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Clinical and Ultrasonographic Evaluation of Polyhydramnios

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OBJECTIVE - To diagnose associated fetal anomalies and to analyse the maternal and fetal complications in polyhydramnios confirmed by ultrasonogram. **METHODS -** One hundred pregnant women between 20-42 weeks gestation clinically detected to have polyhydramnios were evaluated ultrasonographically and followed up till delivery. Symptomatic women received indomethacin upto 34 weeks gestation. Women with acute symptoms also had therapeutic amniocentesis. Mode of and gestational age at delivery were based on obstetric criteria. **RESULTS -** Majority of women (71%) were in the age group of 25-30 years and 63% were multigravidas. Diagnosis of polyhydramnios was made in 40% women only at 37-40 weeks gestation because they never had antenatal check up. Only 12% women were symptomatic and needed treatment. No definite etiology was identified in 40% women, 40% had associated fetal anomalies and 8% had diabetes mellitus. Most common anomalies seen were central nervous system anomalies (52.5%); anemia (27%), hypertension and intrauterine death were common antenatal complications. Over 49% of women had spontaneous vaginal delivery and 34% underwent caesarean section. There were 36 perinatal deaths, fetal anomalies being the commonest cause. **CONCLUSION -** Polyhydramnios is associated with fetal anomalies in about 40% women, it can be evaluated using ultrasonogram. If fetal anomalies are not compatible with life, termination is advocated. In symptomatic women the underlying cause has to be treated; decompressive amniocentosis and indomethacin therapy can help in prolonging pregnancy. When fetal anomalies are diagnosed, it is essential to discuss with paediatric surgeons and to counsel the woman and her husband.

Key words : polyhydramnios, fetal anomalies

Introduction

Polyhydramnios occurs in 0.4 to 1.2% of all preganancies. It is defined as an amniotic fluid volume of more than 2000 ml[1]. It is usually a clinical diagnosis confirmed ultrasonographically. An abnormal increase in amniotic fluid volume has been associated with increased frequency of both maternal and fetal complications with increased perinatal morbidity and mortality due to congenital anomalies and prematurity. So it is important to diagnose this condition in the antenatal period itself to reduce maternal and fetal morbidity and mortality.

Present study was undertaken using a real time ultrasound to measure amniotic fluid volume in a semi - quantitative manner using either a single vertical pocket or amniotic fluid index and also to do a detailed scan to diagnose congenital anomalies and to evaluate maternal and fetal outcome.

Material and methods

This is prospective descriptive study of 100 cases of polyhydramnios over one and half years (1999 – 2000).

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Study group included all women between 20 and 42 weeks of gestation detected to have polyhydramnios clinically and were evaluated by ultrasound. Cases of rupture of membranes and gestational age less than 20 weeks were excluded from the study. The deepest unobstructed and clear pocket of amniotic fluid was measured in all four quadrants. Maximum vertical pocket of 8 cm. or more and or amniotic fluid index of 20 cm. or more was taken as polyhydramnios. A detailed ultrasonographic evaluation of the fetus for structural anomalies was done. Those with major anomalies not compatible with life were terminated. Others were followed up during pregnancy and labour. Maternal and fetal outcome was evaluated.

Observations

Polyhydramnios was seen commonly in the age group of 21-30 years (71%). Most common gestational age at which hydramnios was diagnosed was between 37 to 40 weeks (40%). Diagnosis at this late stage was due to lack of antenatal care and most patients presented only at term. History of consanguinity was present in 11% of cases, 72% were multigravidas and majority were asymptomatic. Few patients had presented with overdistension of abdomen with difficulty in breathing (12%).

Forty percent patients were idiopathic, 40% had associated congenital anomalies, and another 8% were diabetic (Table I).

Table I : Etiological Factors

Factors	No. (Also %)
Idiopathic	40
Diabetes mellitus	8
Multiple pregnancy: monozygotic	4
Multiple pregnancy: dizygotic	2
Rh -ve pregnancy	6
Congenital anomalies	40
Total	100

Table II : Associated Antenatal Complications

Complications	No. (Also %)
Anemia	27
Malpresentation	22
Breech	10
Face	7
Transverse	5
Preterm labour	16
Pregnancy induced hypertension	15
Intrauterine death	10
Premature rupture of membranes	6
Placenta previa	3
Abruption	1
Eclampsia	1
Total	100

Commonest antenatal complications were anemia (27%) and malpresentation (22%) (Table II). Thirteen percent patients had lower segment caesarean section in previous pregnancies.

Majority of the patients were managed conservatively (53%). Indomethacin 25 mg 8 hourly was given to 12% of the patients and 5% required indomethacin as well as amniocentesis. Remaining 30% of the patients came in labour and delivered within 24 hours after admission. Forty four percent patients went into spontaneous labour, labour was induced in 42% and elective cesarean section was done in 14%. Most common indication for induction was congenital anomalies of the fetus (36%) followed by postdating (24%) and intrauterine death (24%) (Table III). Table IV shows indications for lower segment cesarean section. Most common indications were cephalopelvic disproportion and malpresentation. Main intrapartum complications were CPD (14%) and abruption (5%). Three percent patients had postpartum haemorrhage.

Table III: Indications for Induction

Indications	No	%
Congenital anomalies	15	35.71
Postdatism	10	23.8
Intrauterine death	10	23.8
Pregnancy induced hypertension	6	14.28
Antepartum eclampsia	1	2.38
Total	42	100

Table IV: Indications for Lower Segment Cesarean Section

Indications	No.	%
Cephalopelvic disproportion	14	41.18
Malpresentation	11	32.36
a) breech	6	17.64
b) transverse lie	5	14.70
Placenta previa	3	8.82
Cord prolapse	3	8.82
Twins – Monoamiotic twins with single fetal demise	1	2.94
Fetal distress	1	2.94
Abruption	1	2.94

Table V: Congenital Anomalies

Anomalies	No.
Central nervous system	
Hydrocephalus	6
Hydrocephalus + sacral meningocele	2
Hydrocephalus + spina bifida	2
Anencephaly	6
Anencephaly + meningocele + encephalocole	2
Anencephaly + spina bifida	1
Microcephaly + encephalocele + cervical meningocele	1
Microcephaly + exomphalos+ iniencephaly+low set ears	1
Total central nervous system anomalies	21
Gastrointestinal tract	
Tracheoesophageal fistula	2
Diaphragmatic hernia	2
Omphalocele	1
Exomphalos	1
Hepatomegaly	1
Total Gastrointestinal tract anomalies	7
Genitourinary tract	
Polycystic kidneys	1
Micropenis	1
Ambiguous genitalia + hypospadias	1
Micropenis + hypoplastic scrotum + micrognathia + bilateral congenital talipes equinovarus	1
Total Genitourinary anomalies	4
Others	
Hydrops fetalis	5
Pierrer Robin syndrome	2
Micrognathia	1
Total	8
Total	**40**

There were 36 perinatal deaths, commonest cause being associated fetal anomalies (19%); other causes being prematurity (5%) and hydrops fetalis (5%). Table V lists the congenital anomalies seen in 40 new borns. CNS anomalies were more commonly seen. Diagnosis was made by ultrasound examination and clinical examination of the infant after delivery.

Discussion

A pregnancy complicated by polyhydramnios presents a difficult diagnostic and therapeutic dilemma for the obstetrician though the advent of ultrasound makes diagnosis and evaluation of fetal anomalies easy.

In the present study of hundred patients the diagnosis of polyhydramnios was confirmed using ultrasonological criteria of single vertical pocket measurement of 8 cm or more and amniotic fluid index of 20 cm. or more. The commonest age group in our study was 21-30 years (71%) and majority of our patients were diagnosed between 36 to 40 weeks which is because of lack of antenatal care and late referrals. Mamopoulus et al found that the mean age was 28.9 years and the gestational age at diagnosis was 27.4+-2.79 weeks.

Stoll et al found an association of 5.1% consanguinity in their patients. Our study showed 11% association, majority of women were married to their maternal uncle. Seventy two percent of our patients were multigravidas; 93% had mild hydraminos and 5% severe. Many et al[4] found in their study of 275 patients that the incidences of mild, moderate and severe hydraminos were 72.3%, 20% and 7.7% respectively.

The commonest cause of polyhydramnios in our study was associated anomalies (40%) and idiopathic conditions 40%. Most mild cases are idiopathic and in severe cases usually aetiological factor was identifiable. Chetrit et al[5] found that 60% of the cases were idiopathic. In idiopathic cases, excess fluid is believed to be due to an unexplained imbalance in water exchange between the fetal placental unit and the amniotic fluid. Carlson et al[6] reported that only 16% of mild hydraminos had a cause, where as in 91% of severe cases etiological factor was definable.

Forty percent of our cases had associated congenital anomalies. Chetrit et al[5] showed 19% associaton and Queenan and Gadow[7] showed 20% association. The high incidence in our study may be due to referral after being diagnosed. The most commonly affected system in our study was central nervous system (52.5%) followed by gastrointestinal tract (17.5%) and genito urinary tract (10%). Hydrocephalus was found in 10 cases and anencephaly with other associated anomalies in 9 cases.

In a study conducted by Stoll et al[3] the most common congenital defects associated with polyhydramnios were congenital heart disease, musculoskeletal malformations and anomalies of gastrointestinal tract. Prevalence of chromosomal abnormalities was 1.77%. In most cases the excess fluid is believed to be due to the development of an imbalance between the fetal urine production and removal of amniotic fluid by fetal ingestion.

Eight percent of our cases were associated with diabetes mellitus. Thompson et al (1998) found 10.8% of their cases were associated with diabetes mellitus. Carlson et al reported 14% association. Exact aetiology of polyhydramnios in diabetic patients is unknown. One explanation is that maternal hyperglycemia causes fetal hyperglycemia that results in osmotic diuresis. Other causes are rise in amniotic fluid osmolality due to increased glucose level and decreased fetal swallowing.

Hydramnios is associated with increased incidence of maternal complications. Desmedt et al[7] in their study of 537 patients found that 17% had pregnancy induced hypertension, 4% anemia, 2.6% placenta previa, 2.2% abruption and 0.4% Rh-isoimmunization. Our study showed 27% anaemia, 22% malpresentations, 16% preterm labour, 10% intrauterine death, 3% placenta previa and 1% abruption. Twelve patients who presented with pressure symptoms were given indomethacin 25 mg. thrice daily; five of these required amniocentesis also to relieve acute symptoms. There was significant reduction in amniotic fluid index one week after therapy. None of them developed oligohyramnios or fetal complications. Deeny[10] used 25 mg six hourly with a reduction in dosage following ultrasound assessment of amniotic fluid index. Mamopoulus et al[2] proposed that indomethacin can be discontinued when the pretreatment amniotic fluid index is reduced by more than two thirds as continuing treatment can lead to oligohyramnios. Mogilner et al found that long term use of indomethacin especially late in gestation has been associated with fetal hydrops and neonatal persistence of fetal circulation. Morse[11] found in a series of the fetuses, whose mothers were receiving indomethacin for premature labour, 50% had duct constriction with three cases of tricuspid regurgitation. Risk of ductal constriction was found to be approximately 5% before 27 weeks of gestation which increases to 50% by 32 weeks. These studies proved the necessity of fetal echocardiogram in patients who receive indomethacin for long duration.

In our study 44% had spontaneous labour, 42% were induced and 14% had elective cesarean section. Common indications for induction were congenital anomalies of the fetus (36%), and intrauterine death (24%). Desmedt et al[7] reported spontaneous labour in 58% and in 16% labour was induced for major anomaly or fetal death.

our study 49% had spontaneous vaginal delivery, 34% had cesarean section, 13% instrumental deliveries and 4% assisted breech delivery. Three percent patients had PPH. The common indications for cesarean section were cephalopelvic disproportion and malpresentations. Desmendt et al found in their study of 537 patients, that 33% had spontaneous vaginal delivery, 41% had LSCS, 12% assisted vaginal delivery and 12% forceps delivery. Common indications for LSCS were cephalopelvic disproportion, obstructed labour and severe PIH.

There are 36 perinatal deaths in our study, 28 were stillbirths and 8 were neonatal deaths. In 16 perinatal deaths birth weight was less than 1500gms. Forty babies had congenital anomalies of which 10 had hydrocephalus, anencephaly and 7 GI tract anomalies. Biggio et al in their comparative study found that perinatal mortality rate in women with hydramnios was 89 per 1000 births compared to 14 per 1000 births in the control group. They found that women with hydramnios had 5 times more anomalies than controls. They also found that the cesarean section rate was three times higher in women with hydramnios compared with controls. Carlson et al reported similar incidence of congenital anomalies as our 44% of which 27% had fetal aneuploidy. Desmendt et al in their study of 537 cases of polyhydramnios found 18% associated anomalies of which 31% were in CNS of which almost half were anencephaly.

References

1. Queenan J, Gadow E. Polyhydramnios: Chronic versus acute. *Am J Obstet Gynecol* 1971; 109: 349–55.
2. Mamopoulos M, Vasmakopoulos E, Reece EA et al. Maternal Indomethacin therapy in the treatment of Polyhydramnios. *Am J Obstet Gynecol* 1991; 165: 1233–9.
3. Stoll C G, Alembik Y, Dott B. Study of 156 cases of Polyhydramnios and congenital malformations in a series of 118, 265 consecutive births. *Am J Obstet Gynecol* 1991; 165: 586–90.
4. Many A, Hill LM, Lazebnik M et al. The association between polyhydramnios and preterm delivery. *Am J Obstet Gynecol* 1993;86:389–91.
5. Chetrit AB, Celnikier DH, Ron M, et al. Hydramnios in the third trimester of pregnancy: A change in the distribution of accompanying fetal anomalies as a result of early Ultrasonographic prenatal diagnosis. *Am J Obstet Gynecol* 1990; 162: 1344–5.
6. Carlson DE, Platt LD, Me Dearis AL, et al. quantifiable polyhydramnios: Diagnosis and management. *Obstet Gynecol* 1990; 75, 989–93.
7. Thompson O, Brown R, Gunnarson G, et al. Prevalence of polyhydramnios in the third trimester in a population screened by first and second trimester ultrasonography. *J Perinat Med* 1998; 26: 371–7.
8. Wallenberg HC S, Wladimoroff VW. The amniotic fluid II polyhydramnios and oligohydramnios. *J Perinat Med* 1977; 195–206.
9. Desmendt FJ, Henry OA, Berscher MA. Polyhydramnios and associated maternal and fetal complications in singleton pregnancies. *J Obstet Gynecol* 1990; 97: 1115–22.
10. Deny M. Indomethacin use to control gross polyhydramnios complicating triplet pregnancy. *J Obstet Gynecol* 1995; 100: 281–2.
11. Mogilner BM, Ashkenazy M, Borenstein R et al. Hydrops fetalis caused by maternal indomethacin treatment. *Acta Obstet Gynecol Scand* 1982; 61: 183–5.
12. Moise KJ Jr. Polyhydramnios. *Clin Obstet Gynecol* 1997; 40: 268–79.
13. Biggio JR, Wenstrom KD, Du Barb MB et al. Hydramnios - prediction of adverse perinatal outcome. *Obstet Gynecol* 1998; 91: 723