J Obstet Gynecol* 1990;162:1168.
2. Many A, Hill LM, Lazebnic N, et al. The association between polyhydramnios and preterm delivery. *Obstet Gynecol* 1995;86:389-91.

3. Phelan JP, Smith CV, Broussard P, et al. Amniotic fluid volume assessment with the four quadrant technique at 36-42 weeks gestation. *J Reprod Med* 1987;32:540-2.
4. Desmedt EJ, Henry OA, Beischer NA. Polyhydramnios and associated maternal and fetal complications in singleton pregnancies. *Br J Obstet Gynecol* 1990;97:1115-22.
5. Hill LM, Breckle R, Thomas ML, et al. Polyhydramnios ultrasonically detected prevalence and neonatal outcome. *Obstet Gynecol* 1987;69:21-5.
6. Panting-Kemp A, Nguyen T, Chang E, et al. Idiopathic polyhydramnios and perinatal outcome. *Am J Obstet Gynecol* 1999;181:1079-82.
7. Cardwell MS. Polyhydramnios: A review. *Obstet Gynecol Surv* 1987;42:612-7.
8. Many A, Lazebnik N, Hill LM. The underlying cause of polyhydramnios determines prematurity. *Prenat Diagn* 1996;16:55-7.
9. Smith CV, Plambeck RD, Rayburn WF, et al. Relation of mild idiopathic polyhydramnios to perinatal outcome. *Obstet Gynecol* 1992;79:387-9.
10. Chamberlain PF, Manning FA, Morrison I, et al. Ultrasound evaluation of amniotic fluid volume II. The relationship of increased amniotic fluid volumes to perinatal outcome. *Am J Obstet Gynecol* 1984;150:250-4.
11. Phelan JP, Park YW, Ann MO, et al. Polyhydramnios and perinatal outcome. *J Perinatol* 1990;10:347-50.
12. Varma TR, Bateman S, Patel RH, et al. The relationship of increased amniotic fluid volume to perinatal outcome. *Int J Gynecol Obstet* 1988;27:327-33.
13. Lazebnik N, Hill LM, Guzick D, et al. Severity of polyhydramnios does not affect the prevalence of large-for-gestational-age newborn infants. *J Ultrasound Med* 1996;15:385-8.
14. Benson CB, Coughlin BF, Doubilet PM. Amniotic fluid volume in large-for-gestational-age fetuses of nondiabetic mothers. *J Ultrasound Med* 1991;10:149-51.
15. Gonen O, Rosen DJ, Dolfin Z, et al. Induction of labor versus expectant management in macrosomia: a randomised study. *Obstet Gynecol* 1997;89:913-7.