www.jmscr.igmpublication.org Impact Factor 3.79
ISSN (e)-2347-176x

IGM Publication Journal Of Medical Science And Clinical Research
An Official Publication Of IGM Publication

Maternal and Fetal Outcome in Cases of Oligohydramnios Diagnosed By Ultrasonography after 34 Weeks of Gestation

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Abstract

The present study "obstetric outcome in cases of oligohydramnios diagnosed after 34 weeks of gestation" is a prospective study and Silchar Medical College & Hospital, Silchar during the period 1st August 2012 to 31st July 2013.

A total of 200 patients were studied from the antenatal cases attending the OPD, antenatal ward and labour room of SMCH, Silchar. 100 cases and 100 controls were identified and they were confirmed ultrasonographically.

All the cases were singleton pregnancies after 34 weeks of gestation in the age group of 18-32 years. Single deepest liquor pocket was < 3 cm (Crowley and O'herlihy)

All the controls were singleton pregnancies after 34 weeks of gestation in the age group 18-32 years. They had single deepest liquor pocket depth between 3-8 cm.

Majority of cases (60%) and controls (58%) were in the age group 23-27 years. The mean age of incidence was 24.6 years.

Most of the cases (70%) and controls (72%) were primigravidas

88% cases and 86% controls were booked cases.

Patients were monitored carefully for mode of onset – spontaneous or induced, course of labour, mode of delivery, caesarean section etc.

To note the fetal outcome, Apgar score of 1 and 5 min, birth weight, incidence of C.S for fetal distress, NICU admissions, stillbirth, neonatal deaths etc. were monitored. Statistical analysis was done between case and control groups.

Fundal heights and abdominal girths were considerably lower in cases than the control groups. This association was found to be statistically significant ($p<0.05$)

30% cases had associated hypertensive complications as compared to 26% controls. Anemia was seen in 22%, Rh negative in 6% and others in 4%. But most common group was uncomplicated or idiopathic with

38% cases and 43% controls. No significant relationship between oligohydramnios and any of these complications was noted. (p>0.05).
18 % cases and 14% controls had malpresentation at the time of delivery.
Only 16% cases delivered spontaneously among cases whereas 83% controls delivered spontaneously. 22% cases were induced and the corresponding rate among controls was 5%. Oligohydramnios was significantly associated with increased rates of induction (p<0.05).
The rates of C.S were 62% among cases compared to 12% in controls. 70.96% of these was due to fetal distress in the case group whereas only 16.67% in the control was due to fetal distress. The rates of C.S were significantly higher in cases (p<0.05)
Liquor was meconium stained in 48% cases and 12% controls. 93.5% of C.S in cases was emergency compared to 83.3% in controls.
The percentage of cases with birth weight <2.5 kg was 42% compared to only 12% controls. Also, 1-min apgar score <7 was seen in 70.44% of cases and only 19.08 % controls. 5-min apgar score <7 was seen in 37.46% of cases where as it was only 5.14% in controls. The association was found to be statistically significant (p<0.05)
The NICU admissions were required in 33% cases ageist 5% controls.
It was observed that adverse maternal and fetal outcome were associated with the cases i.e., oligohydramnios which was statistically significant.

AIMS AND OBJECTIVES

The study was conducted during a period of 12 months from 1st August 2012 to 31st July 2013. The study included 100 cases of oligohydramnios and 100 controls (of normal amniotic fluid volume) after 34 weeks of gestation. The intrapartum courses of labour and fetal outcome were monitored.

The aims and objectives of the study are :

(i) To know the obstetric outcome in cases of oligohydramnios diagnosed after 34 weeks of gestation.

(ii) To Assess whether oligohydramnios is associated with adverse maternal and fetal outcome.

INTRODUCTION

Amniotic fluid surrounds the fetus after few weeks of gestation and serves several roles during pregnancy. It creates a physical space for the fetal skeleton to shape normally, promotes normal fetal lung development and helps to avert compression of the umbillical cord.

Oligohydramnios is defined as a condition where liquor amnii is deficient in amount.

Sonographically, it is defined as an amniotic fluid index (AFI) of less than 5 cm (10th centile)[1] or maximum vertical pocket of liquor less than 2cm.[2]

It commonly affects 1-5% of all pregnancies and the overall incidence is 3.9% of all pregnancies.[3]

Previously, Manning et al described oligohydramnios as single largest pocket on ultrasound less than 1 cm. Later on it was revised to be 2 cm in both vertical and horizontal planes.[4]

Marks and Divon et al., (1982) have also defined oligohydramnios as an AFI less than 5cm on ultrasound examination.[5]

Amniotic fluid is in a dynamic state with an equilibrium being achieved in its formation and removal.

Aberrations in amniotic fluid volume, both low (oligohydramnios) and high (polyhydramnios) are associated with a multitude of pregnancy related problems. An understanding of the basic physiologic mechanisms that regulate both amniotic fluid volume and composition is required to devise effective strategies for pregnancies complicated by disorders of amniotic fluid volume.

Oligohydramnios is a significant cause of maternal and perinatal adverse outcomes.[6]

Maternal complications[7] :

Oligohydramnios per se does not lead to maternal complications; but it may be a complication of delivery, increased rates of intervention and malpresentation.

- Increased rates of induction
- Prolonged labour due to inertia
- Increased operative interference due to manipulation

Fetal complications[7] :

- Abortions
- Deformity due to intraamniotic adhesions or due to compression. These include alteration in the shape of the skull, wry neck, club foot or even amputation of limb.
- Fetal pulmonary hypoplasia(may be cause or effect)
- Cord compression
- Increased rates of labour induction and operative delivery
- Intrauterine growth retardation (IUGR)
- NICU admissions
- Fetal death

Polyhydramnios means “excess of amniotic fluid”. Traditionally polyhydramnios has been considered to be present when amniotic fluid volume exceeds 2000 ml. Although minor degrees of polyhydramnios are common, more severe cases of polyhydramnios are distinctly less frequent.[75]

Several large studies have reported the incidence to be 0.2-1.6%.[76,117] In recent days, ultrasonography has developed into a new science of fetal medicine, where the fetus is considered as a patient. Since long, the aim of the obstetricians was safety of the mother only. This has resulted in tremendously reduced mortality over years. But now, the recent trend is to not only deliver a live fetus but also a healthy fetus which is structurally normal and not asphyxiated. Only then a healthy adult can grow. It is the obstetricians’ job to unravel every information regarding the fetus in utero. A reasonably good ultrasound machine, a comprehensive training and good clinical practice will enable him to find out all the queries towards delivering a better human being.

The ultrasound machine primarily has a transducer, a signal processing part and a monitoring screen or a cathode ray tube (CRT). A transducer is a part on which several crystals having piezo electric effect are mounted. It is then capable of sending ultrasound signals and receiving echoes. The received echoes are

converted to electrical energy, processed and amplified and finally displayed as bright dots on the screen (B-mode).[79]

A grey scale of 32.64 are used for display which permits the use of 32.64 shades between black and white on the display screen, thus giving details of received echoes. By using more crystals on transducer and with electronically controlled excitation more frames of the picture can be produced continuously. This is real time B- scan.[80]

The unit of frequency of sound is Hertz, which is no. of cycles per second. Human ear is capable of hearing between 20-20,000 Hertz. One million hertz is a mega hertz (MHz). 2-15 MHz frequencies are used in medical ultrasound; abdominal transducers use a frequency of 2-6.5 MHz. while trans vaginal transducers use a frequency of 5-15 MHz. there.[80]

Certain crystals are there which can change the shape when electricity is passed through them, thus producing ultrasound. The same crystal on receiving ultrasound can convert it to electrical energy. This phenomenon of the ability to convert one form of energy to another is known as Piezo-electric effect.[81]

So far as the quality of ultrasonography is concerned, it is important to remember that a sonogram is composed of a number of lines of echo information. The larger the number of lines, the greater is the amount of anatomic details displayed.[82]

Evaluation of amniotic fluid is done by :

- Total intrauterine volume[77]
- Nonquantitative method[78]
- Quantitative :
  a) 1-cm pocket[4]
  b) 2 cm pocket[2]

  c)3 cm pocket[53]

  d) Two diameter fluid pockets[51]

  e) Amniotic fluid index[1]

In the past, maternal and fetal adverse outcomes were quite prevalent due to late diagnosis and inadequate management of condition. But with the advent of ultrasonography, early diagnosis is quite common and it has resulted in significant reduction in maternal and fetal complications.

Ultrasonography is a non invasive, effective and relatively simple tool to asses the amniotic fluid volume. A number of parameters like amniotic fluid index (AFI), single deepest pocket, two diameter amniotic fluid pocket have been devised to asses the liquor volume. Ultrasonographic evaluation of the liquor volume is also included in the biophysical profile (BPP); modified biophysical profile (MBPP) to asses the fetal wellbeing.

About 15-25% of cases of oligohydramnios are associated with congenital anomalies.[8] Ultrasonography has added to the diagnosis of these conditions also.

The prognosis for patients with early onset oligohydramnios remains poor because the two most common etiologies, preterm rupture of membranes and fetal congenital anomalies are not amenable to successful treatment. For patients with oligohydramnios after fetal viability, the management is usually dictated by the

gestational age. Expectant management may require intensive fetal monitoring along with serial ultrasound examinations for amniotic fluid volume, otherwise termination may be considered.

The present study analyses the maternal and fetal outcomes in cases of oligohydramnios as diagnosed by ultrasonography after 34 weeks of gestation with the existing facilities in our institution.

MATERIALS AND METHODS

Source of data :

The present study analyses and fetal outcomes in cases of oligohydramnios, diagnosed after 34 weeks of gestation. The study was carried out at the department of Obstetrics & Gynaecology of Silchar Medical College & Hospital, Silchar over a period of one year from 1st August 2012 to 31st July 2013.

Method of collection of data :

It is a prospective study conducted in the department of Obstetrics & Gynaecology of Silchar Medical College & Hospital, Silchar which is carried out in 100 cases of oligohydramnios diagnosed clinically and confirmed by USG. The cases and controls were studied to compare the maternal and fetal outcome. Maternal outcome was assessed in terms of mode of delivery, operative interventions, days of hospital stay etc. fetal outcome was studied in terms of of fetal distress, birth weight, NICU admissions, stillbirth etc.

The criteria for selection of cases were following detailed history taking regarding duration of amenorrhea, fetal movements, draining per vagina, previous obstetrical history regarding oligohydramnios, congenital anomalies, medical conditions like hypertension,anemia, diabetes etc. The proforma for history taking was duly followed.

Examination scheme included general examination which included evaluation for anaemia, oedema, obesity, B.P. measurement, icterus. Thorough systemic examination of central nervous, cardiovascular, respiratory systems were made.

On abdominal examination, height of fundus, abdominal girth, lie and attitude of fetus, its position and FHR were noted. Clinical palpation for amount of liquor was done and amount of liquor assessed. Presence of IUGR if seen is noted. It is followed by per vaginal examination to note the dilatation, effacement of cervix, the presentation, station, membrane status and type of pelvis. The examinations were done according to the proforma.

Patients were divided into two groups - **Cases (n=100) and Controls (n=100).**

CASES

Inclusion criteria :

The cases included 100 patients seen after 34 weeks of gestation, with oligohydramnios detected clinically and confirmed by single USG scan. The cases with singleton pregnancies after 34 weeks of gestation were included. Patients in the age group 18 – 32 years were included and younger or older were not included due to associated complicating factors with very young and elderly gravidas.

Criteria for lower limit of normal liquor volume was by single deepest pocket depth of 3 cm (as per Crowley and O'Herlihy, 1984) in this study.

CONTROLS

Inclusion criteria :

100 control patients were selected in the age group 18 – 32, after 34 weeks of gestation with singleton pregnancies. They had normal liquor volume as determined clinically and confirmed by single USG scan.

Single deepest liquor pocket of depth 3-8 cm was taken as normal adequate liquor volume.

Exclusion criteria :

For both cases and controls -

- ★ Patients with ruptured membranes
- ★ Postdated pregnancies
- ★ Intrauterine death of fetus
- ★ Congenital anomalies
- ★ Multiple pregnancies

After clinical estimation of liquor and making a presumptive diagnosis, patients were sent for single USG scan. The USG scan was done within prior 7 days of delivery. The criteria for labelling a case as oligohydramnios in this study was a single deepest liquor pocket of depth less than 3 cm (as per Crowley and 'Herlihy 1984).

This was followed by other routine antenatal investigations including :

Hb % , RBS , Routine urine examination including protein, sugar and microscopy, VDRL, HBsAg, HIV (voluntary), ABO and Rh typing etc.

Patients with isolated oligohydramnios without risk factors were managed conservatively with oral hydration, intravenous amino acid infusion, multivitamin supplementation, after ruling out fetal distress and maternal associated complications.

High risk cases of oligohydramnios were managed according to the gestational age, Bishop's score, associated HTN, labour pains etc. FHR monitoring was done. Labour was monitored with partograph. Accordingly, induction- augmentation scheme was employed.

For Bishop's score < 6.

Induction with tab. PGE1 (misoprostol), 25 μm was given 6 hourly to maximum of 6 doses.

For Bishop's score >6.

ARM with titrated oxytocin drip was given.

Strict monitoring of fetal condition during labour was done by FHR monitoring. Colour of liquor, progress and duration of labour and noted for appearance of fetal distress.

Accordingly delivery was conducted by vaginal or caesarean section. In such cases prompt delivery services and good neonatal care were given.

The newborn was immediately assessed in terms of birth weight, Apgar score at 1 min and 5 min, IUGR/ SGA etc. The fetal prognosis was followed in terms of NICU admissions, number of days of NICU stay, stillbirths, neonatal deaths etc.

The maternal and fetal outcomes were compared between the case and control groups and finally conclusions were drawn regarding the maternal and fetal outcome associated with oligohydramnios.

RESULTS AND OBSERVATIONS

The present study -

"OBSTETRIC OUTCOME IN CASES OF OLIGOHYDRAMNIOS DIAGNOSED AFTER 34 WEEKS OF GESTATION", was conducted in the department of Obstetrics & Gynaecology of Silchar Medical College & Hospital, Silchar, Assam during the period of one year from 01-08-2012 to 31-07-2013.

A total of 200 pregnant women, of >34 weeks of gestation were enrolled for the study. The patients were selected from the OPD, antenatal ward and labour room of SMCH. Each patient was subjected to a single USG scan and the single deepest pocket of amniotic fluid was measured.

The patients were divided into :

Cases (n=100) – with single deepest pocket < 3 cm

Controls (n=100) – with single deepest pocket 3-8 cm.

AGE DISTRIBUTION

Table 1: Showing age distribution of both groups

Age in years	Cases (n=100)	Controls (n=100)
18 -- 22	24	20
23 -- 27	60	58
28 -- 32	16	22

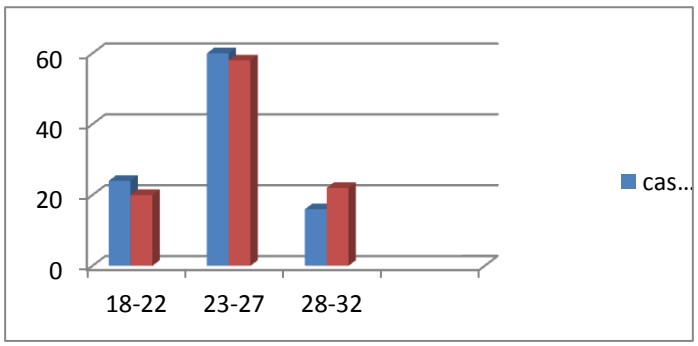


Fig.: A

From the table and the graph, it is clear about the age distribution between cases and controls. They were divided into three groups depending on age. Maximum number of cases(60 %) as well as controls(58%) were in the age group of 23-27 yrs. Very young(<18) and older(>32) patients were excluded from the study. Thus it can be said that the cases and controls were similar in age distribution. The mean age of incidence of oligohydramnios was found out to be 24.6 years with S.D. of 3.15 unit.

GRAVIDA DISTRIBUTION

Table 2 : Showing distribution of gravidas among cases and controls

Gravida	Cases (n=100)	Controls (n=100)
G1 (primi)	70	72
G2	15	17
G3 or more	15	11

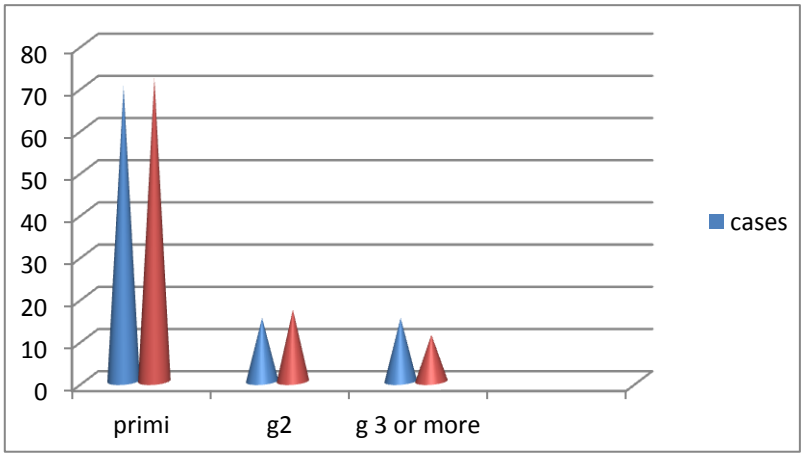


Fig. : B

The above table and graph show the gravida distribution among the cases and the controls. It is seen that the maximum no. of patients i.e, 70% cases and 72% controls were primigravida; whereas 15% of cases were 2nd gravida compared to 17% controls. 15 % cases and 11% controls were 3rd or higher order gravida. Thus it can be said that the cases and the controls were similar in terms of distribution of gravidas.

BOOKING STATUS

Table 3 : Showing booking status of cases and controls

Booking status	Cases (n=100)	Controls (n=100)
Booked	88	86
Unbooked	12	14

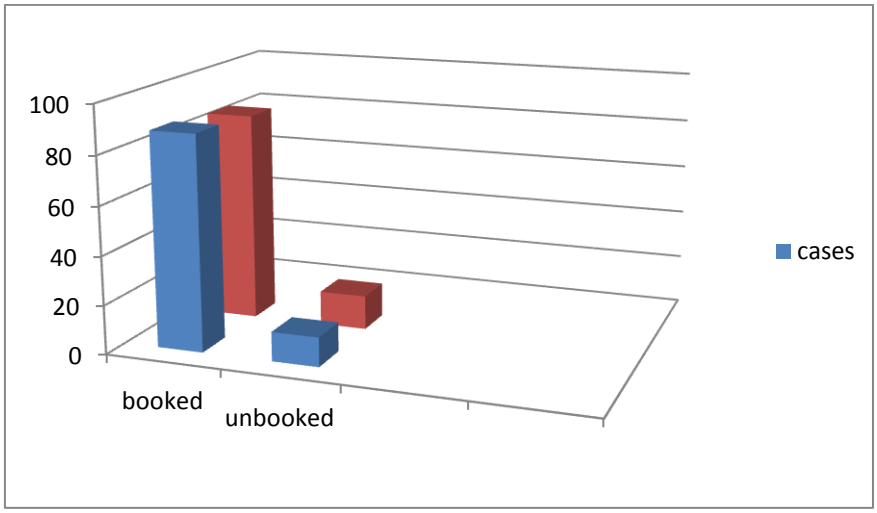


Fig. : C

The above table and graph show that 88% cases and 86% controls were booked cases. Thus the cases and the controls were similar in terms of their booking status.

Fundal heights

Table 4 : Showing distribution of fundal heights among cases and controls

Fundal height (in weeks)	Cases (n=100)	Controls (n=100)
< 32	24	01
32-36	74	38
>36	02	61

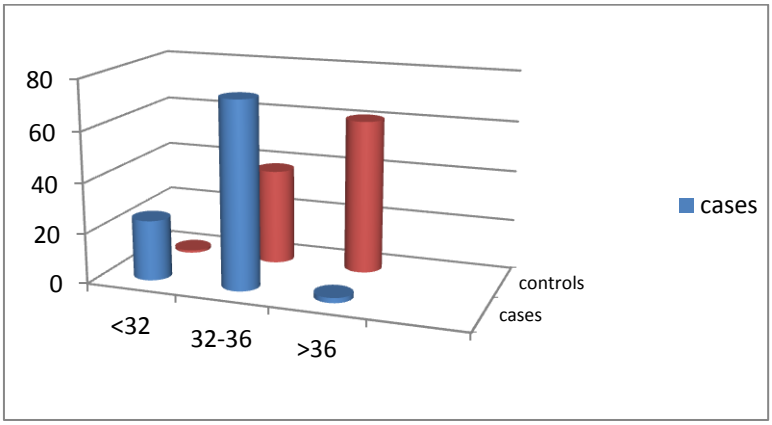


Fig. : D

From the above table and graph it is clear that among cases only 02% had fundal heights >36 weeks, whereas majority 74% were in the range 32-36 weeks and a significant 24% had heights <32 weeks. This is in comparison with the controls where 61% were having heights >36 weeks, 38% between 32-36 weeks and only 01% <32 weeks. The standardised z- test for difference of proportions was applied and the p value was found to be <0.05. Thus we can say oligohydramnios is associated with significant reduction in fundal heights.

ABDOMINAL GIRTH

Table 5 : Showing distribution of abdominal girth among cases and controls

Girth (in cms)	Cases (n=100)	Controls (n=100)
<80	88	07
80-100	12	93
>100	00	00

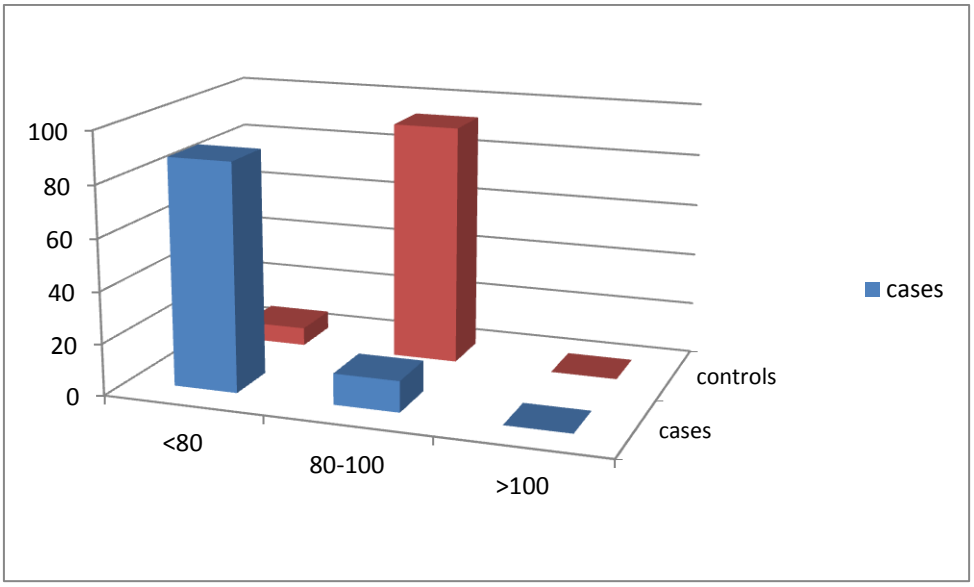


Fig. :E

The above table and graph shows that majority 88 % cases had girth <80 cm whereas only 12% cases had normal girth. Compared to controls where 93% had normal girths and only 07% had below normal girths. None of the cases and controls had girth >100cm. Statistical analysis was done and found that $p<0.05$(p two tailed).Thus it can be said that oligohydramnios is associated with significant risk of reduced abdominal girths.

ANTENATAL COMPLICATIONS

Table 6 : Showing distribution of antenatal complications

Complications	Cases (n=100)	Controls (n=100)
Hypertensive Disorders	30	26
Anemia	22	25
Rh negative	06	02
Others	04	04
Uncomplicated	38	43

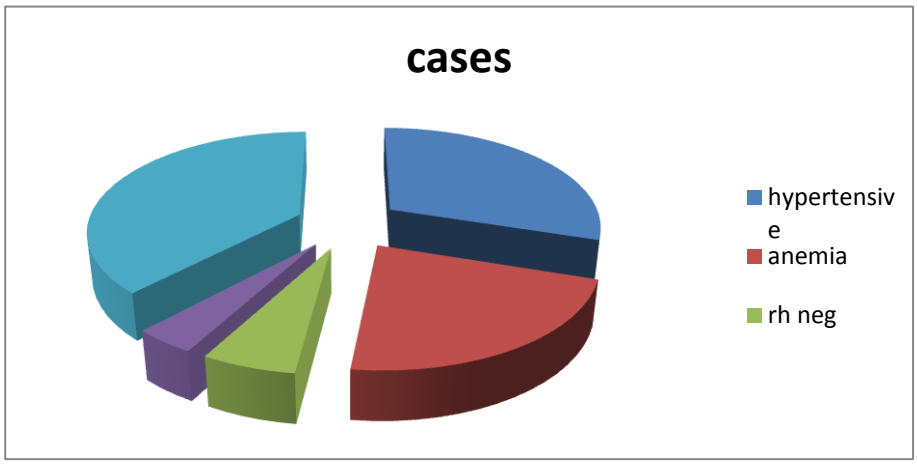


Fig. : F

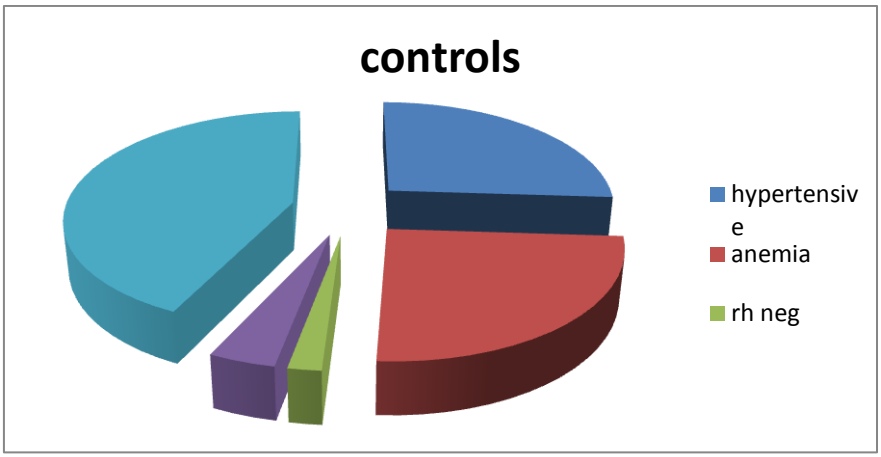


Fig. : G

The table and the pie chart show the associated antenatal complications among the cases and the controls. Maximum of cases (38%) and controls (43%) were uncomplicated. The most common complication was hypertensive disorders of pregnancy which comprised of 30% cases and 26% controls. This was followed closely by anemia which was seen in 22% cases and 25% controls. Rh neg pregnancy was seen in 06% cases and 02% controls. The others were 04% in both groups. Thus maximum of cases and controls were uncomplicated. The statistical analysis revealed that $p>0.05$, which showed that oligohydramnios is not significantly complicated by any of these complications. The p value was calculated for each of these factors and they were found to be insignificant. Thus association of complications was not significant.

DISTRIBUTION OF PRESENTATION

Table 7: Showing distribution of presentations

Presentation	Cases (n=100)	Controls (n=100)
Cephalic	82	86
Breech	17	11
Transverse/unstable	01	03

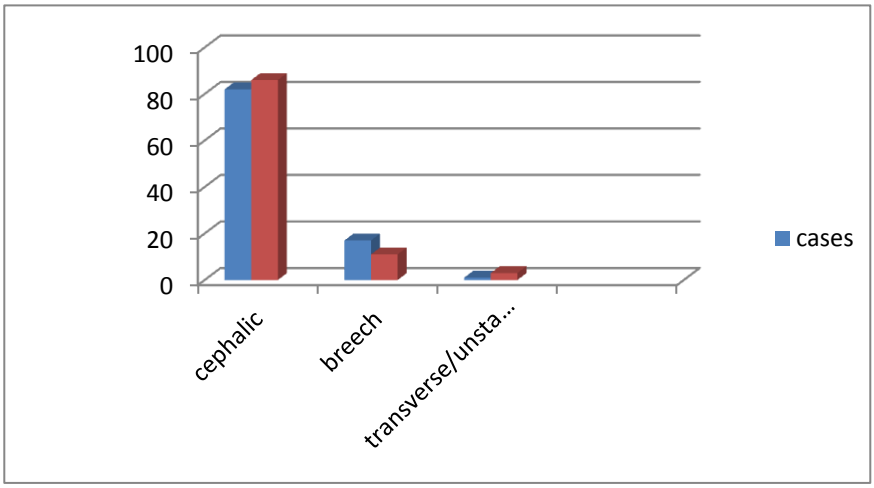


Fig. : H

The above table and graph show that majority (82%) of the cases and 86% of the controls had cephalic presentations at the time of delivery, whereas percentage of cases with breech was higher at 17% as co-pared to 11% of controls. Conversely transverse lie was seen in 03% controls over 01% of cases. Thus it can be said that oligohydramnios is associated with higher rates of malpresentations, especially breech whereas the control group had higher rates of unstable lie.

MODE OF DELIVERY

Table 8 : Showing mode of delivery

Mode of delivery	Cases (n=100)	Controls (n=100)
Spontaneous	16	83
Induced(successfully)	22	05
Caesarean Section (includes induction failures)	62	12

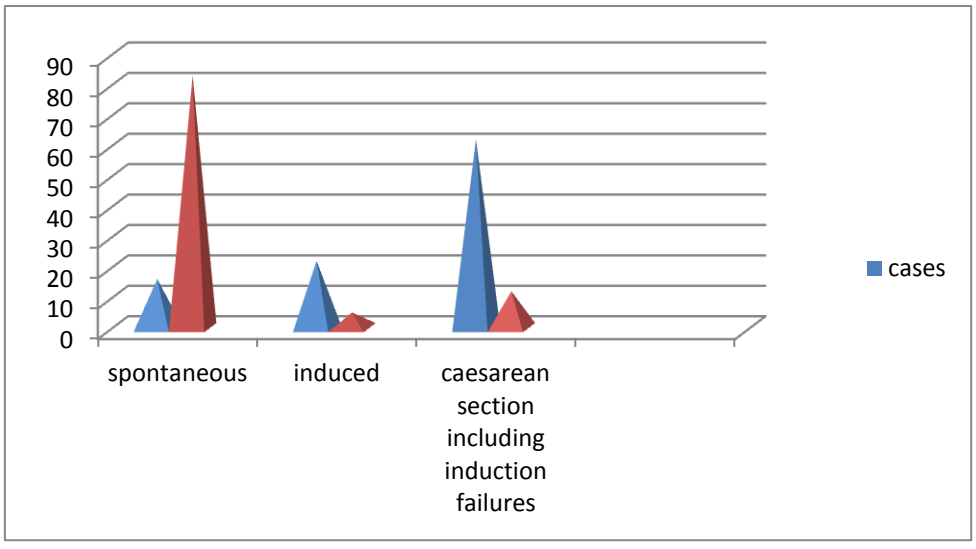


Fig. : I

The above table and chart shows that 62% of cases had C.S compared to only 12% controls. Thus, oligohydramnios is associated with significant risk of C.S ($p<0.05$). Majority of controls83% had spontaneous delivery whereas only 16% cases had spontaneous delivery. In 22% cases induction was reqd. compared to05% controls. Induced group includes the successfully induced ones whereas C.S group includes induction failure ones also. Thus oligohydramnios is associated with increased rates of induction. The standardised z-test for difference of proportions was applied to test for significance.

LIQUOR COLOUR

Table 9 : Colour of liquor at delivery

Colour	Cases (n=100)	Controls (n=100)
Meconium stained	48	12
Clear	52	88

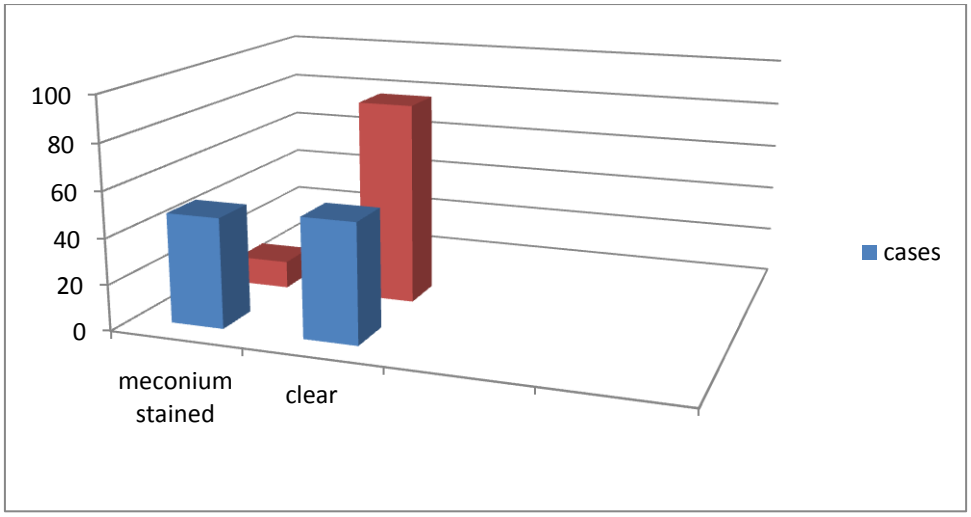


Fig. : J

The above table and graph show the distribution of colour of liquor at the time of delivery. It is seen that 48% cases had meconium stained liquor at delivery as compared to only 12% controls. The p-value two tailed was <0.05. The z statistic was 5.5549. Thus it is clear that oligohydramnios is associated with significant risk of meconium stained liquor.

INDICATION FOR CAESAREAN SECTION

Table 10 : Showing indication of caesarean section

Indication	Cases (n=62)	Controls (n=12)
Fetal distress	44	02
Induction failure	08	02
Obstetrical	04	04

Assoc. maternal complications	02	02
Elective	04	02

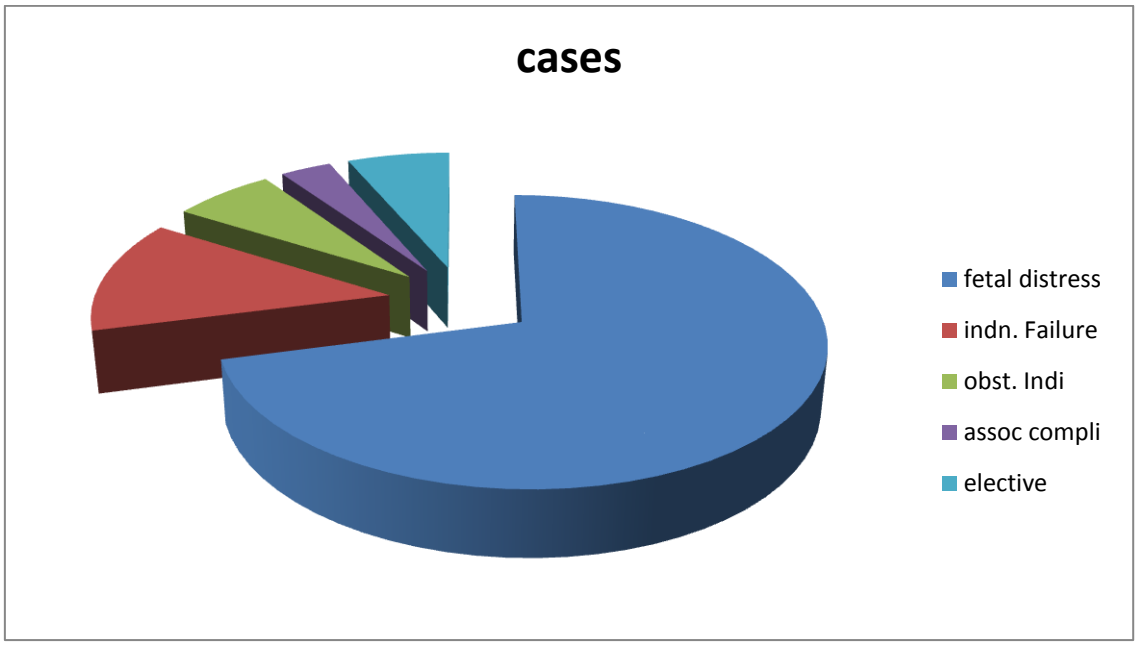


Fig. : K

The above table and figure make it clear that majority 44 cases (70.96%) underwent C.S for fetal distress.

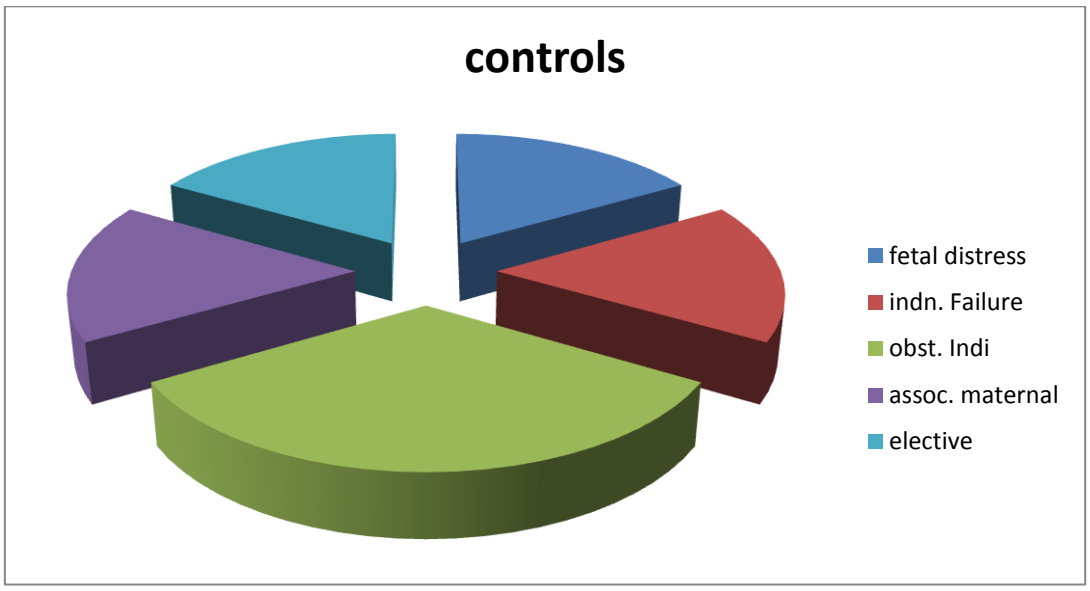


Fig. : L

The major indication among controls was obstetrical which had 04 patients (33.34%). Thus C.S due to fetal distress is significantly raised in cases of oligohydramnios ($p<0.05$).

DISTRIBUTION OF LSCS

Table 11: Showing distribution of LSCS

Type of LSCS	Cases (n=62)	Controls (n=12)
Elective	04	02
Emergency	58	10

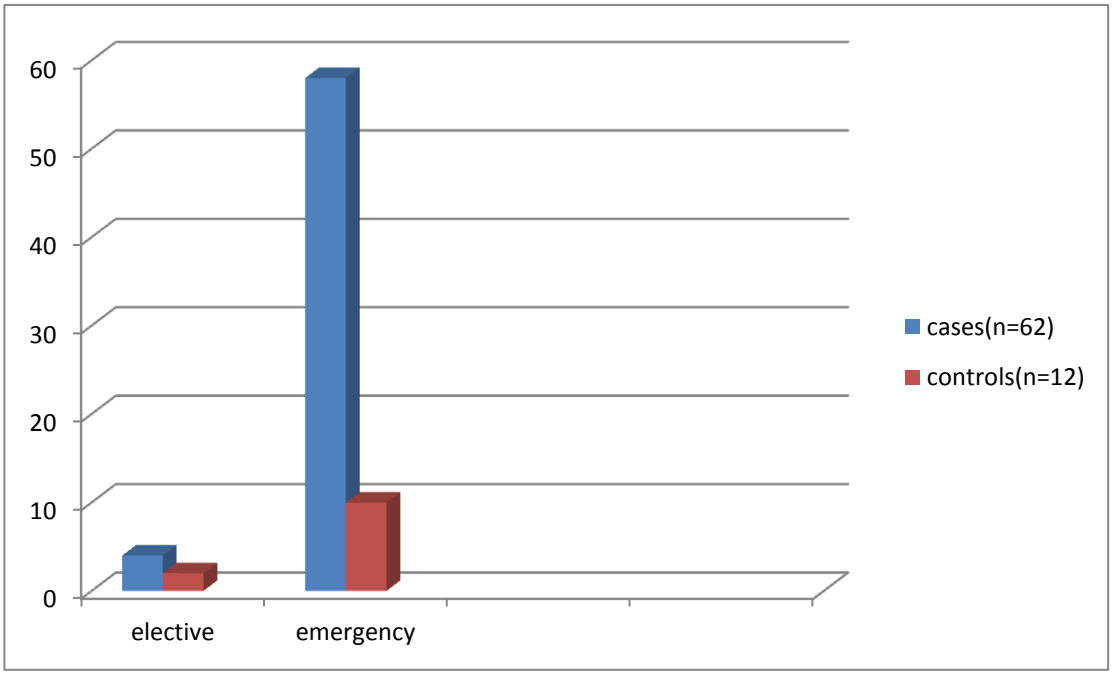


Fig. : M

The above table and the graph below show that majority of both cases (93.54%) and controls (83.33%) underwent LSCS due to emergency reasons which may be due to fetal distress, induction failure, etc. whereas, 6.45% cases and 16.67% controls underwent elective LSCS.

DISTRIBUTION OF BIRTH WEIGHTS

Table 12: Distribution of birth weights

Weight in kgs	Cases (n=100)	Controls (n=100)
<2.5	42	12
2.5-3.5	57	78
>3.5	01	10

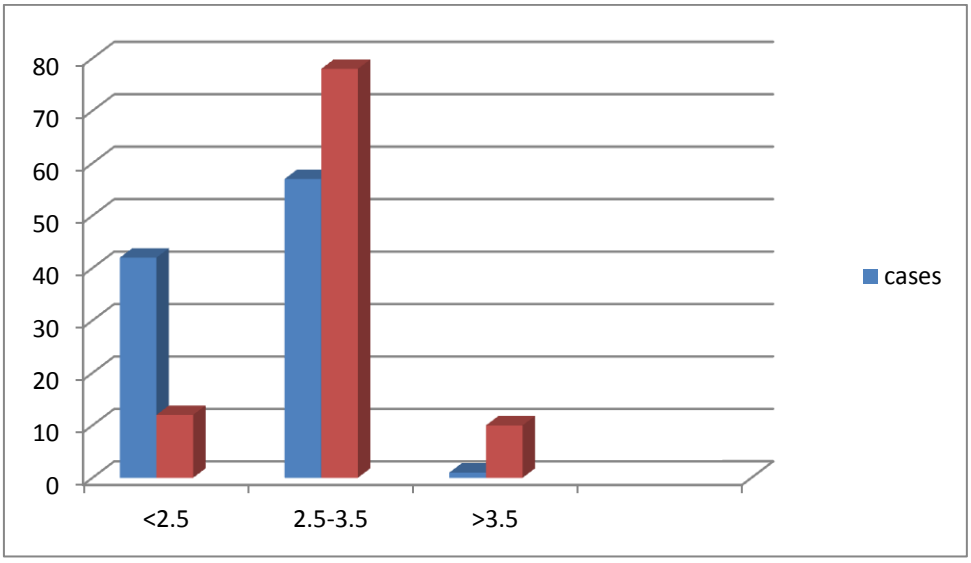


Fig. : N

From the table and diagram above it is clear that whereas 42% cases had their birth weight of babies <2.5 kg only 12% controls were in this category. Thus there is significant association oligohydramnios and low birth weight. Majority of patients in both groups i.e., 57% cases and 78% controls had birth weight between 2.5-3.5 kg.

1 MINUTE APGAR SCORE

Table 13 : Showing distribution of 1-min Apgar scores

1-min score	Cases (n=88)	Controls (n=97)
<5	20	02
5-7	42	17
>7	26	78

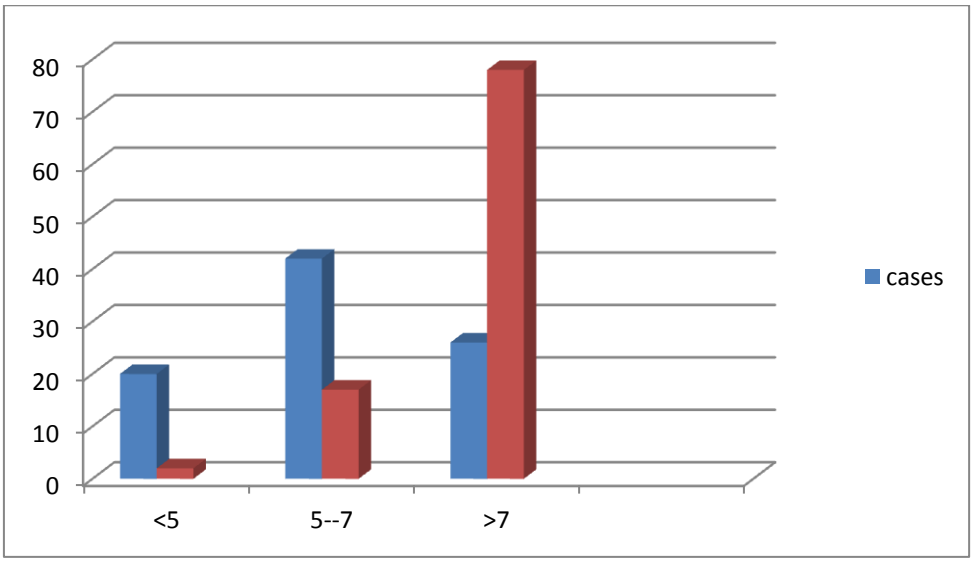


Fig. : O

It is clear from the above table and figure that the no. of newborns with Apgar score <5 was significantly higher in cases (22.72%) compared to controls (2.06%), whereas a score >7 was seen in 78% of controls compared to 26% cases. The standardised z-test of difference of proportions and chi square tests were applied to test for significance ($p<0.05$). Thus it can be said that oligohydramnios is associated with significant risk of low 1-min Apgar score.

5-MINUTE APGAR SCORE

Table 14 : Showing distribution of 5-min Apgar scores

Score	Cases (n=88)	Controls (n=97)
<5	12	00
5-7	21	05
>7	55	92

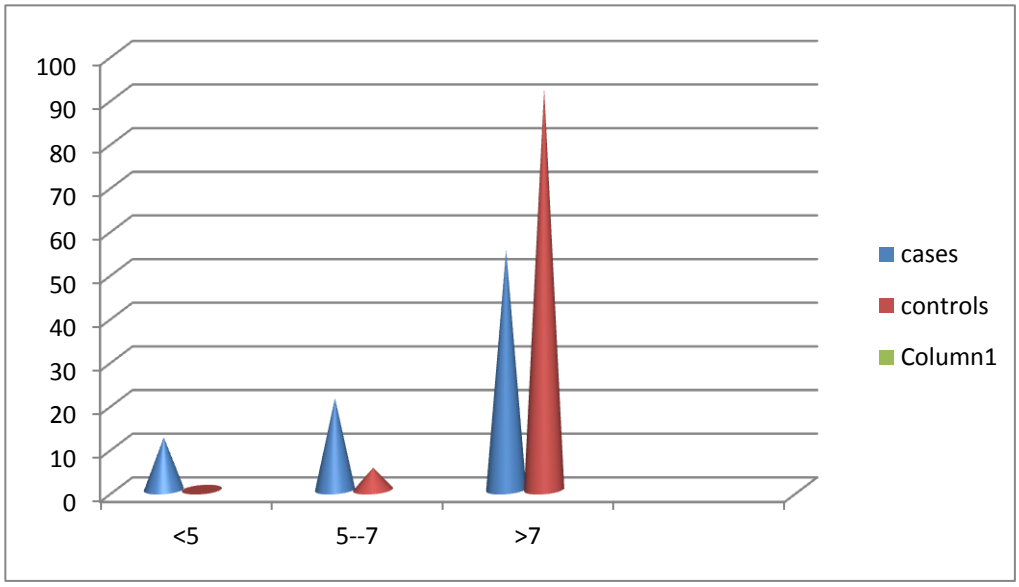


Fig. : P

It is clear from the above table and figure that whereas none of the newborns of the control group had their Apgar score <5 at 5-min, still 12 cases (13.6%) had apgar score <5 at 5-min signifying a poor fetal outcome. After 5-mins, 94.84% of control babies had score >7 whereas among the cases only 62.5% had a score >7. Statistical analysis found this difference to be significant ($p<0.05$, standardised z-test, chi square test). Hence it can be said that oligohydramnios is associated with significant risk of low 5-min Apgar score and hence poor fetal outcome.

ADVERSE FETAL OUTCOME

Table 15 : Distribution of adverse fetal outcome

Outcome	Cases (n=100)	Controls (n=100)
Stillbirth	10	02
Intrapartum death	02	01
NICU admissions	33	05
Neonatal death	02	00

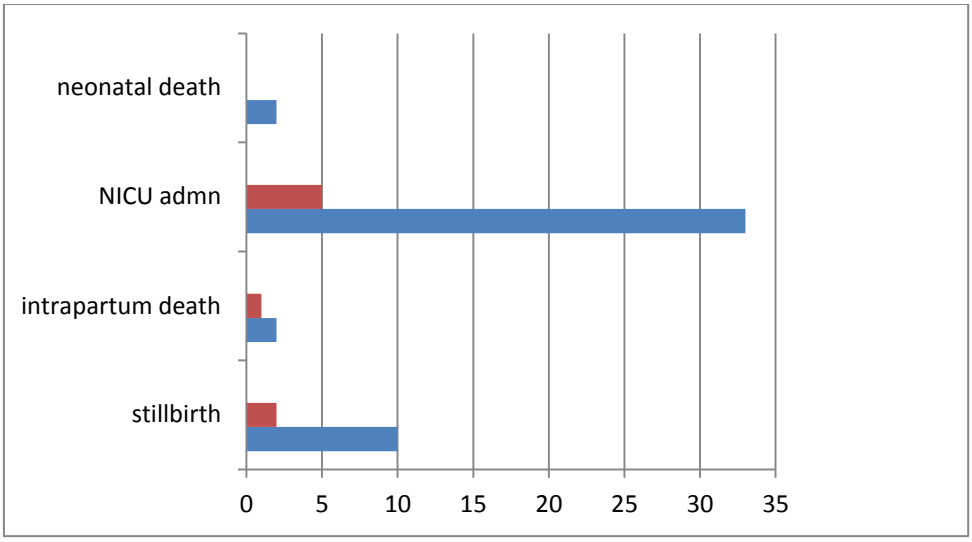


Fig. : Q

The above table and graph show that the percentage of cases requiring NICU admissions for newborns was quite high (33%) when compared to control group (5%). Also a high 10% stillbirth was noted among cases as compared to controls (02%) cases recorded 02 neonatal deaths whereas it was none for the controls. There were 02 cases of intrapartum deaths among the cases and 01 such case among the controls. Statistical analysis with standardised z-test was applied and adverse outcomes were found to be in significant association with case group ($p<0.05$). Thus we can say adverse fetal outcome are significantly higher among cases of oligohydramnios.

MATERNAL HOSPITALISATION

Table16 : Showing duration of maternal hospitalisation days

Duration	Cases (n=100)	Controls (n=100)
<2 days	20	80
2-7 days	18	08
>7 days	62	12

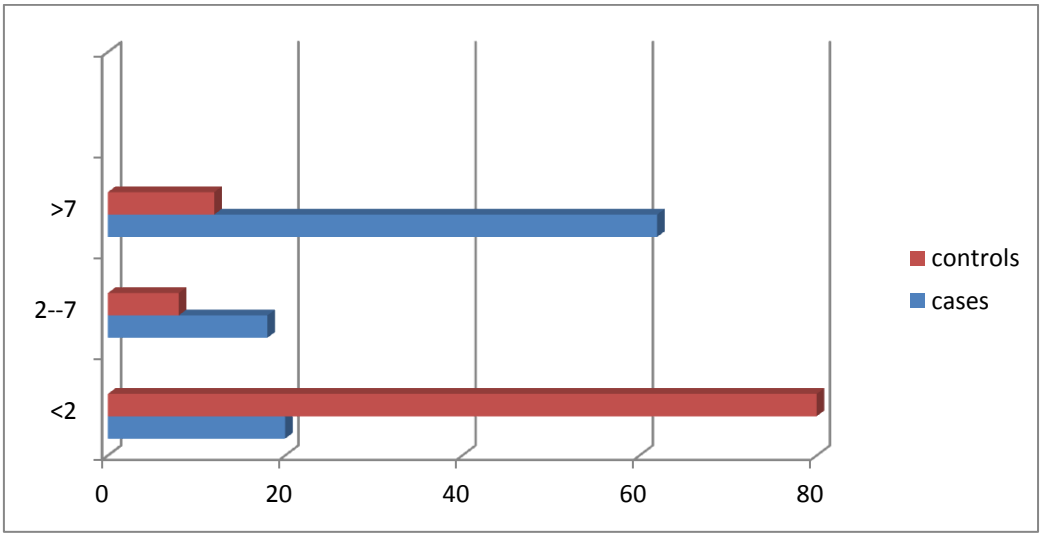


Fig. : R

The above table and graph clearly show that the period of hospitalisation was 48 hrs (2 days) in majority controls (80%) whereas whereas in majority cases (62%) it was >7 days. This was mainly due to increased operative delivery resulting in prolonged hospital stays. The z- statistic was 7.3230 for hospitalisation > 7 days and $p<0.05$. Thus oligohydramnios is associated with significant maternal morbidity in terms of increased duration of hospital stay.

NICU STAY

Table 17 : Showing distribution of NICU stay days

Days	Cases (n=33)	Controls (n=05)
<3	19	04
3-7	14	01

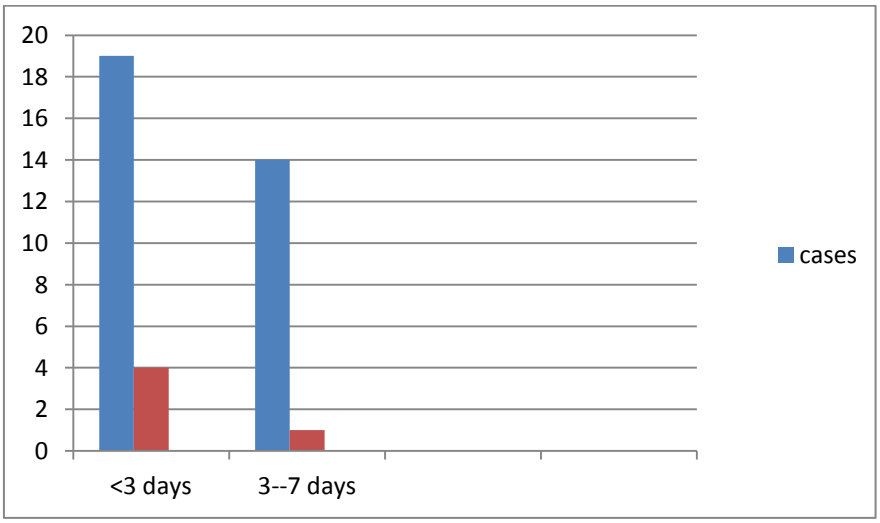


Fig. : S

The above table and diagram show the average duration of NICU stay duration among the cases and the controls. Among the controls those who required NICU admissions were 05. Out of them only one baby required prolonged stay of 3-7 days. Majority 80% of controls were discharged within 3 days. Among the cases, 33 needed NICU admissions. Out of these 19 (57.87%) had a short stay of <3 days, whereas, 14 (42.42%) had prolonged 3-7 days of NICU stay. Thus it can be said that oligohydramnios is associated with longer NICU stay of newborns and hence neonatal morbidity.

DISCUSSION

Liquor amnii plays a crucial role both in the developmental process of the fetus as well as during parturition. Reduction in amniotic fluid levels below normal can be detrimental to the baby and a source of morbidity to the mother. In 1984, a pioneering study was conducted by Chamberlain PF, Manning FA and colleagues on the relationship between marginally decreased amniotic fluid volumes to perinatal outcome. They made a retrospective study into the relationship between last biophysical profiles, postdatism, IUGR, diabetes etc. Out of 7582 cases, in 7096 (93.8%) patients, the largest pocket was between 2-8 cms and were termed normal whereas in 64 (90.85%) patients, the largest pocket as less than 1 cm and they were called decreased liquor volume. In 159 (2.1%) patients the largest pocket was between1-2 cm, they were termed as marginal liquor. Gross and corrected perinatal mortality in association with normal amniotic fluid volume ranged from 4.65/1000 & 1.97/ 1000 respectively whereas it was 187.5/1000 and 109.4/100 respectively in the association with decreased qualitative amniotic fluid volume. The incidence of major congenital anomaly and IUGR were significantly related to qualitative amniotic fluid volume[2].

Prior to this, Garzetti et al., and colleagues had performed a study on longterm measurement of amniotic fluid index (AFI) and its association with intrapartum fetal distress in 1977. A total of 117 patients were studied for fetal distress. It was found that these patients had significantly lower AFI ($p<0.001$) at their last sonographic evaluation than the other cases. Conclusion was made that longitudinal assesment of AFI was an effective method in predicting intrapartum fetal distress[9].

In 1981, Manning, Hill, Platt studied on qualitative AFV determination by ultrasonography and its relationship with IUGR. A total of 120 patients were studied with diagnosis of IUGR. 89 patients delivered a normal baby. AFV was taken to be normal if atleast one pocket of 1 cm in broadest diameter was identified it was concluded that perinatal mortality increased 10 folds in patients with decreased AFV.[4]

In 1989, Albert P Sarno and colleagues conducted a study on the efficacy of intrapartum Doppler velocimetry, amniotic fluid volume and FHR as predictors of subsequent fetal distress. It was found that out of total 109 patients examined, both abnormal initial FHR and AFI of 5cm or less were associated with intrapartum fetal distress. Conclusion was made that the FHR tracing or amniotic fluid volume in early intrapartum period was reasonable predictor of subsequent fetal outcome.[10]

In another study conducted by Sarno and colleagues in 1990 on the association between intrapartum amniotic volume at term, oligohydramnios and increase in fetal risk, 200 cases at term pregnancies were studied, and it was found that an intrapartum AFI of 5 cm or less was associated with considerable increase in caesarean section rates for fetal distress and of Apgar score less than 7 at one minute. It was concluded that AFI of 5cm and less in early intrapartum period was risk factor for perinatal morbidity.

From July 1991 to September 1996, Casey BM et al., conducted a study on association of antepartum oligohydramnios with adverse perinatal outcomes. A total of 6420 cases were studied out of which 147 were

found to have oligohydramnios. The study concluded on increased association with labour induction, stillbirth, NICU admissions, meconium aspiration and neonatal death with oligohydramnios.[12]

In 1993, Fischer and colleagues produced a report on the comparison of techniques for evaluation of amniotic fluid volume in post-dated pregnancies. A total of 198 women at 40 weeks of gestation were studied. Ultrasound confirmed or established dates were evaluated twice weekly with NSTs and amniotic fluid estimation. It was concluded that largest vertical pocket of 2.7 cm had greatest diagnostic value to detect postdatism and hence abnormal perinatal outcome, surpassing the AFI and the 2 cm largest vertical pocket.[13]

In a study by McCurdy Jr and Seeds JW on oligohydramnios and treatment, they found that it is associated with fetal anomalies, premature rupture of membranes, uteroplacental insufficiency(e.g. : growth retardation, postdatism, abruptio placentae, significant maternal illness) abnormalities of twinning and idiopathic oligohydramnios. They found the role of amnioinfusion as an adjunct to continuous fetal monitoring in labour to improve neonatal outcome in such cases.[101]

In 1994, another study was conducted by the Golan and colleagues on maternal complications and fetal outcome in 145 cases of oligohydramnios diagnosed in second and third trimester were studied and it was found to have a higher rate of meconium staining of aqmniotic fluid (29.1%), fetal distress (7.9%), premature separation of placenta (4.2%), asphyxia during labour occurred in 11.5% and caesarean section was needed in 35.2%, intrauterine fetal death occurred in 5.5% cases. The gross and corrected perinatal mortality was 16% and 10.7% respectively. Conclusion was made that oligohydramnios was associated with increased fetal morbidity and mortality and then termination should be considered when fetal pulmonary maturity was present in cases fetal distress.[14]

Nageotte and colleagues in 1994 published a study on the association of perinatal outcome with modified BPP. A total of 2774 patients with singleton gestation and intact membranes were considered and they underwent 17,429 tests. The uncorrected perinatal mortality rate was 2.9 per 1000. The incidents of adverse perinatal outcome in patients with abnormal modified BPP was 7% when compared with patients having persistently normal modified BPP. It was concluded that modified BPP is an excellent means of fetal surveillance and cases of high risk groups for adverse perinatal outcomes.[15]

In 1995, Baron and colleagues took a study on effect of intrapartum assessment of amniotic fluid volume on perinatal outcome. Their aim was to value of routine assessment of amniotic fluid volume on perinatal outcome. Oligohydramnios was defined as AFI 5.1-8 cm (n=261) and normal AFI was 8.1-20 cm (n=336). The two groups were compared for antepartum, intrapartum and postpartum variations. There was significant increase in the rates of meconium stained amniotic fluid, variable deacceleration, caesarean section rates. Conclusion was made that AFI for detecting intrapartum oligohydramnios is a valuable screening test for subsequent fetal distress and caesarean section.[16]

Another study was conducted by Divon and others in 1995 on association of AFI in post-term pregnancies with fetal outcome. Serial AFI was monitored twice weekly in 139 cases of uncomplicated singleton pregnancies at 41 weeks of gestation. Adverse fetal outcomes such as staining of meconium in amniotic fluid, FHR deaccleration, caesarean section for fetal distress, neonatal ICU admissions and perinatal mortality were seen in cases where AFI was < 5cm. However adverse outcomes were not associated with AFI > 5 cm. It was concluded that post-term prognosis with AFI < 5 cm are severely affected by poor perinatal outcome.[17]

In 1996, a study on serial amniotic fluid index measurements in cases of severe pre-eclampsia with adverse outcomes was conducted by Schuker and others. A considerable relationship was found between low AFI and IUGR and non reassuring fetal heart status in patients with severe pre-eclampsia. 136 women with

severe pre-eclampsia were observed conservatively for 48 hours and followed with daily NST and AFI. It was seen that 107 patients had caesarean section but only 42 (39%) were for non reassuring fetal heart status or a BPP of less than 4 and 38 (36%) of patients had infants with IUGR. For those with AFI less than 5 cm both on admission and delivery there was higher incidence of IUGR than those with AFI > 5 cm (p< 0.007 and p=0.029 respectively). Conclusion was made that AFI < 5 cm is predictive of IUGR in severe pre-eclampsia but it lacks sensitivity.[18]

Again in 1996, a study was published by Shipp TD and others as evaluation of the outcome of oligohydramnios diagnosed in second and third trimesters. They evaluated the range of etiologies and also differences in fetal and neonatal outcome. A total of 250 singleton pregnancies were taken up for study with severe oligohydramnios. A bimodal distribution of gestational age was seen with more cases diagnosed at 13-21 weeks than at 34-42 weeks. Fetal anomalies were seen in 50.7% cases in 2nd trimester and 22% of those in 3rd trimester. The baby survival rate was 10.2% and 85.3% respectively in 1st and 2nd trimesters.[99]

In 1997, Chauhan and others conducted a study to evaluate whether intrapartum oligohydramnios affected the perinatal outcome in high risk pregnancies. A total of 490 parturients with medical or obstetrical complications were studied and they found that incidences of caesarean section for fetal distress and umbillical artery pH < 7.00 were 14 % and 1.8 % respectively. They concluded that both criteria for oligohydramnios are poor predictors of adverse outcomes for high risk intrapartum patients.[19]

In the same year i.e., 1997, Hill reported on clinical implications of sonographic diagnosis of oligohydramnios. He stressed that decrease in amniotic fluid volume is often one of the clues to underlying fetal abnormality or maternal distress state. Significant reduction in amniotic fluid usually translates into increased perinatal morbidity and mortality.[20]

Again in 1997, Miyamura and others made a comparative study on the single deepest pocket and AFI in predicting fetal distress in small for gestational age fetus. A total of 69 patients with singleton pregnancies, intact membranes and birth weight < 50 % were followed up. The results demonstrated that single deepest pocket measurement of < 3cm was most useful criterion for oligohydramnios in prediction of fetal outcome.[21]

In 1998, Conway and others published a study on isolated oligohydramnios in term pregnancies between 37–41 6/7 weeks of gestation. They were matched by gestational age and parity with controls having normal AFI, who were in spontaneous labour. Complicated pregnancies were excluded. A total of 183 women underwent induction for isolated oligohydramnios compared to control group, neonatal outcome did not differ in the group induced for oligohydramnios. However induced women had more no. of caesarean section deliveries. They concluded that isolated oligohydramnios in otherwise normal term pregnancy may not be an indicator of fetal compromise.[22]

Again in 1998, another report was made by Roberts and others found that there was a four fold increase in induction of labour for fetal reasons in pregnancies with isolated reduced AFV in 3rd trimester.[23]

In 1999, Banks and associates reported on the association between borderline amniotic fluid and perinatal risks. They conducted a retrospective study over a period of 4 months. AFI >5cm and <10 cm was defined as borderline and AFI 10-24 cm was taken as normal. They concluded that there is a two fold increase in the incidence of adverse perinatal outcome in women with borderline AFI in comparison with the control group. There was a four fold increase in incidence of IUGR in cases with borderline AFI.[24]

Magann and colleagues in 1999 produced a report on the role of estimation of AFV in 1001 patients. They compared the efficacy of AFI and two diameter pocket technique in predicting adverse pregnancy outcomes. They found that out of 1001 patients, significant 46% were found to have oligohydramnios according to two diameter criteria than according to AFI (21%,p<0.0001,RR of 1.7,95% confidence interval-1.5-1.8). No differences were observed between cases and control groups in terms of fetal outcome. They concluded that both AFI and the two diameter pocket techniques are poor diagnostic tests to predict perinatal outcomes.[25]

Shenker and colleagues in 1999 studied a total of 80 pregnant patients, out of them 40 had PROM and of these outcomes were good in 25. 12 of 14 fetuses with oligohydramnios and IUGR survived. All of the fetuses with severe renal anomalies died. In six cases of oligohydramnios, premature separation of placenta was identified resulting fetal or neonatal death in the 2nd trimester.[26]

In 1999, a meta analysis of 18 studies with 10,500 cases complicated with intrapartum AFI <5cm which was compared with controls with AFI >5 cm by Chauhan et al., women with oligohydramnios had significantly higher rates of delivery with fetal distress.[27]

In 2000, another important study on pregnancy outcomes in cases of oligohydramnios diagnosed after 34 weeks of gestation by Casey BM et al., found that out of 6423 cases they examined, 147 (2.3%) were complicated by oligohydramnios. This was associated with increased labour induction (42% v/s 18%, p<0.001), stillbirth (1.4% v/s 0.3%, p<0.03), non-reassuring fetal heart status (48% v/s 39%, p<0.03) admission to NICU(71.02%, p<0.001), meconium aspiration syndrome (1% v/s 0.1%, p<0.001) and neonatal death (5% v/s 0.3%, p<0.001) and concluded that antepartum oligohydramnios is associated with perinatal morbidity and mortality.[102]

In study report published by Kawasaki and others in 2002, a total of 242 pregnant cases were studied and divided into group A (n=99) having AFI <8cm; group B (n=143) with AFI 8.1-20 cm and antenatal, intrapartum and neonatal variables were compared. Statistical analysis was performed using independent student-t test; chi-square analysis; Fischer's test etc. Based on observations, they concluded that adverse neonatal outcomes were higher in patients with reduced AFI.[28]

Voxman and colleagues in 2002 published a report on perinatal outcome of cases with low AFI. A total of 779 women were enrolled for study over a 212 month period. They concluded that AFI of 5 cm or less was associated with abnormal FHR but not with increased caesarean section rates, meconium stained liquor, apgar score <7 or NICU admissions. Subjects with AFI < 5cm had higher rates of caesarean section but this did not reach statistical significance.[29]

In 2003, Magann and colleagues reported on predictability of intrapatum and neonatal outcomes with AFV distribution. The study was conducted between Jan 1997 Jan 2001, 135 women were enrolled for the study. The sum of the p value by AFI (P=0.309), single deepest pocket (P=0.168), dye determined group (p=0.368) were similar. Deacceleration in labour (p=0.597), late deacceleration (p=0.999) and C.S for fetal distress (0.413) and cord blood pH <7.2(p=0.647) were similar. They concluded that all the three methods are not affected by AFV distribution.[30]

In 2004 locatelli and co authors reported on prognostic value of serial estimation of AFI. Between Jan 1997-Dec 2000, all uncomplicated single gestation without congenital anomaly underwent serial monitoring of AFI. Comparison between favourable and adverse outcomes were made using chi-square, Fischer or student-t test. They concluded that sonography diagnosed oligohydramnios carried enhanced risk of adverse perinatal outcomes.[31]

In 2004, Magann reported on comparison of BPP with AFV assessments by single deepest pocket. Based on their observation they concluded that AFI offers no advantage in detecting unfavourable outcomes compared with single deepest pocket when performed with BPP.[32]

In another report published by Volante and others in 2004, the prevalence of oligohydramnios was seen in 3-5 % of pregnancies. They found that most of the cases of oligohydramnios are due to PROM and others are fetal abnormalities such as urinary tract malformations, drugs-NSAIDS, chromosomal aberration etc. oligohydramnios as often found to be complicated by preterm birth and IUGR. Neonatal prognosis was quite poor in cases of severe oligohydramnios which was however improved by amnioinfusion.[33]

Again in 2004, Zhang and colleagues analysed the data from multicentre clinical trial of Routine antenatal diagnostic imaging with USG (RADIUS). A total of 15,151 cases were examined. On the basis of USG examinations, oligohydramnios was diagnosed in 1.5% cases. Almost half of the cases were associated with adverse perinatal outcomes.[34]

In 2004, ACOG did a study on comparison of AFI with single deepest pocket and other components of BPP. About 273 women were monitored using AFI and 264 were diagnosed with single deepest pocket. They concluded that AFI has no advantage over single deepest pocket method in predicting perinatal outcome when performed as a part of BPP.[35]

In 2005, Ek S and colleagues published a study on the outcome of uncomplicated oligohydramnios beyond 40 completed weeks. It was a prospective, randomised, pilot study on neonatal and maternal outcomes. 58 cases were taken up for study, 26 were randomised for expectant management and 28 for induction of labour. Based on the results significant differences were found in maternal and neonatal outcomes.[100]

In 2005, Ott et al., reported on relationship between amniotic fluid volume and perinatal outcome. Two groups of cases- high risk and low risk were evaluated to determine relationship between AFI and gestational age. Statistical analysis was performer using linear regression to evaluate co-relation between AFI and birth weight. A total of 14,747 cases were studied and AFI determined. 4337 high risk cases and 1153 low risk cases were evaluated. There was significant co-relation between non-reassuring FHR during labour and oligohydramnios.[36]

In 2006, Danon and colleagues reported that pregnancies with isolated oligohydramnios was not associated with increased perinatal complications per se but increased rates of induction resulted in higher C.S. rates.[37]

Magann EF, Doherty DA and others in 2010, studied 2597 cases and detected 72 with oligohydramnios. They conducted a prospective longitudinal study and concluded that pregnancies complicated by oligohydramnios had greater risk of labour induction, IUGR and preterm delivery.[38]

In 2007, Chabra and colleagues submitted a report on the role of oligohydramnios as a potential marker for serious obstetrical complications. Analysis of cases that were admitted over 2 years and cases admitted over previous 5 years was done. The incidence of oligohydramnios over 7 years was 4.45%. It was 4.9% in retrospective and 4% in prospective cases; 38% retrospective and 31.5% of prospective cases were complicated by intrapartum meconium. Abruptio placentae was associated in 9.8% retrospective and 8.3% prospective cases. Labour was induced in 18.2% retrospective and 13.9% prospective cases. Perinatal mortality rate in cases of oligohydramnios was 87.7 and 41.5% babies had congenital anomalies. Overall induction was required more often.[95]

In 2007, a report was published by Manzanaras and others on the need for induction in term pregnancies with oligohydramnios. It was a retrospective study and comprised of 412 pregnancies with cephalic presentation and no maternal risk factors or fetal abnormalities. Two groups were compared, 206 with isolated oligohydramnios and 206 with normal Amniotic fluid index. The overall rates of caesarean section, non reassuring fetal heart status, operative vaginal delivery rates were found to be higher in the oligohydramnios group. There was no difference between groups on neonatal outcome, perinatal mortality or morbidity.[96]

Another study was reported by Melaned N and colleagues on the perinatal outcome in oligohydramnios diagnosed before 37 weeks of gestation. It was a retrospective cohort study of singleton pregnancies with isolated oligohydramnios at preterm (n=108). Pregnancy outcome was compared with matched control group of normal amniotic fluid in 3:1 ratio. It was found that cases with oligohydramnios had higher rates of preterm deliveries (26.9% v/s 12.3%, p<0.001), most of which were iatrogenic. Also higher rates of labour induction and caesarean delivery were noted. Neonates were associated with low birth weight and higher neonatal morbidity.[98]

In 2012, Magann EF, Chauhan SP and others compared two normative datasets of amniotic fluid volume. Two classifications were used- i) Brace's and ii) Magann's. It was concluded that Brace's dataset is more likely to categorize patients as having oligohydramnios and Magann's dataset is more useful for oligohydramnios or small for gestational age identification.[39]

In 2012, Visvalingan G and others submitted a report on the perinatal outcome in women with diagnosed anhydramnios at term pregnancies. This was a retrospective study and was conducted at Rotunda hospital from Jan 1st, 2003 to 31st Dec, 2007. All women with only anhydramnios at term were identified. The high risk pregnancies were excluded from the study. The maternal and fetal parameters that were reviewed included maternal age, gestation, parity, method of induction, mode of delivery and requirement of obstetric intervention. The study showed that anhydramnios is associated with 56.6% of LSCS rate in primigravida and 19.0% LSCS rate in multigravida.[108]

Again in 2012, another study was published out by Novikova N and others on the role of prophylactic versus therapeutic amnioinfusion for oligohydramnios in labour. It is registered in the Cochrane pregnancy and childbirth groups trials register (28 Feb '12). It is a randomised control trial comparing prophylactic amnioinfusion in women in labour with oligohydramnios but not fetal heart rate deacceleration in labour with therapeutic amnioinfusion. A total of 116 women were included. No differences in caesarean section rates (RR 1.29, 95% confidence interval 0.60 to2.74). There were no differences in cord arterial pH, oxytocin augmentation, neonatal pneumonia or postpartum endometritis. Prophylactic amnioinfusion was associated with increased intrapartum fever (RR 3.38, 95% confidence interval 1.21 to 10.05).[103]

Another study was reported by Rosi AC and Prefumo F in 2013 on the review of literature and metaanalysis of perinatal outcomes in cases of isolated oligohydramnios at term and post-term pregnancy. A search in Pubmed, Medline, embase and reference lists was done and cases with singleton pregnancies with amniotic fluid index <5 cm were taken up. Those with fetal malformations, premature rupture of membranes, preterm delivery, intrauterine growth retardation were excluded. Comparison in terms of obstetric intervention for non reassuring fetal heart status, meconium stained liquor, Apgar score <7 at 5-min and umbillical artery pH<7.0, adnmission to NICU and perinatal death rate. Metaanalysis was done. It was found that obstetrical interventions were done more frequently in cases with oligohydramnios than the control group. Meta-analysis did not show any difference in relation to meconium staining, Apgar score, pH, small for gestational age, NICU admissions and perinatal death.[97]

Amnion :

Amnion is a tough and tenacious but pliable membrane. It is the innermost layer of fetal membranes. Its innermost surface is smooth and shiny and it is in contact with the liquor amnii. The outer surface consists of connective tissue and it is opposed to the inner aspect of chorion from where it can be peeled off except at the insertion of the umbillical cord.[40]

Structure of amnion (bourne, 1962)[41] :

1. Inner surface - Consists of uninterrupted single layer cuboidal epithelium derived from embryonic epithelium.
2. Basement membrane - Which is connected to the acellular compact layer.
3. Acellular compact layer - Composed primarily of interstitial collagen.
4. Row of fibroblast like mesenchymal cells derived from embryonic mesoderm
5. Acellular zona spongiosa which is contiguous with chorion levae.

Development of amnion[146,147] :

Early during implantation, a space develops between the embryonic cell mass and adjacent trophoblasts. Small cells that line this inner surface of trophoblasts have been called amniogenic cells - precursors of amnionic epithelium. The amnion is first identifiable about the 7^{th} or 8^{th} day of embryo development. It is initially a minute vescicle, which then develops into a small sac that covers the dorsal surface of the embryo. As the amnion enlarges, it gradually engulfs the growing embryo, which prolapses into its cavity.[42]

Functions of amnion[43] :

1. Contributes to the formation of liquor amnii.
2. Intact membranes prevent ascending uterine infection.
3. Facilitates dilatation of cervix during labour.
4. Has got enzymatic activities for steroid hormone metabolism.
5. Rich source of glycerophospholipids for prostaglandin synthesis.
6. Produces- endothelin 1 – vasoconstrictor.

Parathyroid hormone related peptide-vasorelaxant.

Enkephalinase-digests endothelin 1.

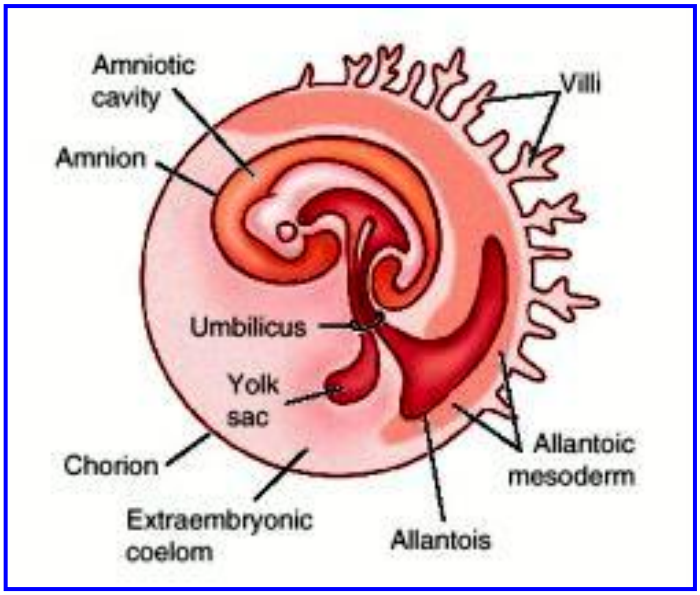


Fig 1 : Anatomy of amniotic cavity

Development of amniotic cavity[82] :

On the 8th day following fertilisation,the embryoblast differentiates into bilaminar germ disc which consists of dorsal ectodermal layer oftall columnar cells and ventral endodermal layer of flattened polyhedral cells. The bilaminar germ disc is connected with the trophoblast by mesenchymal condensation, called the connecting stalk or body stalk which later on forms the umbillical cord.

Two cavities appear-one on each side of the germ disc- one on the 12th post ovulatory day, a fluid filled cavity appears between the ectodermal layer and the cytotrophoblast which is called amniotic cavity.Its floor is formed by the ectoderm and the rest of the wall by primitive mesenchyme. Another cavity-yolk sac appears on the ventral aspect of the bilaminar disc and is lined externally by the primitive mesenchyme and internally by the migrating endodermal cells from the endodermal layer of the germ disc.

Extraembryonic mesenchyme derived from the trophoblast appears to separate the yolk sac from the blastocyst wall and also the amniotic cavity from the trophoblast of chorion.

Small spaces appear in the extraembryonic coelom, which gradually enlarges and fuses to form extraembryonic coelom. Progressive enlargement of the extraembryonic coelom, seperates the amnion from the inner aspect of chorion except at the caudal end of the embryo. The mesenchymal attachment persists to form the body stalk. Umbillical cord develops from this stalk. Subsequently amniotic cavity enlarges at the expense of the extraembryonic coelom. The developing embryo bulges into the enlarged amniotic cavity. The yolk sac becomes partly incorporated into the embryo to form the gut. The part that remains outside is incorporated into the body stalk. Gradually the extraembryonic coelom is completely obliterated. The extraembryonic mesenchyme covering the amnion fuses with the lining of the chorion. The single layer of fused amniochorion is now formed.

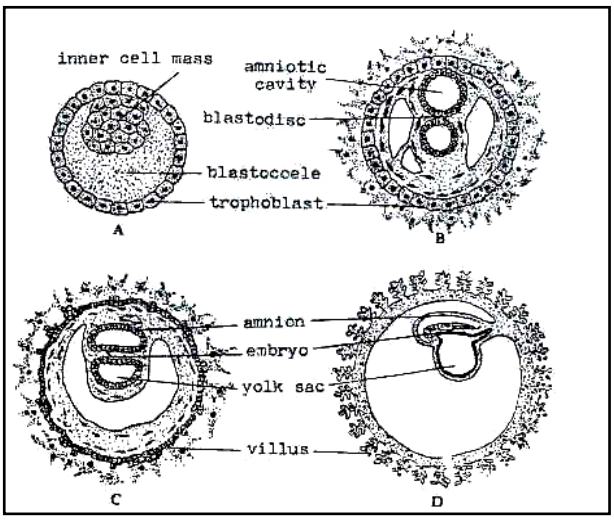


Fig. 2 : Development of amniotic cavity

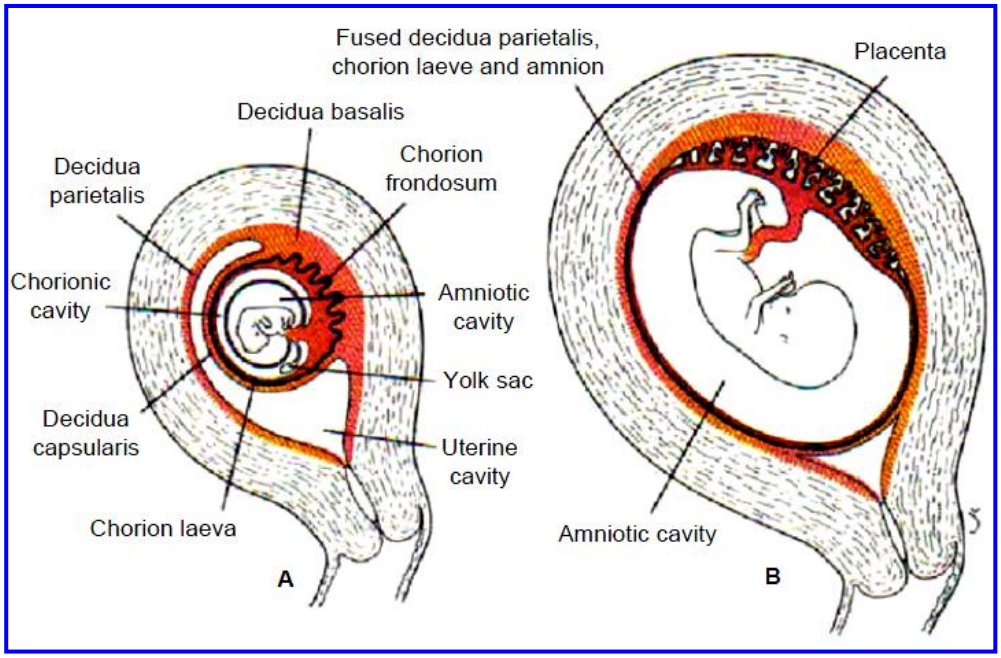


Fig. 3 : Enlargement of amniotic cavity

Source of amniotic fluid :

Amniotic fluid is derived from ano. of fetal and nonfetal sources which the contribution from each source varies according to gestation. Early in gestation, the amniotic fluid is thought to be derived from the mother directly across the amnion and the fetal surface of the placenta along with fetal body surface.[44]

In midgestation, fetal urine production begins to enter the amniotic sac and fetus begins to swallow amniotic fluid; although daily volume flows are quite small. The fetal lungs also secrete liquid into the amniotic fluid at the same time.[45] Despite large variations in current estimates of human fetal urine production,[46] the best estimates of daily amniotic fluid flows in a near term fetus are :

Flow into the amniotic sac :

- Fetal urine production-800-1200ml/day
- Fetal lung liquid secretion-170 ml/min

- Oral nasal secretion-15 ml/day

Flow out of the amnionic sac :

- Fetal swallowing-500-1000ml/day
- Intramembranous flow-200-400ml/day
- Transmembranous flow-10ml/day[47]

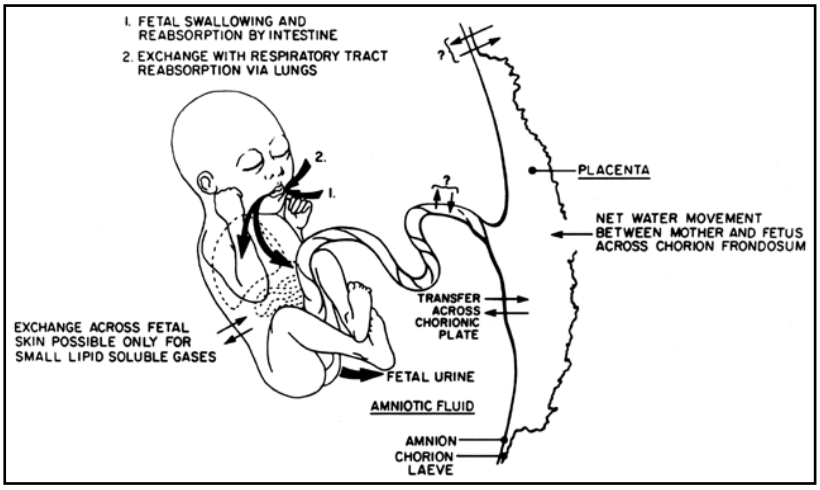


Fig. 4 : Regulation of amniotic fluid formation

In the 1st trimester of pregnancy, amniotic fluid is mainly comprised of the embryo's plasma volume. Embryonic skin is quite permeable and allows diffusion of fluid into the amniotic cavity. Active transport of solute by the amnion can also cause passive movement water into the amniotic cavity.[83]

Prior to 20 weeks, amniotic fluid is identical in its composition to that of maternal plasma.[84]

The amnion and chorion provide large surface area for transport of water and solutes and transfer of solute followed by inward diffusion of water. The fetal nasal and buccal secretions also contribute.[85]

In the second half of pregnancy, fetal urine becomes the major source of amniotic fluid. The fetus passes urine as early as 8-11 weeks of gestation.[85]

Due to prematurity of the fetal kidneys, the fluid is predominantly hypotonic. The amount of urine passed initially is about 10 ml/24 hr. As gestation advances, it increases to about 110 ml/kg/24 hr at 25 weeks to 190 ml/kg/24 hr at 39 weeks. At term the production is about 500-600 ml/day, followed by a subsequent fall as gestation advances.[86]

As renal maturity occurs, the composition of the fluid changes. The sodium and potassium concentration falls while that of urea and creatinine increase. That's why renal abnormalities are frequently associated with IUGR and oligohydramnios.[87]

The fetus also swallows fluid. The swallowing reflex appears concurrently with urine production at 8-10 weeks. The volume of swallowing varies from 200-450 ml/day to 1500ml/day. The estimated average volume swallowed in late gestation is 210-760 ml/day. It occurs mainly with fetal breathing activity[88].

The second important source of amniotic fluid in the 2nd half of pregnancy are the fetal lungs. The rate of amniotic fluid production can be as high as 200-400 ml/day and it is about 10% of body weight /day. It is mediated by active transport of chloride ions in the epithelial lining of the developing lungs.

Amniotic fluid movement :

There are two important pathways of amniotic fluid movement :

1) Intramembranous pathway

2) Transmembranous pathway

The above two pathways are responsible for rapid movement of water and solute between amniotic fluid and the fetal blood across the placenta and membranes called the "intramembranous pathway".[131]

The movement of water and solute between amniotic fluid and maternal blood within the wall of the uterus is an exchange through the "trans membranous pathway".[89]

As gestation advances, cornification and stratification of the skin of the fetus occurs and results in reduction in the diffusion of extracellular fluid into the amniotic cavity. Absence of fetal skin cornification in preterm infants plays a major role in formation of amniotic fluid in pre-term infants.

Also, the fetal gastrointestinal tract,umbillical cord and the fetal surface of the placenta are additional sources of amniotic fluid production.[90]

Hormones like prolactin reduce the amniotic fluid volume by 50% by shift of fluid from the amniotic cavity to the maternal blood. Cortisol and anti diuretic hormone also affect the permeability of amnion.[91]

Studies with radio isotopes have shown that amniotic fluid is completely replace every 3 hours.[92]

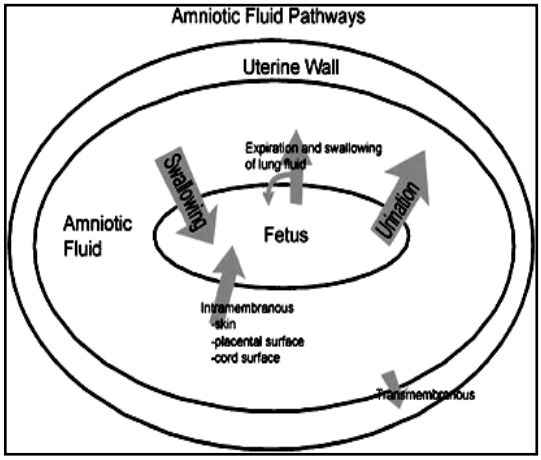


Fig. 5: Amniotic fluid pathways

Composition of amniotic fluid :[48]

In the 1st half of pregnancy, the composition of the fluid is almost identical to a transudate of plasma. But in late pregnancy, the composition is very much altered mainly due to contamination of fetal urinary metabolites. The composition includes – (i) Water-98-99% and (ii) Solid-1-2%. The following are the solid constituents :

(a)Organic : Protein - 0.3mg%, glucose - 20mg%, urea - 30mg%, NPN - 30mg%, uric acid - 4mg%, creatinine - 2mg%, total lipids - 50mg%, hormones - prolactin, insulin and rennin.

(b) Inorganic : The concentration of sodium, chloride and potassium is almost the same as that found in maternal blood. As pregnancy advances, there may be slight fall in the sodium and chloride concentration probably due to dilution by hypotonic fetal urine, whereas the potassium concentration remains unaltered.

(c) Suspended particles include : lanugo, exfoliated squamous epithelial cells, vernixcaseosa, cast of amniotic cells and cells from the respiratory tract, urinary bladder and vagina of the fetus.

Regulation of amniotic fluid volume :[93]

The mechanisms involved are :

- Changes in the membrane permeability across the chorion and amnion.
- Fetal urine production changes by : Arginine
  Vasopressin
  Adrenaline
  Cortisol
- Changes in fetal swallowing rates
- Maternal hydration status[118,119]
-

Regulation mainly occurs at :[126,144,145]

- Placental control : Permeability undergoes significant changes depending on the maternal hydration state, solute changes and hormonal changes.
- Fetal control : The fetal urine flow and the swallowing rates are affected by several factors like arginine, vasopressin, adrenaline, cortisol, aldosterone, angiotensin ii, etc.
- Maternal osmotic status : The amniotic fluid volume and maternal plasma volume have strong interrelationship with a lower plasma volume frequently associated with oligohydramnios and elevated with polyhydramnios.

The removal of amniotic fluid is mainly by fetal swallowing which approximately is about 200-450 ml/day.[88]

FUNCTIONS OF AMNIOTIC FLUID

During pregnancy :

(1) It acts as ashock absorber,protecting the fetus from possible external injury.

(2) Maintains an even temperature

(3) The fluid distends the amniotic sac and thereby allows for growth and free movement of the fetus and prevents adhesions between the fetal parts and amniotic sac.

(4) Its nutritive value is negligible because of small amount of protein and salt content, however, water supply to the fetus is quite adequate.

During labour :

(1) The amnion and chorion are combined to form a hydrostatic wedge which helps in dilatation of the cervix.

(2) During uterine contraction,it prevents marked interference with the placental circulation so long as the membranes remain intact.

(3) It flushes the birth canal at the end of 1st stage of labour and by its aseptic and bactericidal action protects the fetus and prevents ascending infections to the uterine cavity.[48]

Typical amniotic fluid volume :[49] Chart 1

Weeks	Fetus (g)	Placenta(g)	Amniotic fluid(ml)	% fluid
16	100	100	200	50
28	1000	200	1000	45
36	2500	400	900	24
40	3300	500	800	17

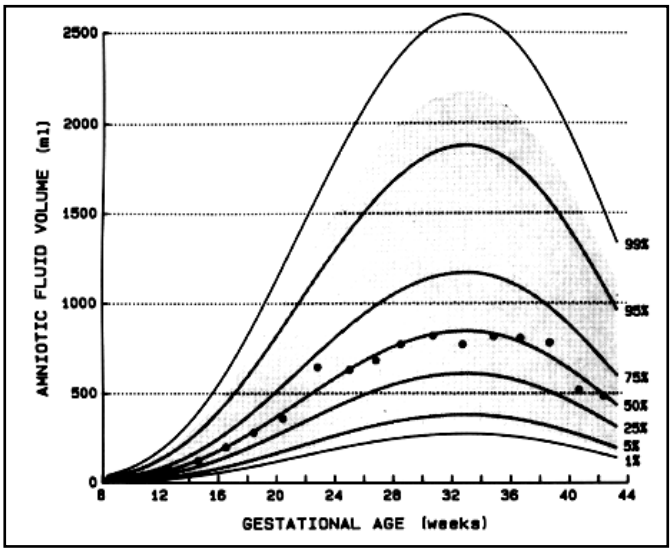


Fig. 6 : AFV

Evaluation of amniotic fluid volume :

Evaluation of amniotic fluid volume can be done by -

(1) Clinical evaluation

(2) Ultrasonography[148-151]—non invasive –

- Amniotic fluid index (AFI)
- Single deepest pocket
- Two diameter fluid pocket

(3) Invasive—dye dilution technique

Clinical assessment of amniotic fluid :

It requires experienced hands and skills to diagnose abnormalities in amniotic fluid volume by palpation of the abdomen. In cases of oligohydramnios, the clinical findings usually obtained are :[50]

(1) Uterine size is much smaller than the period of amenorrhea.

(2) Perception of less fetal movements.

(3) The uterus is "full of fetus" because of scanty liquor.

(4) Malpresentations are common.

(5) Evidence of IUGR of the fetus.

Invasive :

This method involves instillation of aminohippurate sodium[51] into the amniotic cavity and subsequent measuring of dye concentration with spectrophotometry. It gives true amniotic fluid volume but is an invasive procedure and is not practiced usually.

Non invasive :

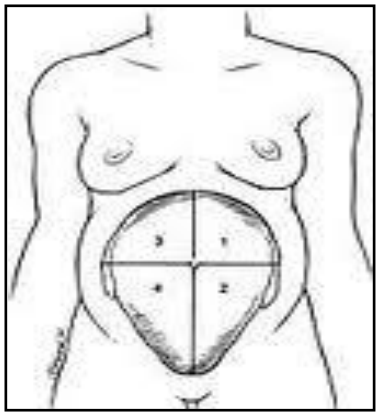
Amniotic fluid index[133-135]**:**

Phelan et al., in 1987 described a four quadrant method for calculation of amniotic fluid index.[1]

It is a semiquantitative method to estimate amniotic fluid volume. Using the umbilicus as one reference point, the uterus is divided into upper and lower halves. The lineanigra is then used to divide the uterus into right and left halves.[139-141]

A linear or curvilinear transducer is placed on the maternal abdomen along the longitudinal axis of the mother. The transducer head is perpendicular to the floor. A perpendicular relationship should be carefully maintained to avoid a falsely enlarged amniotic pocket.[52] Since the patient is supine, amniotic fluid will now flow into the flanks and the ultrasound evaluation must be extended to the lateral margins of the uterus to avoid inadequate measurements of the amniotic fluid.

The vertical diameter of the largest pocket in each quadrant, once identified, is measured. Care is taken to avoid any loop of cord. The numbers obtained from each quadrant are added up. The added up figure represents the amniotic fluid index or AFI of the patient.[132]



ig. 7: Four quadrant method for AFI estimation

Phelan's classification of amniotic fluid indices[1]:

AFI (in cms)	Classification
< 5	Oligohydramnios
5 – 8	Borderline low
8 – 18	Normal
18 – 24	High normal
>24	Polyhydramnios

Chart 2 : Classification of amniotic fluid indices

Quantitative assessment of amniotic fluid volume :[79,80,81]

1. One centimeter pocket :

In 1979, Manning and Platt described the quantitative estimation of amniotic fluid volume as ascreening test for IUGR.[4]

Using ultrasound, the largest cord free pocket of amniotic fluid was identified and the vertical and transverse diameters of this pocket were measured. If the pocket measured 1 cm or more in its broadest diameter, it was considered normal and if measured less than 1 cm it was considered decreased.

2. Two centimeter pocket :

Chamberlain et al., who determined the criteria for classifying liquor volume based on the measurement of single deepest vertical pocket dimensions.[2]

Chamberlain's criteria :

Single deepest pocket (in cms)	Inference
< 1	Oligohydramnios
1 – 2	Marginal
2 - 8	Normal
>8	Polyhydramnios

Chart 3: chamberlain's classification of liquor volume

3. Three centimeter pocket :

In 1984, Crowley and O'Herlihy assessed the efficacy of ultrasound estimation of amniotic fluid volume in detecting fetal compromise in pregnancies beyond 42 weeks. When patients had no single vertical pocket

measuring more than 3 cm there was a statistically significant increase in meconium staining and fetal distress.[53]

4. Two diameter amniotic fluid pockets :

In this technique, the vertical and horizontal dimensions of largest amniotic fluid pocket is measured and it is expressed as a product of vertical and horizontal dimensions.[51]

Two diameter fluid pocket (cm^2)	inference
15 – 50	Normal
< 15	Oligohydramnios
>50	Polyhydramnios

Chart 4 : Classification of two diameter fluid pocket

All the above mentioned techniques are used for assessment of amniotic fluid volume with reasonable accuracy.

The single deepest pocket method using 3 cm as lower limit (Crowley and Herlihy, 1984) has been used in the present study to asses the amniotic fluid volume.

Oligohydramnios :

It is defined as a condition where the amniotic fluid is deficient in amount. Clinically it is diagnosed if the amniotic fluid volume is less than 200 ml at term.[54]

Sonographically, AFI < 5 cm[1] (Phelan et al.,) or single deepest pocket < 2 cm (Chamberlain et al.,)[2] or two diameter fluid pocket < 15 cm^2 (Chauhan et al.,)[51] is used to define oligohydramnios.

It affects 1-5 % of all pregnancies and overall incidence is 3.9 % of all pregnancies.[3]

Aetiology :[54]

Fetal :

- Chromosomal abnormalities
- Congenital abnormalities
- Growth retardation
- Intrauterine demise of fetus
- Postterm pregnancy
- Ruptured membranes

Placenta :

- Abruption
- Twin-twin transfusion syndrome

Maternal :

- Uteroplacental insufficiency

- Hypertension
- Pre-eclampsia
- Diabetes, rarely

Drugs :

- Prostaglandin synthetase inhibitors
- ACE inhibitors
- Idiopathic

Potter's syndrome :[116]

It is the atypical physical appearance of the newborn due to decreased liquor in the uterus.

It classically comprises of :

- Clubbed feet
- Pulmonary hypoplasia
- Bilateral renal agenesis
- Cranial abnormalities
- Parrot beak nose
- Redundant skin
- Skin fold from medial canthus to cheek

Classic potter's syndrome develops when the fetus has bilateral renal agenesis and also agenesis of the ureters.[94]

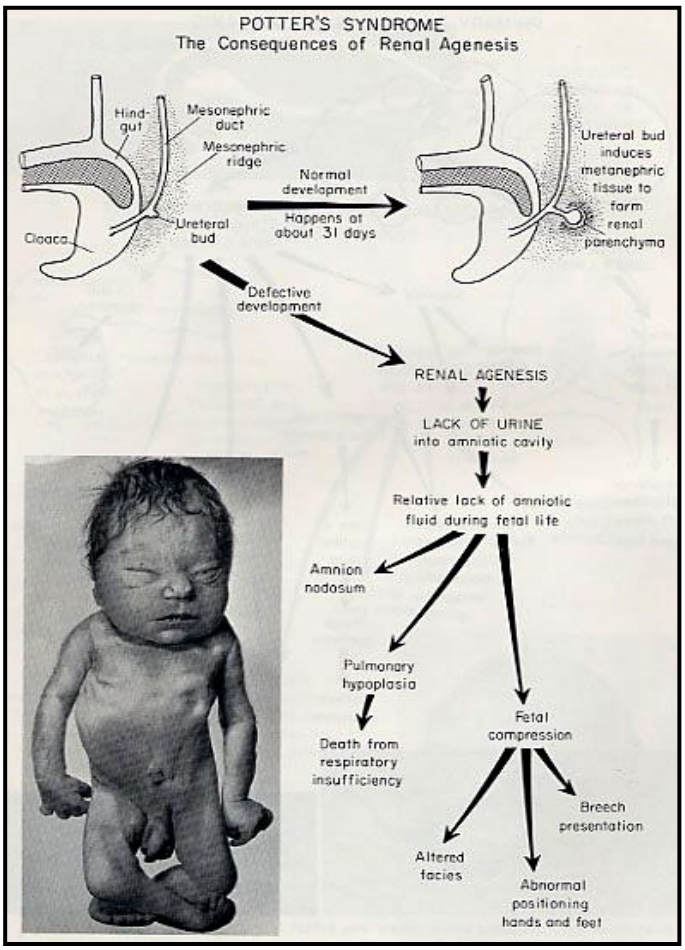


Fig. 8 : Potter's syndrome

Amniotic Band Syndrome :[109]

Amniotic band syndrome is also known as ADAM complex, or amniotic band sequence. It is a congenital disorder caused by entrapment of the fetal parts usually limbs or digits in fibrous amniotic bands while in utero. It affects 1 in 1200 live births.[152]

There are two main theories to explain it's occurrence -

- Amniotic band theory : It occurs due to partial rupture of the amniotic sac. The amnion is ruptured but the chorion remains intact. Fibrous bands of ruptured amnion float in the amniotic fluid and trap some part of the fetus.
- Vascular disruption theory : It occurs as a part of intrinsic defect in blood coagulation.

Fig. 9 : Amnionic band syndrome

Diagnosis :

(i) Careful history taking - period of amenorrhea, last menstrual period, fetal movement counts, history of oligohydramnios, preeclampsia etc. in previous child birth, congenital anomalies etc.

(i) Clinical estimation of liquor volume (as discussed earlier)

(iii) Dagnosis by ultrasound (as discussed earlier)

(iv) Other investigations : Blood : complete haemogram including haemoglobin, blood grouping and typing, VDRL,HIV, HBsAg, blood sugar, urea, creatinine, uric acid

Urine : albumin, sugar, microscopy.

Doppler study, NSTs, BPP etc.[115]

COMPLICATIONS

Maternal :[127-129]

Oligohydramnios per se does not lead to maternal complications; but it may be a complication of delivery, increased rates of interventions and malpresentations. It may lead to -

- Prolonged labour due to inertia.

- Increased operative interference.
- Malpresentations.
- Prolonged hospital stay, morbidity and increased costs due to operative interventions

Fetal :[110-114]

- Abortions.
- Deformity due to intra amniotic adhesions or due to compression. These include alteration in shape of skull, wry neck, club foot or even amputation of limb.[137,138]
- Fetal pulmonary hypoplasia (may be cause or effect).[124,130]
- Cord compression.
- Increased rates of labour induction and operative interference.
- IUGR.
- Neonatal ICU admissions.
- Intrauterine death of fetus, stillbirth or neonatal death.

Casey & co-workers in 2000 cited an incidence of oligohydramnios of 2.3 % in more than 6400 pregnancies undergoing sonography after 34 weeks at parkland hospital. They confirmed oligohydramnios is associated with increased risk of adverse perinatal outcome.[7]

Management of oligohydramnios :

Identify cause (if possible) :[55]

Detailed history to include drug exposure, maternal medical illness, prior screening for aneuploidy, prior USG findings, symptoms of membrane rupture.

Rupture of membranes is ruled out by physical examination.

USG to asses - degree of oligohydramnios, presence of growth deficiency, presence of fetal anomalies, presence and appearance of kidneys, presence of fetal renal artery, fetal and uterine blood flow studies, fetal wellbeing.

Further prognosis and management depends on gestational age, severity of oligohydramnios and diagnosis.

Early oligohydramnios (<34 weeks) :

Fetal outcome is generally poor in early onset oligohydramnios. Shenker and colleagues in 1991 described 80 pregnancies in which only half of the fetuses survived.[26] Mercer and Brown in 1986 described 34 midtrimester pregnancies with oligohydramnios, out of which only 6 infants were delivered at term and did well.[56]

Fetal surgery :

Fetal surgical approaches to the management of oligohydramnios fall into two areas - bladder drainage and tracheal occlusion. Shunt placement usually is done into bladder. More recently, reports of ablation posterior urethral valves by fetoscopy and vesico-amniotic shunts have been seen.[55] Fetoscopic surgery have been undertaken for some anomalies but data upon which to judge is limited. If anomalies are not amenable to treatment, it is better to counsel the patient for termination of pregnancy. In very early PPROM, there is significant dilemma. Given the poor pregnancy outcomes, termination is reasonable.

EXPECTANT MANAGEMENT

1. Thee should be regular assessment of amniotic fluid volume, BPP, and NST wherever applicable

2. Maternal hydration therapy :[136]

Goodlin and colleagues 1st found out that there is increase in AFI with maternal hydration.[107]

Similarly, Sherr and colleagues[57] and klipatrick and colleagues[58] also found an increase in AFI with maternal hydration therapy.

Mechanism is believed to be due to increase in uteroplacental blood flow. Doi and colleagues found out that only hypotonic solutions were effective in hydration therapy.[59]

3.Supplimentation of zinc and other multivitamins :

Zinc is a metalloenzyme and stabilizes the enzymes responsible for degradation of amniotic membrane. Helps in prevention of preterm rupture of membranes. Total dose-100 mg; 2-3 mg/ day.[60]

4. Aminoacid infusion therapy :

Parenteral amino acid infusion showed significant increase in AFI and lesser intervention rates. Usually a balanced solution of both essential and non essential amino acid is used.

Dosage : 200 ml at the rate of 40 drops/min on alternate days of 1 week.[61-62]

5. Extract of salvia miltiorrhiza :

It is a chinese herb whose extract is found to increase the uteroplacental outflow and hence increase the AFI.

Dosage : 50 ml thrice/day.[63]

6.Water therapy :

Physical activity in water is found to insrease AFI. Moderate activity for 50 mins, thrice per day improves the uteroplacental flow.[64]

7.Newer therapies :

Sealing of ruptured membranes with cryoppt., platelets, DDAVP etc. are under investigation.[55,142]

LATE ONSET OLIGOHYDRAMNIOS

Amnioinfusion :[120-123,125,127]

Numerous studies have evaluated whether intra-partum amnioinfusion may prevent fetal morbidity from meconium stained fluid - often associated with oligohydramnios. Pierce and colleagues in 2000 performed a meta-analysis of 13 such studies with more than 1900 women and found that amnioinfusion resulted in significant decrease in adverse outcomes.[70]

Conversely, spong and associates in 1994[71] and Fraser and associates in 2005[72] found that amnioinfusion did not reduce the risk of meconium aspiration syndrome, C.S. delivery or other adverse outcomes. Finally Xu and colleagues in 2007[73] performed a meta-analysis of 12 trials including 4000 women and did not find significant reduction in adverse outcomes.[153]

ACOG in 2006 has concluded that routine prophylactic amnioinfusion for meconium staining is not recommended.[74]

Two routes of amnioinfusion are currently in use, a) transabdominal and b) transvaginal

Transabdominal amnioinfusion :[65]

1. After informed consent and sterile preparation, a 20 G needle is advanced into the gestational sac under USG guidance, aiming for a fluid pocket or area between the limbs, avoiding the placenta.

2. Sterile tubing is attached to the stopcock, large syringe and saline intravenous line is attached to the needle, priming may be done to avoid entry of air.

3. Fluid (usually N.S) is transfused under USG guidance. Pre-warming of saline to 37 degree may be done.[65] FHR must be monitored during the process.

4. Patient should be kept under observation for 24 hours anti D immunoglobulin is administered in Rh negative mothers.

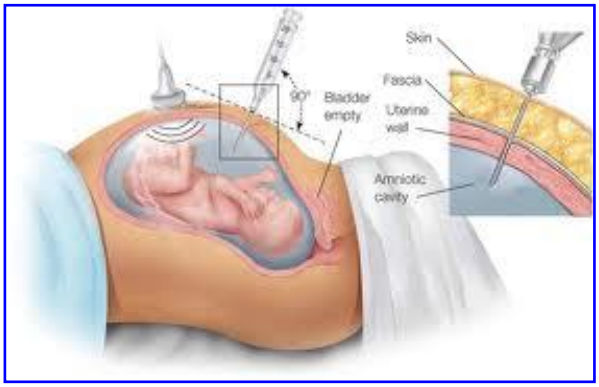


Fig.10 : Transabdominalamnioinfusion

TRANS VAGINAL AMNIOINFUSION[66-67]

Miyazaki and taylor described the technique for trans-vaginal amnioinfusion. Per vaginal examination is done to evaluate dilatation, presentation and exclude cord prolapse. Usually normal saline infused by intrauterine pressure catheter at bolus of 600 ml or at the rate of 10-12 ml/ min for the 1st hour, then at the rate of 3 ml/min. this was continued till delivery or recovery of deacceleration. Alternatively, a bolus of 250- 600 ml was given in 30-60 min period and then infusion is stopped when the AFI reaches 5-8 cm.

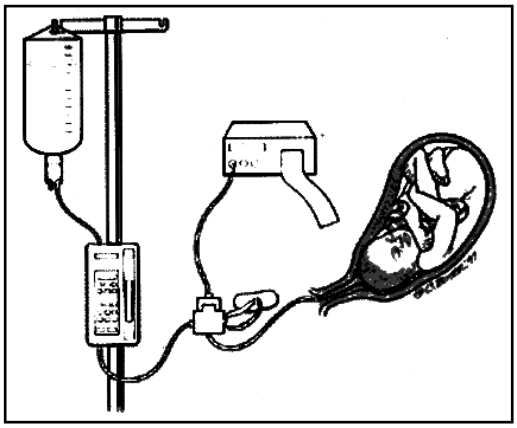


Fig. 11 : Trans vaginal amnioinfusion

Assessment of fetal maturity is done and then decision is taken regarding the mode of termination of pregnancy.

Complications :

- ★ Uterine hypertonus
- ★ Abnormal FHR
- ★ Infection
- ★ Amniotic fluid embolism
- ★ Cord prolapse
- ★ Uterine rupture
- ★ Placental abruption
- ★ Maternal death

INDUCTION OF LABOUR[68,111]

As termination is desired in cases of uncomplicated oligohydramnios, induction is usually employed. Studies have shown greater risk of caesarean section in cases who were induced compared with those who underwent spontaneous labour.

Indications :

- Maternal hypertensive disorders- pre-eclampsia, PIH
- Medical conditions - diabetes mellitus, chronic renal disease, heart disease
- PROM
- Intrauterine demise of fetus

Fetal :

- IUGR
- Postmaturity
- IUD
- Gross fetal anomalies

Augmentation of labour :

Maternal :

- Prolonged labour
- Dysfunctional labour
- Secondary arrest

Fetal :

- To prevent fetal hypoxia
- To prevent fetal infection

Monitoring of labour :

In the present study, partograph was used to monitor the course of labour.

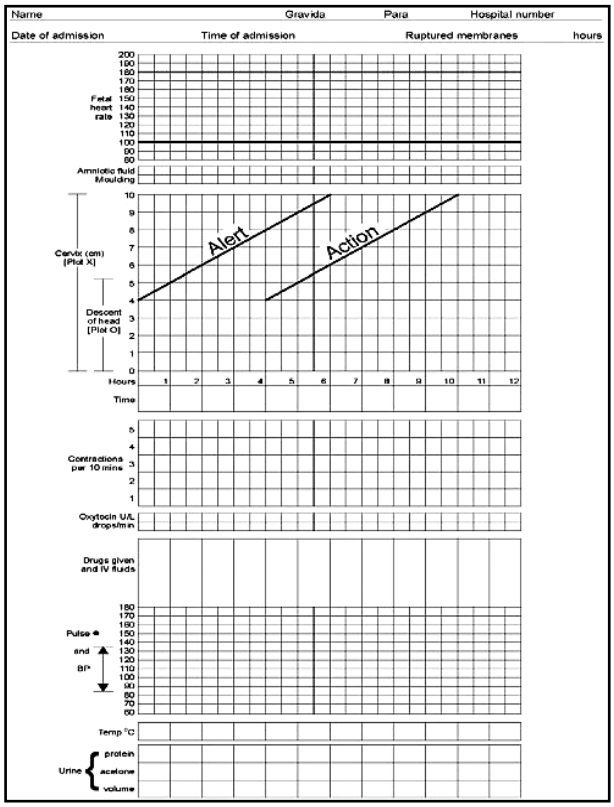


Fig.12 : Partograph

Induction-augmentation of labour in present study :

If Bishop's score < 6, tab. PGE1(misoprostol), 25 microgram, vaginally, 6 hourly.

If Bishop's score > 6, ARM followed by titrated oxytocin infusion was given.

Place of caesarean section :

Indications :

1. Fetal distress
2. Induction failure
3. Associated maternal complications
4. Obstetrical indications.

The present study was conducted to know the maternal and fetal outcome in cases of oligohydramnios diagnosed after 34 weeks of gestation. The study was carried out in the department of Obstetrics & Gynaecology of Silchar Medical College & Hospital, Silchar

A total of 200 pregnant patients (100 cases and 100 controls) were taken up during the period 1st August, 2012 to 31st July 2013.

In the present study majority of patients were in the age group 23-27. 60% of the cases and 58% controls were in this age group. The mean age of incidence was 24.6 years.

Majority of patients in our study were primigravidas i.e., 70% cases and 72% controls.

Among the cases, 88% were booked while among the controls, 86% were booked. The importance of this factor lies in the fact that early detection and management of the cases can minimise the fetal hazards and maternal morbidity.

In our study a single USG scan and estimation of single deepest liquor pocket was done to estimate liquor volume. Those with <3 cm pocket were labelled as oligohydramnios and those between 3-8 cm were classified as normal liquor volume.

The ultrasound diagnosis was supported by clinical findings of abdominal examination. In measurement of fundal heights, 24% cases had fundal heights < 32 weeks compared to only 1 % controls. This association was found to be statistically significant ($p<0.05$). Abdominal girth was significantly less than 80 cm ($p<0.05$) in 88% of cases.

In the present study, 30% cases and 26% controls had associated hypertensive disorders like PIH, pre-eclampsia, imminent eclampsia etc. Anemia was seen in 22% cases and 25% controls. Rh negativity was seen in 06% cases and 02% controls. There was no complicating factor in 38% cases and 43% controls. PROM, congenital anomalies, multiple gestation, IUD were excluded from the study. It cannot be stated clearly whether this conditions resulted in maternal or fetal hazards or decreased amniotic fluid per se is the cause. There is no such study in this regard to differentiate the factors. The association of maternal complications was not found to be statistically significant ($p<0.05$).

In our study, 17 % of the cases showed associated breech presentation compared to only 11% controls, adding support to the fact that oligohydramnios is a cause for malpresentations.

The present study also showed that 16% of the cases delivered spontaneously and 22% required induction. These rates were 83% and 05% respectively for control group. The rates of induction were significantly higher ($p<0.05$) among the cases.

Meconium staining was observed in 48% cases compared to only 12 % controls. This association was also statistically tested and found to be significant ($p<0.05$).

93% of the total caesarean section were for emergency indication among the cases and 6.45% went for elective LSCS. These rates were 83.33% and 16.64% respectively among the control group.This association was statistically significant ($p<0.05$).

The present study noted LBW babies (<2.5 kg) in 42% of cases compared to only 12% controls. This may be due to poor nutritional status and lower socioeconomic status. Statistical analysis revealed the association between LBW babies and oligohydramnios to be statistically significant ($p<0.05$).

The Apgar score at 1 min and 5 min were lower in cases as compared to the controls.After statistical analysis lowering of Apgar scores at 1 and 5 min were found to be significantly associated with oligohydramnios ($p<0.05$).

Among the cases who underwent C.S., 70% were due to fetal distress in case group compared to 16.67% in control group showing significant association between oligohydramnios and fetal distress and hence increased operative interventions. This association was found to be statistically significant ($p<0.05$).

Higher rates of adverse fetal outcome like stillbirth, IUD, NICU admissions, intrapartum death were noted in case group.

The duration of NICU stay and maternal hospitalisation was also considerably higher in case group adding to the morbidity and increased cost of healthcare. This association was also found to be statistically significant ($p<0.05$).

Table A : Showing distribution of LBW babies in various studies :[7,16,105]

Study	Cases	controls
Baron (1995)	4.7%	2.3%
Casey (2000)	16.32%	0.14%
Jandial(2007)	58%	--
Present (2013)	42%	12%

The association of LBW babies in the present study is higher than the studies by Baron (1995) and Casey (2000) but it is lower than that on jandial (2007). The socioeconomic status of Indian people as well as pre-pregnant nutritional status of mothers is quite poor as compared to the western counterparts which may have resulted in higher rates of LBW. Also, facilities for early detection and treatment are also better elsewhere.

Table B : Showing distribution of fetal distress in various studies :[7,16]

Study	Cases	Controls
Baron (1995)	4.1%	0.4%
Casey (2000)	32.65%	0.62%
Present(2013)	44%	02%

The association between fetal distress and oligohydramnios was seen in 44% cases which is comparable to Casey (2000) study, but is quite higher than Baron (1995) study. The higher incidence in Indian setup is due to late referral in most cases.

Table C : Showing distribution of caesarean section in various studies : [7,16,104,105]

Study	Cases	Controls
Mercer (1984)	24%	--
Baron (1995)	12.4%	4.65%
Casey (2000)	3.4%	0.04%
Jandial (2007)	56%	--
Present (2013)	62%	12%

The rates of C.S in the present study were lower than the western studies of Baron (1995) and Casey (2000) but the rate is comparable to the Indian study of Jandial (2007).

Table D : showing distribution of low 5-min Apgar score among the studies'[14,16,104]

Study	Cases	Controls
Mercer (1984)	7%	--
Golan (1994)	4.6%	--
Baron (1995)	0.06%	0.04%
Present(2013)	13.6%	5.14%

The 5-min Apgar score was lower i.e <7 in n13.6% of the cases of the present study which is higher than the studies of Mercer (1984), Golan (1994) & Baron (1995). It depends on the availability of adequate neonatal resucitation facilities and adequate set up for support of these depressed babies.

Table E : Showing distribution of NICU admissions : [7, 16,105,106]

Study	Cases	Controls
Baron (1995)	8.2%	6.9%
Garmel (1997)	18.5%	--
Casey (2000)	4.76%	0.03%
Jandial (2007)	16%	--
Present (2013)	33%	5%

The present study showed higher NICU admissions (33%) than other studies of Baron (1995), Garmel (1997), Casey (2000) and Jandial (2007). This may be attributed to higher meconium staining rates in the present study. Late referral may also be a cause causing delay in management.

A comparative look between the study of Charu Jandial et al., in 2007 with the present study is given below :[105]

	Charu jandial et al., (2007)	Present study (2013)
Induction	58%	22%
Caesarean section	56%	62%
Meconium stained liquor	24%	48%
Low birth weight	58%	42%
NICU admissions	16%	33%

The present study showed higher rates of meconium staining, C.S rates and NICU admissions compared to the study of Charu Jandial et al., but the induction rates and the incidence of LBW babies were lower in the present study.

These results support the fact that oligohydramnios is associated with considerable adverse fetal outcome and maternal morbidity. Early diagnosis, skilful management of cases and good paediatric backup will go a long way in improving the obstetric outcome.

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