SCREENING FOR FETAL ANOMALIES IN 11 TO 13 WEEKS ULTRASONOGRAM - A PROSPECTIVE STUDY

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CERTIFICATE

This is to certify that the dissertation entitled "**SCREENING FOR FETAL ANOMALIES IN 11 TO 13 WEEKS ULTRASONOGRAM - PROSPECTIVE STUDY"** is the bonafide original work of **Dr. R. PADMAPRIYA** under the guidance of **Dr. K. MAHESWHARI MD., DGO.,** Associate Prof. of department of Obstetrics and Gynecology KMCH, Chennai in partial fulfillment of the requirements for MD Obstetrics and Gynecology branch II examination of the Tamil Nadu Dr. M.G.R Medical university to be the held in March 2010. The period of postgraduate study and training was from April 2008 to Feb 2010.

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LIST OF ABBREVIATIONS

1. USG	-	ULTRA SONOGRAM
2. TAS	-	TRANSABDOMINAL SCAN
3. TVS	-	TRANSVAGINAL SCAN
4. NT	-	NUCHAL TRANSLUCENCY
5. CRL	-	CROWN RUMP LENGTH
6. SURUSS	-	SERUM, URINE AND ROUTINE ULTRASOUND SCREENING STUDY
7. AJOG	-	AMERICAN JOURNAL OF OBSTETRICS AND GYNECOLOGY
8. FPR	-	FALSE POSITIVE RATE
9. BUN STUDY	-	FIRST TRIMESTER MATERNAL SERUM BIOCHEMISTRY AND FETAL NUCHAL TRANSLUCENCY SCREENING STUDY
10. FASTER	-	FIRST AND SECOND TRIMESTER EVALUATION OF RISK TRIAL

TABLE OF CONTENTS

INTRODUCTION

INTRODUCTION

Every pregnant women desire a healthy child who is free of anomalies. The incidence of major malformation in newborn is about 3-5%. Ultrasonogram plays a central role in the provision of prenatal screening and diagnosis.

Since *Professor. Ion Donald* introduced ultrasound in to obstetrics in the late 1950's vast improvement has been made in the ultrasound technology. This revolutionary technological improvements and the use of high frequency transvaginal scanning have allowed the resolution of ultrasound imaging in the first trimester to evolve to the stage where detailed early fetal development can be well visualized.

The early first trimester scan was initially introduced with the primary intention of measuring the fetal crown – rump length to achieve accurate pregnancy dating. Besides, imaging technology has also made it possible to accurately diagnose chromosomal abnormalities and structural anomalies before the second trimester.

Based on Radius Trial, the routine anomaly scan was done at 18-22 weeks but now the emphasis for screening for fetal abnormalities have been moved to the first trimester 11-14 weeks.

Until recently, attempt to detect fetal disorders during the first trimester have been confined to high risk & selected population. Evidences prove that routine early ultrasonography in screening low risk pregnancies for fetal defects is beneficial .

The early ultrasound screening has various advantages like

a) It provides reassurance to the patient and also to the obstetrician.
b) It provides information about specific abnormalities or aneuploidy.
c) It provides opportunity for prenatal invasive testing.
d) It helps in deciding for early termination.
e) It provides knowledge of the condition that may affect future pregnancies' and necessitate counseling.

The 11-13 week window of gestation age is the best to assess gestational age, measure Nuchal translucency thickness and obtain a potentially detailed anatomic survey. (Whitlow et al :1998)[1].A more thorough examination of the fetal anatomy is performed when mandated by the patients history of an abnormal finding on a screening examination.

AIM OF THE STUDY

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To assess the value of performing a detailed examination of the fetus for both structural anomalies and markers of aneuploidy as a part of routine 11 to 13 weeks ultrasound scan in a two stage screening process in the general obstetric population.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

ULTRASOUND AND ITS PRINCIPLES

Ultrasound is defined as the sound above the range of human hearing that is above the frequency of 20,000 Hz – Normal human frequency range is between 20 Hz – 20,000 Hz. Ultrasound imaging is based on the same principle involved in the sonar used by bats, ship and fishermen. When a sound wave strikes an object, it bounces back, or echo's. The transducer sends the sound waves and records the echoing waves. When the transducers is pressed against the skin, it directs small pulses of inaudible, high frequency sound wave in to the body. As the sound waves bounces off of the internal organs, fluids and tissues, the sensitive microphone in the transducer records tiny changes in the sounds pitch which are instantly measured and displayed by a computer which in turn create the real time picture on the monitor.

HISTORY OF ULTRASOUND

The History of ultrasound can be traced to *Spallanzovi.* In 1970 he experimented with bats and found that they maneuver through the air using their hearing rather then an sight. Jean – Daniel colladon in 1826 discovered sonography with an under water bell and determined

the speed of sound through water. The major step in the history of ultrasound was the invention of the transducer by Paul Langevin.

ULTRASOUND IN OBSTETRICS

Professor Ion Donald was the first person to use ultrasound as a diagnostic tool in obstetric in 1950 and was called the “Father of Obstetric ultrasound”. In 1972 the first prenatal diagnosis of a congenital anomaly by USG was made by Campbell which altered the Obstetric Management.

Ultrasound has become an integral component of Obstetric care, with the vast majority of patients having at least one ultrasound examination during pregnancy. Recent advances in obstetricUSG have increased its importance in managing pregnancy at risk for aneuploidy, structural anomalies, pre term delivery and blood flow abnormalities. First trimester USG was traditionally used to assess fetal viability and accurately date the pregnancy. More recently the introduction of screening for fetal nuchal translucency thickness has resulted in many more women having a scan in early pregnancy. Once an anomaly is detected, it allows the chance for early termination. Prenatal diagnosis not only allows termination but has profound

implication on antenatal and intrapartum management such as in-utero treatment of diaphragmatic hernia and other fetal therapy.

Prenatal diagnosis of anomaly was routinely performed in 18-22 weeks but now due to advancement in resolution in ultrasound the diagnosis of structural anomaly can be done in the first trimester. The timing of ultrasound examination depends upon the gestational age at which the organs are expected to developed. Most fetal structures are visualized at approximately 12-13 weeks and hence early detailed USG is technically feasible as a screening test for fetal structural defect.

Green and Hobbins (1988)[2] commented that with the greater resolution of modern day ultrasound a detailed fetal anatomy could be visualized with TAS in the first trimesters. Transvaginal sonogram has superior resolution over transabdominal sonogram and gives better quality images in patients with high body mass index or with retroverted uterus.

Rottem et al[3] described the detection of fetal abnormalities in the first trimester using TVS in 1989. Similarly *Quashie et al* in 1992[4] investigated visualization of fetal anatomy with increasing gestation using Transvaginal Sonography. Braithwaite et al[5] examined

fetal anatomy with both TAS and TVA between 12-13 weeks and concluded that the two scan modes were found to be complementary (1996).

Gestation age at visualization of anatomical landmark. Chitty - L, Pandya P. Prenatal Diag -1997[6]

S.No	Landmarks	Gestation age in weeks	
		TAS	TVS
1.	Cranium	11,12-13	11-12
2.	Spine	12-13	11
3.	Long Bones	12-13	10-11
4.	Feet	12-13	13
5.	Four Chamber view	12-13	12
6.	Kidney	12-13	12
7.	Bladder	12-13	13
8.	Ant. Abd. Wall	12-13	12
9.	Face	12-13	12
10.	Stomach	13	11-12

ULTRASOUND EXAMINATION AT 11-14 WEEKS

- Viability
- Determination of GA
- Identification of multiple pregnancy and determining chorionicity.

- Screening for aneuploidy based on Nuchal translucency measurement.
- Assessment of fetal gross anatomy.

DETECTION OF VIABLE PREGNANCY

Ultrasound examination at 11-14 weeks helps to diagnosis early pregnancy failure including missed abortion and anembryonic pregnancies.

DETERMINATION OF GESTATIONAL AGE USING CRL

Ultrasound helps in dating the pregnancy as the CRL up to 14 weeks is the most accurate for determining the gestational age. When gestational age is confirmed the induction rate is reduced by about 40%

DETECTION OF MULTIPLE PREGNANCY AND THE CHORIONICITY

Ultrasound plays a major role in detecting the chorionicity in multiple pregnancy and the ideal time is 11-13 weeks scan. The Lambda sign which is due to chorionicity is a triangular tissue projection at the base of the intertwin membrane *Sepulveda W.et al* [7]. It is actually the extension of two chorion layers within the intertwin membrane that is present only in dichorionic pregnancy.

SCREENING FOR ANEUPLOIDIES

Chromosomal Abnormalities occurs in approximately 0.9% of newborns and it is estimated that at least 10 to 15% of conceptions are chromosomally abnormal and about 95% of them are lost before term. Chromosomal abnormality can either be numeric or structural. Aneuploidy which refers to the presence of abnormal number of chromosomes is the most common clinically significant type of human chromosomal abnormality, and it occurs in 3% to 4% of recognized pregnancies. The risk of trisomy increases with maternal age and monosomy is seen less frequently than trisomy. The most common aneuploidy at birth is Down's syndrome (Trisomy 21)

INCIDENCE

Trisomy 21 - 1 in 700 live births

Trisomy 18 - 1 in 8000 live births

Trisomy 13 - 1in 20,000 live births

Monosomy 45 x, Turner syndrome – 1 in 3000 live births

Sonography plays a major role in detecting chromosomal abnormalises abnormality in 11 – 13 week by using

1. Nuchal translucency
2. Nasal bone Sonography
3. Ductus venons Sonography

TRISOMY 21 / DOWN SYNDROME

J.Langdon Down, in 1866[8] first made the observation of a subgroup of patients with particular facial features and mental handicap in the outpatients department of the London Hospital

Lejeune and Jacobs et al, in 1959, identified Trisomy 21 was the cause of Downs syndrome and the first chromosomal analysis from amniotic fluid was made in 1966. Antenatal screening for Down Syndrome was first performed in the 1970's using advanced maternal age or as previous history of aneuploidy. In the 1980's the association of Down syndrome with abnormal levels of certain specific serum markers were discovered and Triple Test came into role.

The association between Downs syndrome and increased maternal age was noted in 1909 by Shuttle worth. In the past, invasive prenatal diagnosis for Downs syndrome with Amniocentesis and chorionic villous sampling was being offered only to women of advanced maternal age (older than 35 years of age at delivery) or those who previously had an affected child. Subsequently, invasive diagnosis was offered to women younger than 35 years of age who had abnormal second trimester multiple marker serum screening and also to those with abnormal second trimester sonographic sign, so

called soft markers of Down's syndrome. But all the invasive prenatal diagnosis like amniocentesis and chorionic villous sampling had several limitation because it was associated with a risk of fetus loss of 1%

Regarding the first observation made by *J.L.Down* in 1866[8] that the common characteristic of patients with trisomy 21 was poor skin elasticity, which gave the appearance that the skin was too large, it was realized in 1990's that the excess skin could be visualized by Sonography as increased Nuchal translucency in the third month of intrauterine life.

Szabo and Gallen [9,10] the first to report the relationship between accumulated fetal nuchal fluid and fetal abnormalities. In 1992, Nicolaides et al introduced the new term Nuchal translucency which was defined as the thickness of translucent space between the skin and soft tissue over lying fetal spine, measured in millimeters and tenth of a millimeter via ultrasound. *Nicolaides et al* (1992)[11] had gained wide spread use of measurement of Nuchal translucency in conjunction with maternal serum markers in the detection of aneuploidy.

Wapner and colleague (2003)[12] detected 85% of case of Down's syndrome with 9% false positive rate using Nuchal Translucency

measurement along with serum chorionic gonadotropin and pregnancy associated plasma protein *A. Malone and colleagues (2003)* [13] reported a multicenter trial in 33,557 pregnant women who underwent first trimester and second trimester aneuploidy screening and concluded 85% of Down syndrome detected for 7.6% false positive rate.

Nuchal Translucency measurement at 11 - 13 weeks, combined with maternal age, provides an effective method of screening for trisomy 21. *Pandya et al in 1995*[15] concluded a detection rate of 75% for trisomy 21 when the cutoff rate of NT was >2.5 mm at 10-14 weeks. Numerous studies have been done on NT measurement for detection of chromosomal abnormality. The largest study of this form of screening was performed by the Fetal Medicine foundation based in London[14]. The study was done in unselected patient and the overall detection rate for Down syndrome was 77 % for a 5% FPR.

SURUSS Trial-Serum Urine And Routine Ultrasound Screening study detected 63% of Dows syndrome for 5% FPR. Similarly the BUN study and FASTER trial also revealed the strong relationship between increased Nuchal translucency and chromosomal abnormalities.

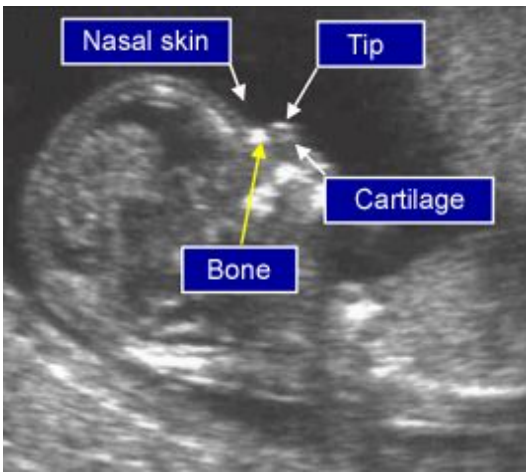
THE PATHOPHYSIOLOGY OF NT

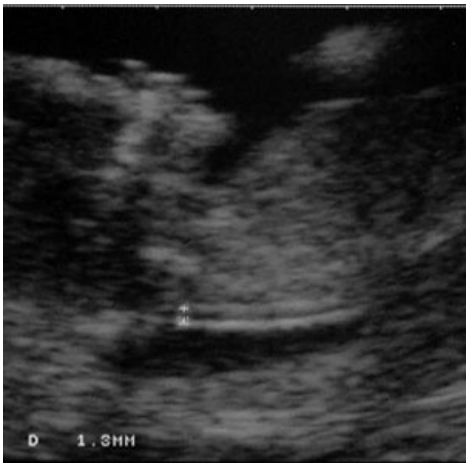
The etiology of increased Nuchal translucency is variable, but it is commonly believed to be due to fluid accumulation in the Nuchal region because of aortic isthmus narrowing or other fetal cardiovascular defects, abnormalities in the extracellular matrix or abnormal or delayed development of lymphatic system.

Measurement of nuchal translucency

Nuchal translucency can be measured successfully by abdominal ultrasound examination in about 95% of cases, in the others, it is necessary to perform Transvaginal Sonography.

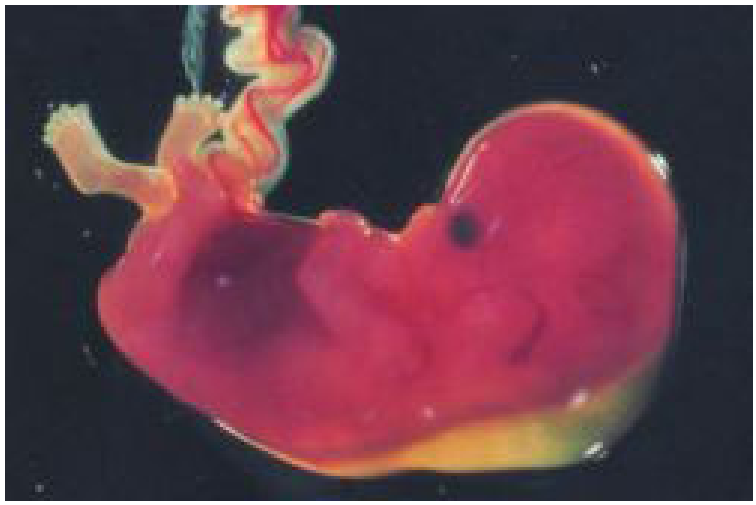
ULTRASONOGRAPHIC PICTURES





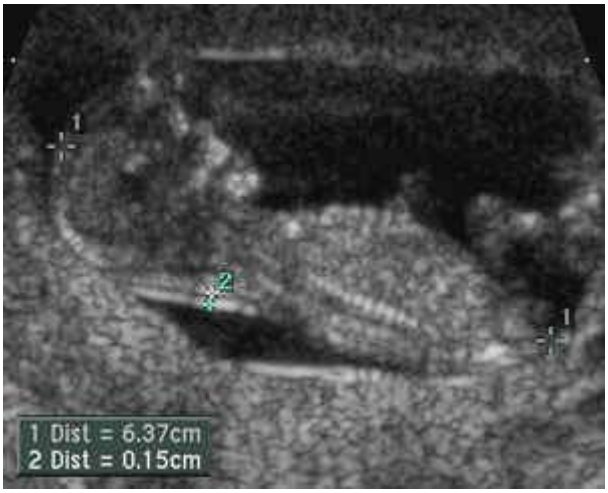
Nuchal Translucency Measurement

3D IMAGE OF NUCHAL FLUID



Criteria for MEASUREMENT

- Strict saggital view appropriate for Crown – rump length
- The minimum crown – rump length should be 45 mm and the maximum 84 mm.
- The optimum gestational age for measurement of fetal nuchal translucency is 11 to 13 + 6 weeks.
- Neutral position of the fetus head.
- Appropriate magnification (>70% image)
- Away from amnion.
- The maximum thickness of the subcutaneous translucency between the skin and the soft tissue overlying at cervical spine should be measured by placing the caliper on the inner borders of the above.



NEUTRAL POSITION OF FETUS AND MID SAGGITAL VIEW

(11to 13.6wks scan)

- More than one measurement must be taken and the maximum one should be recorded. Should be performed by trained sonographer as per Fetal Medicine Foundation guidelines.

Based on various studies conducted in screening for aneuploidy using Nuchal translucency measurement the least cutoff value for predicting abnormalities is a NT measurement of $\geq$ 3mm.

Whenever a screening test in performed, the background risk is multiplied by the factor to calculate a new risk[16]. Therefore fetus with a given CRL, each NT measurement is a likelihood ratio that is multiplied by the background risk to provide a new risk. A woman's

risk of having a fetus with trisomy 21 is higher if the NT measurement is large and lower if is small.

NUCHAL TRANSLUCENCY SCREENING FOR OTHER ANEUPLOIDY.[17,18,19,20]

Based on prenatal diagnosis and neonatal ascertainment the detection rate for trisomy 18 was 84%, Turner's syndrome 80% and 63% for Triploidy because the true prevalence of the condition in the first trimester in uncertain and most affected fetus spontaneously die in utero, true detection rate for these cases are difficult to calculate.

INCREASED NT WITH NORMAL KARYOTYPE[21]

Souka and colleagues have studied that chromosomally normal fetus with increased NT > 3.5mm and concluded that 86% had no structural defects. If the NT thickness exceed 6.5mm then there in one in three chance that the pregnancy will end with a live birth of a infant with no major defects.

INCREASED NT WITH OTHER MALFORMATIONS[22]

An abnormal NT with normal karyotype is mostly associated with cardiac abnormalities.[23] Hence fetal echo is to performed at 16 weeks and a second echo at 18 – 20 weeks. Diaphragmatic hernia is

associated with 37% of fetus with increased NT and this reflects venous congestion in the head and neck as a results of mediastinal shift or compression and impaired venous return.[24]

Exomphalos is 10 times higher in fetus with normal karyotype and increased NT. Increased NT in twin gestation implies Twin to Twin transfusion syndrome. Enlarged Nuchal translucency and cystic hygroma in the first trimesters have an extremely high risk of fetal aneuploidy or other adverse pregnancy outcomes. Septated cystic hygroma are shown to have 50% chance of being associated with fetal aneuploidy, mostly trisomy 21.

According to FASTER trial whenever a simple Nuchal translucency measurement of 3.00mm or greater is noted, CVS should be offered immediately because of a minimum risk of aneuploidy of 1 in 6.

NASAL BONE SONOGRAPHY IN THE FIRST TRIMESTER

According to a study conducted by *Ceciro et al*, [25]701, Fetuses with increased NT were also evaluated for the presence or absence of nasal bond during first trimester ultrasonography. The fetal nasal bone would not be visualized in 75% of Down Syndrome fetus. It was also

found that the absence of fetal nasal bone was not related to NT thickness and therefore the nasal bones screening has to be included in ultrasound screening modality with sensitivity of 85% with 1% FPR.

DETECTION OF FETAL ANOMALIES

The early pregnancy scan was initially introduced with the primary intention of measuring the CRL to achieve accurate dating. However, with improvement in the resolution of the scan machine it is now possible to describe the normal anatomy of the fetus and to diagnose or suspect the presence of a wide range of fetal defects in the first trimesters of pregnancy.

Protocol for first trimesters scan in a low risk population – *Carol et al,*

ANATOMIC SURVEY

CNS

Obtain BPD view

Normal Skull outline

Presence of falx – cerebri

2 choroid plexus

Face

Profile, orbits and nose

Neck

Measure NT

Heart

FHR

Four chamber view

Thorax

Location of stomach

GIT

Stomach

Physiological hernia up to 11 weeks 5 days

***Bladder* < 7MM**

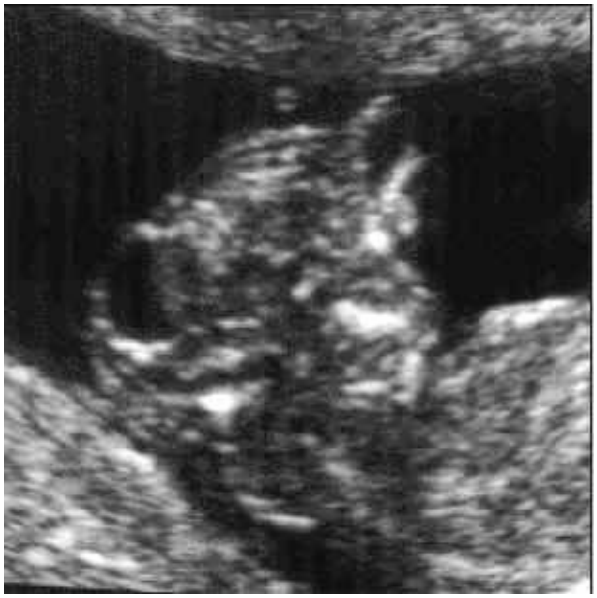
Musculoskeletal system

4 limbs, 2 hands and 2 feet

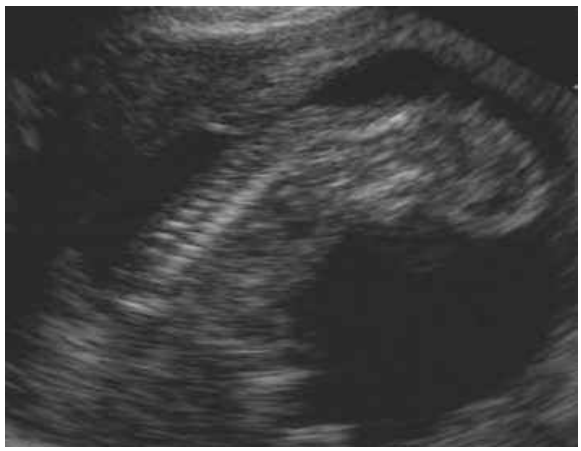
CENTRAL NERVOUS SYSTEM DEFECTS

In normal human fetuses, there is histological evidence that the onset of ossification of the cranial vault is at 10 weeks of gestation

and that, ultrasonographically by 11 weeks, there is hyperechogenicity of the skull in comparison to the underlying tissue. Ultrasound report have demonstrated that in the human there is progression from acrania to exencephaly and finally anencephaly. In the first trimester, the path gnomonic features is acrania, the brain being either entirely normal or at varying degrees of distortion and disruption.



ENCEPHALOCELE



ACRANIA

ACRANIA, ANENCEPHALY AND EXCENCEPHALY

Schmidt and Kubli in 1982[26] demonstrated anencephaly at 13 weeks. *Rotten et al* in 1989[27] reported a fetus at 9 weeks with an abnormal shape of the cephalic pole and cervical spine, and at 11 weeks, the diagnosis of anencephaly and open cervical spina bifida was made.

In first trimester anencephalic fetus neural tissue can be seen but the cranium absent, giving rise to *"Mickey Mouse"* appearance, which is different from "*Frog Eyes"* seen in second trimesters.

Kennedy et al (1990)[28] described a case of acrania at 10 weeks in which the brain was of normal volume but appeared echogenic and disorganized, at 19 weeks the fragmented and degenerative brain was visualized. *Bronshtein and Ornoy et al*,[29] reported a case with no abnormal findings at 9 and 13 weeks there was anencephaly at 14 weeks. The findings from various screening studies clearly demonstrate that anencephaly can be reliably diagnosed at the routine 11- 13 weeks scan, provided the sonographic features for this condition are specially searched for.

ENCEPHALOCELE

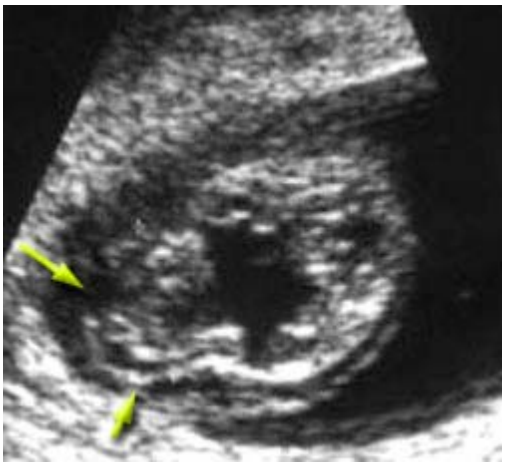
This is a cranial defect with protrusion of meninges (Meningocele) and brain (Encephalocele). In about 75% of cases, the lesion is occipital but alternative sites include the frontoethmoidal and parietal region. It is often associated with microcephaly, hydrocephaly, spinabifida and meckel –gurber syndrome.

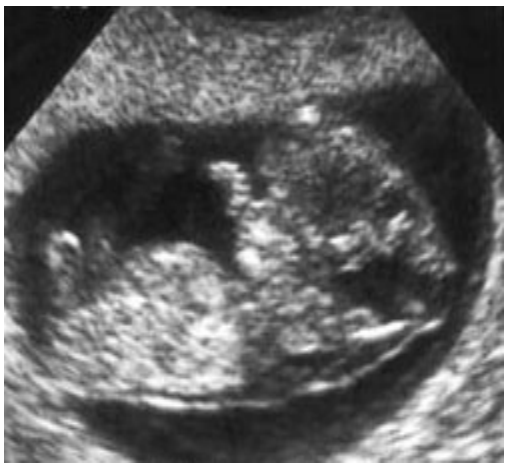
Meckel - Gruber syndrome.

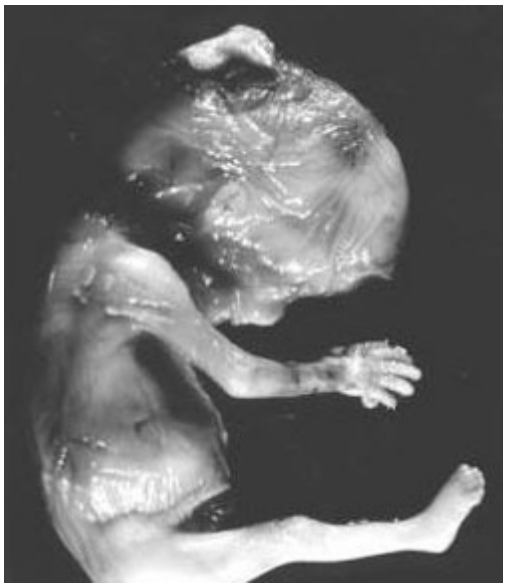
Bronshtein and Zimmer (1991)[30] described a case of occipital encephalocele that was seen as empty occipital sac at 13 weeks and later with brain tissue inside the sac at 14 weeks. Van *Zalen – Sprock et al (1992)*[31] described a fetus at 11 weeks with two translucent areas in the occipital region.

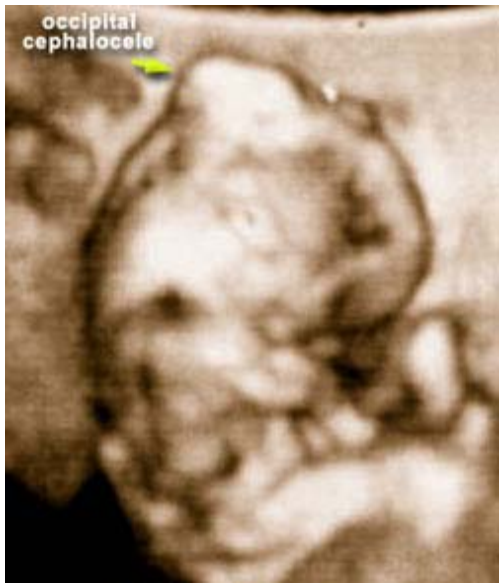
MECKEL – GRUBER SYNDROME

USG Pictures









Meckel – Gruber syndrome, is a lethal autosomal recessive condition characterized by the triad of encephalocele, bilateral polycystic kidney and polydactyly. *Pachi et al,*[32] described the sonographic features of this syndrome at 13 weeks gestation with occipital bony defects accompanied by encephalocele with enlarged kidneys. Similarly *Sepulveda et al* [33] and *Van Zalen – Sprock et al*, while examining High risk pregnancies were able to correctly identify the Meckel –Gruber syndrome at 11-14 weeks scan.

The diagnosis of this syndrome is easier at 11-14 weeks when the amniotic fluid is normal than during the second trimester when the presence of the associated oliogohydramnios could easily cause encephalocele and certain polydactyly to be missed. Additionally, at 11-14 weeks, the fingers are easier to examine because they are

invariably extended, whereas in the second trimesters the hands are often clenched.

HYDROCEPHALUS

Congenital hydrocephalus with a prevalence of about 2 per 1000 are most probably due to a combination of genetic and environmental factors. Antenatal sonographic diagnosis is based on the demonstration of dilated lateral cerebral ventricles. In normal fetuses, the outline of the lateral ventricles, the echogenic choroid plexus and the mid-line echo are visible by ultrasound from 9 weeks, at 10 – 11 weeks, the third and fourth ventricles become visible and at 12 weeks, the cerebellum and thalamus can be seen. Ventriculomegaly usually develops after the 14^{th} weeks of gestation. Hence the detection of hydrocephalus is mostly done in second trimester ultrasound.

Lin et al (1992)[34] reported a 12 week fetus with large head, small hemisphere and a fluid – filled intracranial cavity with no midline echo and was diagnosed to be a lethal sporadic condition called hydranencephally.

HOLOPROSENCEPHALY

It is a spectrum of cerebral abnormalities resulting from incomplete cleavage of the fore brain. There are three types according to the degree of forebrain cleavage. The alobar type, which is most severe, is characterized by a monoventricular cavity and fusion of the thalami. In the semilobar type, there is partial segmentation of the ventricles and cerebral hemispheres posteriorly with incomplete fusion of the thalami. In lobar holoprosencephaly, there is normal separation of the ventricles and thalami but absence of the septum pellucidum. The first two type are often accompanied by facial abnormalities.

Toth et al (1986)[35] observed a floating membranous structure in place of skull at 11 weeks scan. Bronshtein and *Wiener(1991)*[36] described a case of alobar holoprosencephaly during routine ultrasound at 14 weeks.

Gonzalez - Gomez et al (1992)[37] described a 10 weeks fetus with single ventricular cavity, absence of the orbits and mid-facial cleft.

Snijders et al (1999) reported on the sonographic features of 46 trisomy – 13 fetus at 10 – 14 weeks gestation. In 24% there was

holoprosencephaly and hence the most common related chromosomal abnormality was found to be trisomy -13.

INIENCEPHALY

This is a rare malformation of unknown etiology, characterized by cervical dysraphism and occipital (inion) defect with or without an encephalocele.

Sherer et al (1996)[38] reported the diagnosis of iniencephaly in a 13 week fetus with acrania, persistently hyper extended head and spinal dysraphism.

SPINA BIFIDA

In Spina bifida, there is failure of closure of the neural tube, which normally occurs by the 6th week of gestation. In the spine of normal fetuses, there are three ossification centers, two pedicles and the spinal body and these are present from the 10th week gestation, allowing ultrasonographic visualization of the neural canal from this gestation.

Braithwaite et al (1996) reported successful examination of the vertebra and overlying skin in both the transverse and coronal planes at 12-13 weeks of gestation. In 1980's the main method of screening

for open spina bifida was by maternal serum α fetoprotein at around 16 weeks of gestation and the method of diagnosis was amniocentesis and measurement of amniotic fluid α fetoprotein and acelylcholinesterase. Now the observation of scalloping of the frontal bones (the lemon sign) and caudal displacement of the cerebellum (the banana sign) associated with spina bifida has led to the replacement of biochemical assessment with ultrasonography, both for screening and for diagnosis of this abnormality.

Blumen field et al (1993)[39], described the evaluation of the cranial and cerebellar sign of spina bifida that was scanned at 10,12 and 15 weeks of gestation. Similarly Bernard and colleagues reported the diagnosis of spina bifida in a 12 weeds fetus with narrowing of the frontal bones and flattening of the occiput. However, the sonographic finding of spina bifida is difficult before 14 weeks as the prevalence of these signs at the 11-14 week scan remains to be determined.

CARDIAC DEFECTS

Abnormalities of the heart and great arteries are the most common congenital defects and the birth prevalence is 5 – 10 per 1000. In general, about half of the cardiac defects are either lethal or require surgery and half are asymptomatic. The first two groups are

referred to as major defects and specialist echocardiography are done at around 20 weeks. Currently screening is based on examination of the four – chamber view at the 20 weeks scan and this examination of the four chamber view of the heart can now be carried out at the 11 - 14 weeks scan.

Braithwaite et al has proved that at 12 -13 weeks, the four – chamber view can be examined successfully by TAS in 76% of the cases and by TVS in 95%.

Dolkart and Reimers in 1991[40] reported that the earliest defined cardiac structures visible were the mitral and tricuspid valves followed by the aortic and pulmonary valves at 12 weeks.

There are several case report of sonographic diagnosis of cardiac defects at 11 - 14 weeks of gestation. In, *1999, Hyett et al* in a large population based cohort study detected that measurement of Nuchal translucency thickness at 11 – 14 weeks, constitutes the most effective method of screening for cardiac defects. In patients with increased Nuchal translucency it is now possible to undertake detailed cardiac scanning in early pregnancy. The prevalence of major cardiac defects increased with increased NT thickness, from 5.4 per 100 with and NT measurement of 2.5 to 3.9mm to 233 per 1000 with an NT

measurement of greater than 5.5mm. The sensitivity of NT varies from 15% to 5.6% for the diagnosis of major cardiac defects.

ABDOMINAL WALL DEFECTS

Sonographically, the stomach is identified as a sonolucent cystic structure in the upper left quadrant of the abdomen. It is first visualized at 8-9 weeks and it is seen in all cases by 12 – 13 weeks. At 8 -10 weeks, all fetus demonstrate herniation of the midgut that is visualized as a hyperechogenic mass in the base of the umbilical cord and the retraction in to the abdominal cavity occurs at 10 -12 weeks and it is completed by 11 weeks and 5 days.

EXOMPHALOS

Prenatal diagnosis by ultrasound is based on the demonstration of the mid – line anterior abdominal wall defect, the herniated sac with its visceral contents and the umbilical cord insertion at the apex of the sac. Fetal exomphalos is associated with chromosomal defect, usually trisomy 18. *Brown et al (1989)* reported the diagnosis of exomphalos containing liver at 10 weeks.

Van Zalen – Sprock et al (1997)[41] reported the findings of 14 cases with exomphalos diagnosed at 11 -14 weeks of gestation. In eight cases, there was increased Nuchal translucency thickness (3.5 –

10mm) and seven of these had chromosomal abnormalities, mainly trisomy – 18. The prevalence of exomphalos and the associated risk for chromosomal defects increases with maternal age and decreases with gestational age.

GASTROSCHISIS[42]

Evisceration of the intestine which occurs through a small abdominal wall defect located just lateral and usually to the right of an intact umbilical cord. There is sparsity of reports on first trimester diagnosis.

URINARY TRACT DEFECTS

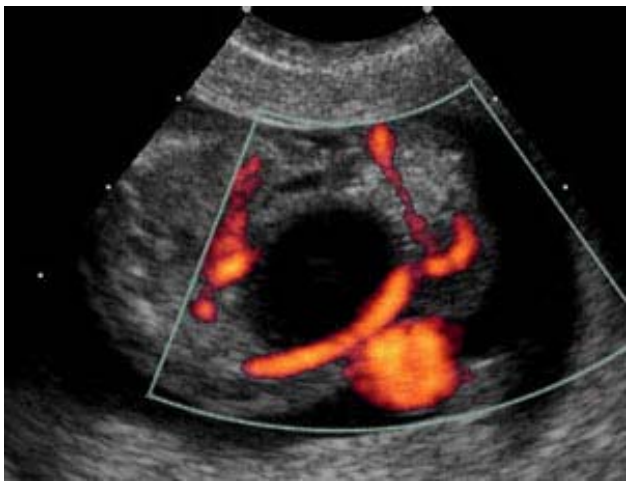
The fetal kidney and adrenals can first be visualized by TAS at 9 weeks and they are seen in all cases from 12 weeks. The fetal bladder can be visualized in about 80% of fetuses at 11 weeks and in more than 90% by 13 weeks. AT 12 -13 weeks, the fetal kidneys can be visualized in 99% of the cases.

MEGACYSTIS

Fetal megacystis is defined as a bladder with a longitudinal diameter of 7mm or more at 10 -14 weeks gestation. *Bulic et al*, (1987)[43] demonstrated megacystis with oligohydramnios at 11 -14

weeks scan and the pathological examination after termination demonstrated urethral atresia's, severe megacystis but normal kidney.

Drugan et al (1989)[44] reported a 12 week fetus with megacystis with normal kidneys and oligohydramnios and vesicoamniotic shunting was carried out and the pregnancy continued and delivered normally. In a recent study of 145 fetuses with megacystis, 25% had a



MEGACYSTIS

chromosomal abnormality, mainly trisomy 13 and 18 . In 75% of fetus with chromosomal abnormalities, NT thickness was also increased, possibly as a result of compression. The extent to which first trimesters diagnosis of megacystis and vesico – amniotic shunting could prevent the subsequent development of renal damage remains to be determined.

BILATERAL RENAL AGENESIS

Bronshtein et al[45], reported the prenatal diagnosis of bilateral renal agenesis at 14 weeks of gestation but it is usually diagnosed in the second trimester of pregnancy by the finding of a hydramnios, absence of urinary bladder and failure to identify the fetal kidneys.

SKELETAL DEFECTS

Limb buds are first seen by ultrasound at about the 8th week of gestation and the length of the humerus, radius/ulna, femur and tibia/fibula are similar at 11 -14 weeks[46].

Skeletal dysplasia are found in 1 in 4000 births. About 25% of the affected fetuses are still born and about 30% die in the neonatal period. The most common dysplasia are thanatotrophic dysplasia, osteogensis imperfecta and achondroplasia. Several cases reports have described skeletal defects in the first trimester of pregnancy and they are usually associated with increased Nuchal translucency.

MATERIAL AND METHODS

MATERIAL AND METHODS

The centre of study was at Department of Obstetric and Gynecology in Kilpauk Medical College.

Study Design : Prospective Study

Period of Study : April 2008 to December 2009

Selection of Patients

300 antenatal mothers who attended our Antenatal clinic at the outpatient department.

INCLUSION CRITERIA

Pregnant women at 11-13.6 weeks GA

- H/o previous anomalies
- H/o anomalies in the family
- H/o diabetes
- H/o Anticonvulsants medication
- H/o. radiation exposure

EXCLUSION CRITERIA

1. GA <11 Weeks
2. GA >14 Weeks
3. Multiple gestation

STUDY DESIGN

Ultrasound screening was performed by experienced radiologist transabdominally using 2 to 6MHZ curvilinear transducer and VITUS Ultrasound machine. Whenever visualization of fetal structure was suboptimal or a structural abnormality was suspected during transabdominal scan, transvaginal scan was always performed.

Ultrasound screening was performed at 11-13.6 Weeks in all 300 pregnant women. For those who had normal scans a follow up scan was done at 18-22 wks for confirmation. Fetal viability was examined and crown rump length was measured.

Evaluation of fetal anatomy was done according to the following check list.

1. Skull and brain
2. Face (Facial Profile, Nasal Bone and orbits)
3. Neck (Nuchal translucency measurement, presence of Cystic hygroma)
4. Spine (Examination of overlying skin and neural tube in longitudinal and transverse planes)
5. Heart (Four chamber view, three vessel view, heart rhythm)
6. Stomach (Its existence in left upper abdomen)

7. Abdominal wall defect
8. Kidney (existence, size, and shape)
9. Urinary bladder (existence, size, and shape)
10. Extremities (existence, size, and shape)

Along with the evaluation of the anatomy, the Nuchal translucency measurement was done according to the guidelines established by the Fetal Medicine Foundation, in fetuses with CRL between 45mm and 84mm at 11 to 13.6 weeks gestational age.

The cut off value of NT measurement was taken as $\geq$ 3 mm. When the NT measurement was $\geq$ 3 mm it was considered to the abnormal and further confirmatory test was combined. The following confirmatory that were offered are, first trimester serum markers (free βhCG, PAPP-A), chorionic villous sampling, amniocentesis and triple screening or quadruple screening and it was left to the patients choice. Women were fully counselled before their ultrasound examination and written informed consent was obtained. Based on the anomalies detected, the patients were counselled regarding termination or continuation of pregnancy. All the patients were followed up till delivery.

Pregnancy outcome was obtained from our maternity unit or the patients themselves. The number of abnormalities that were detected at 11-13 weeks and at 20 weeks were analyzed.

STATISTICAL ANALYSIS

The test statistics used were chi-square, ‘t’ test and binary logistic regression.

RESULTS AND ANALYSIS

RESULTS AND ANALYSIS

During the one year period of 2008-2009. 300 pregnant women who attended our antenatal clinic were enrolled for evaluation for fetal structural and chromosomal abnormalities between 11 and 13.6 weeks of gestational ages. Out of the 300 antenatal mothers who had ultrasonogram, 16 patients were excluded from the study because 11 patients were lost to follow up and not seen during the second trimester, 3 of them had findings suggestive of missed abortion at the time of the 11-13 weeks scan and 2 of them had miscarriages around 16 weeks of gestation.

The remaining 284 antenatal mothers, underwent the second trimester scan and were analysed based on the distribution of age, gravida, risk factors, no of anomalies detected and the outcome of their pregnancies.

Mean Age In The Study Group

Study group	No	Mean	Std. deviation
Normal	275	24.1418	3.98147
Anomalies	9	23.5556	2.12786

P= 0.661

The mean age of the participants was 24 yrs among the normal pregnancies and 23 yrs among the anomalous pregnancies

Mean Gestational Age:

Total	Mean (wks)	Std. deviation
284	12.4 days	0.66102

The mean examination time of the early scan was 12wks and 4 days

Mean CRL in cm

Total	Mean (cm)	Std. deviation
284	6.1	3.60364

The mean crown rump length measurement during the 11-13.6 wks scan was 6.1cm.

AGE GROUP DISTRIBUTION

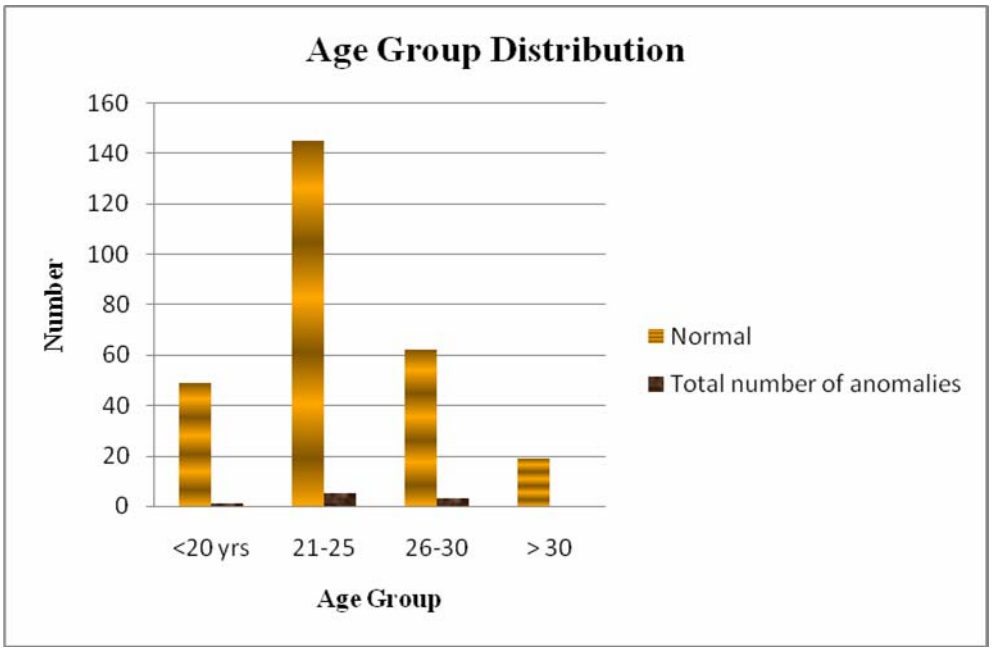
Table :1

Age group	Normal	Anomalies	Total
< 20 yrs	49 17.3%	1 0.4%	50 17.6%
21-25yrs.	145 51.1%	5 51.1%	150 52.8%
26-30yrs	62 21.8%	3 1.1%	65 22.9%
>30yrs	19 6.7%	0 .	19 6.7%
Total	275 96.8%	9 3.2%	284 100%

P= 0.729 not significant

Most of the antenatal mothers in the study group was in the age group of 21-25yrs. (52.8%).

Among the anomalies detected, about 5of them were between 21-25yrs of age (55.6%).



GRAVIDA DISTRIBUTION

Table :2

Gravida	No. of Normal	No. of Anomalies	Total
Primi	128 45%	6 2.1%	134 47.2%
Multi	147 51.8%	3 1.1%	150 52.8%
Total	275 96.8%	9 3.2%	284 100%

P=0.234 not significant

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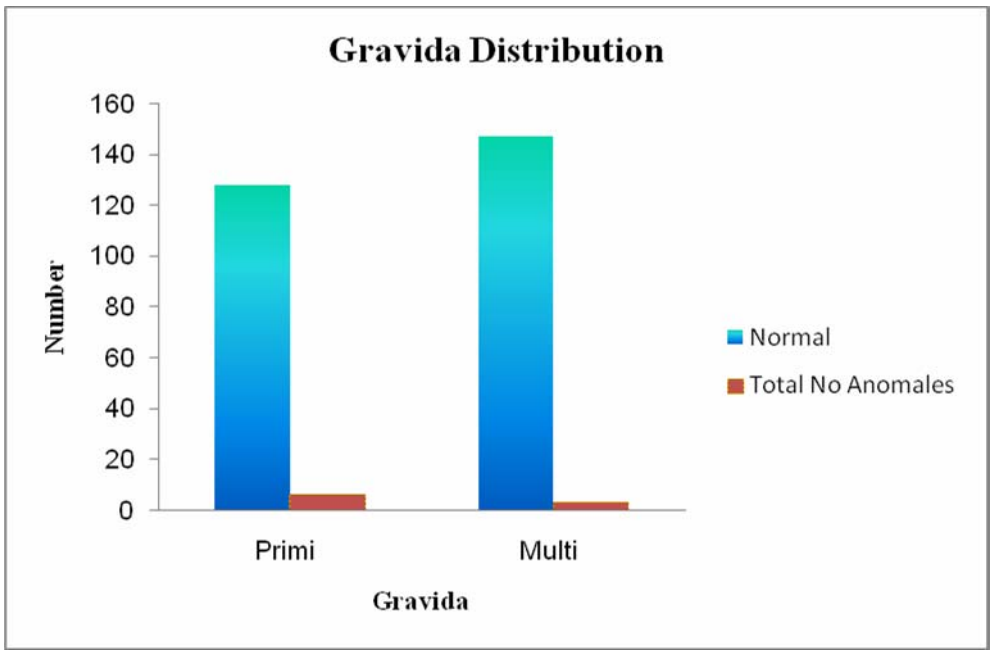
Primigradvida accounted for 47.2% in the study group and multigravida were 52.8%. The anomalies were more among the primi gravida.



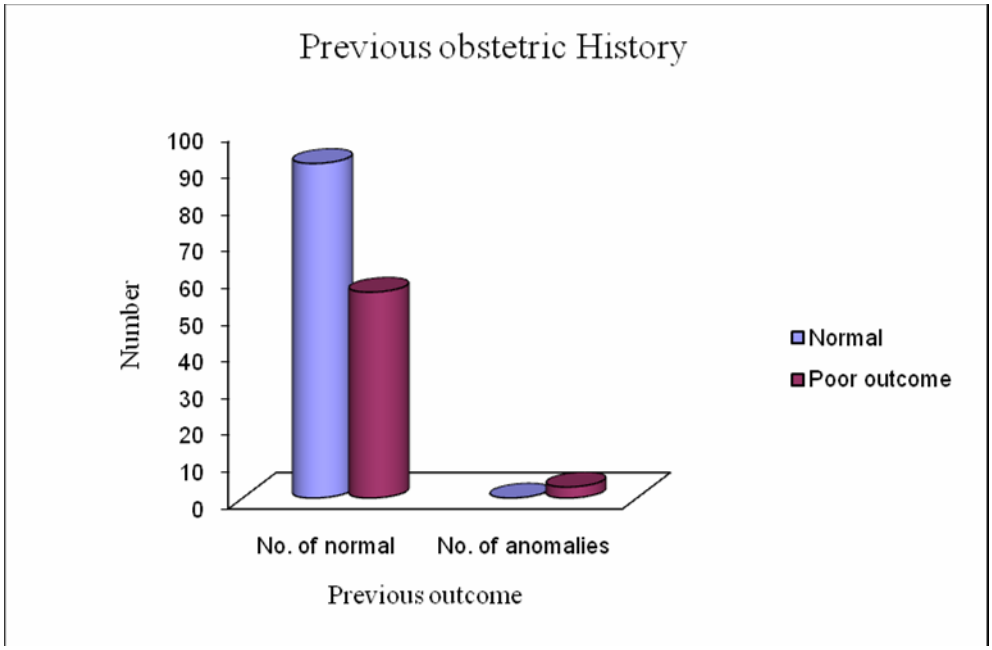
PREVIOUS OBSTETRIC HISTORY AMONG MULTIGRAVIDA

Table :3

Previous obstetric outcome	No. of normal	No. of anomalies	Total
Normal	91	0	91
Poor obstetric out come	56	3	59
Total	147	3	150

P=0.109 not significant

Among the multigravida 59 antenatal mothers had poor obstetric outcome. The mothers of the three anomalous fetuses had previous poor obstetric outcome.



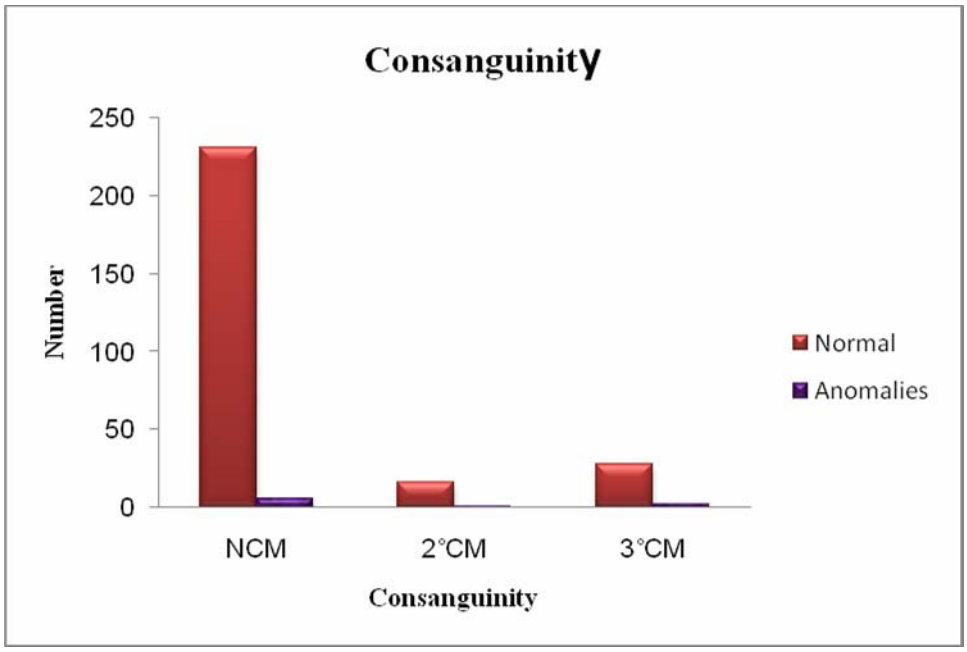
CONSANGUINITY

Table :4

Consanguinity	Normal	Anomalies	Total
NCM	231	6	237 (83.5%)
IIº CM	16	1	17 (6.0%)
IIIº CM	28	2	30 (10.6%)
Total	275	9	284 (100%)

P=0.383 not significant

237 (83.5%) antenatal mothers had non-consanguineous marriage 17 (6.0%) had second degree consanguineous marriage and 30 (10.6%) had third degree consanguineous marriage. Among the 9 antenatal mothers with anomalous fetuses 6 (66.7%) were NCM, one (11.1%) was of second degree and 2 (22.2%) were of third degree consanguinity.



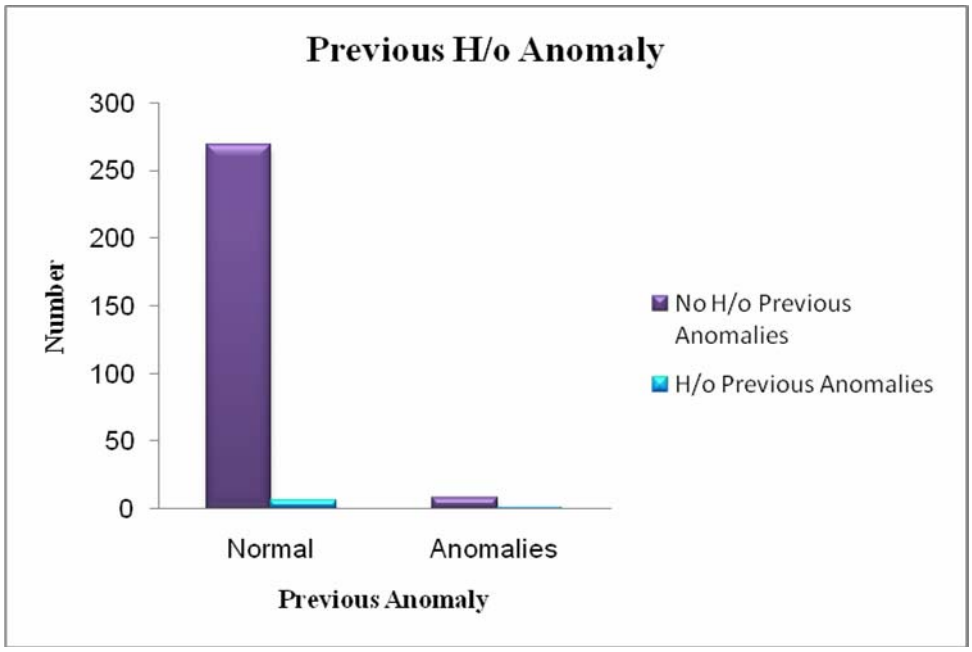
PREVIOUS H/O ANOMALY

Table :5

Study group	Normal	Anomalies	Total
H/o previous anomalies	6	1	7 (2.5%)
No H/o previous anomalies	269	8	277 (97.5%)
Total	275	9	284 (100%)

P= 0.204 not significant

Among the nine anomalies detected only one was a recurrent anomaly .



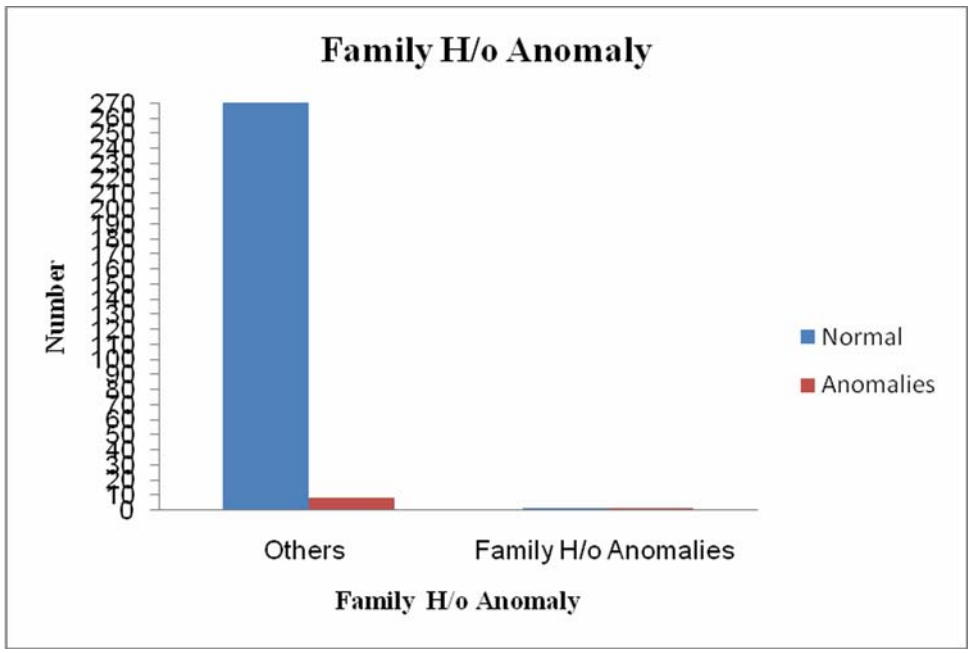
FAMILY H/O ANOMALY

Table :6

Study group	Normal	Anomalies	Total
Family H/o anomalies	1	1	2 (0.7%)
Others	274	8	282 (99.3%)

P=0.000 significant

Of the 9 anomalies there was family History of similar anomaly in one fetus



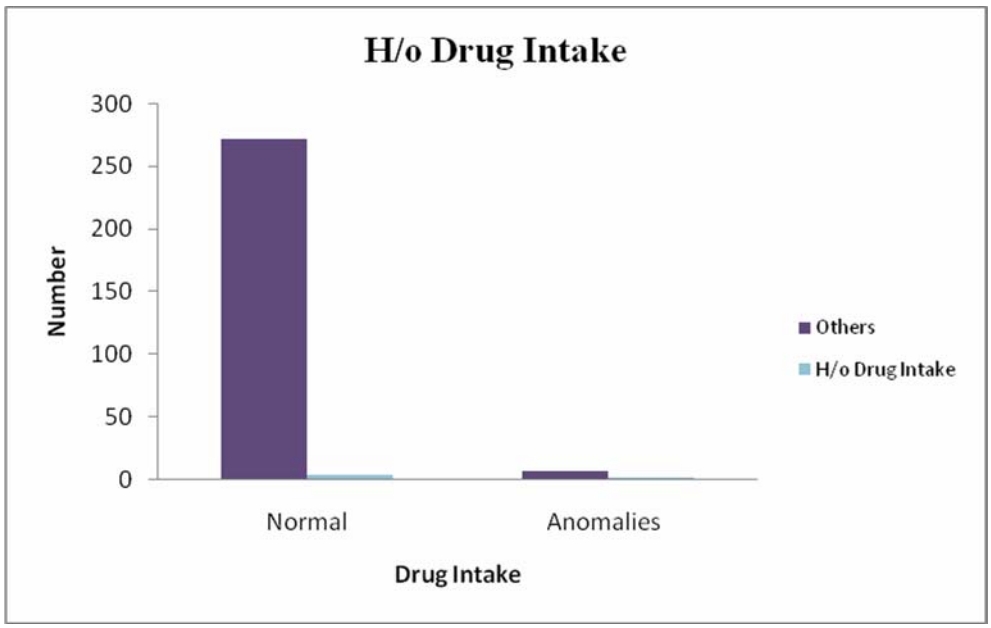
H/O DRUG INTAKE

Table: 7

Study group	Normal	Anomalies	Total
H/o drug intake	4	2	6 (2.1%)
Others	271	7	278 (97.9%)

P=0.000 significant

of the 9 anomalies two of the mother gave a history of drug . in take in the first trimester and carried fetuses with cleft lip and cleft palate and the other had an anencephalic fetus. The particular drug was not known in the case of cleft lip. The patient with anencephaly baby had H/o antiepileptic drug intake.



H/O MEDICAL DISORDER

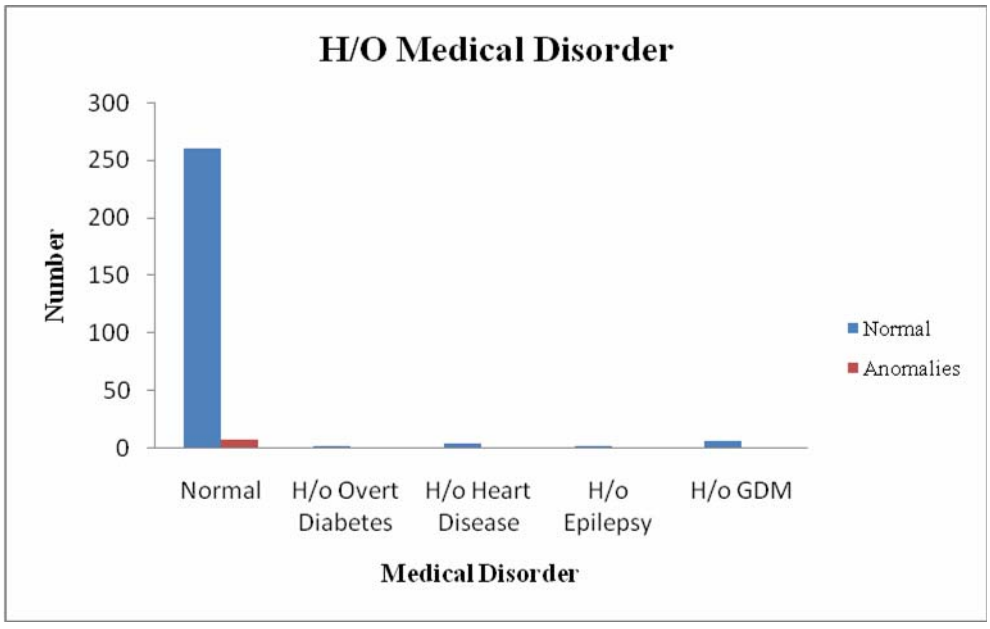
Table: 8

Medical disorder	Normal	Anomalies
Overt diabetes	5	0
Heart disease	4	0
Epilepsy	3	1
GDM	8	0

Mothers with out medical disorders	255	8

P= 0.053 not significant

There was one anomalous fetus among the patients with epileptic disorder who was on anticonvulsant drug.

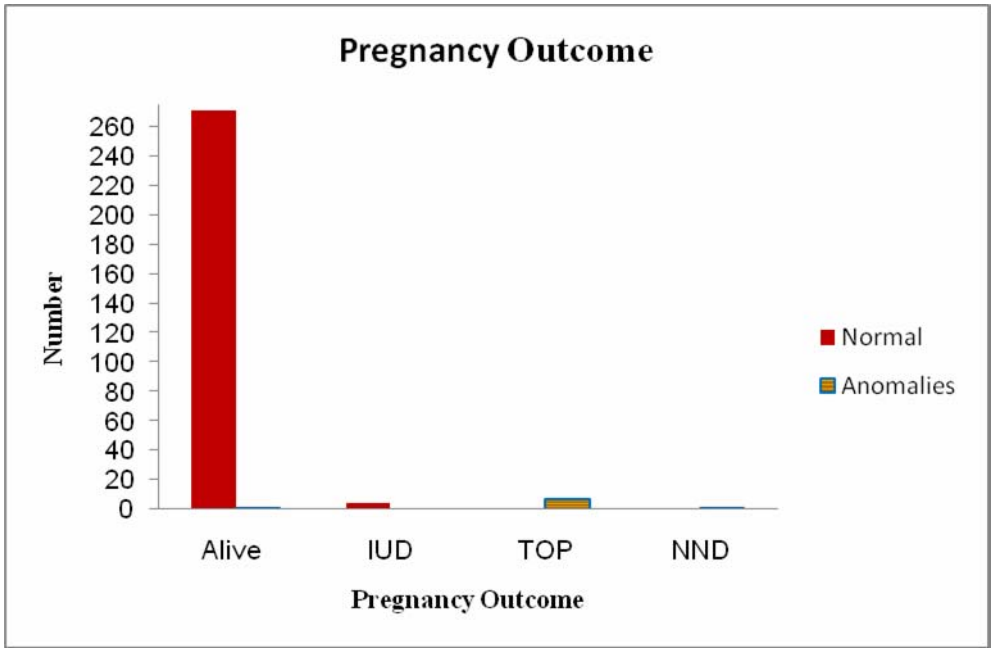


PREGNANCY OUTCOME

Table: 9

Pregnancy outcome	Normal	Anomalies	Total
Alive	271	1	272 (95.8%)
IUD	4	0	4 (1.4%)
TOP	0	7	7 (2.5%)
NND	0	1	1 (0.4%)

272 (95.8%) of the antenatal mothers had delivered alive baby 4(1.4%) had intrauterine fetal death due to medical complication in pregnancy Termination of pregnancies was done in 7 (2.5%) cases with anomalous fetuses and one anomalous baby that was born alive went in for early neonatal death. (0.4%).(it was a cases of meningomyelocele and the mother refused termination of pregnancy)



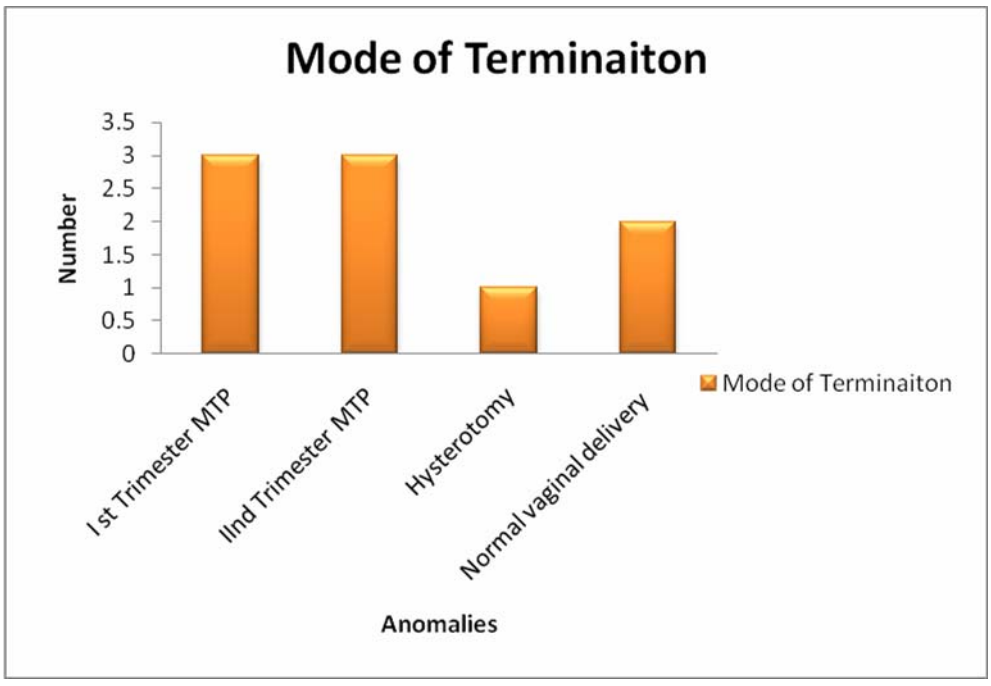
MODE OF DELIVERY

Table: 10

Mode of termination of the anomalous fetuses

Mode of Termination	Anomolous fetuses
I st Trimester MTP	3
IInd Trimester MTP	3
Hysterotomy	1
Normal vaginal delivery	2

7 Mothers with anomalous fetuses underwent medical termination of pregnancy and in one of the case, it went for Hysterotomy

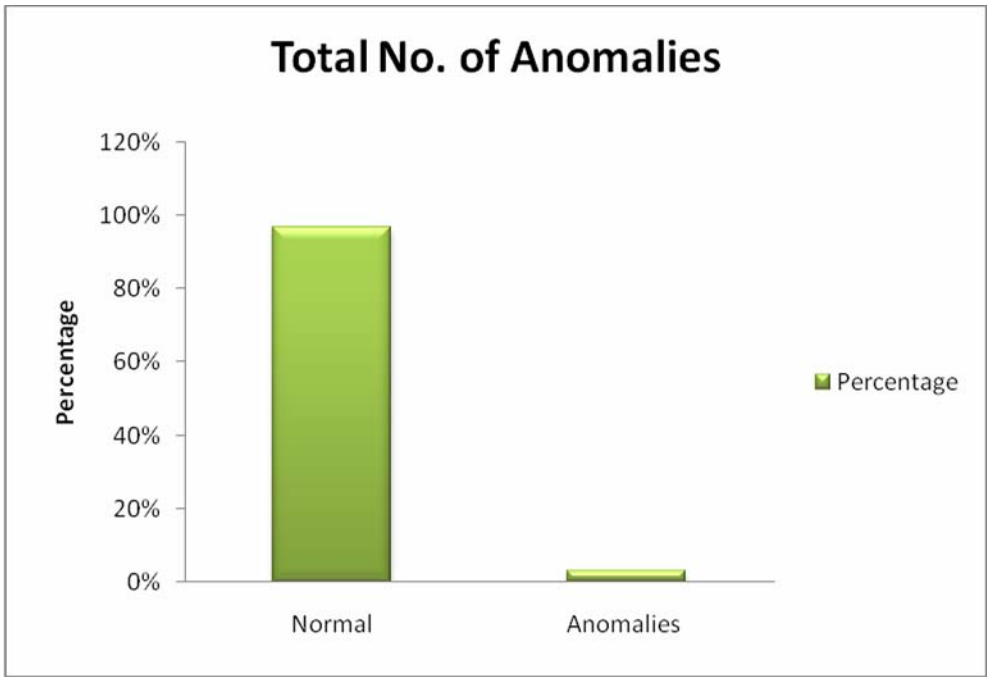


TOTAL NO OF ANOMALIES DETECTED IN THE STUDY GROUP

Table: 11

Study group	Frequency (n)	Percentage
Normal	275	96.8%
Anomalies	9	3.2%
Total	284	100%

There were 9 fetuses with anomalies in the study population (3.2%) and all were detected by ultrasound screening of the total 9 (3.2%) anomalous fetuses, 3 (1.1%) were detected at 11-13 weeks scan and the remaining 6 anomalous fetuses were detected during the followup second trimester scan.

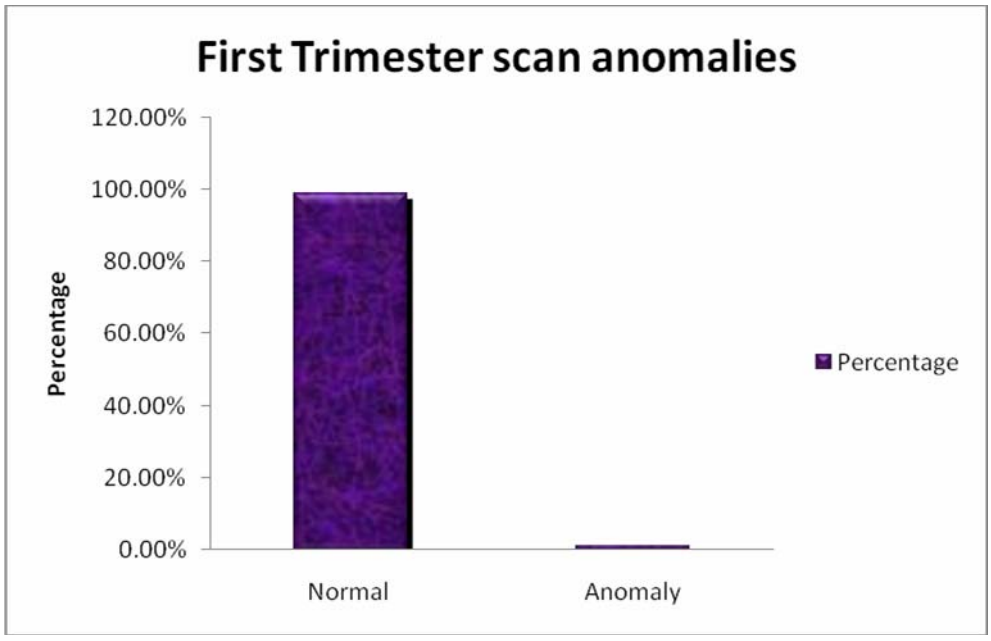


FIRST TRIMESTER SCAN ANOMALIES

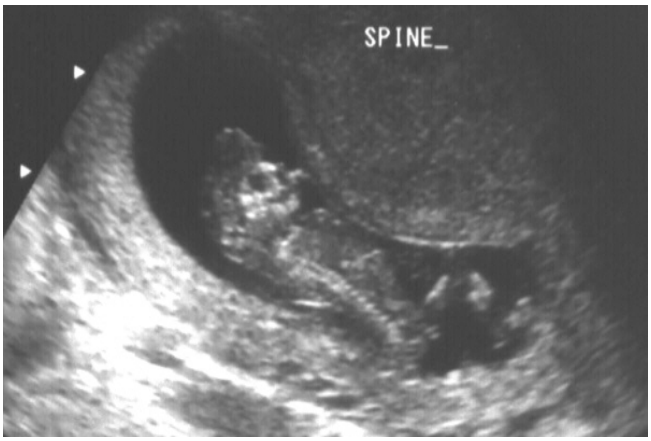
Table: 12

Study Group	Frequency (n)	Percentage
Normal	281	98.9%
Anomaly	3	1.1%
Total	284	100%

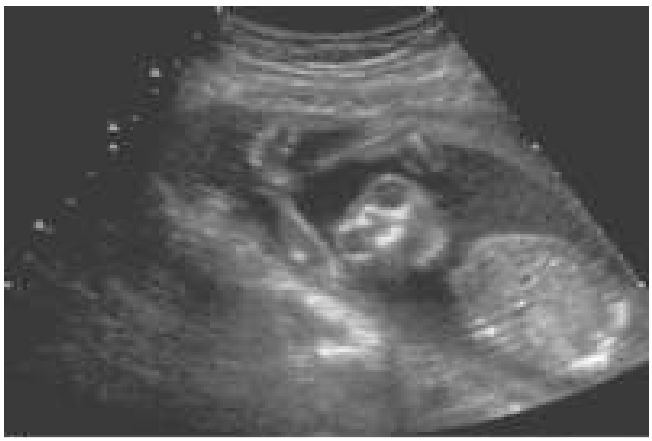
The sensitivity of the 11-13 wks scan in detecting the anomalies was about 33.33%. The overall sensitivity in detecting the anomalies using ultrasound in the study population was about 100%.



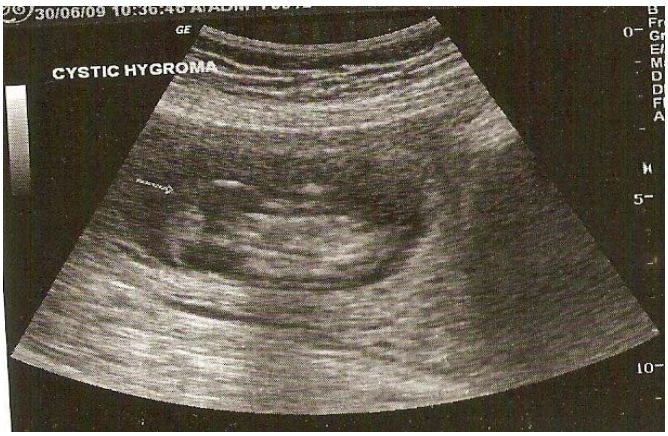
FIRST TRIMESTER SCAN ANOMALIES



ANENCEPHALY :1



ANENCEPHALY :2



CYSTIC HYGROMA

NUCHAL TRANSLUCENCY MEASUREMENT

Abnormal nuchal translucency measurement ie ≥ 3mm was noted in 2 of the 284 cases (0.7%). Only one case with abnormal NT was found to be an anomalous fetus

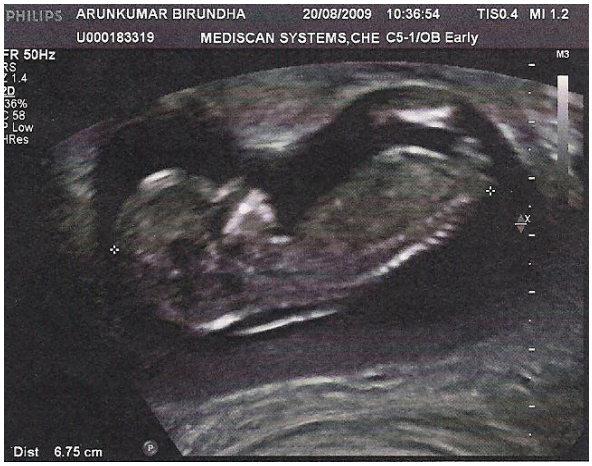
Case I.

The NT measurement was about 3.07 mm and the patient underwent chorionic villous sampling after counseling. The karyotype report was found to be normal and she was followed up regularly and there was no abnormalities detected in the second scan. The patient delivered an alive normal baby.

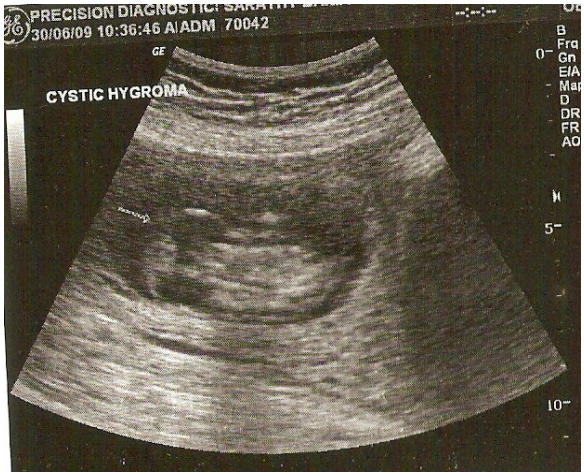
Case II

The NT measurement was about 11.2mm but was diagnosed to be a case of cystic hygroma with septation . The patient was counselled for chorionic villous sampling to detect chromosomal abnormalities but the patient refused and opted for termination of pregnancy.

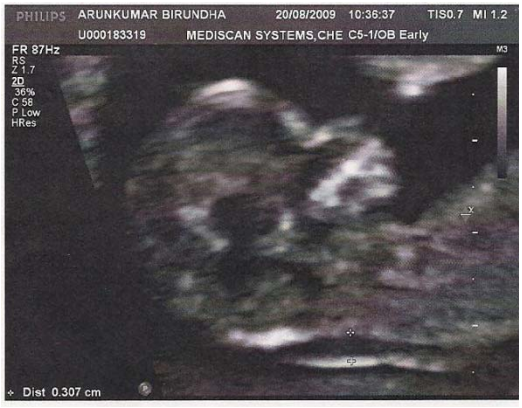
ABNORMAL NT SCANS CASE 1



NT=11.2 mm (Cystichygroma) CASE 2



NT=3.07mm



CYTOGENETIC ANALYSIS

NAME: Brinda Arunkumar

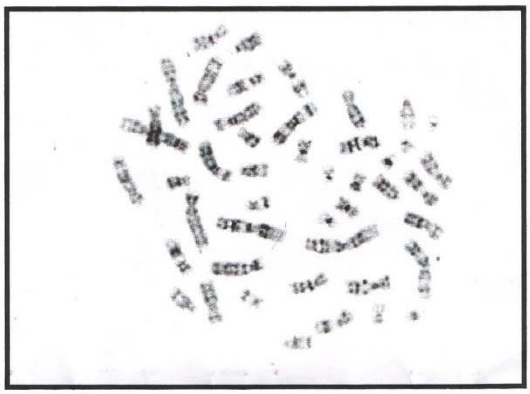
AGE/SEX: 28 YEARS/F

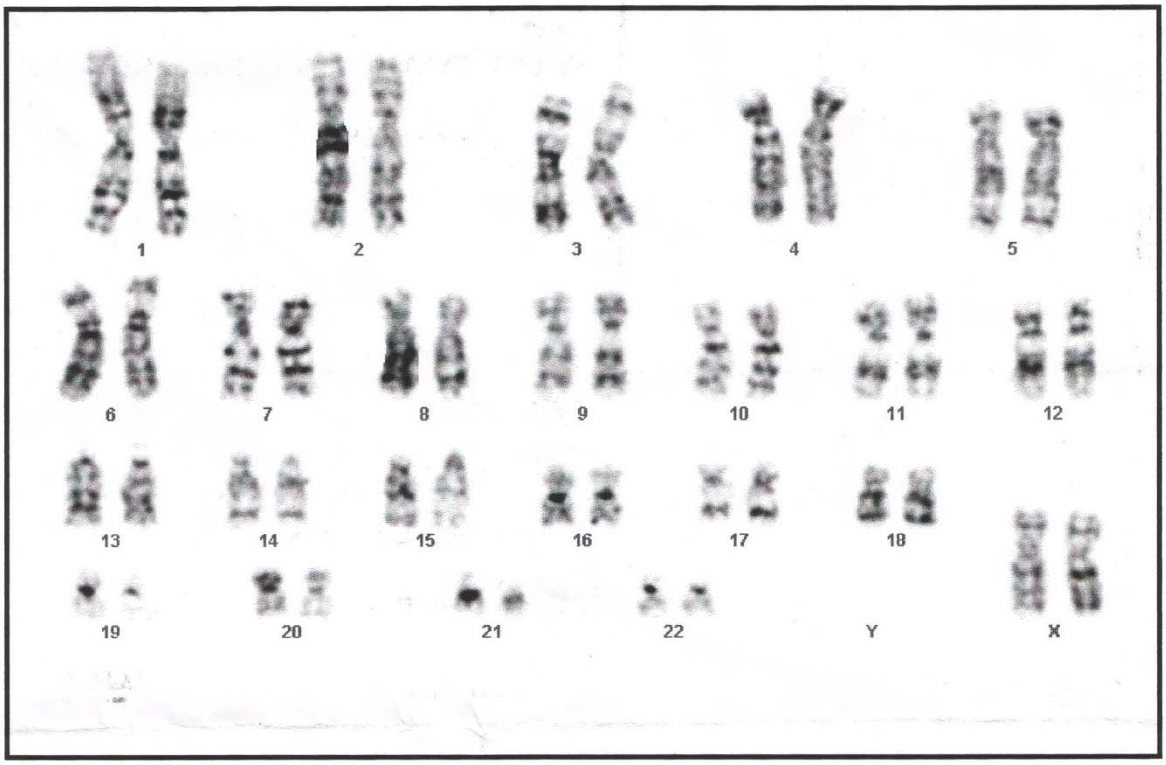
DATE: 9.03.2009

CASE NO/MRDNO: PL1671/*****

SPECIMEN TYPE: CVS

REFERRED BY: DR.PRIYA KANNAN





KARYOTYPE: 46,XX

IMPRESSION: NO CHROMOSOMAL ABNORMALITIES DETECTED

TYPES OF ANOMALIES DETECTED AT THE 11-13 WK SCAN

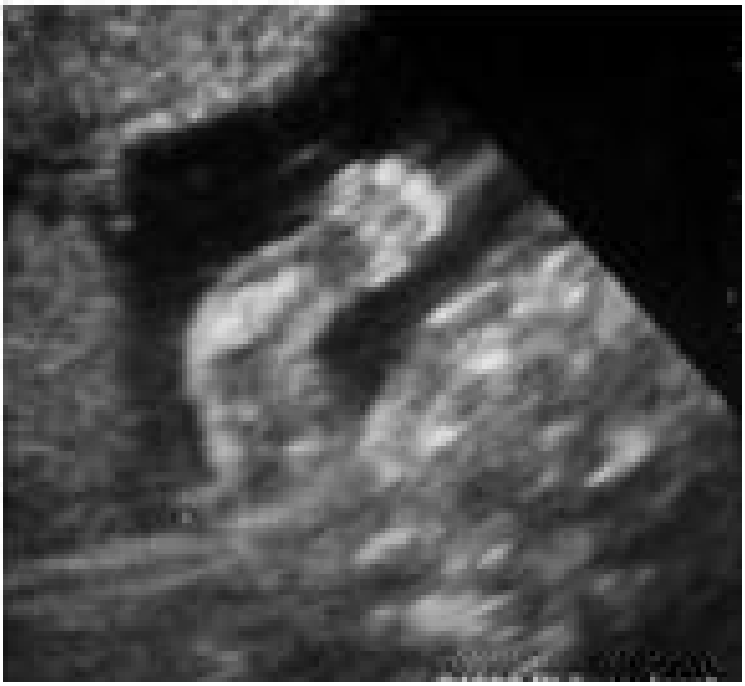
S.No.	Anomalies	GA(wks)	Outcome
1.	Cystic hygroma	13	TOP
2.	Anencephaly	13.2	TOP
3.	Anencephaly	12.2	TOP

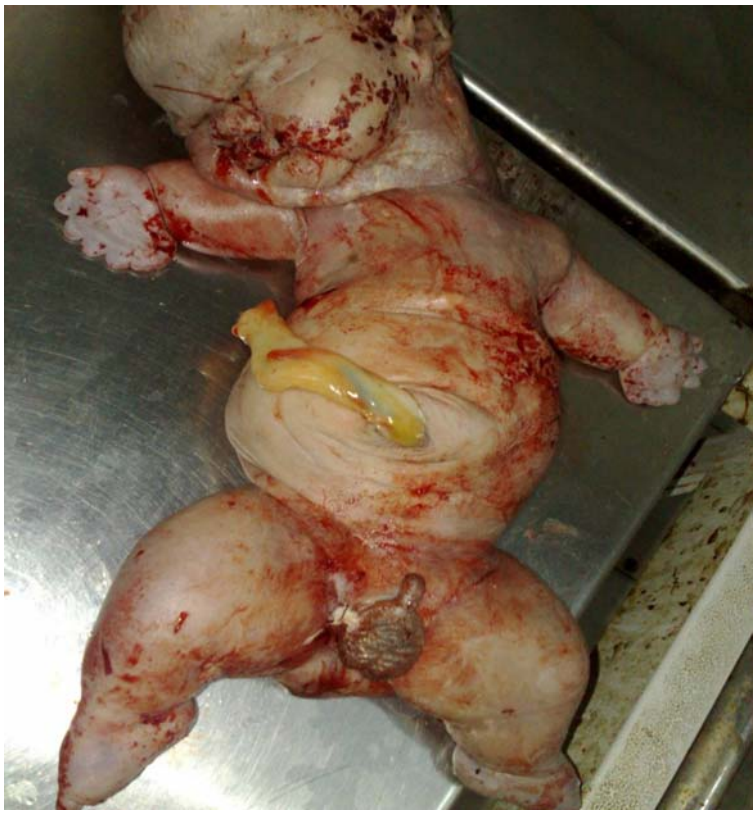
Cranial abnormalities was the most common abnormality detected in the first trimester and all of them were terminated.

TYPES OF ANOMALIES DETECTED AT THE 18-22 WKS SCAN

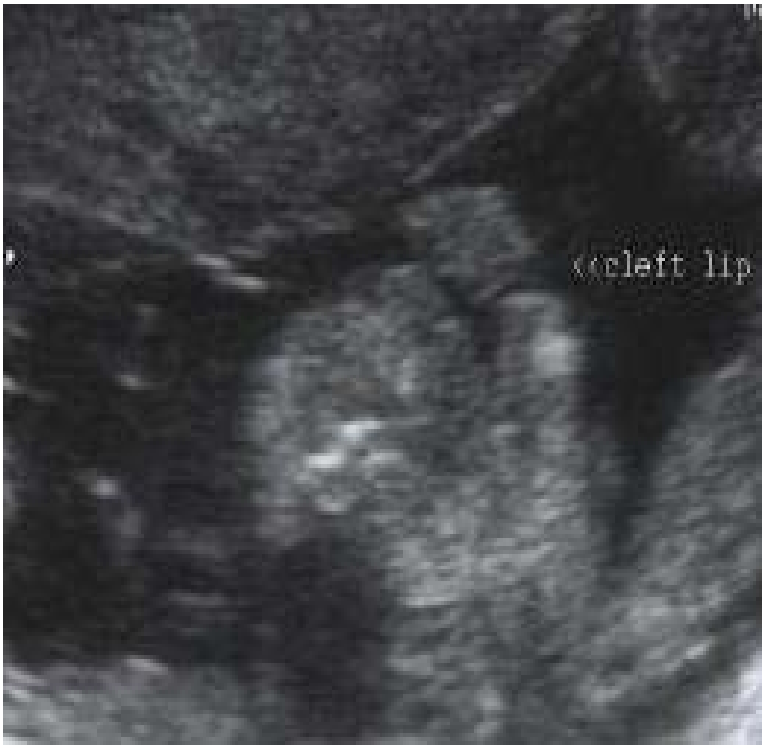
S.No.	Anomalies	Outcome
1.	Hydrocephalus	TOP
2.	Hydrocephalus	TOP
3.	Occipital meningocele	TOP
4.	Meningomyelocele	NND
5.	Cleft lip and cleft palate	Alive
6.	Skeletal dysplasia (achondroplasia)	TOP

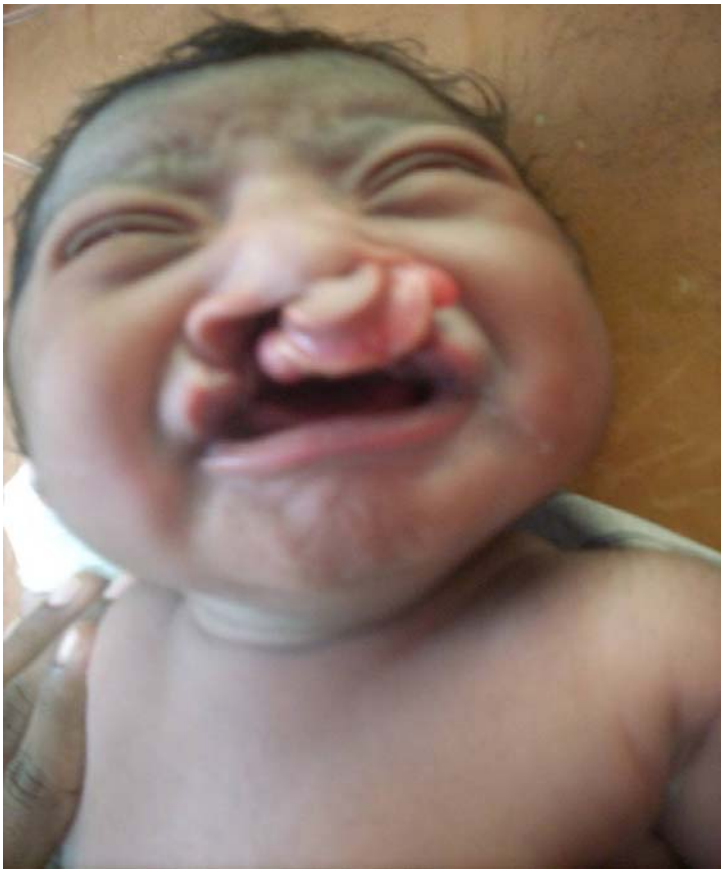
ACHONDROPLASIA USG PICTURE



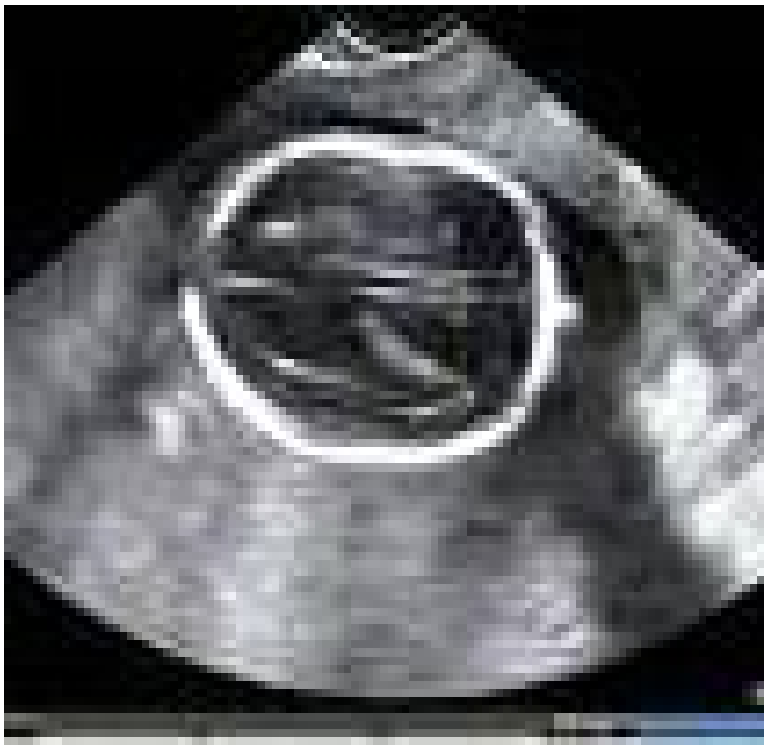


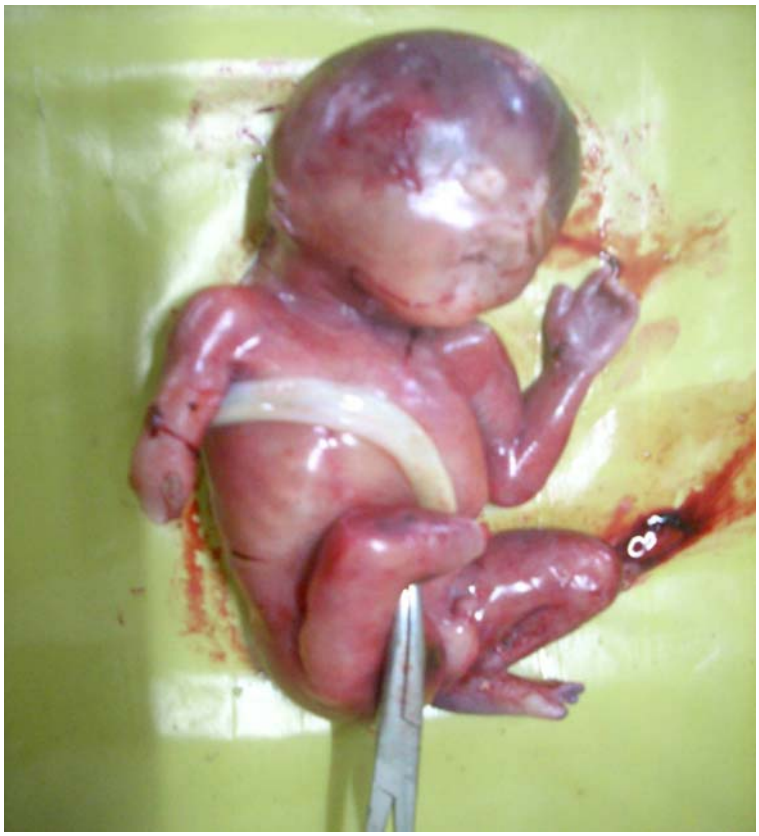
CLEF LIP AND CLEFT PALATE



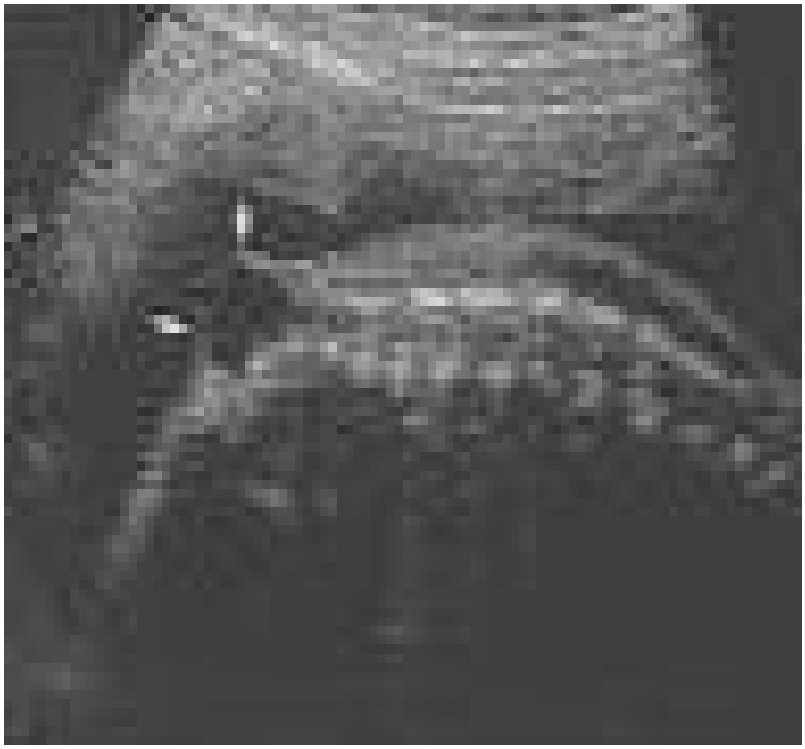


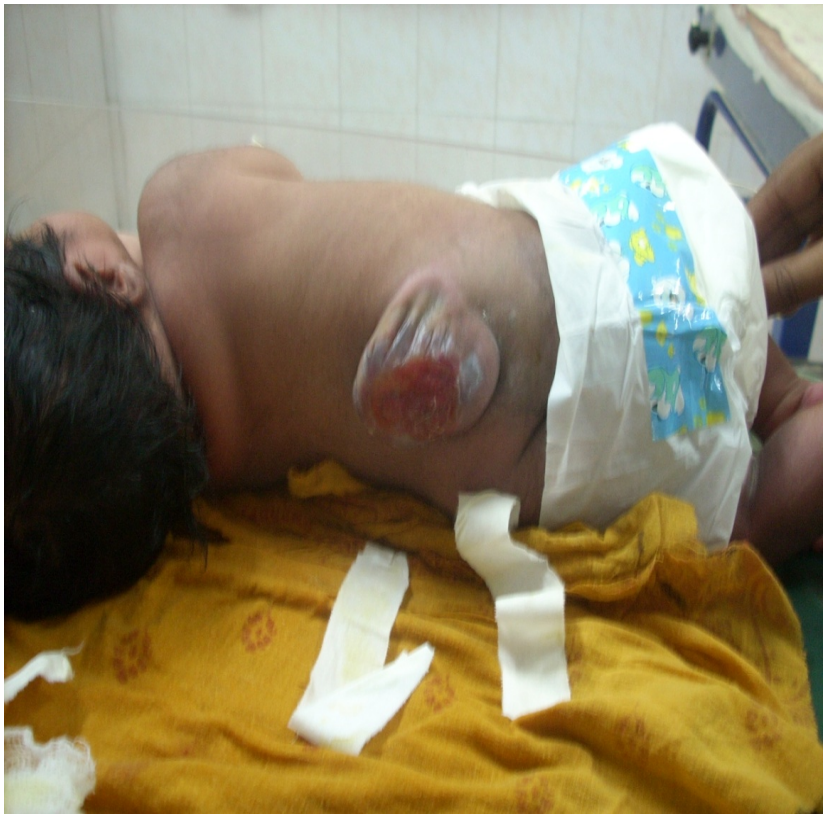
HYDROCEPHLUS



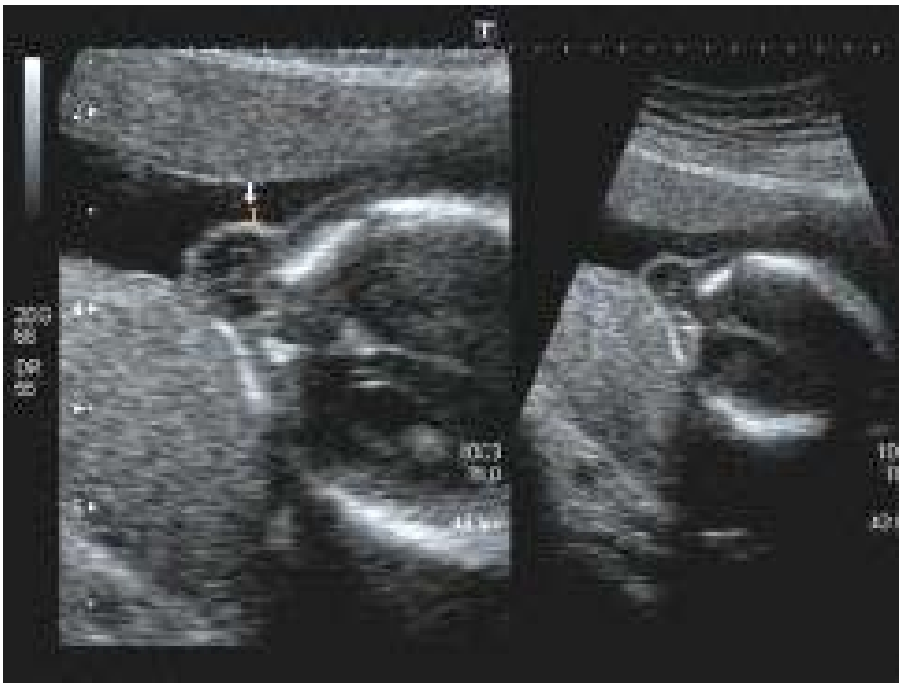


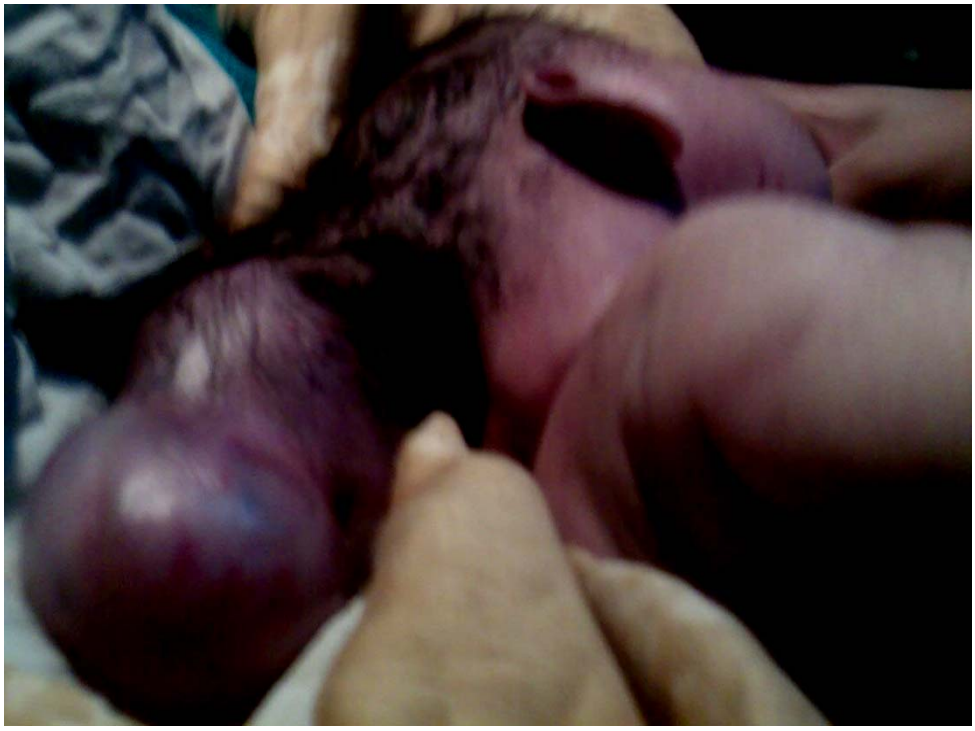
MENINGOMYELOCELE





OCCIPITAL ENCEPHALOCELE





Total no. of 6 anomalous fetuses which were not detected in the 11-13 wks scan were detected in the 18-22wks scan. Four cases were terminated. The anomalous baby with deft lip and deft palate was delivered alive and was referred to pediatric surgery department.

The mother who refused TOP delivered a baby with meningmyelocele which was born alive but went in for early neonatal death due to sepsis.

NICU ADMISSION

Study group	Normal	Anomalies	Total
NICU Admission	42	2	44
Others	229	0	229
Total	271	2	273

44 (16.1%) babies were admitted in NICU.

Both the anomalous babies with cleftlip and cleft palate and meningomyelocele were admitted into the NICU

BINARY LOGISTIC REGRESSION WAS USED TO IDENTITY THE ASSOCIATED RISK FACTORS WITH RESPECT TO TOTAL ANOMALIES

DEPENDENT VARIABLE: TOTAL ANOMALIES

INDEPENDENT VARIABLES : 1. FAMILY H/0 ANOMALJES

2. H/O DRUG INTAKE

VARIABLE NAME	CHI-SQUARE	P VALUE
FAMILY H/O. OF ANOMALIES	5.983	0.014
H/O DRUG INTAKE	10.946	0.001

It was found that family H/O anomalies and positive history of drug intake (anticonvulsants and other teratogenic drugs) had a very high risk for presence of anomalies. Hence first trimester ultrasound is useful in early diagnosis of structural anomalies.

DISCUSSION

DISCUSSION

The incidence of major fetal anomalies in this study was about 3.2% and it falls within the range reported in the literature [47,48].

In this study detailed examination of fetal structures at 11-13.6 wks of pregnancy revealed 33.3% of major structural abnormalities in low risk pregnant women. This result is similar to the results reported from other studies.

Hernadi and Torocsik in 1997[49] reported first trimester detection rate of 36% of structural anomalies in low risk Population.

Carvalho et al., (2002),[51] reported about 38% of structural anomalies detectable in first trimester.

Taipale et al., (2004)[52] had reported a low detection rate of about 18% in first trimester and it was mainly due to poor training and limited examination time.

Souka et al., (2006)[54] and Dane et al (2007) had reported high detection rate of about 50% and 70% respectively. This high detection rate of structural abnormalities at 11-13.6 wks of gestation, reflects the fact that experienced obstetrician or radiologist has a

major role and good training is mandatory with high resolution ultrasound machine

Studies Examining The Sensitivity of First and Second Trimester Ultrasound In Detecting Major Fetal Structural Defects

Authors	N	Population	Method	Major anomalies	First trimester detection rate	Total detection rate
Hernadi and Torocsik	3991	Low risk	TA	35 (0.9%)	36%	72%
Carvalho et al	2853	Low risk	TA+TV	66(2.3%)	38%	79%
Taipale et al	4513	Low risk	TV	33(0.7%)	18%	48%
Souka et al	1148	Low risk	TA+TV	14(1.2%)	50%	92%
Chen et al	1609	High risk	TS+TV	26(1.6%)	54%	77%
Economdies and Braithwaite	1632	Low risk	TA+TV	13(0.8%)	54%	77%

Increased Nuchal translucency is known to be associated with chromosomal defects and major structural abnormalities, in particular cardiac anomalies and genetic syndrome. In our study the abnormal Nuchal translucency measurement was noted in 2 cases (0.7%) and both were found to be false positive cases. This is mainly attributed to the small sample size taken in the study and most of them were among the low risk population.

The introduction of routine first trimester scanning will have important implication for second trimester scan. Most of the chromosomal abnormality are detected in the first trimester scan (11 – 13 weeks) using nuchal translucency thickness and hence it is used for screening for Downs syndrome and other abnormalities. Once it is found to be screen positive, the confirmative test such as chorionic villous sampling, and amniocentesis are done.

The chorionic villous sampling done at 11 to 13 weeks of gestation helps in earlier diagnosis of aneuploidy and early amniocentesis is no longer optimal at 11 – 13 weeks because of its higher association for fetal loss, fetal club foot, and procedure failure. Previously the cell culture and karyotype results took 2 to 3 weeks. But now the FISH technique helps in providing the karyotype results within 48 hrs.

Hence early detection of fetal malformation allows early termination of malformed fetuses. Once an abnormality is diagnosed, parents will choose for elective termination. Prenatal diagnosis not only allows termination but also has profound implication on antenatal and intrapartum management such as in utero treatment of diaphragmatic hernia and other fetal therapies.

SUMMARY

SUMMARY

This study was chosen to detect the incidence of major fetal abnormalities in general obstetric population and to evaluate the performance of ultrasonogram screening during 11 to 13 weeks gestation. 284 antenatal mother were screened for fetal anomalies in a two stage ultrasound scanning at 11 to 13 weeks gestation and follow up scan at 18 -22 weeks.

- The incidence of major fetal abnormalities was 3.2%.
- The sensitivity of first trimester (11-13 wks) scan in detecting the anomalies was 33% in this study.
- The overall sensitivity of ultrasonogram in detecting the anomalies was 100%.
- Increased nuchal translucency measurement was detected in two cases.
- 3 cases of missed abortion was detected at the time of 11-13 weeks scan which was an incidental finding.
- Craniospinal abnormalites were the most common abnormalities detected and hence it indicates the need for periconceptional folic acid intake.

CONCLUSION

CONCLUSION

1. The detection of the major anomalies at this early gestation (11-13 wks) offers to the parents the option of an earlier, safer and psychologically less traumatic termination of the pregnancy.

2. The ultrasound examination at 11-14 wks to screen for fetal abnormalities is effective and can be an adjunct to the routine 18-22 weeks anomaly scan.

3. Hence first trimester ultrasound (11-13wks) must be made mandatory, not only for the detection of major fetal anomalies, but also for diagnosing multiple pregnancy and abnormal pregnancy like missed abortion , molar pregnancy and ectopic pregnancy.

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PROFORMA

THE VALUE OF 11-13 WKS SCAN – IN SCREENING FOR FETAL ANOMALIES

PROFORMA

1. Patient's Name :
2. Age :
3. Husband's Age :
4. Obsteteric code :
5. No.of children with sex :
6. Marital Status and consanguinity :
7. LMP :
8. Weeks of pregnancy :
9. H/O Genetic /Medical Disease in family:
10. High risk Factor :
    a) H/O Previous anomalies :
    b) H/O DM (overt / GDM) :
    c) H/O congenital Heart disease :
    d) H/O epilepsy :
    e) H/O drug intake :
11. USG (11-13.6 wks scan) :

a. CRL :

b. GA :

c. Nuchal Translucency :

d. Skull :

e. Abdominal wall :

f. Bladder :

g. Extremities :

h. Other :

12. Bio –Chemical /Cyto – Genetic :

13. Second trimester USG :

14. Pregnancy Outcome :

MASTER CHART

MASTER CHART

S.No	Age	Gravida	Previous Outcome	Consangunity	H/o Previous anomaly	Family H/o anomalies	H/o Drug intake	H/o medical disorders	Scan at 11 - 13 Weeks			First Trimester Scan (Anomaly)	Second Trimester scan (Anomaly)	Total No. of Anomalies	Pregnancy outcome	Mode of Delivery	Birth Weight	NICU Admission
									GA (wks)	CRL (cm)	NT (mm)							
1	27	Multi	n	Ncm	N	N	Y	N	13	6.6	1.1	Normal	Normal	Normal	Alive	VD	N	N
2	21	Primi	-	Ncm	N	N	N	N	12	65	1.2	Normal	Normal	Normal	Alive	VD	N	N
3	22	Primi	-	Ncm	N	N	N	N	13	6.45	1.5	Normal	Normal	Normal	Alive	CS	N	N
4	20	Multi	n	Ncm	N	N	N	N	13	6.4	1.8	Normal	Normal	Normal	Alive	VD	N	N
5	26	Primi	-	Ncm	N	N	N	N	13	6.6	1.9	Normal	Normal	Normal	Alive	CS	N	N
6	22	Multi	n	Ncm	N	N	N	N	12	5.3	1.2	Normal	Normal	Normal	Alive	VD	N	N
7	34	Multi	n	Ncm	N	N	N	N	11.4	4.6	0.9	Normal	Normal	Normal	Alive	CS	N	N
8	25	Multi	n	3˚cm	N	N	N	N	13	6.4	1.9	Normal	Normal	Normal	Alive	VD	N	N
9	26	Primi	-	Ncm	N	N	N	N	12.3	5.8	1.8	Normal	Normal	Normal	Alive	VD	LBW	Y
10	22	Multi	poor	Ncm	N	N	N	N	13	6.5	1.5	Normal	Normal	Normal	Alive	VD	N	N
11	31	Multi	n	Ncm	N	N	N	N	13	6.4	1.8	Normal	Normal	Normal	Alive	VD	N	N
12	22	Primi	-	3˚cm	N	N	N	N	13	6.6	1.2	Normal	Normal	Normal	Alive	VD	N	N
13	22	Multi	n	3˚cm	N	N	N	N	12	5.3	1	Normal	Normal	Normal	Alive	VD	N	N
14	24	Primi	-	Ncm	N	N	N	N	12.2	5.6	1.1	Normal	Normal	Normal	Alive	VD	N	N
15	23	Multi	poor	Ncm	Y	N	N	N	12.4	5.9	1	Normal	Normal	Normal	Alive	VD	LBW	Y
16	20	Primi	-	Ncm	N	N	N	N	11.6	4.9	1.5	Normal	Normal	Normal	Alive	VD	N	N
17	29	Multi	n	Ncm	N	N	N	N	13	6.4	1.9	Normal	Normal	Normal	Alive	VD	N	N
18	22	Multi	n	Ncm	0	N	N	N	13	6.5	1	Normal	Normal	Normal	Alive	VD	N	N
19	26	Multi	n	Ncm	N	N	N	N	12	5.3	1	Normal	Normal	Normal	Alive	VD	N	N
20	27	Primi	-	Ncm	N	N	N	N	12	5.3	1	Normal	Normal	Normal	Alive	CS	N	N
21	22	Multi	n	Ncm	N	N	N	N	13	6.6	1	Normal	Normal	Normal	Alive	VD	N	N
22	21	Primi	-	Ncm	N	N	N	N	11.6	4.6	0.9	Normal	Normal	Normal	Alive	VD	N	N
23	22	Multi	n	3˚cm	N	Y	N	N	11	4.5	0.9	Normal	Normal	Normal	Alive	VD	N	N
24	23	Multi	n	Ncm	N	N	N	N	13	6.7	1.1	Normal	Normal	Normal	Alive	VD	N	N
25	25	Multi	poor	Ncm	N	N	N	N	12	5.3	1	Normal	Normal	Normal	Alive	CS	N	N
26	32	Multi	poor	Ncm	N	N	N	N	11	4.6	0.5	Normal	Normal	Normal	Alive	VD	N	N
27	23	Primi	-	Ncm	N	N	N	N	12.2	5.6	0.9	Normal	Normal	Normal	Alive	VD	N	N
28	18	Primi	-	Ncm	N	N	N	N	12.4	5.6	0.9	Normal	Normal	Normal	Alive	VD	N	N
29	25	Multi	poor	Ncm	N	N	N	N	12.3	5.7	1	Normal	Normal	Normal	Alive	VD	N	N
30	23	Multi	poor	Ncm	Y	N	N	N	12	5.8	1.1	Normal	Normal	Normal	Alive	VD	N	N

31	19	Primi	-	Ncm	N	N	N	N	12	5.6	1	Normal	Normal	Normal	Alive	CS	N	Y
32	28	Primi	-	Ncm	N	N	N	N	12.4	5.6	1	Normal	Normal	Normal	Alive	For	N	N
33	29	Primi	-	Ncm	N	N	N	N	11	4.5	1.2	Normal	Normal	Normal	Alive	VD	N	N
34	21	Primi	-	Ncm	N	N	N	N	11.2	5.4	1.1	Normal	Normal	Normal	Alive	VD	N	N
35	29	Primi	-	Ncm	N	N	N	N	12	5.4	0.9	Normal	Normal	Normal	Alive	VD	LBW	Y
36	21	Multi	n	Ncm	N	N	N	N	13	6.4	1.1	Normal	Normal	Normal	Alive	VD	LBW	Y
37	25	Primi	-	Ncm	N	N	N	N	11.6	4.8	1.2	Normal	Normal	Normal	Alive	VD	N	N
38	24	Multi	n	Ncm	N	N	N	N	12.3	5.7	1	Normal	Normal	Normal	Alive	CS	N	N
39	23	Multi	n	3˚cm	N	N	N	N	11.6	5	1	Normal	Normal	Normal	Alive	VD	N	N
40	22	Primi	-	Ncm	N	N	N	N	12	5.3	1.2	Normal	Normal	Normal	Alive	VD	N	N
41	22	Multi	2	Ncm	N	N	N	N	12.4	5.8	1.2	Normal	Normal	Normal	Alive	VD	N	N
42	29	Primi	-	Ncm	N	N	N	N	13	6.4	1.5	Normal	Normal	Normal	Alive	VD	N	N
43	20	Primi	-	Ncm	N	N	N	N	11.3	4.1	1.1	Normal	Normal	Normal	Alive	CS	N	N
44	35	Multi	n	3˚cm	N	N	N	N	12	5.1	0.9	Normal	Normal	Normal	Alive	CS	N	N
45	28	Primi	n	Ncm	N	N	N	N	12.4	5.9	0.9	Normal	Normal	Normal	Alive	VD	N	N
46	20	primi	-	Ncm	N	N	N	N	13.2	6.6	1.2	Normal	Normal	Normal	Alive	VD	N	N
47	28	Primi	n	Ncm	N	N	N	N	13.1	6.6	1.1	Normal	Normal	Normal	Alive	CS	N	N
48	21	Multi	-	Ncm	N	N	N	N	12.1	5.3	1	Normal	Normal	Normal	Alive	VD	N	N
49	30	Primi	n	Ncm	N	N	N	N	11.6	5	0.8	Normal	Normal	Normal	Alive	VD	N	N
50	26	Multi	poor	Ncm	N	N	N	N	11.3	4.5	0.9	Normal	Normal	Normal	Alive	VD	LBW	N
51	22	Multi	-	Ncm	N	N	N	N	13.1	6.4	0.5	Normal	Normal	Normal	Alive	For	N	N
52	25	Primi	-	Ncm	N	N	N	HD	13.1	6.6	0.9	Normal	Normal	Normal	Alive	VD	LBW	N
53	29	Primi	-	Ncm	N	N	N	N	11.6	5	1.2	Normal	Normal	Normal	Alive	VD	LBW	N
54	21	Primi	n	Ncm	N	N	N	N	13.5	7.4	1.1	Normal	Normal	Normal	Alive	VD	N	N
55	27	Multi	poor	Ncm	N	N	N	N	13.5	7.4	1.1	Normal	Normal	Normal	Alive	CS	N	N
56	22	Primi	-	3˚cm	N	N	N	N	13.4	7.2	1.2	Normal	Normal	Normal	Alive	VD	N	N
57	21	Primi	-	2˚cm	N	N	N	N	12.1	5.3	1.4	Normal	Normal	Normal	Alive	VD	N	N
58	20	Primi	-	Ncm	N	N	N	N	12.5	6.1	1.5	Normal	Normal	Normal	Alive	VD	N	N
59	20	Multi	poor	2˚cm	N	N	N	N	12.6	6.3	1	Normal	Normal	Normal	Alive	VD	N	N
60	19	Primi	-	Ncm	N	N	N	N	11.5	4.8	1.5	Normal	Normal	Normal	Alive	VD	N	N
61	22	Primi	-	Ncm	N	N	N	N	12.2	5.6	0.8	Normal	Normal	Normal	Alive	CS	N	N
62	21	Primi	-	Ncm	N	N	N	N	12.6	6.2	0.9	Normal	Normal	Normal	Alive	VD	LBW	Y
63	28	Primi	-	Ncm	N	N	N	N	11.4	4.7	0.9	Normal	Normal	Normal	Alive	VD	LBW	Y
64	19	Primi	-	Ncm	N	N	N	N	12	5.3	1.1	Normal	Normal	Normal	Alive	VD	N	N
65	22	Primi	-	Ncm	N	N	N	DM	13.4	7.2	1.3	Normal	Normal	Normal	Alive	CS	N	Y
66	26	Multi	poor	2˚cm	N	Y	N	N	11.4	4.6	1.2	Normal	Abnormal	**occipital encephalocele**	TOP	Hys	-	-
67	27	Primi	-	Ncm	N	N	N	N	12.5	6.1	1	Normal	Normal	Normal	Alive	VD	N	N
68	19	Primi	-	Ncm	N	N	N	N	13.5	7.4	1.2	Normal	Normal	Normal	Alive	VD	N	N
69	20	Primi	-	Ncm	N	N	N	N	12	5.3	1.1	Normal	Normal	Normal	Alive	VD	N	N
70	19	Primi	-	Ncm	N	N	N	N	13.1	6.5	1.2	Normal	Normal	Normal	Alive	VD	N	N
71	22	Primi	-	Ncm	N	N	N	N	11.6	5	0.8	Normal	Normal	Normal	Alive	VD	N	N
72	22	Multi	n	Ncm	N	N	N	N	12.3	5.7	1.5	Normal	Normal	Normal	Alive	CS	N	N
73	22	Primi	-	Ncm	N	N	N	N	13	6.4	1.2	Normal	Normal	Normal	Alive	VD	N	N
74	26	Primi	-	Ncm	N	N	N	N	13	6.8	**11.2**	Ab normal	Normal	**cystichygroma**	TOP	MTP1		
75	21	Primi	-	Ncm	N	N	N	N	12	5.3	0.8	Normal	Normal	Normal	Alive	CS	LBW	Y

76	23	Multi	n	Ncm	N	N	N	N	11.3	4.5	0.9	Normal	Normal	Normal	Alive	CS	N	N
77	20	Primi	-	Ncm	N	N	N	N	12	5.3	0.9	Normal	Normal	Normal	Alive	VD	N	N
78	21	Primi	-	Ncm	N	N	N	N	12.5	6.1	1.2	Normal	Normal	Normal	Alive	VD	N	N
79	20	Primi	-	Ncm	N	N	N	N	13.4	7.2	1.4	Normal	Normal	Normal	Alive	VD	N	N
80	22	Primi	-	3˚cm	N	N	N	N	13.2	6.9	1	Normal	Normal	Normal	Alive	VD	N	N
81	22	Multi	n	Ncm	N	N	N	N	13.1	6.6	1.1	Normal	Normal	Normal	Alive	CS	N	N
82	20	Primi	-	Ncm	N	N	N	N	12.6	6.2	1	Normal	Normal	Normal	Alive	VD	N	N
83	23	Multi	poor	Ncm	N	N	N	N	12.4	5.9	0.9	Normal	Normal	Normal	Alive	CS	N	N
84	21	Multi	n	Ncm	N	N	N	N	12.1	5.3	1.2	Normal	Normal	Normal	Alive	VD	N	N
85	21	Primi	-	Ncm	N	N	N	N	13.4	7.2	1.3	Normal	Normal	Normal	Alive	CS	N	N
86	20	Multi	n	Ncm	N	N	N	N	12.2	5.5	1.1	Normal	Normal	Normal	Alive	VD	N	N
87	22	Primi	-	Ncm	N	N	N	N	13	6.4	1.5	Normal	Normal	Normal	Alive	VD	N	N
88	24	Multi	poor	Ncm	N	N	N	N	12.5	6.1	1	Normal	Normal	Normal	Alive	for	N	N
89	22	Multi	poor	Ncm	N	N	N	N	11.6	5	0.9	Normal	Normal	Normal	Alive	CS	N	N
90	22	Primi	-	Ncm	N	N	N	N	11.4	4.7	0.8	Normal	Normal	Normal	Alive	CS	N	N
91	27	Primi	-	Ncm	N	N	N	DM	11.4	4.8	0.8	Normal	Normal	Normal	Alive	CS	N	N
92	26	Multi	n	Ncm	N	N	N	N	12.5	6.1	1	Normal	Normal	Normal	Alive	VD	N	N
93	28	Multi	n	Ncm	N	N	N	N	12.6	6.3	1.2	Normal	Normal	Normal	Alive	VD	N	N
94	24	Multi	n	ncm	N	N	N	N	13.2	6.2	1.3	Normal	Normal	Normal	Alive	CS	N	N
95	22	Primi	-	Ncm	N	N	N	DM	13.6	7.6	1.5	Normal	Normal	Normal	Alive	VD	LBW	Y
96	28	Multi	poor	3˚cm	Y	N	Y	EPI	13.5	7.6	1.1	Normal	Normal	Normal	Alive	CS	N	N
97	20	Multi	n	Ncm	N	N	N	N	13.6	7.6	1.5	Normal	Normal	Normal	Alive	CS	LBW	Y
98	24	Primi	-	Ncm	N	N	N	N	12.4	5.9	1	Normal	Normal	Normal	Alive	VD	LBW	Y
99	21	Primi	-	2˚cm	N	N	N	N	13.4	7.3	1.3	Normal	Normal	Normal	Alive	VD	N	N
100	25	Multi	n	2˚cm	N	N	N	N	12	5.3	1.2	Normal	Normal	Normal	Alive	VD	N	N
101	28	Primi	-	Ncm	N	N	N	N	11.5	4.8	0.7	Normal	Normal	Normal	Alive	VD	LBW	Y
102	31	Multi	n	Ncm	N	N	N	N	12.5	6.2	1	Normal	Normal	Normal	Alive	VD	N	N
103	24	Multi	n	Ncm	N	N	N	N	11.6	5	1	Normal	Normal	Normal	Alive	VD	N	N
104	23	Primi	-	Ncm	N	N	N	N	12.6	6.3	1.2	Normal	Normal	Normal	Alive	VD	N	N
105	22	Primi	-	Ncm	N	N	Y	EPI	13.5	7.4	1.6	Normal	Normal	Normal	Alive	VD	N	N
106	22	Multi	n	Ncm	N	N	N	N	13.1	6.6	1.2	Normal	Normal	Normal	Alive	CS	N	N
107	22	Primi	-	Ncm	N	N	N	N	13.4	7.2	1.5	Normal	Normal	Normal	Alive	VD	N	N
108	26	Multi	poor	3˚cm	N	Y	N	N	12.4	5.9	1.2	Normal	Abnormal	**skeletal dysplasia**	TOP	MTP2	-	-
109	24	Multi	n	3˚cm	N	N	N	N	13.5	7.4	1.2	Normal	Normal	Normal	Alive	VD	N	N
110	23	Primi	-	Ncm	N	N	N	GDM	13.1	6.6	1.2	Normal	Normal	Normal	Alive	VD	LBW	Y
111	20	Primi	-	Ncm	N	N	N	N	12.5	6.1	1.3	Normal	Normal	Normal	Alive	CS	N	N
112	25	Multi	n	Ncm	N	N	N	N	12.2	5.5	1.6	Normal	Normal	Normal	Alive	VD	N	N
113	26	Multi	n	Ncm	N	N	N	N	12.1	5.3	1.2	Normal	Normal	Normal	Alive	CS	N	N
114	25	Multi	n	Ncm	N	N	N	N	13.1	6.6	1.5	Normal	Normal	Normal	Alive	VD	N	N
115	29	Multi	n	Ncm	N	N	N	HD	12.2	5.5	1	Normal	Normal	Normal	Alive	For	N	N
116	25	Primi	-	Ncm	N	N	N	N	13.1	6.7	1.2	Normal	Normal	Normal	Alive	CS	N	N
117	24	Primi	-	Ncm	N	N	N	N	11.6	5	0.9	Normal	Normal	Normal	Alive	VD	N	N
118	25	Primi	-	Ncm	N	N	N	N	12.4	5.9	1.9	Normal	Normal	Normal	Alive	VD	N	N
119	26	Primi	-	Ncm	N	N	N	N	13.6	7.6	1.3	Normal	Normal	Normal	Alive	VD	N	N
120	30	Multi	poor	Ncm	N	N	N	N	12	6.4	1	Normal	Normal	Normal	Alive	CS	N	N
121	24	Multi	n	Ncm	N	N	N	N	12.6	6.3	1.1	Normal	Normal	Normal	Alive	VD	N	N

122	22	Multi	n	2°cm	N	N	N	N	12.4	5.4	1.2	Normal	Normal	Normal	Alive	VD	N	N
123	24	Primi	-	Ncm	N	N	N	N	12.1	5.8	1.2	Normal	Normal	Normal	Alive	VD	N	N
124	20	Primi	-	Ncm	N	N	N	N	12	5.3	0.9	Normal	Normal	Normal	Alive	VD	N	N
125	25	Primi	-	Ncm	N	N	N	N	13	6.4	1.5	Normal	Normal	Normal	Alive	VD	N	N
126	33	Multi	poor	Ncm	N	N	N	N	12.2	5.6	1.7	Normal	Normal	Normal	Alive	CS	LBW	Y
127	32	Multi	poor	Ncm	N	N	N	GDM	13.6	7.6	1.4	Normal	Normal	Normal	Alive	CS	N	N
128	22	Multi	n	Ncm	N	N	N	N	13.1	6.6	1.1	Normal	Normal	Normal	Alive	VD	N	N
129	24	Primi	-	Ncm	N	N	N	N	12.1	5.3	1.1	Normal	Normal	Normal	Alive	VD	N	N
130	19	Multi	poor	Ncm	N	N	N	N	12	5.3	1	Normal	Normal	Normal	Alive	VD	LBW	N
131	28	Multi	n	Ncm	N	N	N	N	11.6	5	0.9	Normal	Normal	Normal	Alive	VD	N	N
132	24	Primi	-	Ncm	N	N	Y	EPI	12.2	5.5	1.9	Ab normal	Normal	**Anencephaly**	TOP	MTP1		
133	30	Multi	n	Ncm	N	N	N	N	12.1	5.3	1	Normal	Normal	Normal	Alive	VD	N	N
134	23	Multi	n	Ncm	N	N	N	N	13	6.4	1	Normal	Normal	Normal	Alive	VD	N	N
135	25	Multi	n	Ncm	N	N	N	N	12.6	6.3	1.2	Normal	Normal	Normal	Alive	VD	N	N
136	23	Primi	-	3°cm	N	N	N	N	12	5.2	0.9	Normal	Abnormal	**Hydrocephalus**	TOP	MTP2	N	N
137	29	Multi	poor	Ncm	N	N	N	N	11.4	4.7	0.5	Normal	Normal	Normal	Alive	VD	N	N
138	26	Multi	poor	Ncm	N	N	N	N	11.4	4.7	0.8	Normal	Normal	Normal	Alive	VD	N	N
139	28	Multi	n	Ncm	N	N	N	N	12	5.3	1.2	Normal	Normal	Normal	Alive	VD	N	N
140	25	Primi	-	Ncm	N	N	N	N	11.6	5	0.8	Normal	Normal	Normal	Alive	VD	N	N
141	23	Primi	-	Ncm	N	N	N	N	12	5.3	1	Normal	Normal	Normal	Alive	VD	N	N
142	34	Multi	n	Ncm	N	N	N	N	13	6.4	1.2	Normal	Normal	Normal	Alive	VD	N	N
143	25	Multi	n	2°cm	N	N	N	N	12.4	5.9	1.1	Normal	Normal	Normal	Alive	VD	N	N
144	27	Multi	poor	2°cm	N	N	N	N	13	6.4	1.1	Normal	Normal	Normal	Alive	CS	N	N
145	30	Primi	-	Ncm	N	N	N	N	12.5	6.1	1	Normal	Normal	Normal	Alive	VD	N	N
146	30	Multi	n	3°cm	N	N	N	N	12	5.3	1	Normal	Normal	Normal	Alive	CS	N	N
147	23	Multi	n	Ncm	N	N	N	N	13	6.4	1.3	Normal	Normal	Normal	Alive	VD	N	N
148	23	Multi	n	Ncm	N	N	N	N	11.6	5	0.5	Normal	Normal	Normal	Alive	VD	N	N
149	26	Multi	n	Ncm	N	N	N	N	13.5	7.4	1.1	Normal	Normal	Normal	Alive	VD	N	N
150	27	Primi	-	Ncm	N	N	N	N	12.4	5.9	1	Normal	Normal	Normal	Alive	CS	N	N
151	21	Primi	-	Ncm	N	N	N	N	13	6.4	1.1	Normal	Normal	Normal	Alive	CS	N	N
152	22	Primi	-	Ncm	N	N	N	N	11.4	4.7	0.8	Normal	Normal	Normal	Alive	VD	N	N
153	22	Primi	-	Ncm	N	N	N	N	12.5	6.1	1	Normal	Normal	Normal	Alive	VD	N	N
154	20	Primi	-	Ncm	N	N	N	N	11.6	5	0.8	Normal	Normal	Normal	Alive	VD	N	N
155	19	Multi	n	Ncm	N	N	N	N	12	5.3	1.5	Normal	Normal	Normal	Alive	CS	N	N
156	25	Primi	-	Ncm	N	N	N	N	12.6	5.7	1.2	Normal	Normal	Normal	Alive	VD	N	N
157	23	Multi	poor	2°cm	N	N	N	N	13	6.4	1.4	Normal	Normal	Normal	Alive	VD	N	N
158	18	Primi	-	0	N	N	N	N	13.2	6.8	1.4	Normal	Normal	Normal	Alive	VD	N	N
159	20	Multi	poor	0	N	N	N	N	12.6	6.3	1.1	Normal	Normal	Normal	Alive	CS	LBW	Y
160	32	Primi	-	NCM	N	N	N	N	13.4	7.2	1.3	Normal	Normal	Normal	Alive	VD	N	N
161	23	Multi	poor	0	N	N	N	N	12.5	6.1	1.5	Normal	Normal	Normal	Alive	VD	N	Y
162	30	Multi	n	3°cm	N	N	N	N	11.5	4.8	1.3	Normal	Normal	Normal	Alive	VD	N	N
163	25	Multi	poor	0	N	N	N	N	12.5	6.1	1.5	Normal	Normal	Normal	Alive	CS	N	N
164	23	Primi	-	0	N	N	N	N	11.5	4.8	1.3	Normal	Normal	Normal	Alive	VD	N	N
165	20	Multi	poor	0	N	N	N	N	13.1	6.6	1.9	Normal	Normal	Normal	Alive	VD	N	N
166	22	Multi	n	3°cm	N	N	N	N	13	6.4	1.1	Normal	Normal	Normal	Alive	CS	N	Y
167	24	Primi	-	0	N	N	N	N	12.1	5.3	0.9	Normal	Normal	Normal	Alive	VD	N	N

168	31	Multi	n	2	N	N	N	N	11.4	4.9	0.6	Normal	Normal	Normal	Alive	VD	N	N
169	18	Primi	-	3˚cm	N	N	N	N	11.6	5	0.5	Normal	Normal	Normal	Alive	CS	N	N
170	26	Multi	n	0	N	N	N	N	12.5	5.9	0.9	Normal	Normal	Normal	Alive	VD	N	N
171	22	Primi	-	0	N	N	N	N	13.1	6.6	1	Normal	Normal	Normal	Alive	CS	N	N
172	20	Multi	n	0	N	N	N	N	12.1	5.3	1.1	Normal	Normal	Normal	Alive	VD	N	N
173	24	Primi	-	0	N	N	N	GDM	13.4	7.2	1.2	Normal	Normal	Normal	Alive	CS	N	Y
174	26	Multi	n	0	N	N	N	N	12.6	6.3	0.9	Normal	Normal	Normal	Alive	VD	LBW	Y
175	25	Multi	poor	2	N	N	N	N	12.1	5.3	1.1	Normal	Normal	Normal	Alive	VD	N	N
176	21	Primi	-	0	N	N	N	N	11.6	5	0.7	Normal	Normal	Normal	Alive	VD	N	N
177	21	Primi	-	0	N	N	N	N	11.3	4.5	1	Normal	Normal	Normal	Alive	VD	N	N
178	23	Primi	-	0	N	N	N	N	12.2	5.5	1.2	Normal	Normal	Normal	Alive	CS	N	N
179	20	Multi	n	2	N	N	N	N	12.6	6.3	1.4	Normal	Normal	Normal	Alive	VD	N	N
180	27	Multi	poor	0	N	N	N	N	13.4	7.2	1.5	Normal	Normal	Normal	Alive	VD	N	N
181	19	Primi	-	3˚cm	N	N	N	N	13.1	6.6	1	Normal	Normal	Normal	Alive	CS	N	N
182	23	Primi	-	0	N	N	N	N	13.6	7.6	1.8	Normal	Normal	Normal	Alive	VD	N	N
183	25	Multi	n	0	N	N	N	N	12.5	6.1	1.5	Normal	Normal	Normal	Alive	VD	N	N
184	21	Multi	poor	3˚cm	N	N	N	N	13.5	7.4	1.3	Normal	Normal	Normal	IUD	VD		
185	24	Multi	poor	0	N	N	N	N	13	6.4	1.1	Normal	Normal	Normal	Alive	VD	LBW	Y
186	22	Primi	-	0	N	N	N	N	12.6	6.3	0.9	Normal	Normal	Normal	Alive	CS	N	N
187	24	Multi	poor	3˚cm	N	N	N	N	13.1	6.4	1.2	Normal	Normal	Normal	Alive	VD	N	N
188	19	Primi	-	3˚cm	N	N	N	N	12.1	5.3	1	Normal	Normal	Normal	Alive	VD	N	N
189	20	Multi	n	3˚cm	N	N	N	N	13	6.4	1.1	Normal	Normal	Normal	Alive	VD	N	N
190	23	Multi	poor	0	N	N	N	N	13.4	7.2	1.2	Normal	Normal	Normal	Alive	VD	N	N
191	22	Multi	poor	0	N	N	N	N	13.2	6.8	1.5	Normal	Normal	Normal	Alive	CS	N	N
192	25	Multi	poor	3	N	N	N	N	12.4	5.8	1.1	Normal	Normal	Normal	Alive	VD	N	N
193	20	Primi	-	0	N	N	N	N	12.6	6.3	1.6	Normal	Normal	Normal	Alive	VD	N	N
194	21	Primi	-	0	N	N	N	GDM	12.4	5.9	1.5	Normal	Normal	Normal	Alive	VD	N	N
195	23	Primi	-	0	N	N	N	N	12.5	6.1	1.2	Normal	Abnormal	**Meningiomyelocele**	NND	VD	N	Y
196	25	Multi	n	0	N	N	N	N	11.3	4.5	0.9	Normal	Normal	Normal	Alive	VD	N	N
197	20	Primi	-	0	N	N	N	N	12.6	6.4	1.1	Normal	Normal	Normal	Alive	VD	N	N
198	37	Multi	n	0	N	N	N	N	13.3	7	1	Normal	Normal	Normal	IUD	VD		
199	24	Primi	-	0	N	N	N	N	12	5.3	0.9	Normal	Normal	Normal	Alive	VD	N	N
200	23	Multi	n	0	N	N	N	N	13.1	6.7	1.2	Normal	Normal	Normal	Alive	VD	N	N
201	19	Primi	-	0	N	N	N	N	12.5	6.1	1.2	Normal	Normal	Normal	Alive	VD	N	N
202	20	Multi	poor	3˚cm	N	N	N	N	11.3	4.6	1.1	Normal	Normal	Normal	Alive	CS	N	N
203	24	Multi	n	0	N	N	N	N	11.6	5.2	1	Normal	Normal	Normal	Alive	CS	N	N
204	23	Multi	poor	0	N	N	N	GDM	11.6	5.2	0.8	Normal	Normal	Normal	Alive	CS	N	Y
205	24	Multi	poor	2˚cm	Y	N	N	N	12.6	6	1.5	Normal	Normal	Normal	Alive	VD	N	N
206	36	Multi	n	0	N	N	N	N	13.6	7.6	1.4	Normal	Normal	Normal	Alive	CS	N	Y
207	19	Multi	poor	0	N	N	N	N	13.3	7.2	0.9	Normal	Normal	Normal	Alive	VD	N	N
208	27	Multi	n	0	Y	N	N	N	12.1	5.4	1.1	Normal	Normal	Normal	Alive	VD	N	N
209	22	Multi	poor	0	N	N	N	N	12.1	5.5	1.2	Normal	Normal	Normal	Alive	VD	N	N
210	28	Primi	-	0	N	N	N	N	13.4	7.1	1.7	Normal	Normal	Normal	Alive	for	N	Y
211	22	Multi	poor	0	N	N	N	N	12.3	5.6	0.2	Normal	Normal	Normal	Alive	VD	N	N
212	36	Multi	n	3˚cm	N	N	N	N	12	5.1	0.9	Normal	Normal	Normal	Alive	VD	N	N
213	21	Multi	n	0	N	N	N	N	12.4	5.8	0.9	Normal	Normal	Normal	Alive	VD	N	N

214	26	Primi	-	0	N	N	N	N	13.1	6.7	1.2	Normal	Normal	Normal	Alive	VD	N	N
215	38	Primi	-	0	N	N	N	N	13.6	7.1	1.5	Normal	Normal	Normal	Alive	VD	N	N
216	23	Primi	-	0	N	N	N	N	11.6	5.2	1.7	Normal	Normal	Normal	Alive	VD	N	N
217	29	Primi	-	0	N	N	N	N	11.6	5.2	1.1	Normal	Normal	Normal	Alive	VD	N	N
218	35	Multi	poor	0	N	N	N	N	13	6.4	1.9	Normal	Normal	Normal	Alive	VD	N	N
219	35	Multi	n	0	N	N	N	DM	13.6	7.6	1.2	Normal	Normal	Normal	Alive	CS	N	N
220	27	Primi	-	0	N	N	N	GDM	11.4	4.6	0.8	Normal	Normal	Normal	Alive	VD	N	N
221	25	Multi	poor	0	N	N	N	GDM	12.1	5.6	1.8	Normal	Normal	Normal	Alive	CS	N	N
222	25	Primi	-	0	N	N	N	N	11.6	5.2	0.9	Normal	Normal	Normal	Alive	VD	N	N
223	18	Multi	poor	0	N	N	N	HD	11.4	4.6	0.8	Normal	Normal	Normal	Alive	CS	N	N
224	20	Primi	-	0	N	N	Y	N	12	5.1	0.9	Normal	Abnormal	**cleftlip and cleftpalate**	Alive	VD	N	Y
225	25	Primi	-	0	N	N	N	HD	12.1	5.3	1	Normal	Normal	Normal	Alive	For	N	Y
226	25	Primi	-	0	N	N	Y	GDM,EPI	13.2	6.8	1.2	Normal	Normal	Normal	IUD	CS		
227	26	Primi	-	3˚cm	N	N	N	N	12.1	5.3	1.1	Normal	Normal	Normal	Alive	VD	N	N
228	28	Primi	-	0	N	N	N	N	13.1	6.75	**3.07**	Normal	Normal	Normal	Alive	CS	N	N
229	23	Multi	n	0	N	N	N	N	12.6	6.3	1.2	Normal	Normal	Normal	Alive	CS	N	N
230	22	Primi	-	0	N	N	N	N	13.2	6.8	1.4	Normal	Normal	Normal	Alive	CS	N	N
231	20	Primi	-	3˚cm	N	N	N	N	12.5	6.1	1	Normal	Normal	Normal	Alive	VD	N	N
232	22	Multi	poor	2˚cm	N	N	N	N	12.1	5.3	0.9	Normal	Normal	Normal	Alive	VD	N	Y
233	22	Multi	poor	0	N	N	N	N	13.2	6.8	1.8	Abnormal	Normal	**Anencephaly**	TOP	MTP1		
234	32	Multi	n	0	N	N	N	N	11.6	5	0.9	Normal	Normal	Normal	Alive	VD	Y	Y
235	22	Multi	n	0	N	N	N	N	12.4	5.9	1.2	Normal	Normal	Normal	Alive	VD	Y	Y
236	19	Primi	-	3˚cm	N	N	N	N	13.4	7.2	1.4	Normal	Normal	Normal	Alive	VD	N	N
237	25	Multi	poor	0	N	N	N	N	13.6	7.6	1.7	Normal	Normal	Normal	Alive	VD	N	N
238	21	Primi	-	3˚cm	N	N	N	N	13.2	6.8	1.2	Normal	Normal	Normal	Alive	VD	Y	Y
239	23	Primi	-	0	N	N	N	N	12.5	6.1	1.3	Normal	Normal	Normal	Alive	VD	N	N
240	21	Primi	-	0	N	N	N	N	13	6.4	1.5	Normal	Normal	Normal	Alive	VD	N	N
241	19	Multi	poor	0	N	N	N	N	12.4	5.9	1.1	Normal	Normal	Normal	Alive	CS	N	N
242	30	Multi	poor	0	N	N	N	N	12.5	6.1	1.1	Normal	Normal	Normal	Alive	CS	N	N
243	23	Multi	n	0	N	N	N	N	12.6	6.3	1.2	Normal	Normal	Normal	Alive	VD	N	N
244	20	Primi	-	0	N	N	N	N	12	5.3	1.1	Normal	Normal	Normal	Alive	VD	N	N
245	21	Primi	-	0	N	N	N	N	12.4	5.9	1.2	Normal	Normal	Normal	Alive	CS	N	N
246	19	Primi	-	0	N	N	N	N	13	6.4	1.5	Normal	Normal	Normal	IUD	VD		
247	27	Multi	poor	0	N	N	N	N	13.6	7.6	1.6	Normal	Normal	Normal	Alive	VD	N	N
248	23	Multi	n	3˚cm	N	N	N	N	11.6	5	0.9	Normal	Normal	Normal	Alive	CS	N	N
249	22	Primi	-	0	N	N	N	N	12.3	5.7	1	Normal	Abnormal	**Hydrocephalus**	TOP	MTP2		
250	23	Primi	-	0	N	N	N	N	12.2	5.5	1	Normal	Normal	Normal	Alive	VD	N	N
251	20	Multi	poor	0	N	N	N	N	12.6	6.3	1.1	Normal	Normal	Normal	Alive	CS	N	Y
252	25	Multi	poor	0	N	N	N	N	13.1	6.6	1.4	Normal	Normal	Normal	Alive	CS	LBW	Y
253	22	Primi	-	0	N	N	N	N	13.2	6.8	1.3	Normal	Normal	Normal	Alive	CS	LBW	Y
254	23	Multi	n	0	N	N	N	N	13	6.4	1.5	Normal	Normal	Normal	Alive	VD	N	N
255	20	Multi	n	0	N	N	N	N	12.4	5.9	1.1	Normal	Normal	Normal	Alive	VD	N	N
256	30	Multi	n	0	N	N	N	N	12.2	5.5	1.1	Normal	Normal	Normal	Alive	VD	N	N
257	26	Multi	poor	2˚cm	N	N	N	N	12	5.3	1.2	Normal	Normal	Normal	Alive	CS	N	N
258	23	Multi	n	0	N	N	N	N	12.1	5.3	1.1	Normal	Normal	Normal	Alive	VD	N	N
259	23	Multi	poor	0	N	N	N	N	11.6	5	0.9	Normal	Normal	Normal	Alive	VD	N	N

260	26	Multi	n	0	N	N	N	N	11.5	4.8	0.7	Normal	Normal	Normal	Alive	CS	N	N
261	28	Multi	n	2˚cm	N	N	N	N	12	6.1	1.1	Normal	Normal	Normal	Alive	VD	N	N
262	23	Multi	n	0	N	N	N	N	12.5	6.1	1.2	Normal	Normal	Normal	Alive	VD	LBW	Y
263	25	Multi	n	3˚cm	N	N	N	N	13.5	7.4	1.1	Normal	Normal	Normal	Alive	VD	N	N
264	26	Multi	poor	0	Y	N	N	N	13.1	6.6	1.3	Normal	Normal	Normal	Alive	VD	N	N
265	20	Primi	-	0	N	N	N	N	13.6	7.6	1.5	Normal	Normal	Normal	Alive	CS	N	N
266	19	Primi	-	0	N	N	N	N	13	6.4	1.1	Normal	Normal	Normal	Alive	VD	N	N
267	20	Multi	n	0	N	N	N	N	12.5	6.1	1.2	Normal	Normal	Normal	Alive	CS	N	N
268	28	Primi	-	0	N	N	N	N	12.4	5.9	1.3	Normal	Normal	Normal	Alive	VD	N	N
269	29	Multi	poor	0	N	N	N	N	12.3	5.7	1	Normal	Normal	Normal	Alive	CS	N	N
270	22	Primi	-	0	N	N	N	N	12.1	.5.3	1.1	Normal	Normal	Normal	Alive	VD	N	N
271	28	Multi	n	0	N	N	N	N	12	6.1	1.1	Normal	Normal	Normal	Alive	VD	N	N
272	20	Primi	-	0	N	N	N	N	12.2	5.5	1.2	Normal	Normal	Normal	Alive	CS	N	N
273	22	Primi	-	0	N	N	N	N	13	6.4	1.4	Normal	Normal	Normal	Alive	VD	N	N
274	22	Multi	poor	0	N	N	N	N	11.6	5	0.9	Normal	Normal	Normal	Alive	CS	N	N
275	28	Primi	-	0	N	N	N	N	13.4	7.2	1.6	Normal	Normal	Normal	Alive	VD	N	N
276	38	Multi	poor	0	N	N	N	N	12.6	6.3	1.4	Normal	Normal	Normal	Alive	CS	N	N
277	32	Primi	-	0	N	N	N	DM	12.1	5.3	1.1	Normal	Normal	Normal	Alive	CS	N	Y
278	25	Multi	n	0	N	N	N	N	12.5	6.1	1.2	Normal	Normal	Normal	Alive	VD	LBW	Y
279	26	Primi	-	0	N	N	N	N	11.6	5	0.9	Normal	Normal	Normal	Alive	CS	LBW	Y
280	22	Primi	-	0	N	N	N	N	12.2	5.5	1.2	Normal	Normal	Normal	Alive	VD	N	N
281	28	Multi	n	2˚cm	N	N	N	N	13.2	6.8	1.6	Normal	Normal	Normal	Alive	CS	N	N
282	28	Multi	n	0	N	N	N	N	13	6.4	1.5	Normal	Normal	Normal	Alive	VD	N	N
283	25	Multi	n	0	N	N	N	N	12.2	5.5	1.1	Normal	Normal	Normal	Alive	VD	N	N
284	22	Multi	n	0	N	N	N	N	12.5	6.1	1	Normal	Normal	Normal	Alive	vD	N	N

KEYS TO MASTER CHART

n Normal
IUD- intrauterine death
poor bad obstetric history
TOP- Termination of Pregnancy
N- No
NND- Neonatal death
Y- Yes
LBW- Lowbirth weight
VD- Vaginal delivery
MTP 1 First trimester medical termination of pregnancy
CS- cesarean section
MTP2 Second trimester medical termination of pregnancy
For - forceps delivery
HYS-Hysterotomy