A PROSPECTIVE STUDY OF INTRAVENOUS HYDRATION THERAPY IN CASES OF OLIGOHYDRAMNIOS IN THIRD TRIMESTER AND ITS EFFECTS ON PERINATAL OUTCOME

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BONAFIDECERTIFICATE

This is to certify that this dissertation is a bonafide work of **Dr. SHARMILA.P** on **''A PROSPECTIVE STUDY OF INTRAVENOUS HYDRATION THERAPY IN CASES OF OLIGOHYDRAMNIOS IN THIRD TRIMESTER AND ITS EFFECTS ON PERINATAL OUTCOME"** during her M.S., (Obstetrics and Gynaecology) course from July 2013 to July 2016 at the Government Stanley Medical College and Government Raja Sir Ramasamy Mudaliar Lying-in Hospital, Chennai.

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DECLARATION

I, Dr. SHARMILA. P. Solemnly declare that the dissertation **''A PROSPECTIVE STUDY OF INTRAVENOUS HYDRATION THERAPY IN CASES OF OLIGOHYDRAMNIOS IN THIRD TRIMESTER AND ITS EFFECTS ON PERINATAL OUTCOME"** is a bonafide work done by me at Government R.S.R.M Lying in Hospital, under supervision and guidance of **Associate Prof. Dr. ARASI SRIVATHSAN, M.D., OG** in Department of Obstetrics and Gynaecology, Government Stanley Medical College, Chennai. This thesis is submitted to The Tamil Nadu Dr. M.G.R. Medical University in partial fulfilment of the rules and regulations for the M.S. Degree examinations in Obstetrics and Gynaecology to be held in April 2016.

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CONTENTS

INTRODUCTION

Amniotic fluid forms an aquatic pond inside the amniotic cavity surrounding the fetus.

It provides a protected milieu for the growing fetus, supplying nutrients facilitating growth, movement and cushioning the fetus against mechanical and biological injury.

It promotes normal development of fetal lungs and musculoskeletal and gast rointestinal systems. It helps to avert compression of umbilical cord and creates a physical space for the fetal skeleton to shape normally. It has antibacterial properties,and serves as a reservoir of water and nutrients.

The quantity of liquor amnii increases from 25ml at 10 weeks to 400ml at 20 weeks. The volume increases to about 800 – 1000ml at 28 weeks. Plateaus near term and declines to about 400ml at 42 weeks.[1]

Reduction in the volume of amniotic fluid is called oligohydramnios, affecting 3% to 5% of pregnancies. Amniotic fluid index is a tool used for semi quantitative measure of amniotic fluid and is a third trimester technique useful

after the fundus is above the umbilicus. Amniotic fluid index is a more objective and reproducible method of assessment of amniotic fluid volume as it estimates the amniotic fluid in four quadrants of the uterus. An AFI of 5-18cm is considered normal.

Phelan[2] who described Amniotic Fluid Index defined oligohydroamnios as AFI less than 5cm.Using amniotic fluid index of less than 5cm the incidence of oligohydrmnios was found to be 2.3% after 34 weeks.oligohydramnios is associated with increased risk of adverse perinatal outcome.The umbilical cord compression during labour is common with oligohydramnios which increases the risk for caesarean delivery for fetal distress and 5minutes APGAR score less than 7.

Oligohydroamnios in third trimester is associated with non-reassuring fetal heart pattern, increased labour induction, meconium aspiration syndrome, still birth and neonatal death – casey & co workers 2004.[3]

Sherer etal in 1990[4] confirmed a significant increase in amniotic fluid volume after oral or intravenous hydration in women with oligohydramnios. This

present study is undertaken to assess the effect of intra venous maternal hydration therapy in third trimester oligohydramnios and to assess the perinatal outcome.

AIM OF THE STUDY

1. To study the impact of intravenous maternal hydration therapy on amniotic fluid index in cases with oligohydramnios in third trimester.

2. To study the perinatal outcome in these patients.

REVIEW OF LITERATURE

Amniotic fluid is normally the clear fluid that collects within the amniotic cavity. This fluid provides several important benefits.Amniotic fluid has a number of important roles in embryo/fetal development like,

1. Permitting fetal movement and the development of the musculo skeletal system.
2. Swallowing of amniotic fluid enhances the growth and development of gastrointestinal system.
3. Amniotic fluid volume maintains amniotic fluid pressure there by reducing the loss of lung liquid an essential component to pulmonary development.(Nicolini,1989)
4. The ingestion fluid provides some fetal nutrition and essential nutrients.
5. Amnioticfluid protects the fetus from external trauma.
6. Its constant temperature helps to maintain the embryo's body temperature.
7. Its bacteriostatic properties reduce the potential for infection.
8. Protects the umbilical cord from compress*ion.*

The Amnion;

The amnion is first identifiable about 7^{th} or 8^{th} day of embryo development. To start with a minute vesicle, amnion develops into a small sac that covers the dorsal surface of the embryo. Amnion engulfs the growing embryo when it enlarges.

Amnion, that contains amniotic fluid is a avascular membrane which is a tough but pliable membrane at term. For the successful pregnancy outcome, development of components of amniotic membrane that protect against its rupture is vitally important.

Bourne (1962) [5] described the layers of amnion.

The inner surface is an uninterrupted single layer of cubical epithelium, which is believed to be derived from embryonic ectoderm. This inner surface is bathed by amniotic fluid.

This epithelium is firmly attached to a distinct basement membrane. This basement membrane is connected to a acellular, compact layer. This acellular layer is composed of interstitial collagens. Row of fibroblast like mesenchymal cells are present on the outer side of the compact layer, which are probably derived from embryonic disc mesoderm. Contiguous with the second fetal membrane chorionic leave Vs the outer most layer of amnion which is the relatively acellular Zona spongiosa.

The human amnion contains few fetal macrophages, but it lacks smooth muscle cells, nerves, lymphatics and importantly blood vessels. During early implantation a space develops between the embryonic cell mass and the adjacent trophoplasts. Inner surface of trophaplasts is lined by small cells have been called amniogenic cells which are precursors of ammonic epithelium.

Amnion Epithelial Cells

The epithelial cells of the amnion are derived from fetal ectoderm of the embryonic disc. It is an important consideration from both functional and embryological perspectives. ie, HLA class I gene expression in amnion is more akin to that in cells of the embryo than that in trophoblasts.

Early in human embryogenesis, the amniotic measenchymal cells lie immediately adjacent to the basal surface of the epithelium. At this period of development the amnion surface is a two cell layer structure with approximately equal numbers of epithelial and measenchymal cells. As interstitial collagens are deposited between these two layers of cells, there is a formation of the compact layer of the amnion, which also brings about a distinct separation of the two layers of amnion cells.

Amniotic epithelial cells have highly developed microvilli in the apical surface that are consistent with a major site of transfer between amniotic fluid and amnion. These cells syathesize tissue inhibitor or metallo proteinase – 1, PGE_2 and fetal fibronectin.[6] Amniotic epithelium participates in the final common pathway of labour initation by prostaglandin production.

Amniotic epithelial cells may respond to variety of endocrine or paracrine modulators derived from fetus or the mother, oxytocin and vasopressin are the examples both of which increase the PGE_2 production in vitro[7]

Amniotic epithelium also synthesizes vaso active peptides like endothelin and para thyroid hormone related protein [8]

Brain natriuretic peptide and corticotrophin – releasing hormone are also produced which are the smooth muscle relaxants [9.] vaso active peptides produced in amnion gain access to adventitial surface of chorinic vessels and may be involed in modulating chorionic vessel tone and blood flow. Amnion derived vasoactive peptides, after their secretion, enter amniotic fluid and thereby are available to the fetus by swallowing and inhalation.

These peptides function in other tissues in diverse physiological processes. Amnionic epithelial cells also produce cytokines such as IL-8 during initiation of labour [10]

Amnion Mesenchymal Cells

Measenchymal cells of the amnion are responsible for other major functions. Synthesis of interstitial collagens – the major source of its tensile strength takes place in measenchymal cells.. cytokines, IL – 6, IL-8, and monocyte chemoattractant protein – 1 (MCP – 1) are synthesized by mesenchymal cells. In response to bacterial toxins and IL– 1 cytokine synthesis will increase.

This functional capacity of mesenchymal cells of the amnion is an important consideration in amniotic fluid study for evidence of labour – associated accumulation of inflammatory mediators [11]

Mesenchymal cells may be a greater source of prostaglandin E_2 than amniotic epithelial cells. [12]

Formation and sources of amniotic fluid;

The amniotic fluid is derived from the mother directly across the amnion and the fetal surface of the placenta and fetal body surface in the first trimester.

During embryogenesis amniotic fluid volume increases faster than embryonic size. There is a rapid diffusion across the embryonic skin as it is only four cell layers thick.

During the embryo-fetal development,the metanephri begin to develop by seven weeks menstrual age and are functional by 10-11 weeks menstrual age.

Because glomerular filtration precedes tubular function fetal urine is initially relatively hypotonic.As the fetus matures,resorption of sodium,chloride and water occurs and the excretion of urea and cretinine increases.

These alterations in fetal renal function in part explain the changes that are seen in the composition of amnioticfluid with advancing gestational age.

Amnioticfluid volume increases from 25ml at 10 weeks to about 400ml at 20 weeks.Its composition is similar to fetal plasma during this period.

Not yet keratinized fetal skin allows a rapid bi directional diffusion between the fetus and amniotic fluid. The surfaces of the amnion, placenta and core umbilical cord each being freely permeable to water and solutes during this period of

pregnancy .The amniotic fluid serves both as a physiologic buffer and an extension of fetal extra cellular compartment.

Even though, fetal kidneys, make urine by 8weeks of gestation, and swallowing begins shortly thereafter, neither fetal urination nor swallowing contributes significantly to the volume or content of amnioticfluid until second half of pregnancy. The fetal skin begins to keratinize at 20 weeks, process being completed by 25 weeks of gestation.

After the fetal skin is fully keratinized, production of AF is predominantly accomplished by excretion of fetal urine (300ml / kg fetal weight / day or 600 to 1200ml / day near term) and the secretion of oral nasal, tracheal and pulmonary fluids 60-100ml kg fetal weight / day) [13]

Near term the major sources of amniotic fluid production are fetal urine and lung liquid. The major routes for resorption of fluid are fetal swallowing and the intra membranous pathway (from amniotic fluid to fetal circulation).The minor sources of amniotic fluid production and clearance include secretions from the fetal oral, nasal cavities and the transmembranous pathway (amniotic fluid to maternal circulation.)

Based on current concepts of amniotic fluid dynamics[14,] there are six routes for fluid entry into and exit from the amniotic sac for the late gestation fetus;

1. Fetal urine
2. Fetal swallowing
3. Oral- Nasal secretions
4. Secretion from respiratory tract
5. Intra membranous pathway
6. Trans membranous pathway

Flow in to the amniotic sac;

Fetal urine production -800 to 1200mL/day

Fetal lung liquid secretion -170 mL/day

Oral-nasal secretions -25mL/day.

Flow out of the amniotic sac;

Fetal swallowing-500 to 1000mL/day

Intramembranous flow -200 to 400mL/day

Transmembranous flow -10mL/day

Fetal urine is the major contributor of the volume of amniotic fluid in the later half of pregnancy. The volume of urine produced by human fetus per day during later half of pregnancy is 35% of its body weight[15.]

The fetal swallowing of amniotic fluid is evidenced by presence of epidermal debris including lanugo hairs in the meconium. The fetus swallows amniotic fluid which is equivalent to 15% of its body weight[16].

Fetal oral and nasal secretions also enter the amniotic fluid, but less than 1% of the body weight per day [17]

The pathway for exchange between amniotic fluid and the fetal blood within the fetal surface of placenta is referred to as intramembranous pathway. Osmotic flow of water and diffusion of solutes occur in this pathway, ie, water and solutes can move in opposite directions.

During late gestation, 400ml of water is absorbed daily intramembranously from amniotic fluid.

Fetal lungs secrete large volumes of fluid each day and less than 1% of this secreted fluid is needed to expand the lungs, and the remainder flows out of the lungs and may either enter the amniotic fluid or be swallowed as it exits the trachea. The amniotic fluid from the trachea is the source of surfactants, used as an indicator of fetal lung maturity. Only under condition of fetal asphyxia or in severe distress fetal lungs absorb fluid. Although meconium staining of amniotic fluid is common, aspiration of meconium into the lungs of the new born is relatively uncommon. Lung secretions rates are approximately are 10% of fetal body weight daily[18].

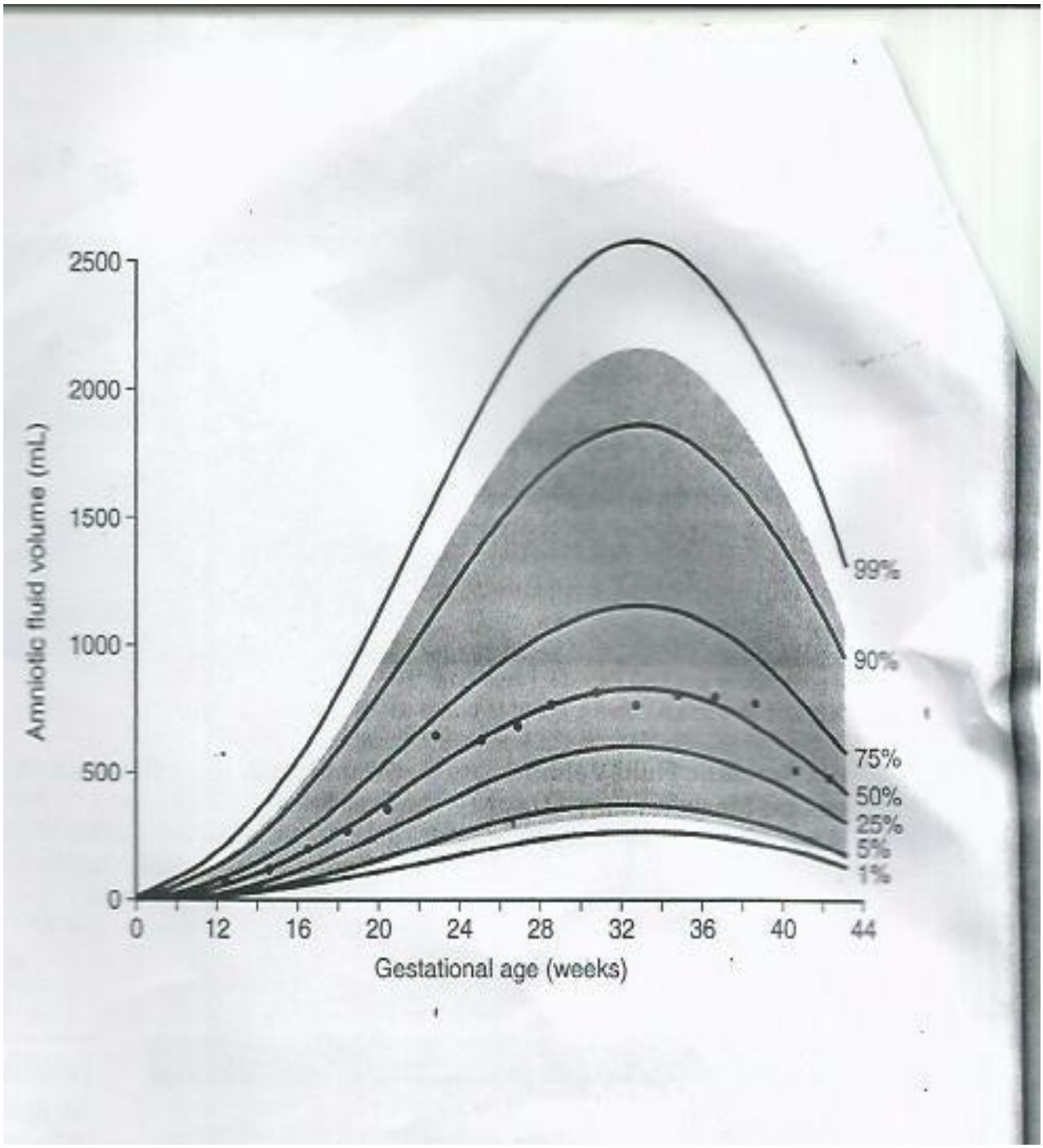
The passive exchange of amniotic fluid between the amniotic fluid and the maternal blood within the uterine wall, is known as trans membranous pathway. It is estimated to be as little as 10ml daily near term under normal conditions[19].

CHANGES IN AMNIOTIC FLUID VOLUME ACROSS GESTATION

In human pregnancy amniotic fluid volume increases dramatically during the first two trimesters,from 25ml at 10 weeks to 800 ml at 28 weeks.Amniotic fluid volume changes in pregnancy were studied by Brace and wolf [20],and observed that , amniotic fluid volume rises progressively during gestation until approximately 32 weeks.From 32 weeks to term the mean amniotic fluid volume is relatively constant in the range of 700-800ml.Although the average amniotic fluid volume in the third trimester is 700 to 800ml ,the range of normal is very wide and a normal fetus at 32 weeks may have more than 2000ml,or less than 500ml of amniotic fluid.This wide range of normal causes problem of assessing the amniotic fluid volume in pregnancy.

After 40 weeks, there is a progressive decline in amniotic fluid volume at the rate of 8% per week, with amniotic fluid volume averaging only 400ml at 42 weeks.

The variation in normal fluid volume below the mean value is smaller than the upper variation in the third trimester oligohydramnios,defined as the 5^{th} percentile which is approximately 300ml.Variations in the upper range is almost threefold greter,so that hydramnios –more than 95^{th} percentile varies from 1700-1900ml.


2500
2000
1500
1000
500
0
Amniotic fluid volume (mL)
99%
90%
75%
50%
25%
5%
1%
0
12
16
20
24
28
32
36
40
44
Gestational age (weeks)

REGULATION OF AMNIOTIC FLUID VOLUME

Late in gestation when the amniotic fluid volume averages 700-800ml, 100ml daily leaves the amniotic compartment. Only minor or moderate aberrations in flows readily lead to oligohydramnios or polyhydramnios. There are no known sensors for amniotic fluid volume, could be part of a control loop to return amniotic fluid volume towards normal whenever it becomes too high (or) too low.

In late gestation fetal lung and urine are the two major sources of amniotic fluid. Removal of fluid depends on fetal swallowing and intra membranous transport via skin, placental and cord surfaces.

Fetal urine flow, lung liquid secretion and swallowing are known to be regulated. Intra membranous absorption is controlled by factors that regulate the intra membranous permeability and surface area.

Recent studies[21] suggest that even slight changes in intra membranous permeability can have very large effects on intra membranous fluid rates.

Substances like prostaglandins excreted by fetal kidneys are released by amnion or chorion which enters the amniotic fluid could alter intra membranous permeability and thus lead to alterations in amniotic fluid volume.

Hormonal factors play a role in amniotic fluid regulation. Cortisol and antidiuretic hormone affect the permeability of amnion. Intra amniotic injection of prolactin shown to reduce the amniotic fluid by 50% by stimulating the transport of water from the fetus to maternal compartment [22]

OLIGOHYDRAMNIOS

Definition:

Oligohydramnios is a condition in which the amount of a amniotic fluid is decreased less than normal level.(normal level of AFI is 8-15cm.)

The definition has varied with different investigators.

Manning et al [23] defined oligoamnios when the largest pocket on ultrasound in its largest diameter measured less than 2cm.

Phelan [2] who described amniotic fluid index, defined AFI – less than 5cm as oligohydramnios.

More recently, oligohydramnios is defined as less than 3rd and 5th percentile for the gestational age.[24]

Definition for oligohydramnios given by various authors

	Technique	Definition	Reference
1.	Dye dilution	200ml	Horsager etal (1994)
2.	Dye dilution	500ml	Maann etal (1992)
3.	12-studies Direct Measurement or dye Dilution	318ml	Brace and wolf (1989)
4.	Ultrasound	Single deep vertical Pocket < 5cm	Mercer ret al (1984)
5.	Ultrasound	Single deep vertical Pocket <1cm	Manning et al (1981)
6.	Ultrasound	Single deep vertical Pocket <2cm	Manning et al (1990)
7.	Ultrasound	Single vertical Pocket <3cm	Halpenin et al and Crawley et al (1984-85)

8.	Ultrasound	Two diameter Pocket (Vertical X horizontal) < 15cm	Magnann et al (1992)
9.	Ultrasound	AFI < 5^{th} Percentile for gestationalage	Moore (1990)
10.	Ultrasound	AFI < 5cm	Phelan (1987)
11.	Ultrasound	AFI <7cm	Dizon – Townson (1996)
12.	Ultrasound	AFI <8cm	Jeng et al (1992)

Clinically the diagnosis of oligohydramnios is considered when the fundal height is small for gestational age or if the fetal parts can be easily palpated and the uterus feels as if it is clamped over the fetal parts,with little ballottment.

Breech like malpresentations are common as there is no enough liquor for the fetus to turn spontaneously.

Methods Of Quantification Of Amniotic Fluid

Measurements of amniotic fluid can be made directly, indirectly (or) estimated sonographically.

During cesarean section (or) hysterotomy [25] amniotic fluid volume can be directly measured.

Indirect measurements are done by dye – dilution techniques via amniocentesis. Para – amino hippurate used dye dilution technique has been shown to be representative of actual amniotic fluid volume obtained by direct measurement [26]

The dye injection technique is considered the gold standard for measuring actual amniotic fluid volume, but it is impractical to use this invasive test to measure amniotic fluid volume in clinical practice at risk for loss and infection.

These techniques are invasive time consuming and may require laboratory support, hence amniotic fluid volumes are usually estimated by ultrasound.

The sonographic assessment of amniotic fluid volume is semi quantitative.

Sonographic evaluation of amniotic fluid volume includes four methods:

1. Subjective assessment
2. Assessment of a single pocket (eg.2 x 2 measurement)
3. Single deepest pocket or maximum vertical pocket (SDP)
4. Amniotic fluid index(AFI)

Amniotic fluid may be described subjectively, but accuracy is dependent on operator experience and gestational age. Goldstein and Filly (1988) reported good intraobserver and interobserver agreement between subjective assessment and single largest pocket determination of amniotic fluid volume. Disadvantage is an inability to compare results from serial examinations as the fetal or maternal conditions.[27]

In practice, the amniotic fluid volume is measured by semi quantitative measures like amniotic fluid index (AFI), single deepest vertical fluid pocket (SDP) ,or assessment of a single pocket (eg.2x2cm)

Amniotic fluid index first proposed by phelan et al[2] is the summation of the vertical diameter of the largest pocket in each of four quadrants. The maternal umbilicus is considered as a central reference point. The transducer should be

oriented in the longiudinal plane. A minimum horizontal measurement of 1cm is considered. Oligo hydramnios was defined as AFI <5 cm.

Jeng and co workers [28] have defined oligohydramnios as an AFI <8 cm. They demonstrated increased incidence of meconium staining, caesarean delivery for fetal distress, abnormal fetal heartrate pattern and APGAR <7 at one minute when AFI was less than 8cm.

Chamber lain [29] proposed the single deepest vertical pocket, and is simply found by identifying the largest pocket of amniotic fluid after a global assessment and selecting the largest vertical measurement with a minimum horizontal measurement of 1cm.

A two – diameter pocket (cm^2) is calculated by multiplying the depth and width of the largest single pocket [30].

Magann and associates found that AFI was reasonably reliable in estimating normal or increased amniotic fluid but in case of oligohydramnios it was inaccurate.

Morris and collegues (2003) [31] found that the AFI was superior to the single deepest pocket of amniotic fluid.

AFI may be modulated by several factors.

Yancey and Richards (1994) reported that high altitude was associated with an increased amniotic fluid index (600 ft) [32].

Maternal hydration increased the amniotic fluid index [33]

Brace and wolf [20] identified that from 22 through 39 weeks of gestation despite an increase in fetal weight from 500gm to 3500gm the average volume of amniotic fluid remained unchanged and this suggests that amniotic fluid volume is carefully regulated.

Aetiology Of Oligo Hydramnios

A number of conditions [34] are associated with oligohydramnios.

1. Fetal causes;

- Congenital anomalies
- Chromosomal abnormalities
- Fetal demise
- Growth restriction

- Post term pregnancy
- Ruptured membranes

2. Maternal causes;

- Utero placental insufficiency
- Hypertension
- Pre eclampsia

-Diabetes insipidus

3. Drugs

- Prostaglandin Synthesis inhibitors
- Angiotension converting enzyme inhibitors

4. Placental factors;

- Abruption
- Twin twin transfusion

5. Idiopathic

Congenital anomalies associated with Oligohydramnios[35]

I. Genito urinary:

a) Renal agenesis,

b) Bilateral multicystic kidneys

c) Infantile poly cystic kidney disease

d) Renal dysplasia

e) Urethral obstruction

f) Bladder extrophy,

g) Meckel – Gruber syndrome,

h) Urethro pelvic junction obstruction

i) Prune-belly syndrome.

II. Central Nervous system:

a) Holo prosencephaly,

b) meningocele,

c) encephalocele,

d) microcephaly

III. Chromosomal abnormalities:

a) Triploidy,

b) trisomy 18,

c) Turner syndrome

IV. Cardiac

a) Fallot tetralogy

b) septal defects

V. Cloacal dysgenesis

VI. Cystic hygroma

VII. Diaphragmatic hernia

VIII. Skeletal:

a) Sireno melia,

b) sacral agenesis,

c) absent radius

d) facial clefting

IX. Twin Reverse Arterial Perfusion sequence

X. Twin –twin transfusion

XI. VACTERL (vertebral, anal, cardiac, tracheo – esophageal, renal, limb) association.

The distribution of abnormalities associated with oligohydramnios in third trimester is different.In study by shipp et al [36]of patients greater than 28 weeks

described a 28% rate of fetal growth restriction ,9% rate of abruption ,and a 4% rate of congenital anomalies.This study excluded premature rupture of membrane patients ,14% of patients with oligohydramnios patients ruptured membranes after the diagnosis had been made.

The incidence of aneuploidy and structural malformations with oligohydramnios range between 4.4 – 30.7% and 7 – 37% respectively

Oligohydramnios in the presence of IUGR, or preeclampsia has markedly worse perinatal outcomes .

Intrauterine Growth Restriction and Oligohydramnios;

The association between FGR and oligohydramnios has been attributed to decreased fetal urine production due to decreased utero placental perfusion.

Fetal hypoxia causes redistribution of cardiac output to fetal brain diverting the blood supply away from kidney and lungs.This results in reduced fetal urinary production and decreased lung secretions both of which contribute to amniotic fluid volume. The risk of FGR progressively increases as the severity of oligohydramnios increases.

The prevalence of IUGR is 5%,20%, and 37% respectively when a single pocket of amniotic fluid is >2cm,between 1&2 cm and <1 cm [37.]

Besides congenital malformations and chromosomal defects the increased perinatal mortality can be largely explained by the conditions associated with oligohydramnios like fetal growth restriction, hypertension, pre mature rupture of membranes and post maturity.

Prolonged Pregnancies and Oligohydramnios;

After 38 weeks fluid volume declines by approximately 125 ml / week, to an average volume of 800ml at 40 weeks, and has a massive reduction by 33% per week after 42 weeks of gestation [38]. The drop is attributed to reduced urine output by the fetus, which in turn is caused by the redistribution of fetal circulation and reduction in renal perfusion. The fluid becomes thick due to an increased amount of vernix caseosa. So, the volume is reduced and the density increased. Addition of meconium in such amniotic fluid leads to respiratory complications for the new born baby .

Preterm Rupture of Membranes and Oligohydramnios;

Spontaneous rupture of membranes between 24 and 34 weeks gestation occurs in 1.7% pregnancies.[36]

Maternal Hypovolemia and Oligohydramnios;

The other factors that affect amniotic fluid are maternal hydration and fetal presentation. Oligohydramnios in association with acute maternal hypovolaemia improved with intravenous hydration.[39]The changes in amniotic fluid volume is mediated by changes in intra membranous flow. A significant increase in amniotic fluid volume in women with oligohydramnios have confirmed after oral or intravenous hydration. [40]

Iatrogenic Oligohydramnios;

First trimester chorionic villus sampling and second trimester amniocentesis are associated with oligohydramnios. The neonatal outcome is good if amnioticfluid volume returns to normal subsequently.[41]

Drugs like prostaglandin synthetase inhibitors inhibit renal vascular flow & thereby reduce amniotic fluid volume. They also cause closure of ducts arteriosus[42] .Amniotic fluid reaccumulates when these drugs are discontinued. When late second or third trimester women are given prostaglandin synthetase inhibitors, serial ultrasound examinations are warranted.

Angiotensin converting enzyme inhibitors are associated with oligohydramnios and growth restriction and anuria.[43]

These drugs cause fetal renal damage, including renal tubular dysgenesis and renal failure.

Isolated Oligohydramnios;

Isolated oligohydramnios may occur in late pregnancy may be incidentally diagnosed on a routine ultrasound in patients with no other high risk factors associated with oligohydramnios. RADIUS trial in which low risk women had a routine ultrasound at 15-22 and at 31-35 weeks, 0.8% had oligohydramnios, of which about 50% had isolated oligohydramnios. The perinatal outcomes were similar to those with normal amniotic fluid volume [44]

Labour induction is attempted in these cases in spite of their cervix is unfavourable for induction .This can often result in caesarean section delivery for failed induction.

Maternal effects of oligohydramnios

Oligohydramnios may be associated with an increase in maternal complications.Premature rupture of membranes is associated with increased risk of

chorioamnionitis and maternal morbidity.Obstetric risks observed are an increased risk for caesarean delivery and increased risk for labour induction.

Fetal effects of oligohydramnios

Reduced amniotic fluid in the first trimester is an ominous finding, the pregnancy often aborts. In one study 94% with smallsac and a normal heart rate spontaneously abort compared with only 8% with normal sac size.[45]

In the second trimester the prognosis depends on the underlying etiology and severity of the oligohydramnios. Pregnancies with isolated mild or moderate idiopathic oligohydramnios have a good prognosis, but in contrast severe second trimester oligohydramnios results in fetal or neonatal death.

Early onset oligohydramnios has significant perinatal morbidity and mortality due to associated congenital anomalies, pulmonary hypoplasia.

Oligohydramnios concurrent with early onset fetal growth restriction and hypertension, and PPROM contributed to perinatal morbidity and mortality.

In particular, severe oligohydramnios due to PPROM in the second trimester is associated with increased perinatal mortality when the duration of the latency

period is more than 14 days.[45] The key element for survival is the presence of atleast some amniotic fluid between 18 and 23 weeks gestation. Absence of amniotic fluid during this interval regardless of the mechanism is highly associated with increased perinatal mortality.

Preterm delivary occurs in >50% of pregnancies with severe second trimester oligohydramnios either spontaneously or indicated by maternal or fetal complications.

Surviving infants of second trimester severe oligohydramnios cases may have significant morbidities. Infants may have anatomic and functional abnormalities.

Fetal deformities with oligohydramnios include potter facies (low set ears,epicanthic folds, receding mandible and flattened nose) and abnormal position of feet and hands [45]

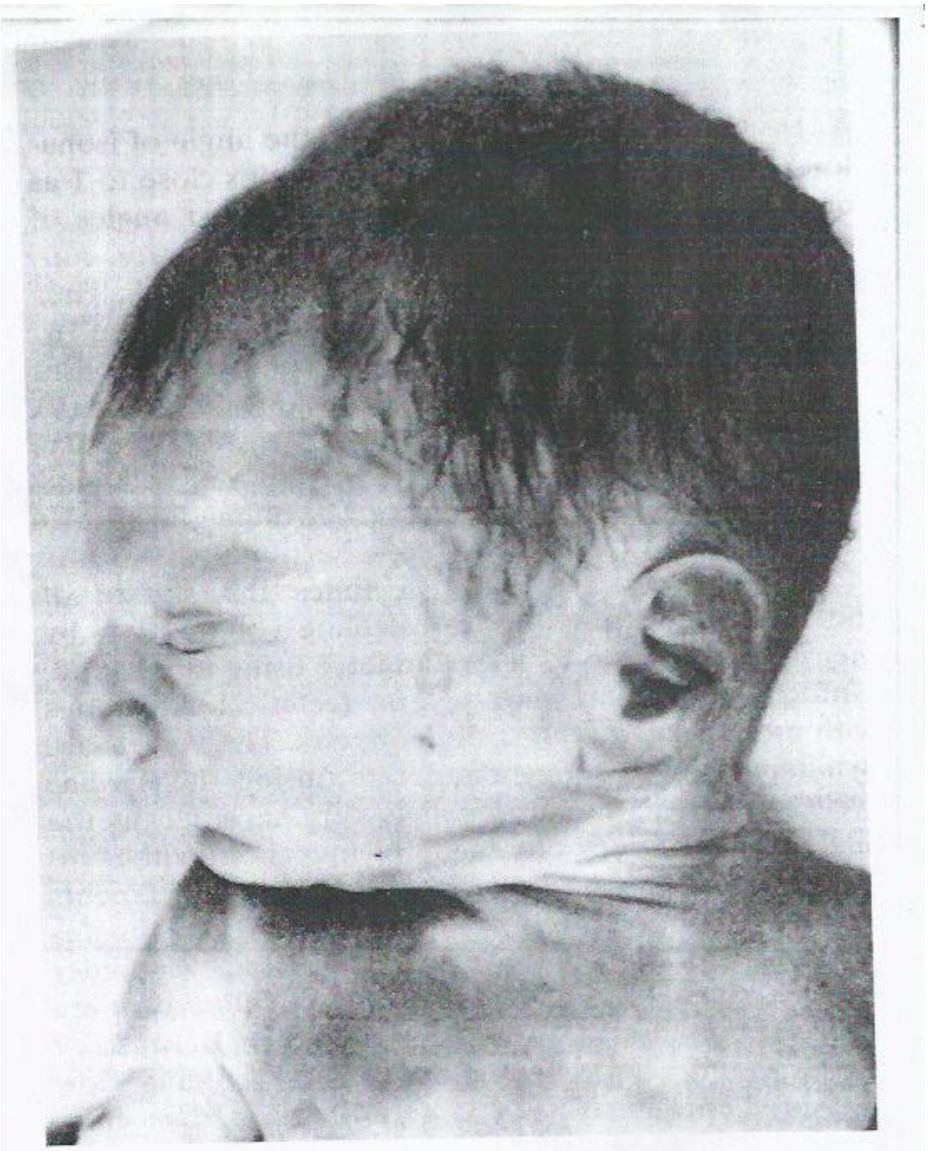


FIGURE 41–18. Infant with typical "Potter face," which is characteristic of renal agenesis and early oligohydramnios. (From Zerres and colleagues, 1984, with permission.)

The presence of oligohydramnios in second trimester markedly increases the fetal risk for pulmonary hypoplasia. There are 3 possibilities that account for pulmonary hypoplasia. First one thoracic compression may prevent chest wall excursion and lung expansion. Second, lack of breathing movements decreases

lung inflow. The third and the most widely accepted model involves a failure to retain amniotic fluid or increased outflow with impaired lung growth and development [46]

Although newer interventions such as inhaled nitric oxide and high frequency ventilation have improved survival in this condition, pulmonary hypoplasia presents a threat to the survival of the fetus.[45]

The prognosis of third trimester oligohydramnios is not fearful as second trimester oligohydramnios. Most cases of severe oligohydramnios in the third trimester occurred at or near term, and they are not associated with Potter-like effects ,but they are often associated with fetal growth restriction. It was shown that an inverse relationship between amniotic volume in the third trimester and the incidence of adverse pregnancy outcome. Adverse outcomes are related to umblicalcord compression , utero placental dysfunction ,and meconium aspiration. Women with oligohydramnios have a significantly increased risk for caesarian delivery for fetal distress and for a 5 minute APGAR score of less than 7 due to associated cord compression and uteroplacental dysfunction [47]

There is a 50% increase variable decelarations during labour and a severalfold increase in caserean delivery rate in woman with oligohydramnios[48]

Variable Deceleration

They are decelerations which are variable in their relation to uterine contraction; they may (or) may not come with every contraction. They may come variably during any phase of contraction – 'M' shape of trace is usually found in CTG, in variable deceleration [49]

Intensity of uterine contraction is unrelated to the decelerations. Decelerations usually vary in degree of fall of FHR and also in their duration.

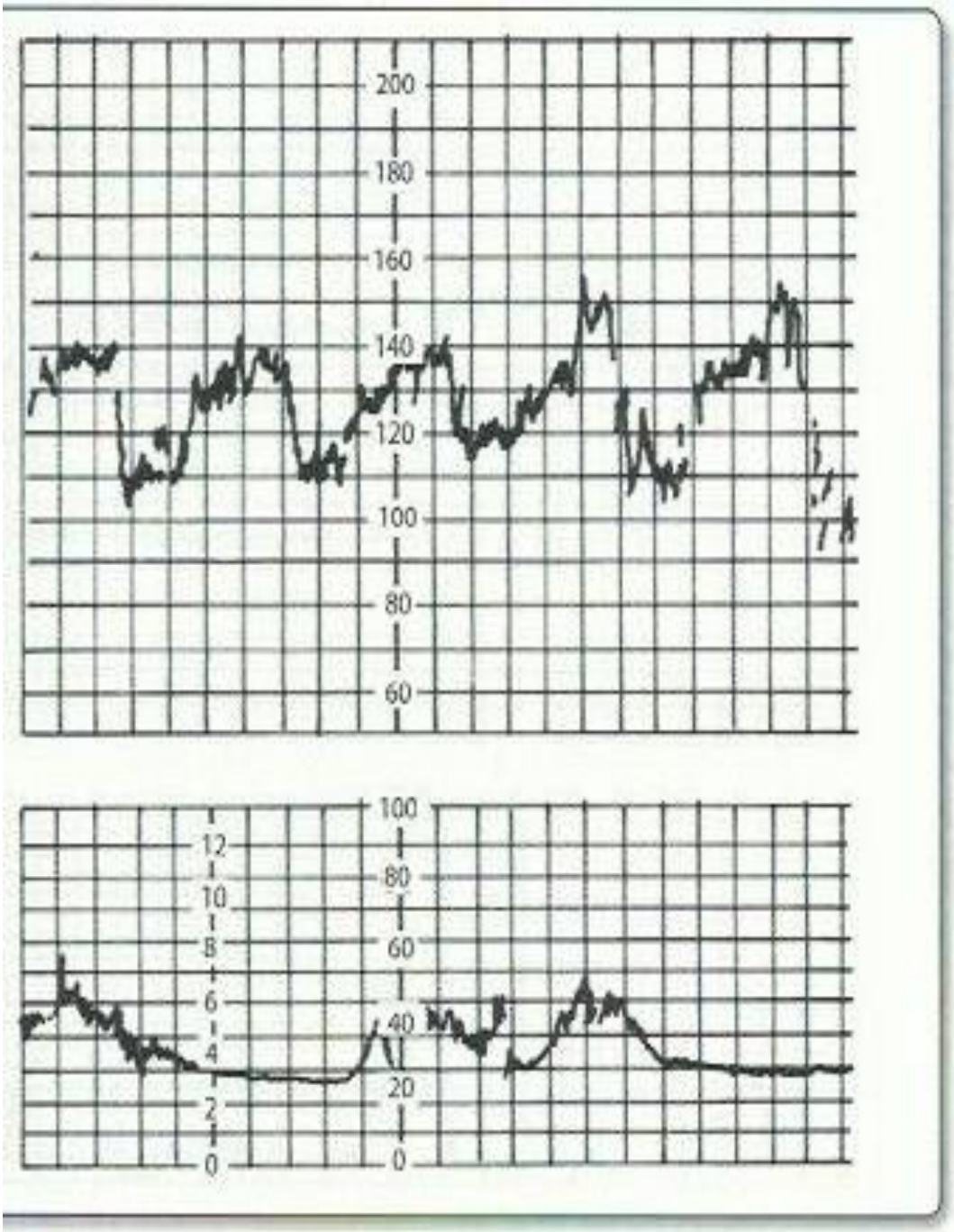
Variable decelerations arising from a disturbance in blood flow through the umbilical cord are due to its varying degrees of cord compression.

Events in the earlier or milder stages of cord compression are due to obstruction in venous blood flow, which leads to decreased venous return to fetal heart. There will be a compensatory tachycardia in order to maintain the blood pressure. The first peak of the 'M' shape tracing co-insides with this spurt of tachycardia.

Events in more severe or late stage of cord compressions are due to arterial blood flow obstruction which leads to increased peripheral resistance and rise of blood pressure. Activation of vagal centre in the brain by activation of aortic stretch receptors causes tachycardia. The middle dipped portion of ‘M’ is due to this crust. It is how the fetus attempts to restore its blood pressure to normal level.

2nd peak of ‘M’ is caused by reactive tachycardia once the occlusion of cord was over. Oligo hydramnios causes higher chances of cord compression and it is the basis of giving amino infusion in these cases.

In



3.21 Variable deceleration

Doppler examination, cord – compression is found to cause the end diastolic blood flow through the umbilical artery to be absent or even reversed.

Absent (or) reversed end diastolic flow develops when 60-70% of placental villous vasculature is abnormal.

Fetuses with absent end – diastolic flow should be delivered as a general guide like, if at or near should be intensively monitored by daily CTG and biophysical profiles.

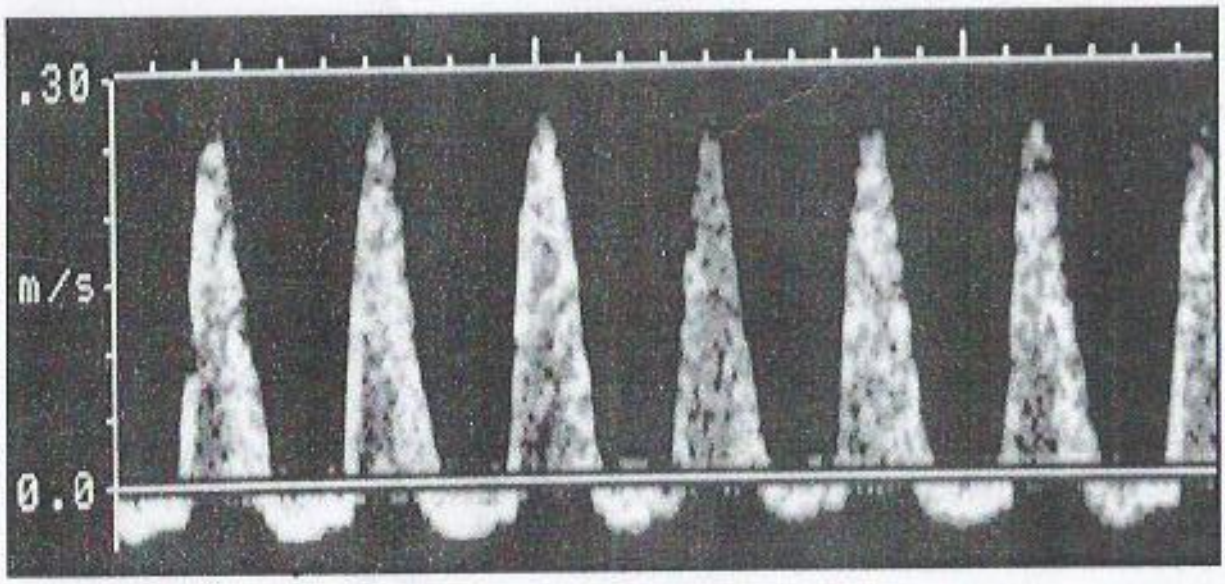


FIGURE 41-22. Doppler velocimetry demonstrating reversed diastolic flow. (Courtesy of Dr. Diane Twickler.)

Oligohydramnios was associated with increased risk of meconium aspiration syndrome, stillbirth and neonatal death.[3]

It is widely believed that any insult causing hypoxia in fetus will cause fetal hyperperistalsis and relaxation of the fetal anal spincter [50]

Hon suggested parasympathetic stimulation from cord compression may stimulate meconium passage without concomitant hypoxia. Whatever be the stimulus ,once meconium has been passed , either fetal gasping or respiratory attempts by the fetus can cause aspiration of amnioticfluid containing meconium into the fetal tracheo bronchial tree.Meconium aspiration thus occurring can obstruct the airway;interfere with gas exchange and cause severe respiratory distress.

The incidence of small for gestational age babies is significantly higher in the oligohydramnios group.

Management of oligohydramnios

The management of oligohydramnios depends on the gestational age and on the associated pregnancy complication.

Maternal hydration oral (or) intravenous has been shown to increase amniotic fluid Volume. Maternal hydration is commonly used, in an attempt to increase amniotic fluid volume in patients without fetal urinary tract abnormalities. Intravenous hydration with two liters of isotonic ringer lactate was effective in increasing aminotic fluid volume [51]Amnio infusion a technique by which normal saline is instilled into the amniotic cavity by the trans abdominal or trans cervical route.

Ante partum amino infusion showed a benefit in cases a preterm premature rupture of membranes. The neonatal survival, pulmonary hypoplasia and PPROM delivery interval were significantly better with serial amino infusion. Risk of abnormal neurological outcome in survivors was significantly less in pregnancies where amino infusion was successful [52]

Intrapartum amnio infusion for meconium stained liquor showed that amino infusion was associated with a significant reduction in heavy meconium staining of liquor, variable fetal heart decelarations and reduced caesarean section rate [53]

Fetal surgical approaches to the management of oligohydramnios are for bladderdrainage and tracheal occlusion.In cases of fetal urinary outflow

obstruction and associated oligohydramnios, a vesico amniotic shunt may be inserted although there is significant risk of PPROM. There is no confirmed value in preserving renal function despite the use of fetal urinary electrolytes to select patients with functioning kidneys for treatment.

Fetoscopic tracheal occlusion has been employed to alleviate or prevent pulmonary hypoplasia associated second trimester oligohydramnios.

Ross and colleagues [54]1996 found that DDAVP –l-deamino-8-D arginine vasopressin increases the amniotic fluid index in cases of oligohydramnios.The increase in AFI is attributed to the maternal serum hypo osmolality caused by DDAVP.

Oligohydramnios

Management Options	Evidence Quality Recommendation
Identify Cause (If Possible)	
Detailed history to include drug exposure, maternal medical illnesses, prior prenatal screening for aneuploidy, prior ultrasound findings, symptoms of membrane rupture.	—/GPP
Rupture of the membranes is ruled out by physical examination.	—/GPP
Ultrasonography to assess:	III/B
• Degree of oligohydramnios (AFI). • Presence of growth deficiency. • Presence of fetal anomalies. • Presence and appearance of the kidneys. • Presence of fetal renal arteries. • Fetal and uterine blood flow studies. • Fetal well-being.	
Amnioinfusion may have a role in diagnosing or confirming suspected PPROM or fetal anomaly.	III/B
Patient Counseling	
Prognosis depends on gestational age, severity of oligohydramnios and diagnosis.	III/B
• Severe oligohydramnios in the second trimester is often associated with fetal death and congenital abnormalities. The patient may wish to consider pregnancy termination.	+/GPP
• Third-trimester oligohydramnios is more often associated with PPROM and IUGR. Delivery may be indicated.	—/GPP
Management (Depends on Etiology)	
PPROM	
Growth deficiency	
Prolonged pregnancy	
Fetal renal anomalies	
Treatments to increase AF volume:	
• Maternal hydration with hypotonic solution	Ib/A
• Serial transabdominal therapeutic amnioinfusions with PPROM remote from term, selected patients (not in labor, retain fluid after infusion).	Ib/A
• Vesicoamniotic shunting in obstructive uropathies may be of value in preventing pulmonary sequelae	III/B
• Sealing of membrane leak: investigational	III/B
• DDAVP: investigational.	III/B

AF, amniotic fluid; AFI, amniotic fluid index; DDAVP, 1-deamino-[8-d-arginine] vasopressin; GPP, good practice point; IUGR, intrauterine growth restriction; PPROM, prenatal premature rupture of membranes.

HYDRATION THERAPY GENERAL CONSIDERATIONS

Intravenous hydration (or) oral hydration significantly increases the amniotic fluid volume [40] in women with oligohydramnios. This increase in amniotic fluid volume whether results from an improved utero placental circulation and increased fetal urination or as a direct effect of decreased oncotic pressure in the maternal circulation is uncertain.

Changes in maternal intravascular volume alter the fetal urine output, amniotic fluid volume and intra vascular volume to some extent.[55]

Maternal hydration acts by 2 mechanisms,

Decrease in maternal plasma osmolality results in fetal hypoosmolality and increased fetal urine output.

Intra membranous amniotic fluid resorption is reduced in response to fetal plasma hypoosmolality.

Magann et al reported that intravenous hydration therpy with isotonic solution causes elevated levels of amniotic fluid index.[56]

Hydration of mothers in term pregnancy and oligohydramnios caused the amniotic fluidvolume to increase with mean change of AFI 4.5cm.[57]

Improvements of utero placental perfusion due to plasma volume increase in mothers would increase renal blood flow and fetal oxygenation is improved. The decrease in vasopressin and increase in urinary output would be observed.[58]. Fetus is compensating by reducing plasma osmolality and increasing urinary output which inturn increases the amniotic fluid volume for the acute changes in maternal plasma osmolality.

Doi et al showed maternal hydration modified their osmolality and thus increased amniotic fluid volume.[59]

Kilpatrick and saford [33] showed after maternal hydration there is a significant increase in the mean arterial flow velocity. Hydration may increase amniotic fluid index by improving blood flow of the placenta.

Intravenous hydration therapy with 6500ml of an isotonic solution increased amnioticfluid volume in dehydrated women. (sherer 1990).[45]

MATERIALS AND METHODS

A prospective study on the effect of intravenous hydration therapy in cases of third trimester oligohydramnios and its effects on perinatal outcome was carried out in,

Government Stanley Medical college, Chennai – 1, during the period of January 2015 to August 2015.

Inclusion Criteria:

1. The pregnant women Whose amniotic fluid index <5cm
2. Third trimester
3. Singleton pregnancy
4. Intact membranes

Exclusion Criteria;

1. Multiple gestations
2. Rupture of membranes
3. Any evidence of fetal structural abnormalities in ultrasound studies
4. Multiple gestations

Maternal complications like,

a) Diabetes mellitus
b) Cardiovascular disease
c) Hyper thyroidism
d) Chronic obstructive pulmonary disease
e) Renal disease
f) First & Second trimester oligohydramnios
g) IUD

Sample Size

About 150 cases in third trimester with amniotic fluid index- AFI <5cm (Study group)

And 150 cases in Control Group (AFI >5cm)

Period of study: 1year

Design of syudy: Case-control study

History regarding age, parity, gestational age, menstrual history, obstetric history and co-morbidities in present pregnancy, H/o premature rupture of membranes was taken.

The patient was informed about my study and consent was taken.

150 patients in third trimester pregnancy with oligohydramnios in one group and 150 normal pregnancy patients in third trimester were taken as control group.

General clinical examination was done. Pulse rate, Blood pressure, temperature, Height of patient, Weight of the patient were noted. Symphysio fundal height and adequacy of amniotic fluid were clinically noted. Fetal heart rate was counted. Speculum, examination was done to rule out draining plv and confirmed the presence of membranes.

All routine investigations done

A non-stress test was done.

Amniotic fluid index was measured by the technique described by phelan et al[2].

Ultrasound examination was carried out after instructing the patient to empty her bladder. Patient was asked to lie down in supine position.

A curvilinear transducer is used

By marking the uterus is divided into four quadrants using the maternal sagital midline vertically and an arbitrary transverse line approximately half way between the symphysis pubis and upper edge of uterine fundus.

The transducer was kept parallel to the maternal sagittal plane and perpendicular to the maternal coronal plane throughout.

The deepest, clear pocket of amniotic fluid excluding the cord loops and small fetal parts visualized and image was frozen. The ultrasound calipers are manipulated in such a way to measure the pocket in a strictly vertical direction. The process is repeated on each of four quadrants and measurements are summed up to calculate the AFI. Caution was exercised to avoid excessive pressure on the transducer as if can alter AFI measurements.

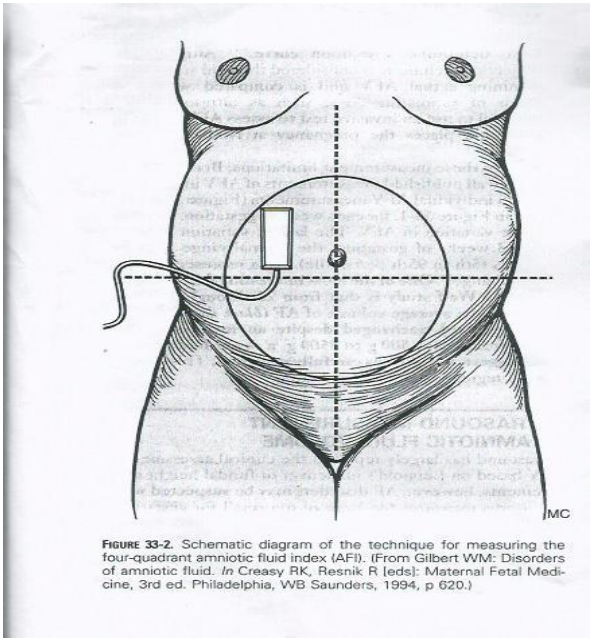


FIGURE 33-2. Schematic diagram of the technique for measuring the four-quadrant amniotic fluid index (AFI). (From Gilbert WM: Disorders of amniotic fluid. *In* Creasy RK, Resnik R [eds]: Maternal Fetal Medicine, 3rd ed. Philadelphia, WB Saunders, 1994, p 620.)

Patients are grouped according to their AFI 5cm or less as cases and control group as their AFI > 5cm.

Starting from 34 weeks till 40 weeks the mean (standard deviation) of AFI values (in cms) were,

34 weeks : 14.59 (1.79)

35 weeks : 14.25 (1.57)

36 weeks : 13.17 (1.56)

37 weeks : 12.48 (1.52)

38 weeks : 12.2 (1.7)

39 weeks : 11.37 (1.71)

The 5th percentile cut off was 8.7CM AT 40 weeks. [60]

Oligo hydramnios group of patients are admitted. Admission test (CTG) was done. On admission USG done to rule out renal tract and other abnormalities. Doppler study of umbilical artery performed and the S/D ratio recorded. An increased S/D ratio in oligohydramnios cases identified the fetuses at risk of perinatal outcome. Continous intra partum monitoring was offered to these cases to detect early signs of hypoxia and performed timely intervention.

Maternal hydration in the form of isotonic Ringer lactate solution 1000 ml/day was given intravenously every day to the oligo hydramnios group of patients.

The mothers vital parameters were monitored to identify early signs and symptoms of over load. Non – stress test was performed everyday for fetal well being. AFI was repeated on every 3rd day. Doppler study of umbilical artery and middle cerebral artery done every week. If any abnormality in Doppler study was diagnosed, pregnancy is terminated.

Maternal hydration is continued till 37 completed weeks or tests indicate serious fetal compromise or the Fetus has reasonable chances of survival.

Labour induction is done according to the Bishop score of individual patients. Cases are followed up with observing mode of delivery, indication for LSCS, colour of the liquor, APGAR, reason for NICU admission, Birth weight, and condition of the baby on discharge.

The following study outcomes are noted: Effect of intravenous hydration by means of increase in amniotic fluid index(AFI) ,and the perinatal outcome between

intravenously hydrated oligohydramnios cases, and normal pregnant cases are noted.

Data analysis was done with use of SPSS, version 18.

The results were expressed in terms mean ± Standard deviation; analysis of the result was done using paired t test, chi-square test.

ANALYSIS OF STUDY OBSERVATIONS

TABLE – 1

AFI after Hydration Therapy

	Mean	N	Std Deviation	P-Value
Pre Treatment AFI	4.965	150	2326	< 0.001
Post Treatment AFI	6.844	150	3500	

In this study there is a significant increase in AFI after intravenous hydration therapy in third trimester oligohydramnios cases.

In our study the mean duration of hydration therapy is 1.2 weeks.

Mean increase in amniotic fluid index in hydrated group is from 4.965 to 6.844. Mean difference is 3.0cm 95% CI (P=<0.001)

Age in Years

Table – 2

Age Group in years		Group		Total	P value
		Control	Cases		
<= 20	Count	22	12	34	
	% within Age Group in years	64.7%	35.3%	100.0%	
	% within Group	14.7%	8.0%	11.3%	
21-30	Count	125	131	256	
	% within Age Group in years	48.8%	51.2%	100.0%	
	% within Group	83.3%	87.3%	85.3%	
> 30	Count	3	7	10	
	% within	30.0%	70.0%	100.0%	0.096

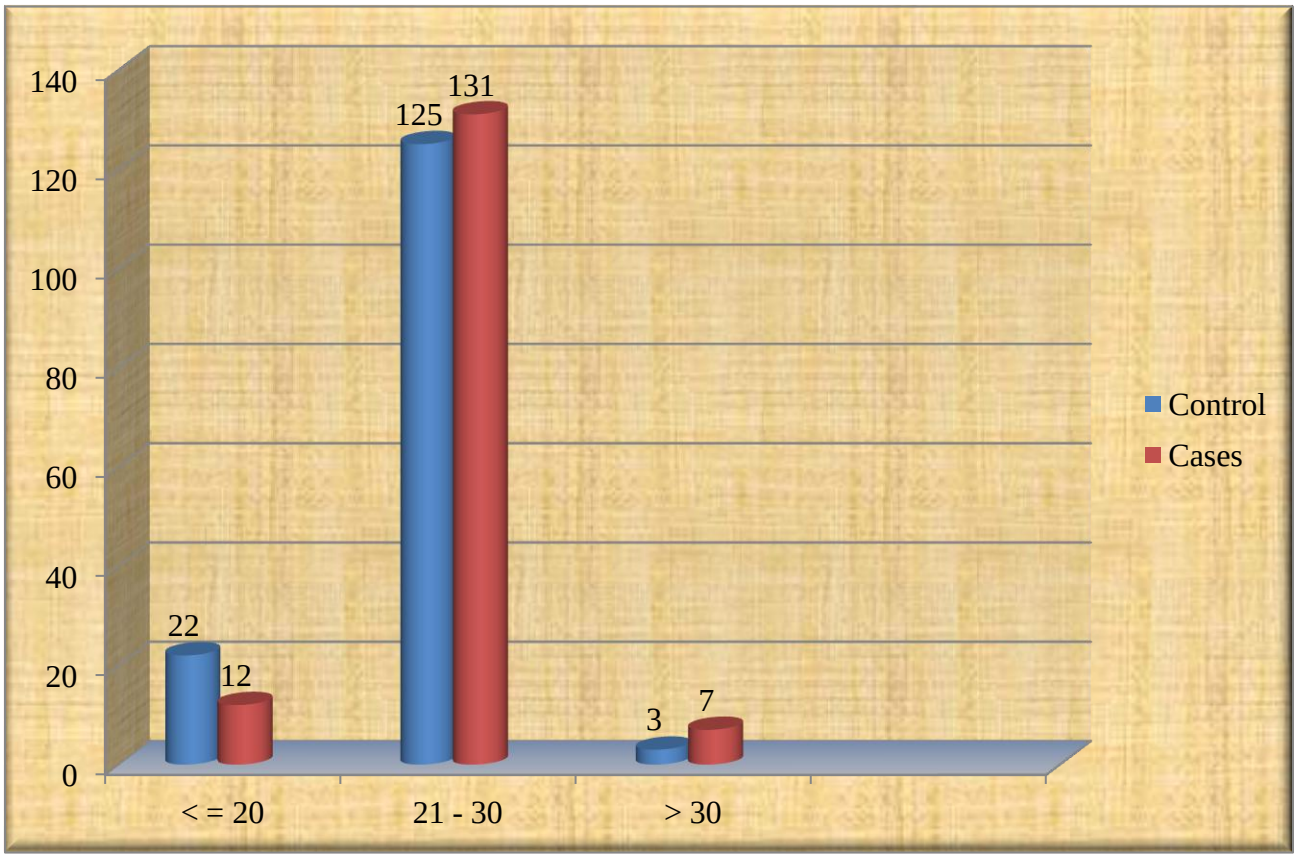
	Age Group in years				
	% within Group	2.0%	4.7%	3.3%	
Total	Count	150	150	300	
	% within Age Group in years	50.0%	50.0%	100.0%	
	% within Group	100.0%	100.0 %	100.0%	

P=0.96

In this study majority of cases are in the age group of 21 – 30 yrs.

Age in Years

Table – 2



Distribution of Cases with Parity

Table - 3

			Group		Total
			Control	Cases	
Parity	Primi	Count	84	81	165
		% within Parity	50.9%	49.1%	100.0%
		% within Group	56.0%	54.0%	55.0%
	Multi	Count	66	69	135
		% within Parity	48.9%	51.1%	100.0%
		% within Group	44.0%	46.0%	45.0%
Total		Count	150	150	300
		% within Parity	50.0%	50.0%	100.0%
		% within Group	100.0%	100.0%	100.0%

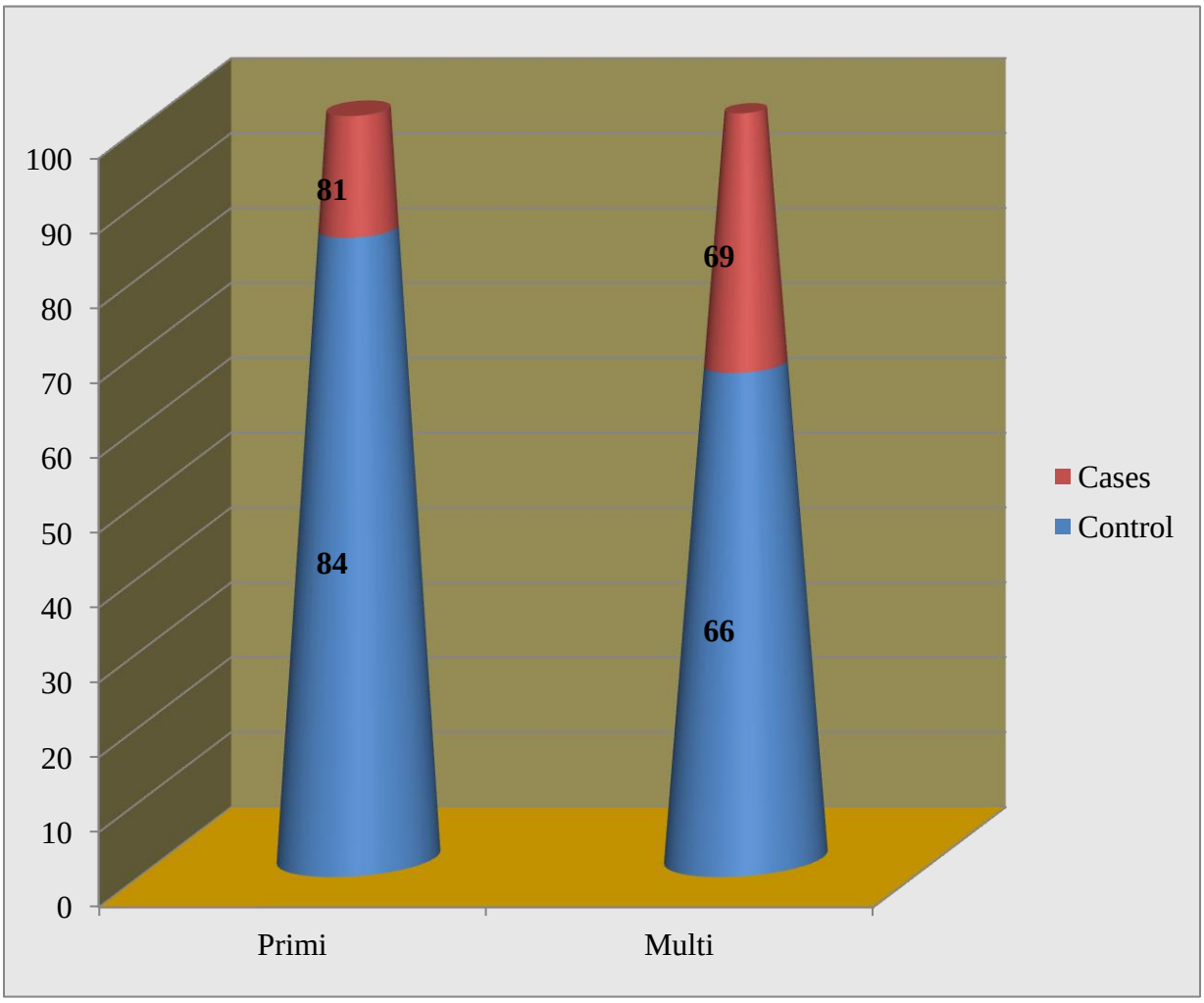
P=0.184

In hydration therapy received group 81Cases were primi gravida, 69 Cases were multi gravida.

In Control Group 84 Cases were primi gravida and 66 cases were multi

Distribution of Cases with Parity

Table - 3



Non – Stress test

Table - 4

NST		Group		Total
		Control	Cases	
Reactive	Count	142	136	278
	% within NST	51.1%	48.9%	100.0%
	% within Group	94.7%	90.7%	92.7%
Non Reactive	Count	8	14	22
	% within NST	36.4%	63.6%	100.0%
	% within Group	5.3%	9.3%	7.3%
Total	Count	150	150	300
	% within NST	50.0%	50.0%	100.0%
	% within Group	100.0%	100.0%	100.0%

P=0.163

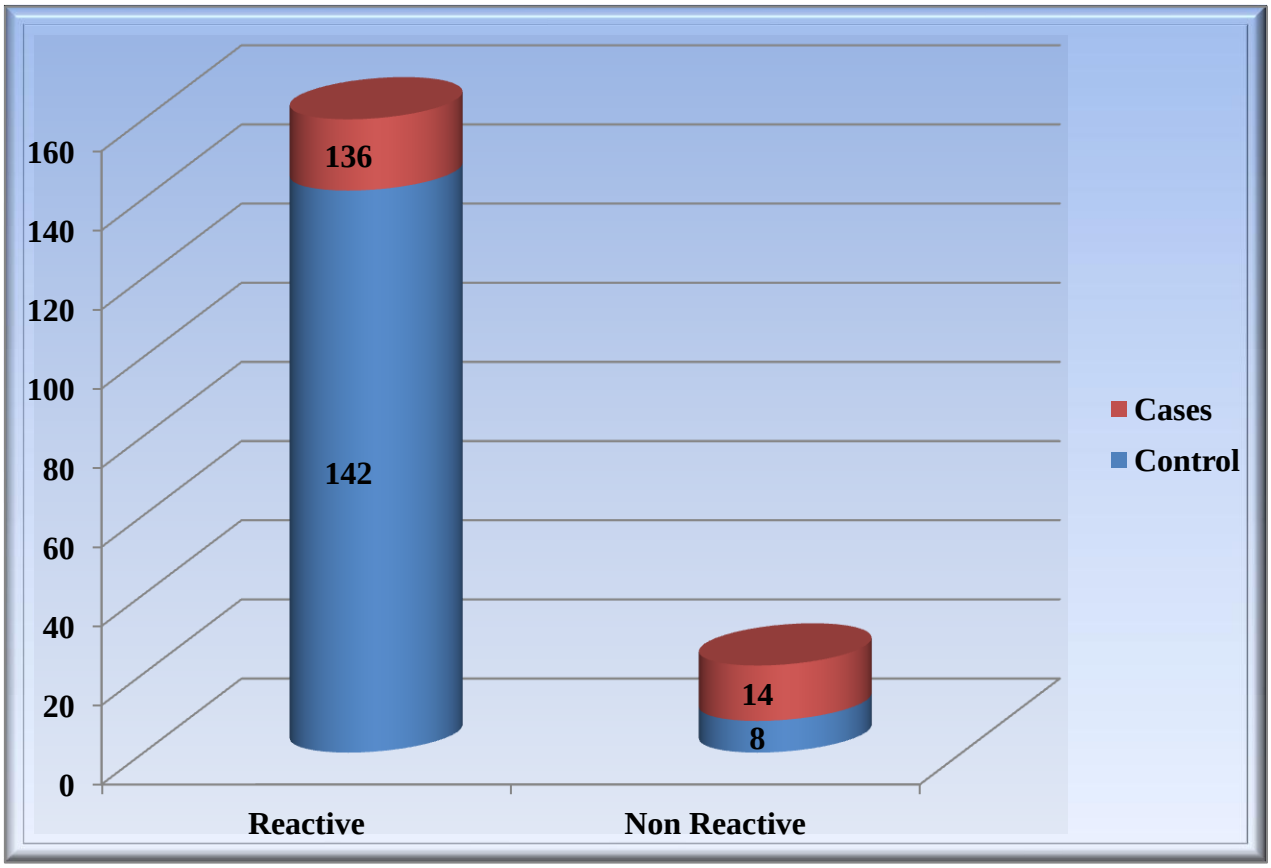
In this hydration therapsy received group had 9.3% non –Reactive CTG..

In Control Group 5.3% had Non – reactive CTG

The difference is non – significant.

Non – Stress test

Table - 4



Mode of Delivery
Table – 5

Mode of Delivery		Group		Total
		Control	Cases	
Normal	Count	73	62	135
	% within Mode of Delivery	54.1%	45.9%	100.0%
	% within Group	48.7%	41.3%	45.0%
Outlet	Count	25	22	47
	% within Mode of Delivery	53.2%	46.8%	100.0%
	% within Group	16.7%	14.7%	15.7%
LSCS	Count	52	66	118
	% within Mode of Delivery	44.1%	55.9%	100.0%
	% within Group	34.7%	44.0%	39.3%
Total	Count	150	150	300
	% within Mode of Delivery	50.0%	50.0%	100.0%
	% within Group	100.0%	100.0%	100.0%

P=0.489

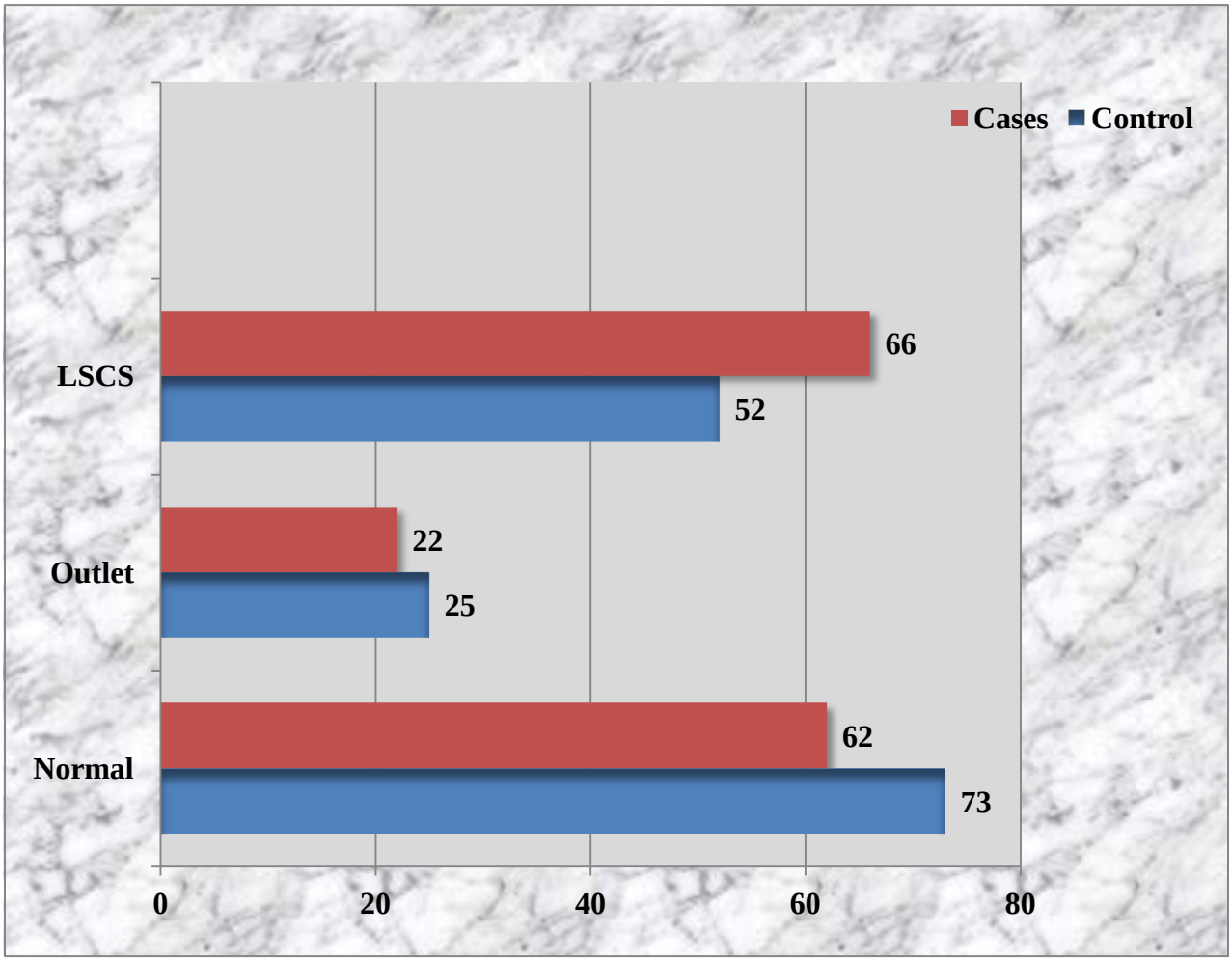
In Hydration therapy group 62 cases had normal delivery, 22 cases had outlet forceps delivery 66 cases had Caesarean Section.

In control group, 73 cases had normal delivery, 25 cases had outlet forceps, delivery, 52 cases had Caesarean Section delivery.

The difference was found to be non – significant.

Mode of Delivery

Table – 5



LSCS Indication

Table – 6

LSCS Indication		Group		Total
		Control	Cases	
FD	Count	13	23	36
	% within LSCS Indication	36.1%	63.9%	100.0%
	% within Group	25.0%	34.8%	30.5%
FI	Count	15	18	33
	% within LSCS Indication	45.5%	54.5%	100.0%
	% within Group	28.8%	27.3%	28.0%
Others	Count	24	25	49
	% within LSCS Indication	49.0%	51.0%	100.0%
	% within Group	46.2%	37.9%	41.5%
Total	Count	52	66	118
	% within LSCS Indication	44.1%	55.9%	100.0%
	% within Group	100.0%	100.0%	100.0%

P=0.489

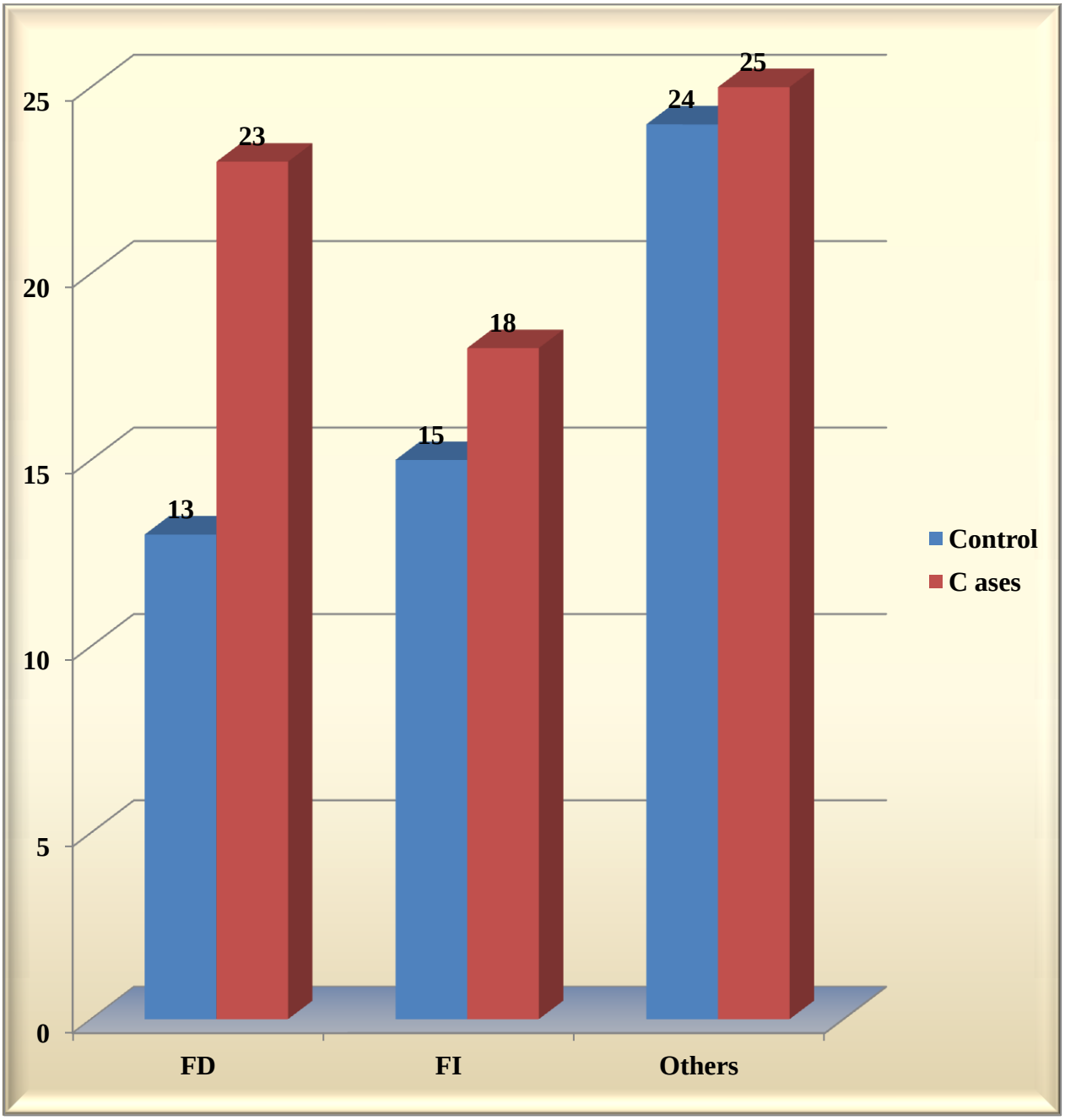
In hydration therapy received group, 23 cases underwent Cesarean Section for fetal distress,

In control group 13 cases underwent Caesarean Section for fetal distress.

The difference was in significant.

LSCS Indication

Table – 6



Liquor

Table – 7

	Group			Total
	Control		Cases	
Clear	Count	134	127	261
	% within Liquor	51.3%	48.7%	100.0%
	% within Group	89.3%	84.7%	87.0%
Moderate MSAF	Count	0	3	3
	% within Liquor	.0%	100.0%	100.0%
	% within Group	.0%	2.0%	1.0%
Thin MSAF	Count	16	20	36
	% within Liquor	44.4%	55.6%	100.0%
	% within Group	10.7%	13.3%	12.0%
Total	Count	150	150	300
	% within Liquor	50.0%	50.0%	100.0%
	% within Group	100.0%	100.0%	100.0%

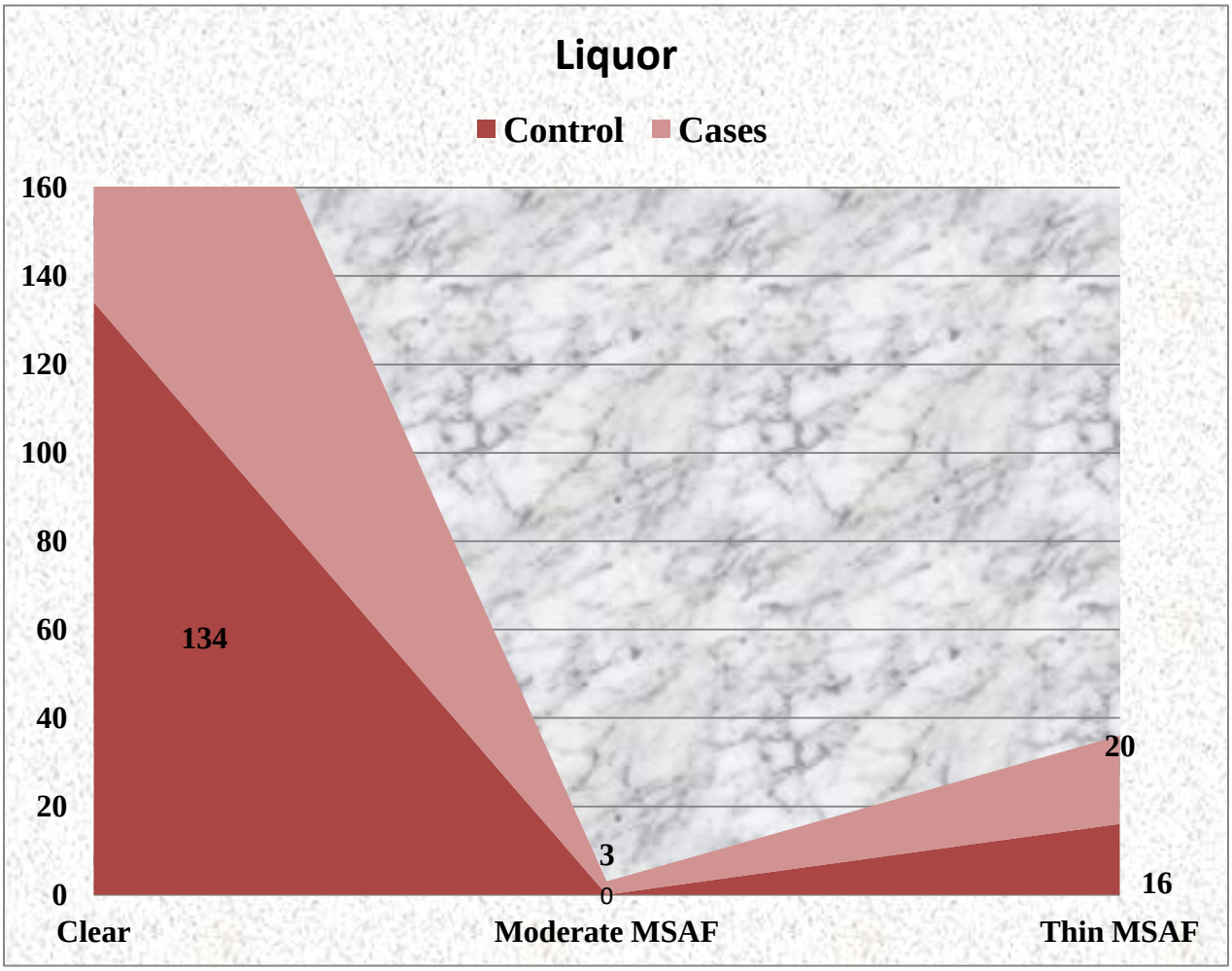
In Hydration therapy received group 127 cases had clear liquor ,20 cases had thin Meconium Stained amniotic Fluid,3 cases had Moderate Meconium Stained amniotic Fluid.

In control group 134 cases had clear liquor, 16 cases had thin Meconium Stained amniotic Fluid and No cases had Moderate Meconium Stained amniotic Fluid.

The difference was found to be non – significance. (P=0.163)

Liquor

Table – 7



1 min APGAR

Table – 8

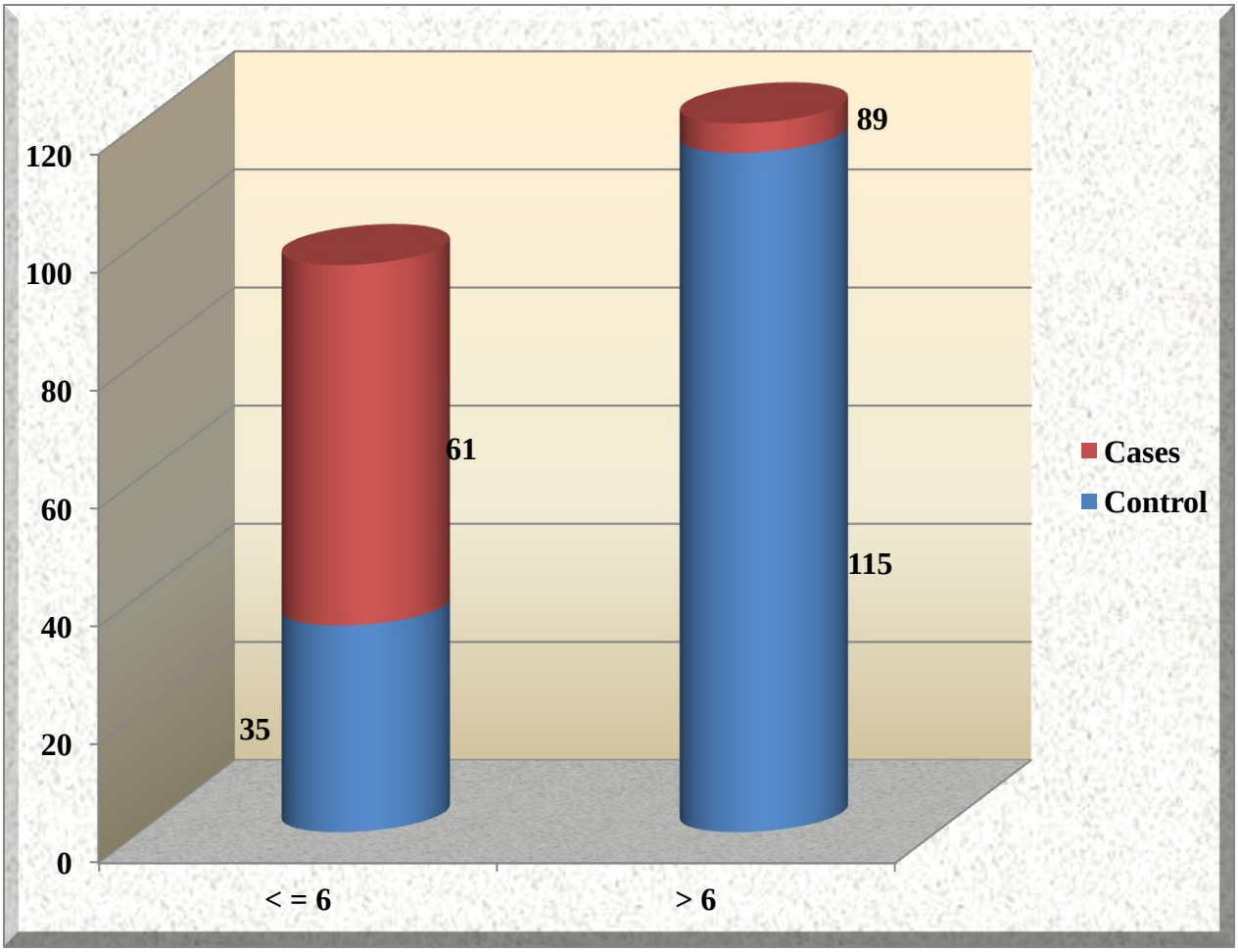
1 min APGAR	Group			Total
	Control		Cases	
<= 6	Count	35	61	96
	% within 1 min APGAR	36.5 %	63.5%	100.0%
	% within Group	23.3 %	40.7%	32.0%
> 6	Count	115	89	204
	% within 1 min APGAR	56.4 %	43.6%	100.0%
	% within Group	76.7 %	59.3%	68.0%
Total	Count	150	150	300
	% within 1 min APGAR	50.0 %	50.0%	100.0%
	% within Group	100.0 %	100.0%	100.0%

In hydration therapy received group 48 cases had 5min APGAR < 7

In Control group 26 cases had 5min APGAR > 7

1 min APGAR

Table – 8



5 min APGAR

Table – 9

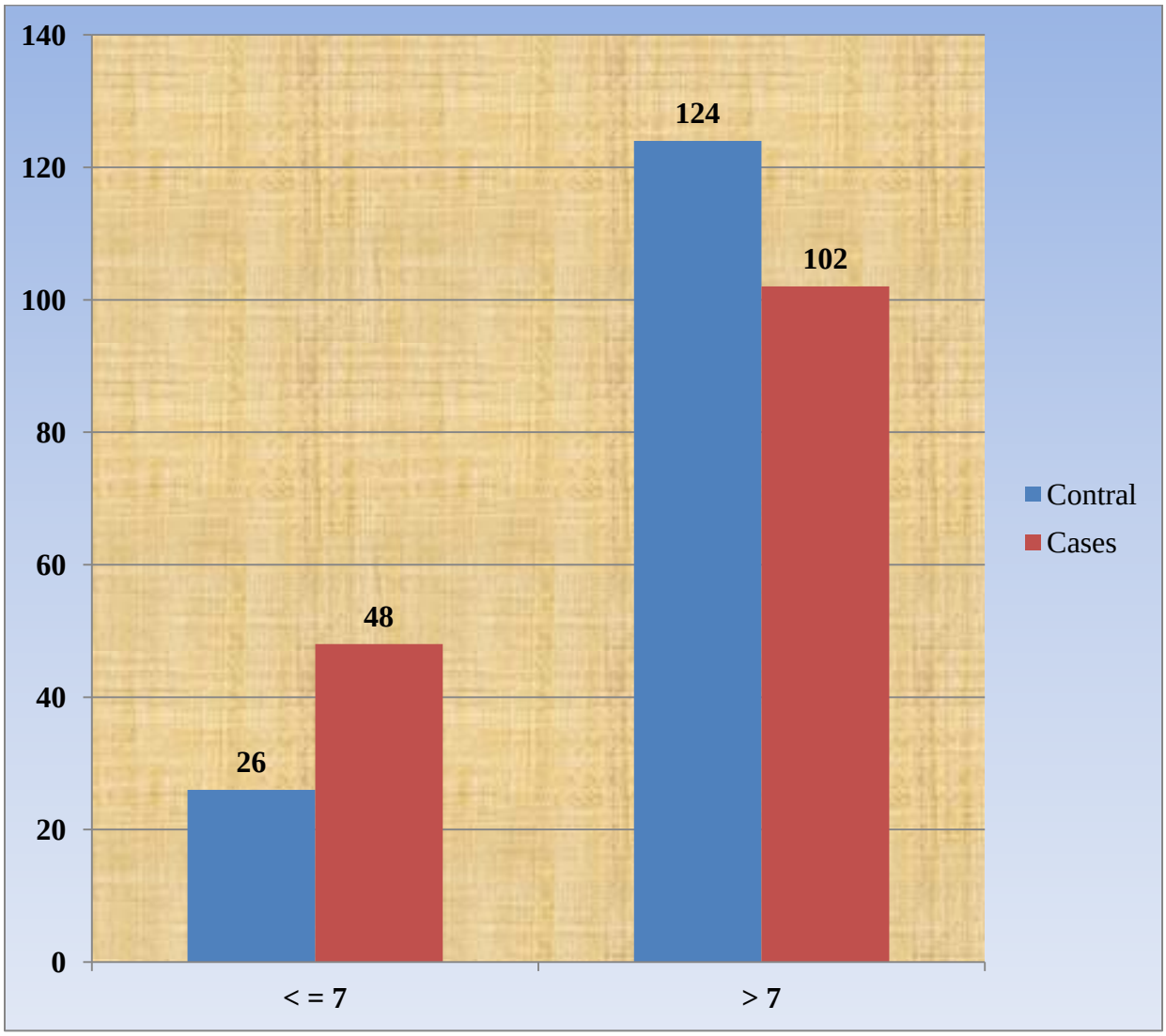
5min APGAR	Group			Total
	Control		Cases	
<= 7	Count	26	48	74
	% within 5 min APGAR	35.1%	64.9%	100.0%
	% within Group	17.3%	32.0%	24.7%
> 7	Count	124	102	226
	% within 5 min APGAR	54.9%	45.1%	100.0%
	% within Group	82.7%	68.0%	75.3%
Total	Count	150	150	300
	% within 5 min APGAR	50.0%	50.0%	100.0%
	% within Group	100.0%	100.0%	100.0%

In a hydration therapy received group 48 cases had 5 minutes APGAR < 7

In control group 26 cases had 5 minutes APGAR < 7

5 min APGAR

Table – 9



Birth Weight in kg

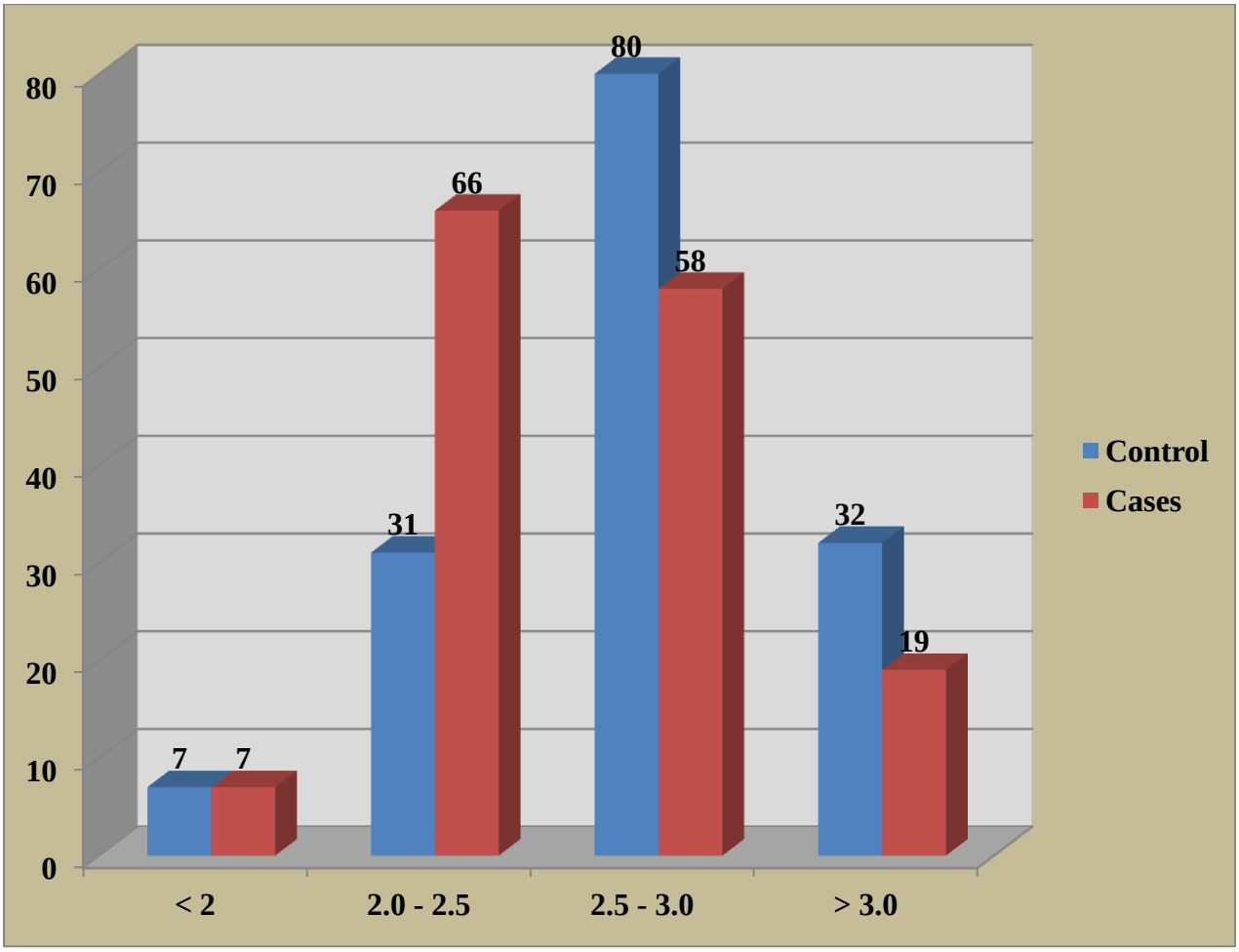
Table – 10

Birth Weight in kg		Group		Total
		Control	Cases	
< 2	Count	7	7	14
	% within Birth Weight in kg	50.0%	50.0%	100.0%
	% within Group	4.7%	4.7%	4.7%
2.0-2.5	Count	31	66	97
	% within Birth Weight in kg	32.0%	68.0%	100.0%
	% within Group	20.7%	44.0%	32.3%
2.5-3.0	Count	80	58	138
	% within Birth Weight in kg	58.0%	42.0%	100.0%
	% within Group	53.3%	38.7%	46.0%
> 3.0	Count	32	19	51
	% within Birth Weight in kg	62.7%	37.3%	100.0%
	% within Group	21.3%	12.7%	17.0%
Total	Count	150	150	300
	% within Birth Weight	50.0%	50.0%	100.0%
	in kg % within Group	100.0%	100.0%	100.0%

In maternal hydration received group 66 babies were birthweight between 2.0 to 2.5 kg,but in control group 31 babies were birth weight between 2.0 to 2.5 kg.But the difference is insignificant.

Birth Weight in kg

Table – 10



Admission in NICU

Table – 11

Admissiomn in NICU		Group		Total
		Control	Cases	
Yes	Count	19	29	48
	% within Admissiomn in NICU	39.6%	60.4%	100.0%
	% within Group	12.7%	19.3%	16.0%
	Count	131	121	252
No	% within Admissiomn in NICU	52.0%	48.0%	100.0%
	% within Group	87.3%	80.7%	84.0%
	Count	150	150	300
Total	% within Admissiomn in NICU	50.0%	50.0%	100.0%
	% within Group	100.0%	100.0%	100.0%

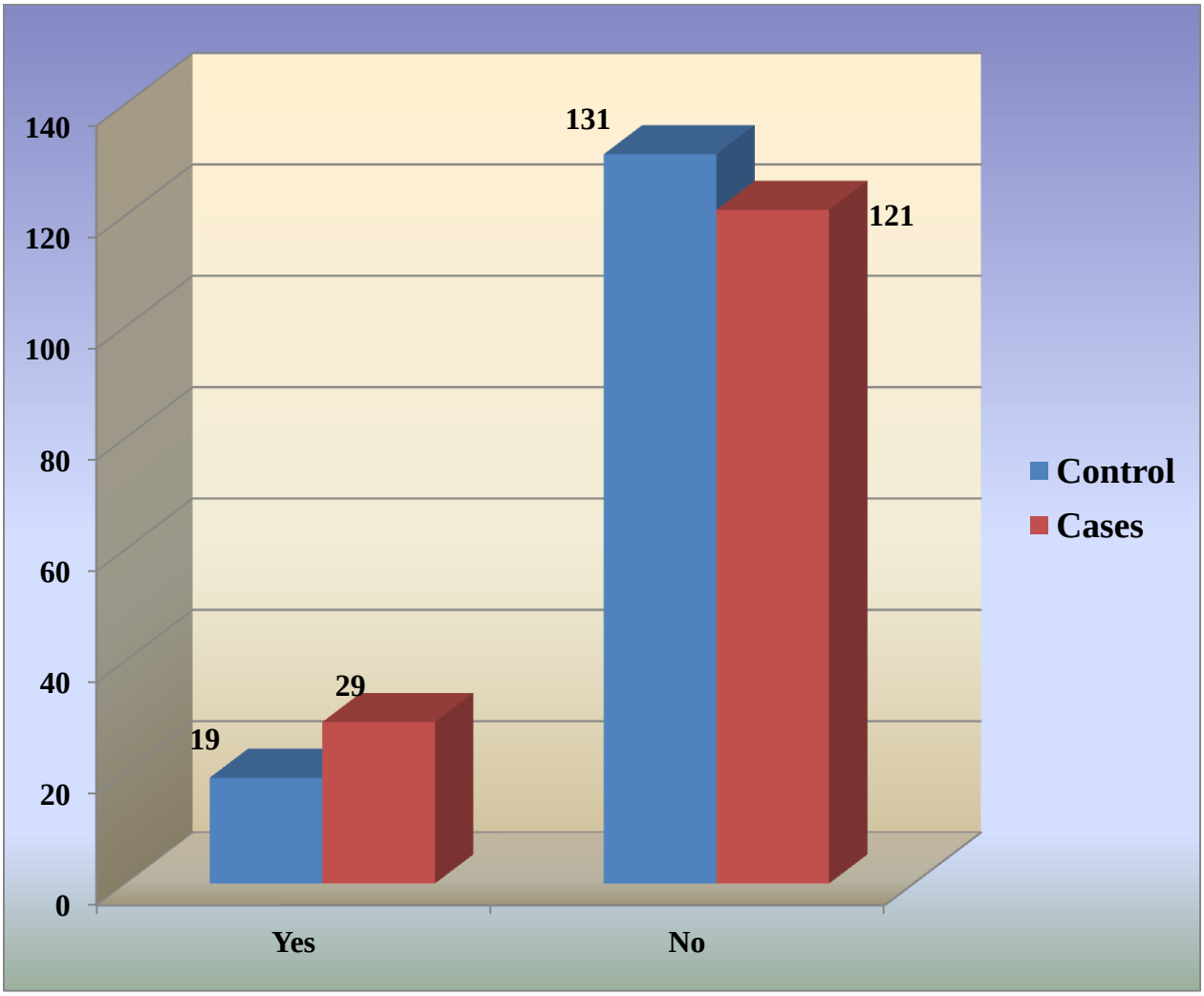
P=0.656

Admission in NICU in Hydration therapy received cases were – 29 – ie 19.3%

In control group only 19 cased ie 12.7% were admitted in NICU but the difference was non –significant.

Admission in NICU

Table – 11



Condition at Discharge

Table – 12

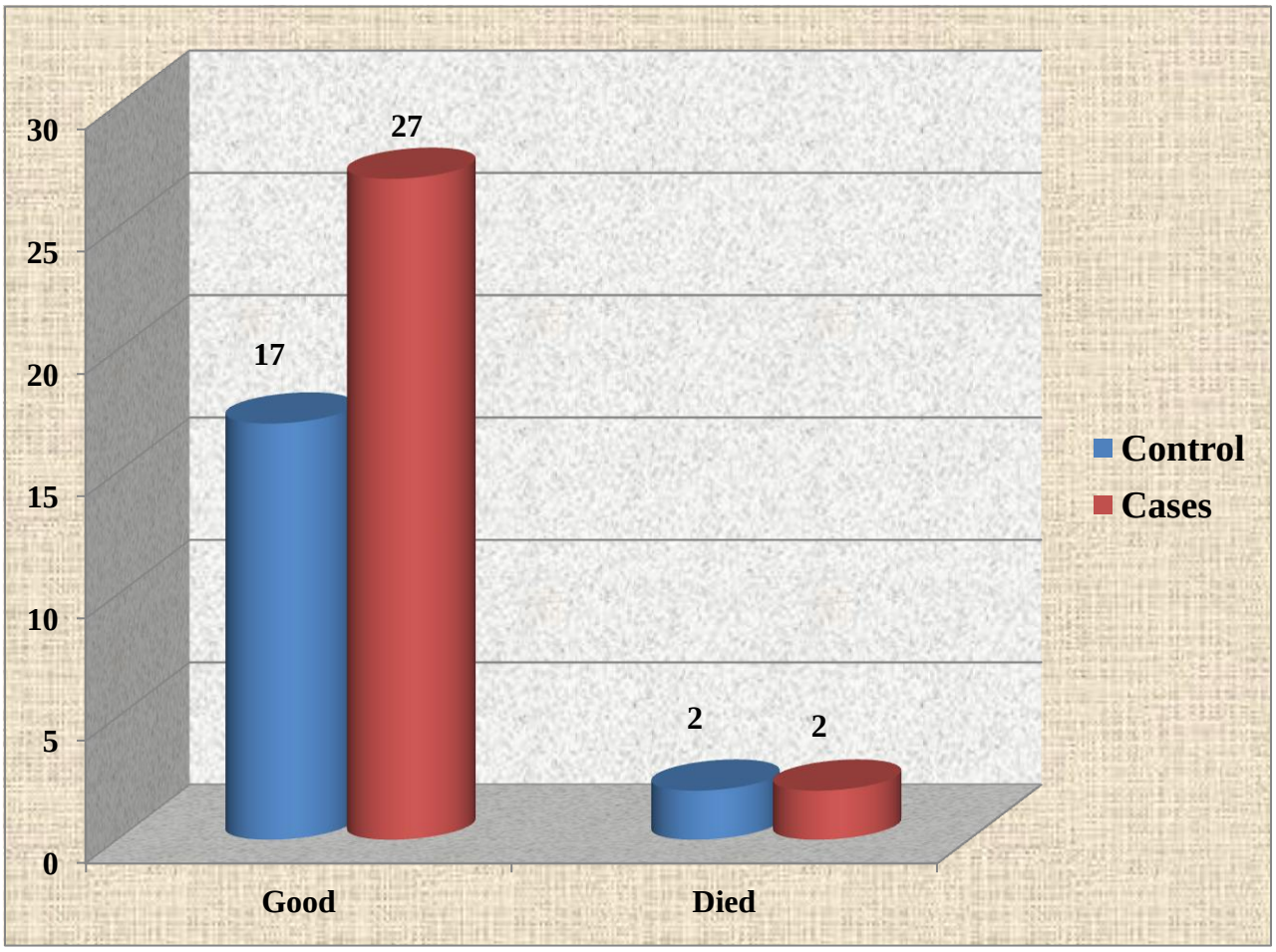
Condition at Discharge		Group		Total
		Control	Cases	
Good	Count	17	27	44
	% within Condition at Discharge	38.6%	61.4%	100.0%
	% within Group	89.5%	93.1%	91.7%
Died	Count	2	2	4
	% within Condition at Discharge	50.0%	50.0%	100.0%
	% within Group	10.5%	6.9%	8.3%
Total	Count	19	29	48
	% within Condition at Discharge	39.6%	60.4%	100.0%
	% within Group	100.0%	100.0%	100.0%

In this study, 2 Neonatal deaths observed in both study group and the control group

The difference is non – significant. (P=0.656)

Condition at Discharge

Table – 12



Maternal Morbidity

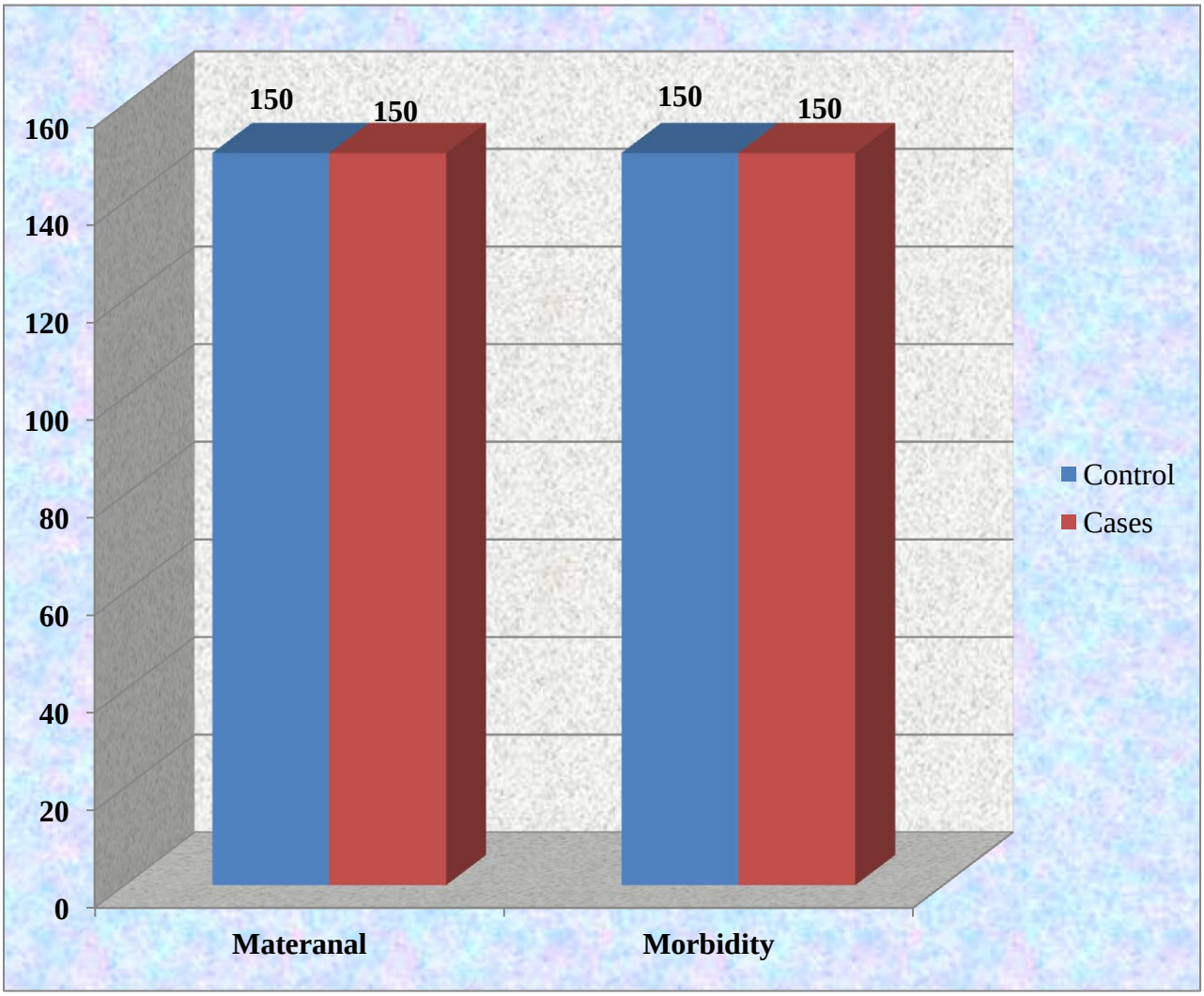
Table – 13

Maternal Morbidity		Group		Total
		Control	Cases	
Maternal	Count	150	150	300
	% within Maternal Morbidity	50.0%	50.0%	100.0%
	% within Group	100.0%	100.0%	100.0%
Morbidity	Count	150	150	300
	% within Maternal Morbidity	50.0%	50.0%	100.0%
	% within Group	100.0%	100.0%	100.0%

There was no Maternal Morbidity in both the groups observed.

Maternal Morbidity

Table – 13



Perinatal Out Come

Table 14

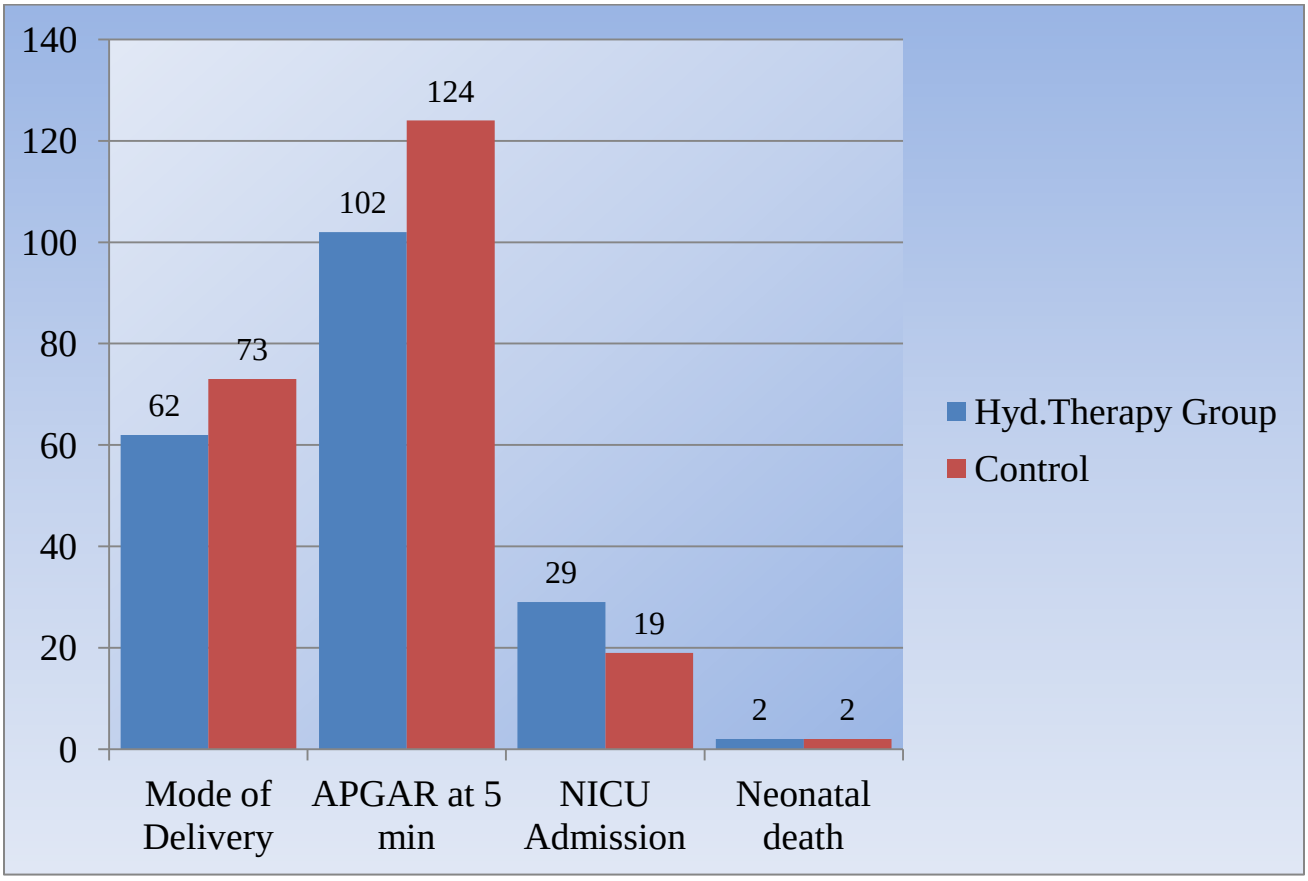
Hydration Therapy Group			Control	
	No	%	No	%
Mode of Delivery				
Vaginal	62	41.3%	73	48.7%
Outlet	22	14.7%	25	16.7%
LSCS	66	44.0%	52	34.7%
APGAR at 5 min				
< 7	48	32.0%	26	17.3%
< 7	102	68%	124	82.7%
NICU Admission	29	19.3%	17	12.7%
Neonatal death	2	6.9%	2	10.5%

In this study there is no statistical difference between mode of delivery, APGAR at 5 minutes, number of NICU admission and number of neonatal deaths in between the groups.

In this present study there is no statistical difference in perinatal outcome between the hydration therapy received study group with AFI <5cm and the control group who's AFI were within normal limits.

Perinatal Out Come

Table 14



DISCUSSION

Third trimester oligohydramnios with AFI less than or equal to 5cm is associated with increased perinatal morbidity and mortality.

Hydration status and maternal plasma osmolality can alter amniotic fluid volume[61].

Megann et al reported elevated levels of amniotic fluid index after intravenous hydration using isotonic solution.

In this study 150 patients with oligohydramnios in third trimester with

AFI – 5 cm received intravenous hydration with 1000ml of isotonic solution per day with mean duration of hydration therapy 1.2 weeks and were compared with 150patients with normal AFI.

Both the group were matched with respect of age, parity and gestational age.

In a study by patreli T. S et al. AFI <5cm isolated oligohydramnios group in third trimester received 6 days of intravenous hydration therapy and AFI measured.

At recruitment it was 39.68 ± 11.11mm.

In a hydrated group AFI was ± 112.45 ($P < 0.001$).

There was no statistical difference in the caesarean delivery rates between groups.

From the analysis of indication for Caesearean Section, in study group 25% of them were performed due to non-reactive NST.

There is significant increase in AFI in study group in this study.

In our present study also there is no statistically significant difference in the caesarean delivery rates between groups. This is correlating with the study by patrili et el [62]

Effect of Maternal Hydration

	Pre treatment AFI – (cm)	Post treatment AFI (cm)	P – Value
Present study	4.965 ± 2.32	6.844 ± 3.35	< 0.001
Tito silvio patreli and co workers (2012)	3.968 ± 1.11	7.77 ± 1.503	< 0.001

In present study there is significant increase in AFI, after maternal hydration (P - < 0.001)

Effect of maternal Hydration

20-30% improvement in AFI and a reduction in cesarean delivery is evidenced after either oral or intravenous maternal hydration in singleton pregnancies with isolated oligohydramnios[63].

This study by Brain S carter et al correlates with our present study.

66 cases had caesarean delivery in study group and 52 cases had caesarean delivery in control group.

There is no statistical difference in rate of caesarean delivery in maternal intravenous hydration received study group and the control group.

In a study conducted by Mahnaz shahnazi et al [64] found significant increase in mean amniotic fluid index in a hydrated group from 4.7 to 6.25cm.mean difference 1.5cm 95%CI (P=<0.001).meconium staining liquor was observed in 20% of cases and 36.4% of the controls which showed no significant difference.

Caesarean section was performed in 30% of the hydrated group and 45.5% of the control group. The difference between the two groups was not statistically significant.

In our present study there is significant increase in post treatment AFI in a hydrated group.(P=<0.001).

Mean increase in amniotic fluid index in hydrated group is from 4.965 to 6.844. Mean difference is 3.0cm 95% CI (P=<0.001)

In this present study meconium staining liquor was observed in 15.3% of cases and 10.7% of the controls. there is no significant difference.

Caesarean section was performed in 34.7% of the hydrated group and 25% of the control group. The difference between the groups was statistically non significant.(P=0.489).

This is correlating with the study by Mahnaz shahnazi et al.

Fait et al (2003) studied effect of one week of maternal hydration.There was increase in AFI .They conclude that maternal intravenous hydration appers to increase both the actual and ultrasound estimated amniotic fluid volume[65]

In our present study also after a mean duration of 1.2 weeks of intra venous hydration therapy there is a significant increase in AFI.

SUMMARY

In this study effect of intravenous maternal hydration therapy in patients in third trimester with oligohydramnios AFI <5 cm was studied and perinatal out come in this study group is compared with the control group who were patients in third trimester of pregnancy with normal AFI.

About 150 cases were studied in each group.

The following points were summed up after analyzing the antepartum period during which hydration therapy was given to the oligohydramnios group ,the intra partum course and the perinatal outcome.

1.87.3%in study group and 83.3% in control group were in the age group between 21-30 yrs.

In study group 54% were primi gravida and 46 % were multi gravida.

In control group 56% were primi gravidaand 44% were multi.

2. In a study group post treatment AFI was found to be significantly increased (P =<0.001)

3. In a study group 9.3% had non- reactive CTG during treatment and 5.3% in control group patients with normal AFI had non Reactive CTG on admission. The difference was not significant (P=0.18)

4. In a study group 15.3% had meconium stained liquor and 10.7% in control group had meconium stained liquor and the difference was found to be non – significant (p=0.163)

5. In a study group41.3% had normal delivery 14.7 had forceps, and 44% had normal delivery, 16.7% had out let forceps delivery and 34.7% had LSCS. The difference was found to be non significant. (p = 0.255).

6. In study group 34.8% were taken up for LSCS for fetal distress and 25% of control group were taken up for LSCS for fetal distress and the difference was non- significant (P =0.489)

7. In a study group32.0% had APGAR score <7 @ 5 min VS 17.3% in control group the difference was found to be non- significant(P =003)

8. The NICU admission in study group was 19.3% vs 12.7% in control group. The difference was found to be not significant (P = 0.115)

9. IN NICU admissions In study group 93.1% were in good condition at discharge and 2 cases died.

In control group 89.5% were in good condition at discharge and 2cases died and the difference was non significant.

CONCLUSION

Intravenous maternal hydration therapy with isotonic solution significantly increases AFI in third trimester oligohydramnios.

Intravenous maternal hydration therapy is not associated with any adverse maternal outcomes in third trimester oligohydraminos patients.

Intravenous maternal hydration therapy in third trimester oligohydramnios is associated with as similar perinatal out come as in patients with normal AFI in third trimester.

Intravenous maternal hydration therapy needs further studies, with large sample size, regarding materials and perinatal outcome.

This study was not designed for the mechanism of increase in AFI.

SUGGESTION

Oligohydramnios and its complications cause the mother and the fetus to suffer. Intra venous hydration therapy to the mother is a low cost method with no complications to the mother. More research with higher number of cases is necessary to confirm the benefits. If confirmed the need for preterm termination of the pregnancy due to isolated oligohydramnios will be eliminated.

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PROFORMA

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OBSTETRIC CODE:

LMP: EDD: GA Weeks:

PRESENTING COMPLAINTS:

MENSTRUAL HISTORY :

MARITAL HISTORY :

OBSTETRIC HISTORY:

PAST HISTORY:

GENERAL EXAMINATION:

HT: WT: TEMP: PR: BP:

PALLOR: PEDAL EDEMA:

CVS: RS:

P/A:

S/E:

P/V:

INVESTIGATIONS

- Haemoglobin level
- Blood grouping and typing
- Oral glucose challenge test
- HIV, VDRL HBs Ag
- Urine for Albumin, Sugar and deposits
- Obstetric Scan
- Pre treatment AFI

-post treatment AFI

- Duration of Hydration therapy
- NST
- Doppler study of UA/MCA:
- Delivery at Weeks GA Date Time

Mode of Delivery: Natural/outlet forceps/LSCS

Indication for LSCS: Foetal distress/others

BABY

: APGAR 1min 5Min

: Admission to NICU: Yes/No

: Reason for Admission: Observation/MAS/Foetal distress

: Condition on discharge

: Maternal Complications

ABBREVIATIONS

AF	=	Amniotic Fluid
AFV	=	Amniotic Fluid Volume
AFI	=	Amniotic Fluid Index
FHR	=	Fetal Heart Rate
LSCS	=	Lower Segment Caesarian Section
PPROM	=	Preterm Premature rupture of membranes
CTG	=	Cordio Toco Graph
NICU	=	Neonatal Intensive Care Unit

INSTITUTIONAL ETHICAL COMMITTEE,
STANLEY MEDICAL COLLEGE, CHENNAI-1

Title of the Work : A Prospective study of intravenous Hydration therapy in cases of oligohydramnios in third trimester and its effects on prenatal outcome.

Principal Investigator : Dr Sharmila.P

Designation : P G, M S (O & G)

Department : Department of O & G
Stanley Medical College
Chennai -01

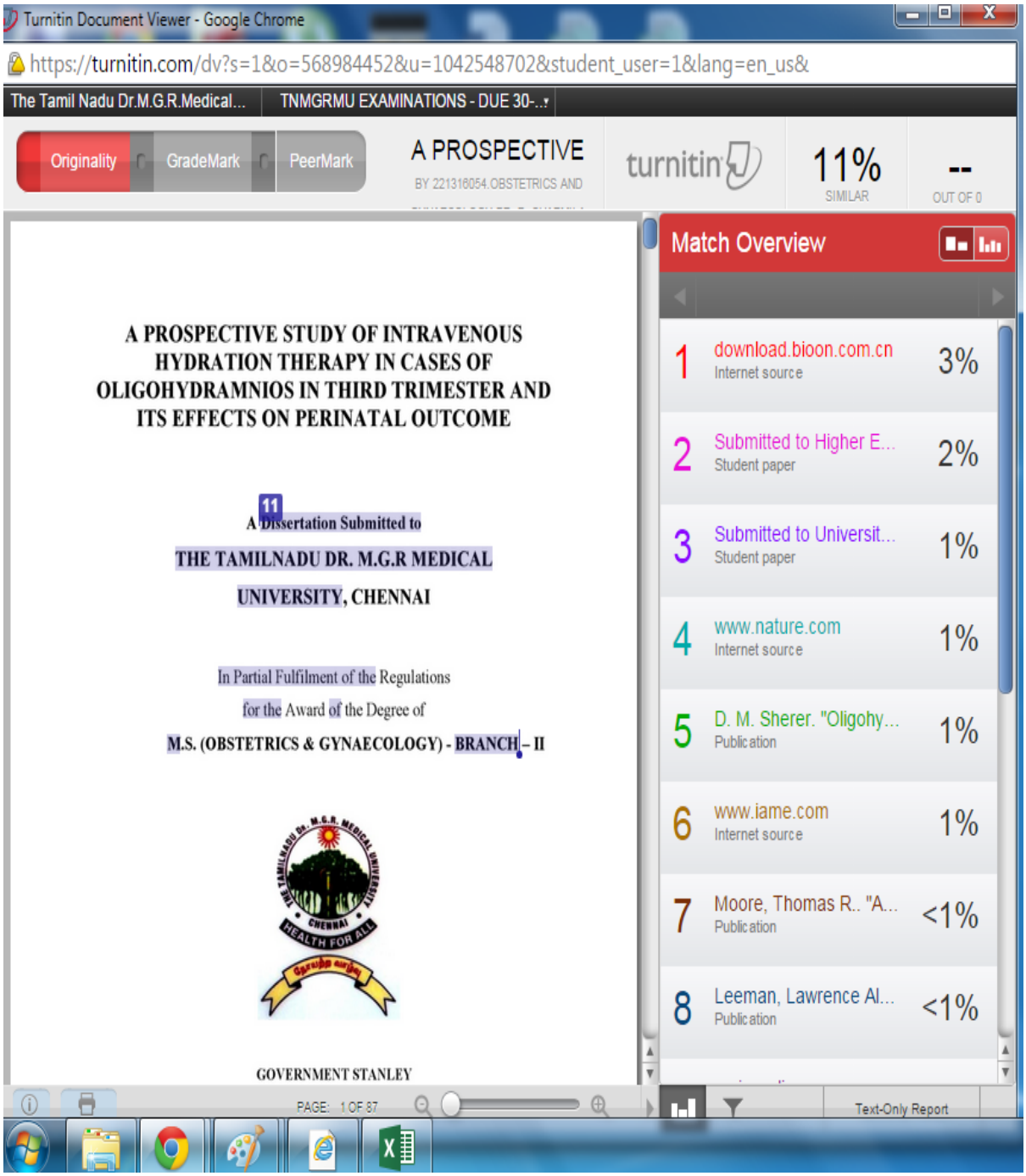
The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 25.03.2015 at the Council Hall, Stanley Medical College, Chennai-1 at 2PM

The members of the Committee, the secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

The Principal investigator and their team are directed to adhere to the guidelines given below:

1. You should inform the IEC in case of changes in study procedure, site investigator investigation or guide or any other changes.
2. You should not deviate from the area of the work for which you applied for ethical clearance.
3. You should inform the IEC immediately, in case of any adverse events or serious adverse reaction.
4. You should abide to the rules and regulation of the institution(s).
5. You should complete the work within the specified period and if any extension of time is required, you should apply for permission again and do the work.
6. You should submit the summary of the work to the ethical committee on completion of the work.

K Vasanthamani
MEMBER SECRETARY,
IEC, SMC, CHENNAI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A PROSPECTIVE STUDY OF INTRAVENOUS HYDRATION THERAPY IN CASES OF OLIGOHYDRAMNIOS IN THIRD TRIMESTER AND ITS EFFECTS ON PERINATAL OUTCOME
A Dissertation Submitted to
THE TAMILNADU DR. M.G.R MEDICAL UNIVERSITY, CHENNAI
In Partial Fulfilment of the Regulations
for the Award of the Degree of
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PATIENT CONSENT FORM

STUDY TITLE: **‘‘A PROSPECTIVE STUDY OF INTRAVENOUS HYDRATION THERAPY IN CASES OF OLIGOHYDRAMNIOS IN THIRD TRIMESTER AND ITS EFFECTS ON PERINATAL OUTCOME”**

STUDY CENTRE: Government RSRM lying in hospital, Chennai.

PARTCIPANT NAME: **AGE:** **I.D.NO:**

I confirm that I have understood the purpose of the above study. I have the opportunity to ask the question and all my questions and doubts have been answered to my complete satisfaction.

I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving any reason, without my legal rights being affected.

I understand that investigator, the institution, regulatory authorities and the ethics committee will not need my permission to look at my health records both in respect to the current study and further research that may be conducted in relation to it, even if I withdraw from the study. I understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from the study.

I hereby consent to, undergo complete physical examination, and diagnostic tests including haematological, and ultra-sonogram examinations for me.

I hereby consent to participate in this study on **‘‘A PROSPECTIVE STUDY OF INTRAVENOUS HYDRATION THERAPY IN CASES OF OLIGOHYDRAMNIOS IN THIRD TRIMESTER AND ITS EFFECTS ON PERINATAL OUTCOME”**

Place: Signature of the Patient:

Date: Address:

Signature of the witness: Signature of Investigator:

ஒப்புதல் படிவம்

திரு/திருமதி .....

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என்ற விலாசத்தில வசிக்கும் நான் எனக்கு அளிக்கப்பட்ட தகவல் படிவத்தில் உள்ள விவரங்களை படித்தும் கேட்டும் புரிந்து கொண்டேன்.

இந்த ஆய்வின் போது எனக்கு நீரேற்ற சிகிச்சை (Intravenous Hydration Therapy) செய்துகொள்ள சம்மதிக்கிறேன். ஆய்வின் முடிவினை சொந்த அடையாளங்களை வெளியிடாமல் மருத்துவ ஆராய்ச்சிக்காக பயன்படுத்திக்கொள்ள சம்மதிக்கிறேன்.

கையொப்பம்

நாள் :

இடம் :

தகவல் படிவம்

ஸ்டான்லி மருத்துவமனையின் ஆர். ஏஸ். ஆர். எம். மருத்துவமனையில் மகப்பேறு மற்றும் பெண்கள் நல மருத்துவ துறையில் மேற்கொள்ளப்படும் ஆய்வு தொடர்பான தகவல் படிவம் இது.

இந்த ஆய்வு அனுபவம் வாய்ந்த மருத்துவர்களின் உதவியோடு நடத்தப்படுகிறது.

28 வாரங்கள் கருக்காலத்திற்குபின் பனிக்குட நீர் குறைவாக உள்ள கர்ப்பிணி பெண்களுக்கு நீரேற்ற சிகிச்சை (Intravenous Hydration Therapy) அளிக்கும் ஆய்வு இது.

இது பனிக்குட நீர் குறைவாக உள்ள கர்ப்பத்தில் உள்ள குழந்தைக்கு மூச்சுத்திணறல் போன்ற விரும்பத்தகாத விளைவுகளை குறைக்கிறது என்பதனை பல்வேறு ஆய்வுகள் கூறுகின்றன.

எனவே இந்த ஆய்வு இந்த மருத்துவமணையில் கர்ப்பிணி பெண்களுக்கு மேற்கொள்ளப்பட்டு ரிங்கர் லேக்டேட் (Ringer Lactate) எனப்படும் திரவம் ஊசி மூலம் செலுத்தப்பட்டு அதன்மூலம் பனிக்குட நீர் அதிகமாவது மற்றும் கர்ப்பத்தில் உள்ள குழந்தைக்கு மூச்சுத்திணறல் ஏற்படுவது தவிர்க்கப்படுவது ஆகியவை கண்டறியப்படுகிறது.

இந்த திரவத்ததை கர்ப்பிணி பெண்களுக்கு இரத்தக்குழாய் மூலம் ஏற்றுவதால் எந்தவித பின் விளைவுகளும் தாய்க்கும், சேய்க்கும் இல்லை.

இந்த ஆய்வு கர்ப்பிணி பெண்கள் தங்கள் சுய விருப்பத்துடன் வந்தால் மட்டுமே மேற்கொள்ளப்படுகிறது.

HYDRATION THERAPY MASTER CHART

S.No	Name	Age	IP.No	Parity	GA on Admission in Weeks	Pre Treatment AFI	Post Treatment AFI	NST	Doppler Study	Liquor	GA Before Delivery	Duration of hydration therapy In Weeks	Mode of Delivery	LSCS Indication	Birth Weight in Kg	1 min APGAR	5 min APGAR	Admissiomn in NICU	Reason	Condition at Discharge	Meternal Morbidity
1	Sathya	19	2880	Primi	35	5.00	7.00	R	N	Clear	36.5	1.5	Normal		2.90	5	7	-			-
2	Gowri	21	2911	Primi	35	5.00	6.80	R	N	Clear	36.3	1.3	Normal		2.90	6	7	-			-
3	Anjali	23	3074	Multi	36	4.80	7.30	R	N	Clear	37	1	LSCS	FD	2.80	6	7	+	Observation	Good	-
4	Dhanam	22	3175	Primi	35	5.00	7.20	R	N	Clear	37	2	Outlet		2.70	7	9				-
5	Deepa	21	3235	Primi	36	4.80	8.00	R	N	Clear	37	1	Normal		2.80	7	8				-
6	Saranya	24	3294	Multi	37	4.70	7.00	NR	N	Thin MSAF	38	1	LSCS	FD	3.10	5	7	+	Observation	Good	-
7	Vanitha	19	3382	Primi	36	4.60	6.80	NR	N	Clear	37	1	LSCS	FD	2.60	7	9				-
8	Geetha	29	3399	Multi	35	5.00	6.90	R	N	Clear	37	2	LSCS	FD	2.70	5	7	+	Observation	Good	-
9	Meena	21	3485	Primi	35	5.00	7.30	R	N	Clear	37	2	Normal		2.30	7	8				-
10	Kavitha	23	3499	Multi	36	4.80	6.80	R	N	Clear	37	2	LSCS	FD	2.30	5	7	+	Observation	Good	-
11	Deepa	17	3502	Primi	34	5.00	7.00	R	N	Clear	36.5	2.5	Normal		2.70	4	5	+	RI	Good	-
12	Tamilselvi	23	3511	Primi	36	5.00	6.00	NR	Ab	Thin MSAF	37	1	LSCS	FD	2.90	6	7				-
13	Keerthana	23	3527	Primi	35	5.00	6.90	R	N	Clear	37	1	LSCS	Others	2.40	7	9				-
14	Kavitha	23	3572	Primi	36	4.00	6.80	R	N	Thin MSAF	37	1	LSCS	FD	2.80	5	7	+	Observation	Good	-
15	Dhanalakshmi	22	3579	Primi	35	5.00	7.00	R	N	Clear	37	1	Normal		2.70	6	9				-
16	Sharmila	24	3598	Multi	36	4.90	6.50	R	N	Moderate MSAF	37	1	LSCS	FD	2.40	6	6	+	RD		-
17	Vasanthi	18	3603	Primi	36	5.00	6.50	R	N	Clear	37	1	Normal		2.40	7	8				-
18	Chithra	21	3604	Primi	36	4.80	6.80	R	N	Thin MSAF	37	1	Outlet		2.40	7	8				-
19	Hema	23	3631	Multi	35	5.00	6.80	R	N	Clear	37	2	Normal		2.37	7	8				-
20	Kalpana	24	3666	Multi	36	4.50	6.30	R	N	Clear	37	1	LSCS	FD	2.40	7	9				-
21	Devi	26	3669	Multi	33	5.00	6.80	R	N	Clear	36.3	3.3	Normal		2.30	7	7				-
22	Subha	28	3680	Multi	36	5.00	6.80	R	N	Clear	37	1	Normal		2.60	7	9				-
23	Sandhiya	18	3908	Primi	35	5.00	6.80	R	N	Thin MSAF	36.5	1.5	Outlet		2.60	7	9				-

24	Nandhini	25	3942	Multi	34	5.00	7.00	R	N	Clear	36.6	2.6	Normal		2.40	7	8				–
25	Kasthuri	26	3962	Multi	37	5.00	7.00	R	N	Clear	37	1	LSCS	Others	3.10	7	9				–
26	Lela	27	3969	Multi	35	5.00	7.10	R	N	Clear	37	2	LSCS	FD	2.60	6	7	+	Observation		–
27	Prema	22	4101	Primi	36	4.80	7.10	R	N	Clear	37	1	LSCS	Others	2.40	6	8				–
28	Sudha	23	4192	Multi	35	5.00	6.80	R	N	Clear	37	2	Normal		2.40	6	7				–
29	Sri Devi	23	4280	Multi	35	5.00	6.90	R	N	Clear	37	2	Normal		2.30	7	8				–
30	Nadhiya	23	4282	Multi	36	4.60	7.00	R	N	Clear	37	1	LSCS	Others	2.40	7	8				–
31	Shanthi	255	4320	Multi	37	5.00	7.20	R	N	Clear	38	1	LSCS	Others	2.90	7	9				–
32	Suganthi	36	4410	Multi	35	5.00	6.90	R	N	Clear	37	2	Normal		3.20	7	9				–
33	Parimala	23	4442	Multi	35	5.00	6.80	R	N	Clear	37	2	Normal		2.40	7	9				–
34	Ranjani	22	4458	Primi	35	5.00	6.90	R	N	Clear	37	2.1	LSCS	Others	2.60	7	8				–
35	Karthika	21	4469	Primi	36	5.00	6.50	NR	Ab	Thin MSAF	36.6	0.6	LSCS	FI	2.60	4	5	+	RD	Died (Sepsis)	–
36	Meena	23	4498	Multi	36	5.00	6.50	R	N	Clear	37	1	LSCS	Others	2.75	6	7				–
37	Sarasu	23	4565	Multi	35	5.00	6.80	R	N	Clear	37	1	Normal		2.40	7	8				–
38	Sathya	22	4624	Primi	36	4.80	6.00	NR	N	Clear	37	1	LSCS	FD	2.60	7	8	+	Observation	Good	–
39	Janaki	18	4698	Primi	36	5.00	6.80	R	N	Clear	347	1	Outlet		2.75	7	8				–
40	Gomathi	26	4786	Multi	35	5.00	6.36	R	N	Clear	37	2	Normal		2.40	7	8				–
41	Geetha	33	4855	Multi	35	5.00	6.80	R	N	Clear	37	2	Normal		2.40	7	9				–
42	Rashiya	25	4901	Multi	35	5.00	6.30	R	N	Clear	37	2	Normal		2.40	7	8				–
43	Parveen	24	4914	Multi	35	5.00	6.80	R	N	Thin MSAF	37	2	Normal		2.30	6	7				–
44	Bharati	22	4927	Primi	35	4.50	6.80	R	N	Clear	36.6	2	Outlet		2.30	7	8				–
45	Salma	23	4932	Primi	36	4.60	6.80	R	N	Clear	37	1	Normal		2.75	6	9				–
46	Monika	19	4946	Primi	36	4.80	6.80	R	N	Clear	37	1	Normal		2.90	7	9				–
47	Divya	24	4950	Multi	37	5.00	6.80	R	N	Clear	38	1	LSCS	FD	3.10	6	9	+	Observation	Good	–
48	Jeevitha	24	4956	Multi	35	5.00	6.80	R	N	Clear	37	2	Normal		2.40	7	8				–
49	Manju	23	4988	Primi	36	4.90	7.00	R	N	Thin MSAF	37	1	Outlet		2.80	7	8				–
50	Muthulakshmi	19	4999	Primi	35	5.00	6.90	R	N	Clear	37	2	Outlet		2.40	6	7				–
51	Kuppammal	24	5004	Multi	35	5.00	6.80	R	N	Clear	37	2	Normal		2.90	6	7				–
52	Jerina	23	5119	Primi	36	5.00	6.80	R	N	Clear	37	1	LSCS	FD	2.90	7	9				–
53	Bakyalakshmi	19	5128	Primi	36	5.00	6.80	R	N	Clear	37	1	LSCS	FD	2.80	6	7	+	Observation		–

54	Anitha	24	5267	Multi	35	5.00	6.80	R	N	Clear	37	2	Normal		2.40	7	8				–
55	Eswari	23	5233	Primi	36	5.00	6.80	R	N	Clear	37	1	Outlet		2.70	7	8				–
56	Malini	26	5299	Multi	35	5.00	7.00	R	N	Clear	37	2	Normal		2.40	6	7				–
57	Kaliammal	19	5302	Primi	36	5.00	7.00	R	N	Clear	37	1	LSCS	Others	2.70	7	8				–
58	Nirosha	25	5354	Multi	35	5.00	7.10	R	N	Clear	37	2	Normal		2.60	6	7				–
59	Gayathri	24	5452	Primi	36	5.00	7.00	R	N	Clear	37	1	LSCS	Others	2.90	7	9				–
60	Santhiya	23	5495	Primi	36	5.00	7.00	R	N	Clear	37	1	LSCS	Others	2.90	7	9				–
61	Vanishree	22	5527	Primi	36	5.00	7.00	R	N	Clear	37	1	LSCS	Others	2.40	7	8				–
62	Rukshana	18	5544	Multi	36	6.00	6.80	R	N	Thin MSAF	37	1	LSCS		2.80	7	8				–
63	Rosi	24	5587	Multi	34	5.00	6.80	NR	N	Clear	36	2	Outlet		2.80	6	7				–
64	Mythili	23	5659	Primi	35	5.00	7.30	R	N	Clear	37	2	Normal		2.40	6	7				–
65	Indira	22	5662	Primi	35	5.00	7.20	R	N	Clear	37	2	Normal		2.40	6	7				–
66	Shamili	18	5697	Primi	36	5.00	7.00	R	N	Clear	37	1	LSCS	Others	2.70	6	9				–
67	Ajimunisha	23	5724	Primi	36	5.00	8.00	R	N	Clear	37	1	Outlet		2.90	7	9				–
68	Raji	22	5736	Primi	35	5.00	7.30	R	N	Clear	37	2	Normal		2.40	7	8				–
69	Aruna	23	5766	Primi	36	5.00	7.20	R	N	Clear	37	1	LSCS	Others	2.80	7	9				–
70	Ramani	23	5798	Primi	36	5.00	7.80	R	N	Thin MSAF	37	1	Outlet		2.90	6	7	+	Observation	Good	–
71	Sudha	24	5802	Multi	35	5.00	7.10	R	N	Clear	37	1	Normal		2.30	6	7				–
72	Suseela	23	5816	Primi	36	5.00	7.30	R	N	Clear	37	1	Normal		2.30	6	7				–
73	Jothika	24	5841	Primi	34	5.00	7.40	R	N	Clear	36.2	2.2	Outlet		1.90	6	7	+	SGA/LBW	Good	–
74	Monika	25	5918	Multi	35	5.00	7.00	R	N	Clear	37	2	Normal		2.60	6	7				–
75	Katheeja	24	5997	Multi	37	5.00	7.00	R	N	Clear	38	1	LSCS	FI	3.10	7	9				–
76	Radha	23	6012	Primi	35	4.50	6.80	R	N	Clear	37	2	LSCS	FD	2.40	6	7	+	Observation	Good	–
77	Maha	26	6098	Multi	36	6.90		R	N	Clear	37	2	LSCS	Others	2.30	7	8				–
78	Samanthi	25	6112	Multi	35	5.00	6.90	R	N	Clear	37	2	LSCS	Others	2.30	7	8				–
79	Vanitha	25	6127	Multi	36	4.50	6.50	R	N	Thin MSAF	37	1	LSCS	FD	2.80	7	9				–
80	Shanthi	25	6186	Multi	35	4.80	6.50	R	N	Clear	36.3	1.3	Normal		1.80	6	7	+	SGA/LBW	Good	–
81	Sunitha	25	6212	Multi	36	4.80	7.00	R	N	Clear	37	1	LSCS	FI	2.70	7	9				–
82	Priya	24	6218	Multi	35	5.00	6.80	R	N	Clear	37	2	Normal		2.40	7	8				–
83	Rejina	32	6267	Multi	35	4.80	6.80	NR	N	Clear	37	2	LSCS	FD	2.30	7	8				–

84	Revathi	23	6292	Primi	36	5.00	7.00	R	N	Clear	37	1	LSCS	Others	2.60	6	9				–
85	Sarala	24	6304	Multi	34	5.00	6.90	R	N	Clear	36.3	2.3	Normal		1.80	7	8	+	SGA/LBW	Good	–
86	Malathi	23	6312	Primi	35	4.60	6.90	R	N	Clear	37	2	LSCS	FI	2.40	7	8				–
87	Pnitha	33	6327	Multi	36	5.00	6.80	R	N	Moderate MSAF	37	1	LSCS	FD	2.80	6	8	+	Observation	good	–
88	Chithra	23	6334	Primi	35	5.00	8.00	R	N	Clear	37	2	Normal		2.30	6	7				–
89	Jothi	22	6342	Primi	36	5.00	7.20	R	N	Thin MSAF	37	1	Outlet		2.70	6	9				–
90	Devi	26	6410	Multi	35	5.00	7.00	R	N	Clear	37	2	Normal		2.75	6	9				–
91	Ranjitha	25	6421	Primi	36	5.00	7.20	R	N	Clear	37	1	LSCS	FD	2.40	7	8				–
92	Rasul	26	6482	Multi	36	4.80	6.80	R	N	Thin MSAF	37	1	LSCS	FD	2.40	7	8	+	Observation	Good	–
93	Shakila	32	6543	Multi	35	5.00	6.80	R	N	Clear	36.3	1.3	Normal		1.95	6	7	+	SGA/LBW	Good	–
94	Shabana	24	6633	Multi	68	5.00	6.80	R	N	Clear	36.6	1.6	Outlet		3.00	6	8				–
95	Indra	25	6688	Multi	35	5.00	6.80	R	N	Clear	37	2	Normal		2.30	7	8				–
96	Priya	25	6714	Multi	35	4.80	6.80	R	N	Clear	37	2	Normal		2.30	7	8				–
97	Asha	26	6728	Primi	36	5.00	6.80	R	N	Clear	37	1	LSCS		2.70	7	9				–
98	Divya	25	6734	Multi	35	4.80	6.80	R	N	Clear	37	2	Normal		2.40	7	8				–
99	Raji	24	6741	Multi	35	4.80	6.70	R	N	Clear	37	2	Normal		2.40	7	8				–
100	Sudha	25	6948	Primi	36	5.00	6.90	R	N	Clear	37	1	Normal		2.60	7	9				–
101	Amudha	23	7009	Primi	36	5.00	6.70	NR	Ab	Moderate MSAF	37	1	LSCS	FD	2.40	3	4	+	MAS(Died)		–
102	Vasanthi	22	7111	Primi	35	5.00	7.20	R	N	Clear	37	2	Normal		2.45	6	7				–
103	Alen Mary	21	7128	Primi	36	4.80	6.80	NR	N	Thin MSAF	37	2	LSCS	FD	2.40	5	7	+	Observation	Good	–
104	Gulsar Bee	22	7214	Primi	36	5.00	7.00	R	N	Clear	37	1	Normal		2.30	6	7				–
105	Kuman	22	7216	Primi	36	5.00	7.00	R	N	Clear	37	1	LSCS	Others	2.40	7	9				–
106	Sathya	34	7288	Multi	37	5.00	7.10	R	N	Clear	38	1	LSCS	FI	3.10	7	8				–
107	Latha	22	7307	Primi	36	4.80	7.00	R	N	Clear	37	1	Normal		2.45	7	9				–
108	Gomathi	23	7328	Primi	36	5.00	7.00	R	N	Clear	37	1	LSCS	FI	2.20	6	7				–
109	Suguna	24	7356	Multi	35	5.00	7.20	R	N	Clear	37	2	Normal		2.30	6	8				–
110	Komala	32	7384	Multi	35	5.00	6.40	NR	N	Clear	36	1	Normal		1.80	6	7	+	SGA/Observation	Good	–
111	Anbuselvi	24	7401	Multi	36	5.00	7.20	NR	N	Thin MSAF	37	1	Outlet		2.30	5	7	+	Observation	Good	–
112	Poornima	27	7418	Multi	36	5.00	6.70	R	N	Clear	37	1	LSCS	FI	2.40	6	8				–
113	Vanitha	28	7427	Multi	36	4.50	6.50	R	N	Clear	37	1	LSCS	FI	2.40	6	7				–

114	Delli Rani	28	7432	Multi	35	5.00	7.00	R	N	Clear	37	2	Normal		2.30	6	7				–
115	Samundi	23	7440	Primi	36	5.00	6.80	NR	N	Clear	37	1	LSCS	FD	2.40	5	7	+	Observation	Good	–
116	Baby	22	7492	Primi	5	5.80	7.30	R	N	Clear	39.1	0.1	Outlet		3.50	7	9				–
117	Aswathi	24	7517	Multi	37	4.90	6.30	R	N	Clear	37.3	0.3	LSCS	Others	3.10	7	9				–
118	Renuka	24	7528	Multi	35	5.00	6.40	R	N	Clear	36.5	1.5	Normal		2.40	7	8				–
119	Beula	23	7540	Primi	366	5.00	6.40	R	N	Clear	37	1	LSCS	FI	2.30	7	8				–
120	subha	23	7613	Primi	36	5.00	6.70	R	N	Clear	37	1	LSCS	Others	2.40	7	9				–
121	Ameena	24	7700	Multi	35	5.00	6.20	R	N	Clear	37	2	Normal		2.40	7	8				–
122	Mubina	23	7812	Primi	36	4.90	6.90	R	N	Thin MSAF	37	1	Outlet		2.30	7	8				–
123	Divya	23	7918	Primi	36	5.00	7.10	R	N	Clear	37	1	Normal		2.40	7	8				–
124	Valarmathi	23	7984	Primi	33	5.00	6.00	NR	Ab	Thin MSAF	35	2	LSCS	FI	1.70	6	7	+	Observation	Good	–
125	Fatima	24	8004	Multi	37	5.00	6.30	R	N	Clear	38	1	LSCS	FI	3.10	6	9	+			–
126	Ambiga	25	8017	Multi	35	5.00	6.90	R	N	Clear	37	2	Normal		2.10	6	7				–
127	Gayathri	24	8119	Primi	36	5.00	6.80	R	N	Clear	37	1	Outlet		3.00	7	9				–
128	Radhika	24	8127	Primi	35	5.00	6.80	R	N	Clear	36.5	1.5	LSCS	FI	1.90	5	7	+	Observation	Good	–
129	Nadhiya	23	8131	Primi	36	5.00	7.20	R	N	Clear	37	1	Normal		2.40	7	9				–
130	Sharmila	22	8204	Primi	37	5.00	6.80	R	N	Clear	37.3	0.3	Normal		3.05	7	9				–
131	Sangeetha	21	8212	Primi	37	4.80	6.30	R	N	Clear	37.02	0.2	Normal		3.02	7	9				–
132	Vijaya	21	8221	Primi	37	5.00	6.90	R	N	Clear	37.1	0.1	Outlet		3.10	7	9				–
133	Kalaivani	21	8294	Primi	37	4.50	7.00	R	N	Clear	37.3	0.3	Normal		3.10	7	9				–
134	sumathi	21	8304	Primi	37	5.00	6.90	R	N	Thin MSAF	37.3	0.3	Outlet		3.10	7	8	+	Observation	Good	–
135	Rihana	21	8327	Primi	37	4.80	6.80	R	N	Clear	37.3	0.3	Normal		3.00	7	8				–
136	Uma	21	8336	Primi	37	5.00	6.30	R	N	Clear	37.2	0.2	LSCS	FI	2.90	6	7				–
137	Lilli	22	8415	Primi	37	5.00	6.20	R	N	Clear	37.1	0.1	Normal		2.90	7	8				–
138	Nalini	26	8487	Multi	34	5.00	6.37	R	N	Clear	36	2	Normal		2.20	6	7				–
139	Gomathi	23	8532	Primi	37	5.00	6.60	R	N	Thin MSAF	37.02	0.02	Outlet		3.00	7	8				–
140	Parvathi	26	8577	Multi	37	5.00	5.80	R	N	Clear	37.2	0.2	LSCS	Others	3.00	7	8				–
141	Jothi	25	8618	Multi	37	5.00	6.00	R	N	Clear	37.3	0.3	LSCS	FI	3.00	7	8				–
142	Jansirani	23	8636	Primi	37	5.00	6.20	R	N	Thin MSAF	37.3	0.3	Outlet		3.00	7	8				–
143	Lawanya	24	8673	Multi	37	5.00	6.80	R	N	Clear	37.2	0.2	LSCS	Others	2.90	6	8				–

144	Tamilarasi	23	8714	Primi	37	5.00	7.00	R	N	Clear	37.2	0.2	LSCS	FI	3.00	7	9				–
145	Devi	24	8745	Multi	37	5.00	6.30	R	N	Clear	37.2	0.2	LSCS		3.30	7	9				–
146	Shyamala	22	8797	Primi	37	5.00	6.70	R	N	Clear	37.5	0.5	LSCS	FI	3.00	7	9				–
147	Shakira	22	8838	Primi	38	5.00	6.70	NR	N	Clear	38	0	LSCS	FI	3.40	7	9				–
148	Deivanai	22	8902	Primi	37	5.00	6.80	R	N	Clear	37.3	0.3	LSCS	FI	3.10	7	9				–
149	Salma	22	8917	Primi	37	5.00	6.90	R	N	Clear	37.2	0.2	LSCS	Others	3.01	7	8				–
150	Farida	22	8999	Primi	37	5.00	7.00	R	N	Clear	37.2	0.2	Normal		3.10	7	8				–

NST = Non Stress test
R = Reactive
NR = Non-reactive
N = Normal
Ab = Abnormal
MSAF = Meconium Stained Amniotic fluid
FD = Fetal Distress
FI = Failed Induction
RD = Respiratory Distress
SGA = Small For Gestational Age
LBW = Low Birth Weight
NICU = Neonatal Intensive Care Unit

CONTROL GROUP

S.No	Name	Age	IP.No	Parity	GA	AFI	NST	Liquor	Mode of Delivery	Birth Weight	1 min APGAR	5 min APGAR	LSCS Indication	Admissiomn in NICU	Reason	Condition at Discharge	Meternal Morbidity
1	Kavitha	19	1036	Primi	36.0	8.0	NR	ThinMSAF	LSCS	2.80	6	7					-
2	Lakshmi	22	1039	Primi	38.0	8.3	R	Clear	LSCS	3.10	6	9					-
3	Samsath Begum	22	1040	Multi	37.0	7.4	R	Clear	Normal	2.80	6	8					-
4	Ruksana	21	1043	Multi	37.0	9.6	R	Clear	Normal	2.80	7	8					-
5	Ramya	22	1045	Multi	37.0	9.0	R	Clear	Normal	2.80	7	9					-
6	Mala	25	1178	Multi	37.0	8.0	R	Clear	Normal	2.80	6	7					-
7	Rasma	22	1132	Primi	39.0	7.4	R	Clear	Outlet	3.50	7	8					-
8	Yogalakshmi	21	1371	Primi	374.0	1.3	R	Clear	Outlet	2.80	7	8					-
9	Deepalakshmi	25	1231	Multi	37.0	9.3	R	Clear	Normal	2.80	7	9					-
10	Kaveri	29	1230	Multi	37.0	9.4	R	Clear	Outlet	2.80	7	8					-
11	Karthika	21	1243	Primi	37.0	8.7	R	Clear	LSCS	3.00	7	9					-
12	Priyanka	27	1248	Multi	37.0	8.0	R	Clear	LSCS	2.80	6	7					-
13	Kavitha	26	1252	Multi	37.0	8.0	R	Clear	LSCS	2.80	7	8					-
14	Gowthami	28	1269	Multi	37.0	8.0	R	Clear	Normal	3.00	7	9					-
15	Suganya	25	1273	Primi	37.0	7.4	R	Clear	Outlet	3.00	7	9					-
16	Yuvarani	16	1284	Multi	37.0	8.8	R	Clear	Normal	3.00	7	9					-
17	Sumithra	23	1300	Primi	37.0	8.7	R	Clear	Normal	3.00	7	9					-
18	Rajathi	25	1323	Multi	36.6	8.8	R	Clear	LSCS	1.90	6	7		+	SGA/LBW	Good	-
19	Nilofar	22	1418	Multi	36.6	9.3	R	Clear	Normal	3.00	6	7					-
20	Sarata	24	1490	Multi	36.6	8.4	R	Clear	Normal	3.00	6	9					-
21	Devi	29	1512	Multi	36.6	8.0	R	Clear	Normal	2.40	7	8					-

22	Priya	21	1514	Multi	36.6	9.2	R	Clear	Normal	2.30	7	8					-
23	Dilshath	22	1610	Multi	36.6	9.4	R	Clear	Normal	3.00	6	9					-
24	Manjula	18	1612	Primi	37.0	8.7	R	Clear	Outlet	3.00	7	8					-
25	Mohana	23	1800	Multi	37.0	9.3	R	Clear	Normal	2.60	7	8					-
26	Kanchana	25	1892	Multi	37.0	9.4	R	Clear	Normal	2.80	7	9					-
27	Sarala	20	1901	Primi	36.2	9.0	R	Clear	Outlet	2.70	6	7					-
28	Sangeetha	21	1912	Multi	36.1	8.8	R	Clear	Normal	2.70	7	8					-
29	Mahalalkshmi	24	1923	Multi	37.0	7.9	R	Clear	Normal	2.80	7	9					-
30	Akila	21	1936	Primi	37.0	9.0	R	Clear	Outlet	2.60	6	7					-
31	Lkamatchi	27	1937	Multi	36.5	8.9	R	Clear	Normal	2.40	7	8					-
32	Radhika	21	1939	Primi	36.6	7.7	R	ThinMSAF	Normal	2.70	6	7					-
33	Sudha	27	1978	Multi	37.0	9.3	R	Clear	Normal	2.75	7	9					-
34	Selvi	19	1987	Primi	37.0	9.3	R	Clear	LSCS	2.75	6	9	FD				-
35	Jothi	25	1999	Multi	27.0	9.3	R	Clear	Normal	2.75	7	8					-
36	Noori	21	2004	Multi	37.0	10.4	R	Clear	Normal	2.75	7	8					-
37	Lakshmi	21	2010	Primi	37.0	11.0	R	Clear	Outlet	2.70	7	8					-
38	Josephin	32	2018	Multi	38.0	12.3	R	Clear	LSCS	3.50	7	9					-
39	Nithya	21	2104	Primi	37.0	11.7	NR	ThinMSAF	LSCS	2.80	6	9					-
40	Shyamala	29	2110	Multi	36.6	13.0	R	Clear	Normal	2.50	7	8					-
41	Mibina	21	2121	Primi	35.5	10.2	R	Clear	Outlet	1.80	6	7					-
42	Meena	22	2124	Multi	36.6	17.4	R	Clear	Normal	2.50	7	9					-
43	Sridevi	24	2126	Primi	37.0	14.0	R	Clear	LSCS	3.40	6	9	FD				-
44	Geetha	24	2128	Primi	37.1	10.0	NR	ThinMSAF	LSCS	2.90	6	9	FD	+	Observation	Good	-
45	Rajeswari	28	2131	Multi	37.1	8.3	R	Clear	Normal	2.85	7	8					-
46	Priya	27	2142	Multi	34.3	8.7	R	Clear	Normal	2.25	6	7		+	preterm	Good	-
47	Madavi	20	2168	Primi	37.1	9.0	R	Clear	Normal	2.65	7	9					-
48	Kanchana	24	2202	Primi	37.0	9.0	NR	ThinMSAF	LSCS	1.80	6	7	FD	+	SGA/LBW	Good	-
49	Saranya	21	2212	Primi	35.9	9.3	R	Clear	LSCS	2.60	5	6	FD	+	preterm	Good	-

50	Ramya	24	2224	Multi	35.5	8.0	R	Clear	LSCS	2.70	7	9					-
51	Gomathi	22	2248	Primi	37.0	7.4	R	Clear	Normal	2.75							-
52	Maha	21	2253	Primi	37.0	7.8	R	ThinMSAF	Outlet	3.30							-
53	Rajeswari	21	2256	Primi	37.0	7.7	R	Clear	LSCS	3.20	7	9	FI				-
54	Kanmani	20	2274	Primi	36.6	7.8	R	Clear	LSCS	3.30	7	9	FI				-
55	Anandhi	21	2292	Primi	37.0	9.3	R	Clear	LSCS	3.40	7	9	Others				-
56	Bagavathi	21	2301	Primi	37.0	9.3	R	Clear	Outlet	3.40	7	8					-
57	Arul Mozhi	25	2334	Multi	37.0	12.0	R	Clear	Outlet	3.40	7	9					-
58	Jothi	20	2314	Primi	37.0	10.3	R	Clear	Outlet	3.40	7	8					-
59	Jen itha	20	2321	Primi	37.0	11.8	R	Clear	Normal	3.20	7	9					-
60	Thenmozhi	25	2334	Multi	37.0	12.0	R	Clear	Outlet	3.40	7	9					-
61	sudha Rani	25	2336	Multi	37.0	8.3	R	Clear	LSCS	3.20	7	9	FI				-
62	Revathi	22	2338	Primi	36.6	8.4	R	Clear	Normal	2.70	7	8					-
63	Meenatchi	25	2342	Multi	36.5	8.5	R	Clear	Normal	2.90	7	8					-
64	Divya	22	2510	Primi	37.0	8.6	R	Clear	Normal	2.90	7	9					-
65	Ramish Bee	22	2612	Primi	37.0	9.3	R	Clear	Normal	2.90	7	8					-
66	Suhasini	22	2688	Primi	37.1	10.1	R	Clear	LSCS	3.00	7	9	FI				-
67	Nirmala	20	2914	Primi	37.2	9.8	R	Clear	LSCS	3.00	7	9	FI				-
68	Shanthi	23	3003	Multi	33.5	8.3	R	Clear	Normal	1.80	7	8		+	preterm Observation	Good	-
69	Kanchana	31	3121	Multi	37.0	9.0	R	Clear	LSCS	3.00	7	8					-
70	Nandhini	22	3140	Primi	37.5	8.7	R	Clear	Outlet	3.50	7	8					-
71	Booma Devi	21	3208	Primi	37.5	8.8	R	Clear	Outlet	3.50	7	9					-
72	Samanthi	29	3400	Multi	37.2	7.8	R	Clear	LSCS	3.50	7	8	FI				-
73	Parveen	22	3432	Primi	37.0	8.9	R	Clear	Normal	3.20	7	9					-
74	Abibunisa	22	3488	Primi	37.0	9.2	R	Clear	Normal	3.10	7	8					-
75	Reena	20	3565	Primi	37.0	8.7	NR	Clear	LSCS	3.10	6	6	FD	+	Observation	Good	-
76	Yasmin	25	3590	Multi	37.0	8.3	R	ThinMSAF	Outlet	3.00	7	9					-
77	Sangeetha	19	3595	Primi	37.0	8.4	R	ThinMSAF	Outlet	2.80	7	9					-

78	Samshath	23	3674	Multi	37.0	8.6	R	Clear	Normal	3.20	7	8					-
79	Mehala	21	3679	Primi	36.5	8.5	R	Clear	Outlet	2.40	7	8					-
80	Deepa	21	3712	Primi	36.3	8.4	R	Clear	Outlet	2.50	7	8					-
81	Bharati	24	3866	Multi	36.2	9.2	R	Clear	Normal	2.50	7	8					-
82	Mobeen	21	3912	Multi	36.1	9.3	R	Clear	LSCS	2.50	7	8					-
83	Sathya	22	3920	Multi	36.6	9.0	R	Clear	Normal	2.70	7	9					-
84	Suganya	21	3922	Primi	36.8	8.8	R	Clear	Normal	2.40	7	8					-
85	Thoulath	22	3948	Multi	37.0	11.0	R	Clear	Normal	2.60	7	8					-
86	Komala	21	3955	Primi	3.3.6	8.8	R	Clear	Normal	1.70	7	8					-
87	Deepa	21	3968	Primi	37.1	8.7	R	Clear	LSCS	2.70	7	9	FD	+	preterm Observation	Good	-
88	Geetha	20	3969	Primi	37.2	8.7	R	Clear	LSCS	3.50	7	9					-
89	Sulaika Banu	22	3998	Primi	37.3	8.0	R	Clear	LSCS	3.60	6	7	FD				-
90	Priya	21	4002	Primi	37.0	8.3	R	Clear	Normal	2.50	7	9					-
91	Deepa	32	4012	Multi	37.0	8.9	R	Clear	Normal	2.80	6	8					-
92	Meena	20	4015	Primi	3.7	9.7	R	Clear	Normal	2.60	7	9					-
93	Vanishree	21	4028	Primi	36.6	14.0	R	Clear	Outlet	2.50	7	8					-
94	Jerina	21	4099	Multi	36.6	11.7	R	Clear	LSCS	2.40	7	8					-
95	Anitha	18	4124	Primi	36.5	13.0	R	Clear	LSCS	2.50	6	7	FI				-
96	Kuppammal	21	4132	Primi	37.0	13.0	R	Clear	Normal	2.60	6	7					-
97	Malimni	27	5001	Multi	37.5	9.3	R	Clear	Normal	3.00	7	9					-
98	Nirosa	22	5007	Primi	33.0	14.0	R	Clear	Normal	2.30	6	7		+	preterm	Good	-
99	Vasanthi	19	5123	Primi	37.0	9.0	R	Clear	LSCS	2.80	7	9	FI				-
100	Gayathri	24	5148	Multi	37.0	9.4	R	ThinMSAF	Normal	2.60	7	9					-
101	Indu	22	5208	Primi	34.0	13.2	R	Clear	Normal	2.00	6	7		+	preterm	Good	-
102	Poonghodi	24	5250	Multi	36.6	10.4	R	Clear	LSCS	2.40	7	8					-
103	Indira	21	5256	Primi	32.0	11.0	R	Clear	Normal	1.60	6	7		+	preterm/LBW	Good	-
104	Satya	24	5341	Multi	37.6	8.4	R	Clear	LSCS	3.60	7	9	Others				-
105	Poonghodi	24	5444	Primi	37.6	9.0	R	ThinMSAF	Normal	2.90	7	9		+	Observation	Good	-

106	Indira	20	5528	Primi	37.0	8.3	R	Clear	LSCS	2.80	7	9	FI				-
107	Nagavalli	21	5612	Primi	32.0	14.0	R	Clear	Normal	1.40	6	7		+	preterm /LBW	Died (Sepsis)	-
108	Maheswari	23	5725	Primi	40.0	7.4	R	ThinMSAF	LSCS	3.60	7	9	FI				-
109	Renuka	22	6014	Primi	38.2	7.3	R	Clear	LSCS	3.20	6	7	FD				-
110	Jansi	19	6126	Primi	37.4	8.3	NR	ThinMSAF	LSCS	3.30	6	6	FD	+	MAS	Died	-
111	Arul	27	6156	Multi	34.0	14.3	R	Clear	LSCS	2.00	6	7	Others	+	preterm	Good	-
112	Vimala	27	6455	Multi	37.4	13.8	R	Clear	LSCS	3.00	7	9	FI				-
113	Rajalakshmi	22	6680	Primi	37.1	13.2	R	Clear	Outlet	21.80	7	9					-
114	Rosy	22	6788	Primi	37.4	14.0	R	Clear	Normal	3.00	7	9					-
115	Chithra	24	6825	Multi	37.2	11.6	R	Clear	LSCS	3.00	7	9	Others				-
116	Gandhimathi	24	6988	Multi	37.3	12.1	R	Clear	LSCS	3.00	7	8	Others				-
117	Ramani	23	7011	Multi	37.0	11.6	R	Clear	LSCS	2.80	7	8	Others				-
118	Sudha	21	7098	Primi	34.0	10.8	R	Clear	Normal	2.40	6	7		+	preterm	Good	-
119	Manjula	22	7135	Primi	37.0	10.7	R	Clear	Normal	3.10	7	9					-
120	Jothika	20	7168	Primi	36.5	9.3	R	Clear	Normal	2.40	6	7					-
121	Nithya	27	7233	Multi	36.4	9.8	R	Clear	LSCS	2.50	7	8	Others				-
122	Saraswathy	21	7348	Primi	37.0	11.4	R	ThinMSAF	Outlet	3.00	7	8		+	Observation	Good	-
123	Sameem	24	7400	Multi	36.3	11.2	R	Clear	LSCS	2.49	7	8	FD				-
124	Arokiamary	23	7412	Multi	36.6	11.4	R	Clear	LSCS	2.49	6	7	FD				-
125	Sureka	20	7428	Primi	36.3	12.7	R	ThinMSAF	LSCS	2.49	7	9	FI				-
126	Radha	25	7532	Multi	37.0	10.8	NR	Clear	Outlet	3.40	7	9					-
127	Devi	23	7612	Primi	36.2	10.8	R	Clear	Outlet	2.50	7	8					-
128	Sudha	23	7614	Primi	34.0	10.5	R	Clear	LSCS	2.00	7	9	FD	+	preterm/LBW	Good	-
129	Katheja	22	7715	Primi	37.6	11.0	R	Clear	Normal	2.80	7	9					-
130	Aruna	27	7718	Multi	37.6	11.2	R	Clear	LSCS	2.70	7	8	FI				-
131	EmiliDevi	23	7814	Primi	37.0	9.6	R	Clear	Normal	2.75	7	9					-
132	Rajakumari	21	7847	Primi	37.3	8.0	R	Clear	Normal	2.70	7	8					-
133	Sowmiya	27	7864	Multi	37.3	9.8	R	Clear	LSCS	2.65	7	8	FI				-

134	Geethalakshmi	24	7877	Multi	37.3	9.8	R	ThinMSAF	LSCS	2.70	7	9	FI				-
135	Seetha	29	7912	Multi	37.5	9.8	R	Clear	LSCS	2.60	7	8					-
136	Anu	25	7918	Multi	37.6	15.2	R	Clear	LSCS	2.80	7	8					-
137	Prema	24	7923	Primi	37.6	14.0	R	Clear	Normal	2.70	7	9					-
138	Durga	21	7927	Primi	37.6	13.2	R	Clear	Normal	2.75	7	9					-
139	Leela	20	7954	Primi	37.0	12.0	R	Clear	Normal	2.60	7	8					-
140	Valarmathy	25	7955	Multi	37.0	11.2	NR	ThinMCAS	LSCS	2.70	7	8					-
141	Ashadevi	22	7956	Primi	37.0	9.9	R	Clear	Normal	2.80	7	9					-
142	Priya	21	7964	Primi	37.4	10.4	R	Clear	Normal	3.10	7	9					-
143	Elakiya	22	7972	Primi	37.3	9.0	R	Clear	Normal	2.40	7	8					-
144	Aswini	22	7974	Prime	36.6	9.8	R	Clear	Normal	2.30	7	8					-
145	Vinitha	22	7976	Prime	37.5	10.0	R	Clear	Normal	3.10	7	8					-
146	Manjula	22	7982	Multi	33.0	11.4	R	Clear	Normal	2.00	7	8					-
147	Thangamani	20	7983	Prime	37.2	11.4	R	Clear	Normal	3.00	7	8					-
148	Meena	21	7984	Prime	36.0	10.2	R	Clear	Normal	25.60	7	8					-
149	Mala	22		Multi	40.0	8.3	R	Clear	Normal	3.40	7	8					-
150	Kousalya	21		Prime	34.0	11.4	R	Clear	Normal	2.40	7	8		+	pretern/LBW	Good	-

NST = Non Stress test
R = Reactive
NR = Non-reactive
N = Normal
Ab = Abnormal
MSAF = Meconium Stained Amniotic fluid
FD = Fetal Distress
FI = Failed Induction
RD = Respiratory Distress
SGA = Small For Gestational Age
LBW = Low Birth Weight
NICU = Neonatal Intensive Care Unit