PREVALENCE OF CONGENITAL ANOMALIES AND ASSESSMENT OF ASSOCIATED RISK FACTORS: A ONE-YEAR HOSPITAL BASED STUDY AT KLE DR. PRABHAKAR KORE HOSPITAL

BY

REG.NO. BJ0116007

Dissertation

Submitted to the
KLE Academy of Higher Education and Research,
Belagavi,Karnataka

In Partial Fulfillment
of the requirements for the degree of

MASTER OF SURGERY
in
OBSTETRICS AND GYNAECOLOGY

DEPARTMENT OF OBSTETRICS AND GYNAECOLOGY,
JAWAHARLAL NEHRU MEDICAL COLLEGE,
BELAGAVI, KARNATAKA

APRIL – 2019

KLE ACADEMY OF HIGHER EDUCATION AND RESEARCH, BELAGAVI, KARNATAKA

ENDORSEMENT BY THE HOD, PRINCIPAL/ HEAD OF THE INSTITUTION

This is to certify that this dissertation entitled **"PREVALENCE OF CONGENITAL ANOMALIES AND ASSESSMENT OF ASSOCIATED RISK FACTORS: A ONE-YEAR HOSPITAL BASED STUDY AT KLE DR. PRABHAKAR KORE HOSPITAL"** is a bonafide research work done by **REG.NO.BJ0116007.**

Dr. M.B.BELLAD **M.D,FICOG**
Professor & Head
Department of Obstetrics
and Gynaecology
Belagavi – 590010

Date:
Place: BELAGAVI

Dr. N.S.Mahantshetti **M.D.(Paed)**
Principal
J.N.Medical College
Belagavi - 590010

Date:
Place: BELAGAVI

LIST OF ABBREVIATIONS USED

WHO	:	World Health Organization
CVS	:	Cardiovascular system
CNS	:	Central nervous system
NTD	:	Neural Tube defects
ICD	:	International Classification of Diseases
CA	:	Congenital Anomalies
LMIC	:	Low and Middle Income Countries
BMI	:	Body Mass Index
IMR	:	Infant Mortality Rate
NA	:	Not Applicable
CHD	:	Congenital Heart Disease

ABSTRACT

Title:

Prevalence of Congenital anomalies and assessment of associated maternal risk factors: A One- year hospital based study at KLES Dr. Prabhakar Kore Charitable Hospital, Belagavi.

Introduction:

Congenital anomalies are an important cause of perinatal mortality and lifelong disability. In India, congenital anomalies have been reported to be responsible for 15% of perinatal mortality, and are now known to be the third most common cause of perinatal mortality following infections and hypoxia, and it is likely to become the leading cause. Some of these congenital anomalies can be prevented. Therefore, it is important to understand the etiological factors and plan the preventive strategies. Early diagnosis is important and early surgical treatment when required can prevent neonatal death and help in better survival of the child.

Aim and objectives:

To study the prevalence of congenital anomalies among the total births occurring in department of OBG at KLES Dr. Prabhakar Kore Charitable Hospital Belagavi and to assess the significance of the associated risk factors.

Materials and methods:

The study undertaken is a hospital based study. All live born and still born babies diagnosed with congenital anomalies, delivering at the department of OBG of the hospital between January 2017 to December 2017 were included. System wise categorization of the anomalies was done using ICD 10. The prevalence is calculated

and summarized data was analysed using descriptive statistical measures. Measures of frequency including percent are used. Prevalence was calculated. Assessment of each risk factor was done. Statistical analysis was done using Independent t test and Chi-square test.

Results:

Out of the total 5755 deliveries, total number of babies with congenital malformations was 111(1.92 %). CNS anomalies constituted 25.2% of the total anomalies, followed by genitourinary and CVS anomalies.

Conclusion:

CNS, genitourinary and cardiovascular abnormality constitutes the majority of cases. Antenatal evaluation of CNS and cardiovascular system in high risk mothers is essential. Awareness about preventable risk factors is also equally important.

Keywords: congenital anomalies, malformations, birth defects, risk factors

LIST OF CONTENTS

LIST OF TABLES

LIST OF GRAPHS

INTRODUCTION

As per WHO, Congenital anomalies are defined as structural or functional anomalies that occur during intrauterine life and can be identified prenatally, at birth or later in life.[1] Birth defects are of prenatal origin which result from defective embryogenesis or intrinsic abnormalities during the process of development. They can either be isolated abnormalities or can be part of a syndrome. Birth defects have been a major cause of neonatal and infant morbidity and mortality.

Congenital anomalies contribute significantly to infant and childhood deaths, chronic illness and disability. [1] Not only causing mortality and fetal loss, but they can also result in preterm birth, childhood and adult morbidity as well as a considerable repercussion on the mothers and their families.

As per the global estimates, congenital anomalies affect 2-3% of births[33.] Every year, an estimated 303 000 newborns die within 4 weeks of birth, worldwide, due to congenital anomalies.[1]

Serious birth defects can most often be lethal. For those who survive, these disorders can cause lifelong mental, physical, auditory or visual disability. Annually at least 3.3 million children less than 5 years of age die because of serious birth defects and the majority of those who survive are mentally and physically disabled for life. [2]

Due to lack of national birth defects surveillance, the true magnitude of the number of births affected by congenital anomalies in India is unknown.

There is a need for accurate data on congenital anomalies in India as currently there is no data on the impact of congenital anomaly affected pregnancies or births on the health service utilization, and also to derive estimates of the number of children born with disabling conditions.

In India, the incidence of Congenital anomalies is around 2.5% and they account for 8-15% of perinatal deaths and 13-16% of neonatal deaths. [29,30] Community based study by Indian council of Medical Research (ICMR) reported that 6.6% of neonatal deaths in the rural as well as urban slum communities are accounted for by congenital malformations. [28] Till the past decade, the highest contributors to neonatal deaths were preterm births (34.7%), intrapartum complications (19.6%), pneumonia (16.3%) and neonatal sepsis (15%), and congenital anomalies constituted the fifth largest cause, accounting for 9% of neonatal deaths. [32]

In India, congenital malformations are now known to be the third common cause of perinatal mortality after infections and hypoxia. [31] However, incidences in death due to congenital malformation are increasing, with decreasing mortality due to infection and nutritional disorders as a result of reduction of mortality due to the latter causes owing to the improvement in perinatal and neonatal care. Thus, India is undergoing an epidemiological transition.

A recent study showed that as compared to 9% of perinatal deaths a decade back, congenital malformations now contributed to a big number, that is 13.4 %. Major malformations accounts for nearly 15% of neonatal death. [9] With the passing decades, congenital anomalies are likely to become a leading cause of morbidity and mortality in centres providing good neonatal care.

According to RCOG, malformations are classified into lethal, severe and moderate ones. Lethal defects include anencephaly, bilateral renal agenesis, giant hygroma, osteochondrodysplasia, ichthyosis congenita. Severe defects include hydrocephalus, spina bifida, esophageal atresia, TOF, ectodermal dysplasia, PUV, ASD, PDA. Moderate defects are septal deviation, choanal atresia, craniosynostosis, eyelid defects.[13]

The International Statistical Classification of Diseases and Related Health problems 10th revision given by WHO in 2010 has a Chapter XVII under section Q00-Q99 for Congenital malformations, deformations and chromosomal abnormalities. Various studies show different prevalence based on the type of study, inclusion of minor defects, still births, and follow up. Data available from Birth Defect Registry of India (BDRI) show that the common systems involved are central nervous system, musculoskeletal system and cardiovascular system, with neural tube defects being the most common. [34]

However, about 40-60% of the anomalies are caused by unknown etiologies, referred to as sporadic birth defects. These have low risk of recurrence in the subsequent pregnancies and may imply a random occurrence. The cause of another 20-25% of congenital anomalies seem to be multifactorial, with a complex interaction of genetic, infections, metabolic diseases, drugs and environmental factors. [13]

The genetic causes may be numerical, structural defects and mosaicisms. Downs syndrome is the commonest, followed by Edwards, Patau. Structural anomalies are deletion, translocation and inversion. Turners and Klinefelters are sex chromosomal abnormalities.

Infections like toxoplasmosis, CMV, Rubella, Herpes, Syphilis can cause anomalies. Uncontrolled diabetes in the period of organogenesis increases the risk to the fetus up to 5-6%. High HbA1c levels in first trimester directly correlate with the incidence. Teratogenic drugs include Thalidomide, antiepileptics, Warfarin, Retinoic acid. Maternal exposure to alcohol, smoking also play a role.

It has been proved that deficiency of folate leads to neural tube defects in fetus. Nervous system becomes evident on day 18 post ovulation. Neural tube closes by day 22-28. Folate deficiency causes developmental delay through disturbances of DNA biosynthesis and/or methylation cycle, during the embryogenesis [36] However, some anomalies may not show a definitive etiology, for example hydrocephalus, renal anomalies, abdominal wall defects.

Some congenital anomalies can be prevented. Identification of various risk factors and modifying them can prevent these anomalies to some extent. Vaccination, adequate antenatal care, adequate intake of folic acid or iodine through fortification of staple foods or supplementation, are to be considered[1.] Therefore, it is important to understand the etiological factors and plan the preventive strategies. Also, early diagnosis and early surgical treatments when required can prevent neonatal death and help in better survival.

OBJECTIVES

1. To know the prevalence of Congenital anomalies among the total births at KLES Dr. Prabhakar Kore Charitable Hospital.
2. To assess the significance of associated risk factors.

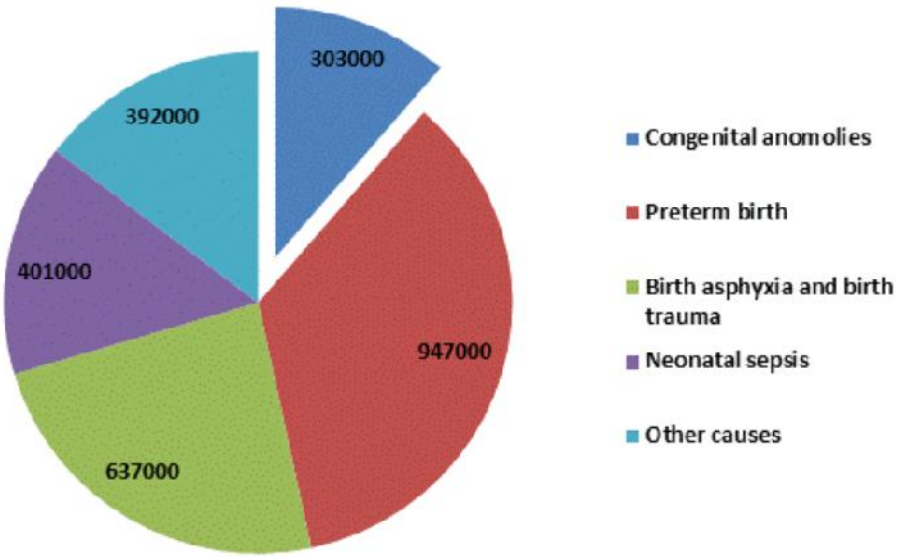
REVIEW OF LITERATURE

Congenital anomalies can be defined as per WHO as structural or functional anomalies (for example, metabolic disorders) that occur during intrauterine life and can be identified prenatally, at birth, or sometimes may only be detected later. Congenital anomalies are also known as birth defects, congenital disorders or congenital malformations [1]

Globally, 2-3 per 100 children are born with birth defects. Every year, congenital anomalies affect approximately 1 in 33 infants and result in approximately 3.2 million birth defect-related disabilities. Worldwide, an estimated 303000 newborns die during first 28 days of life every year from congenital anomalies. [1]

Birth defects are a global problem, but their impact is however severe in middle- and low- income countries. [2]

Figure 1: Causes of 2.68 million deaths during the neonatal period in 2015, worldwide (WHO 2000-2015 child causes of death)



The Global Burden of Disease study (2013) noted congenital anomalies to be among the top ten causes of mortality in children less than five years of age. While congenital anomalies are the leading cause of death in children in this age group in the high-income countries, they are not generally considered to be significant public health problems in low- and middle-income countries (LMICs). [16]

Globally, child mortality trends are showing a decrease, with reduction in mortality due to infections and malnutrition. However, this reduction is likely to be accompanied by a transition in the causes of child mortality in these countries, with a proportionate increase in no communicable conditions like congenital anomalies. The epidemiological transition is being clearly witnessed in the differences in the urban and rural mortality causes and rates. For example, in India , urban infant mortality rate (IMR) is 27 as compared to rural IMR of 44 per 1000 live births, with sepsis, pneumonia and diarrhoea being major causes of mortality in rural areas. [16] With the availability of standard maternal and paediatric services, the transition in causes of mortality in urban areas is likely to be accelerated, resulting in increasing contribution of congenital anomalies to neonatal deaths.

Not all congenital anomalies are lethal. Babies born with several types of non-lethal anomalies would survive with disability or need lifelong care, often leading to large expenditure for affected families.

There is scant data on the number of live born children with birth defects. Congenital anomalies are not yet considered to be a priority health problem in India. In 2010, congenital anomalies were estimated to be the fifth largest cause of neonatal deaths in India after preterm births (34.7%), intra partum complications (19.6%), pneumonia (16.3%) and neonatal sepsis (15%). in 2013, Congenital anomalies were estimated to contribute to 60 699 neonatal deaths in India, which accounted for the

highest global burden of neonatal mortality due to congenital anomalies. India lacks a national birth defects surveillance, so there is no clear data on the magnitude of congenital anomalies in the country. But it is time to realise that congenital malformations are emerging as important perinatal problem contributing largely to the perinatal mortality and morbidity with considerable repercussion on the mother and the families affected. [16]

Data on the magnitude of congenital anomalies are also needed as some of these conditions can be prevented, through primary care interventions targeted towards women in the preconception, intra-conception and antenatal periods. Strategies targeting the prevention of births affected by congenital anomalies also target the shared risk factors for other adverse pregnancy outcomes, effectively aiming at reduction of reproductive wastage, and improving pregnancy outcome [17]

The temporal trends in the occurrence of birth defects are even more concerning. The occurrence of birth defects, has not decreased for many decades. Few localized exceptions are for neural tube defects in countries that implemented folic acid fortification. Birth defects might even increase worldwide, with the alarming increase of known risk factors such as maternal diabetes and obesity. New threats such as the Zika epidemic are also emerging. Unless progress is made in identifying and preventing the root causes of birth defects, these conditions will continue to have significant effects on the survival and health of individuals, families, and countries. Progress in detecting and characterizing risk factors for birth defects has come mainly from epidemiologic studies. Such studies have in fact produced many associations between risk factors and the groups of birth defects. However, translating these associations to actual causes has been difficult. [20]

There are differences in birth defect rates in different countries and studies. This could be attributed to the true differences among different populations or to different definitions of birth defects, different methods, different time periods for ascertainment. [22]

Conditions that may contribute to higher incidence of birth defects in Developing countries: [25]

1. Inadequate periconceptional intake of folic acid

2. Iodine deficiency in mothers diet

3. Lack of vaccination against Rubella

4. Women giving birth after 35 years of age

5. Consanguineous marriage

6. Alcohol consumption during pregnancy

7. The use of teratogenic medications and oral contraceptives

8. Low birth weight

Interventions to reduce the impact of Birth defects in India**:**

If there is a lack of public health support for treatment, there's going to be lifelong suffering. Many of the tools to prevent birth defects are present in the existing reproductive and child health programme. Some additions may help in modelling a prevention programme in order to address this invisible public health problem. Prevention of birth defects depends on risk identification and management through community and health service personnel education, population screening, genetic counselling and the availability of appropriate services. Effective preventive services should include basic reproductive health services and medical genetic screening. [25]

The recommendations to reduce the impact of birth defects in India are : Firstly, education of the public about congenital anomalies. Second, to conduct population based studies or surveys. And third, but most importantly, prevention. Prevention strategies include primary, secondary and tertiary levels of prevention. These strategies can be applicable in various stages of pregnancy including pre-conception, antenatal and postnatal period.

To discuss the national data, a community based study by Indian Council of Medical Research (ICMR) reported that congenital malformations accounted for 6.6% of neonatal deaths in the rural as well as urban slum communities. The true incidence of congenital malformations however depends upon several factors. No two studies are strictly comparable. It depends upon the population sample (hospital or community based), nature of study (prospective or retrospective), age at the time of diagnosis, duration of follow up , autopsy rate, diagnostic facilities available and accuracy of diagnosis.

A study conducted in Maharashtra in 2014 showed a prevalence rate of 2.69% at birth, and out of that, malformations involving CVS was highest (29.6%). They noted that by improvement in antenatal, postnatal diagnosis, early referral to tertiary hospital and early intervention most of these newborns can be saved. [3]

A hospital based study conducted in Central India in 2005-2007 showed that congenital malformations were significantly more in stillbirths ($p<0.01$) as compared to live births, the frequency being 4.68% and 1.84%, respectively. The mothers of babies with congenital anomalies were mostly between 20 and 30 years (90.49%). History of parental consanguinity was present in 14 cases. History of oligohydramnios was seen in 7.26% and polyhydramnios in 3.91%. 10.61% had history of previous abortions, 3.35% were diabetic mothers and 2.79% had history of congenital heart

disease in previous child or malformed babies. Cardiovascular malformations were most common in live births, followed by musculoskeletal malformations. The CNS defects were most commonly seen in still born. [4]

According to a study conducted in 2011-2012 in Eastern India, prevalence rate of congenital anomalies was 2.22%. [5] The predominant system involved was musculoskeletal system (33.2%) followed by gastro intestinal system (15%) and Central nervous system (11.2%). [5] A study conducted in South India between 2011-2014 showed the major Congenital anomalies were Central Nervous System (CNS) followed by renal anomalies. Maternal age (<=25 years), Paternal age (<30 years), primigravida and consanguinity contributed to the burden of Congenital anomalies in high risk pregnancies. Consanguinity was found to be a predisposing factor for congenital anomalies in high risk pregnancies with previous BOH while Toxoplasma seropositivity conferred risk for pregnant women with Congenital anomalies in present pregnancy with previous normal pregnancies. [7]

A study conducted in North India in 2008 showed cardiovascular congenital malformations to be the commonest (27.1%), followed by gastrointestinal (23.1%), genitourinary (14.2%), CNS (12.8%). Non intake of folic acid in pregnancy was found to be significantly associated with congenital malformations. History of drug usage (pain killers, paracetamol, drugs for morning sickness, antibiotics) was found to be significantly associated with anomalies. Maternal age at conception <20 years to be significantly associated with congenital malformation. [6] Blencowe et al concluded that folic acid supplementation and fortification are effective in reducing neonatal mortality from NTDs. [38]

Mao B, Qiu J conducted a cohort study in 2010-2012 in China, and concluded that folic acid supplementation before pregnancy was associated with a reduced risk

of congenital heart diseases. A lower dietary folate intake during pregnancy had an increased risk for the same.[37] Aggarwal et al study and Chaturvedi and Banerjee study found the previous history of abortion to be the most significantly associated with Congenital malformations.[30] Ronya et al found higher incidence of congenital malformation with previous history of congenital malformation.[35]

Henry and Varma showed positive relation between threatened abortion and life threatening congenital malformation. Women with pre-existing diabetes had a much higher prevalence overall of offspring with major defects than did women without diabetes (5.88% *vs*1.34%), and those with gestational diabetes had no appreciable excess of defects (1.38%). In this centre of study, there has been no study conducted to assess the maternal risk factors associated with the Congenital anomalies. Hence, the study has been taken up to assess the prevalence and the associated maternal risk factors.

METHODOLOGY

Source of data and materials

Patients attending Department of OBG at KLES Dr. Prabhakar Kore Charitable Hospital and MRC, Belagavi

Study design: Cross sectional study

Duration of the study: One year.

Period of study: January 2017 to December 2017

Selection of cases:

Inclusion criteria:

- All live born and still born babies diagnosed with congenital anomalies, delivering at Department of OBG at KLES Dr. Prabhakar Kore Hospital.

Exclusion criteria:

- Patients who do not give consent for participation in the study.

Methodology:

All the women delivering babies with congenital anomalies, diagnosed antenatally or detected at birth or post delivery at KLES Dr. Prabhakar Kore Charitable Hospital, were identified.

With the approval of the Ethical committee, written informed consent was taken from all the participants enrolled for the study. Participation was voluntary and

they were assured that confidentiality and anonymity would be maintained throughout the study after explaining the purpose of study.

They were interviewed, relevant history noted and data collected from clinical records.

Information regarding the newborn was collected from clinical records. Diagnosis of congenital anomalies was based on clinical evaluation of newborn babies by the pediatrician and other appropriate investigations. In cases of stillbirths or abortions, autopsy report was collected and the findings noted. System wise distribution of the anomalies was performed.

Factors studied included type of conception, non intake of folate by mother in pregnancy, antenatal check-ups, history of fever in pregnancy, contraception use, previous history of congenital malformation, history of abortion, history of drug intake, age of mother at marriage , age of mother at first pregnancy, history of consanguinity, alcohol, smoking and tobacco habituation in father/mother, family history of congenital malformations, diabetes, Preeclampsia.

Sample size:

All babies born with Congenital anomalies at KLES Dr. Prabhakar Kore Hospital in one year.

Statistical Analysis plan:

The collected data was summarized using descriptive statistical measures and prevalence was calculated. System wise distribution was done. The data analysis was done to find whether there is a statistical association between each risk factor included in the study and the congenital anomaly. Statistical analysis was done using Independent t test and Chi-square test, with significance level at 5% ($p<0.05$ considered to be statistically significant).

RESULTS

The present one year hospital based study was conducted in the labour room of KLE'S Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum during the period of January 2017 to December 2017.

Out of the total 5755 deliveries, total number of babies with congenital malformations was 111.

The data collected was entered into the Microsoft Excel spreadsheet. The data was analysed and the final results and observations were interpreted as follows.

Total number of congenital anomalies:

Total No of deliveries	**5755**
Total No of congenital anomalies	111
	1.92%

Out of the total 5755 deliveries, total number of babies with congenital malformations was **111** (**1.92%**).

Maternal characteristics:

Table 1: Age wise distribution

Age groups	Number	Percentage
<=20yrs	18	16.22%
21-30yrs	80	72.07%
>=31yrs	13	11.71%
Total	**111**	**100.00%**

Majority of the congenital anomalies were present in maternal age group of **21-30 years,** n=80 (72.0%)

Graph 1: Distribution of patients by age groups

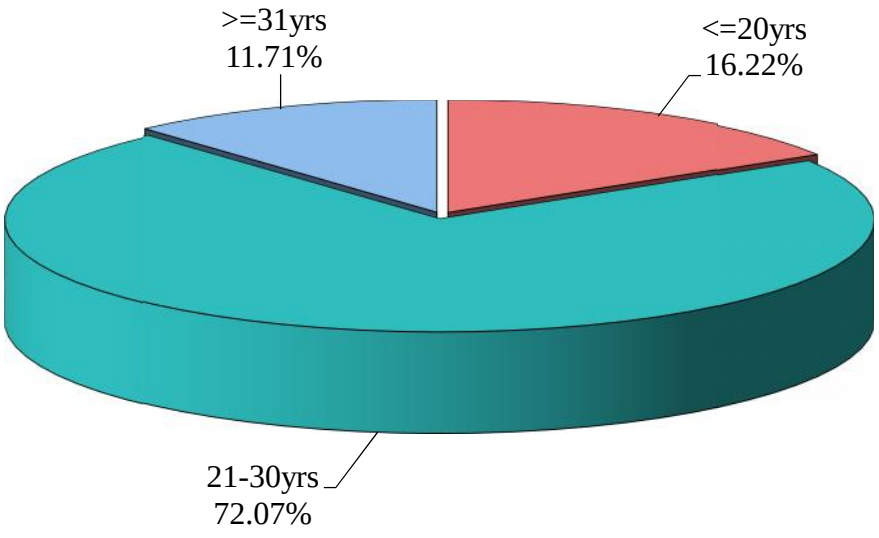


Table 2: BMI distribution

BMI (kg/m^2)	Number	Percentage
<= 20	9	8.1%
21-30	101	84.1%
>30	1	0.9%
Total	**111**	

Most of the women had BMI between 21-30 kg/m^2, n= 101. Nine cases had BMI <=20 kg/m^2. One case had BMI of more than 30kg/ m^2 .

Graph 2: BMI distribution (in kg/m$^{2)}$

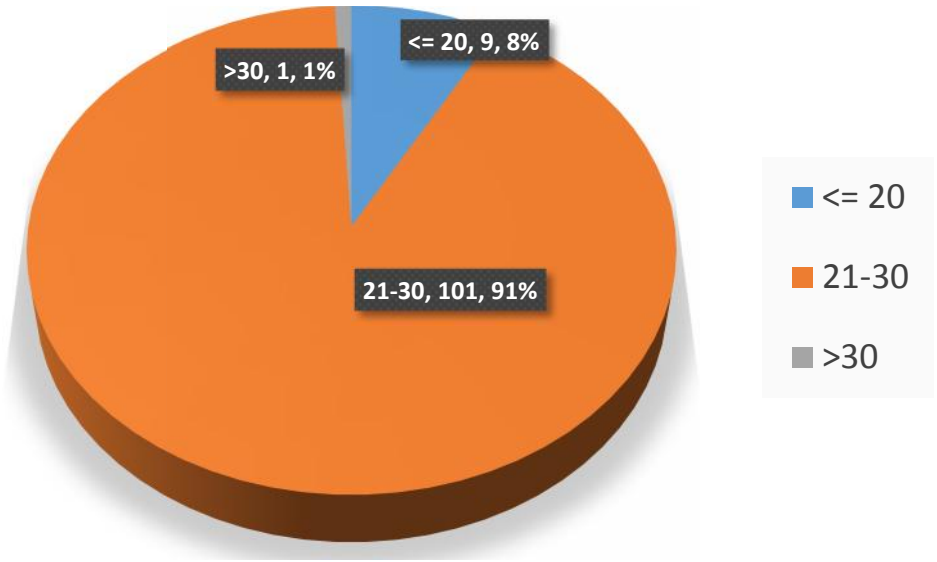


Table 3: Parity distribution

Parity	Number	Percentage
Primi	40	36%
Multi (G2-3)	56	50%
Multi (G4 and more)	15	13.5%
Total	**111**	100%

50% of the cases were of multigravida (G2-3), total of 56 cases. 40 cases were of primis. 15 cases were of gravida 4 or more.

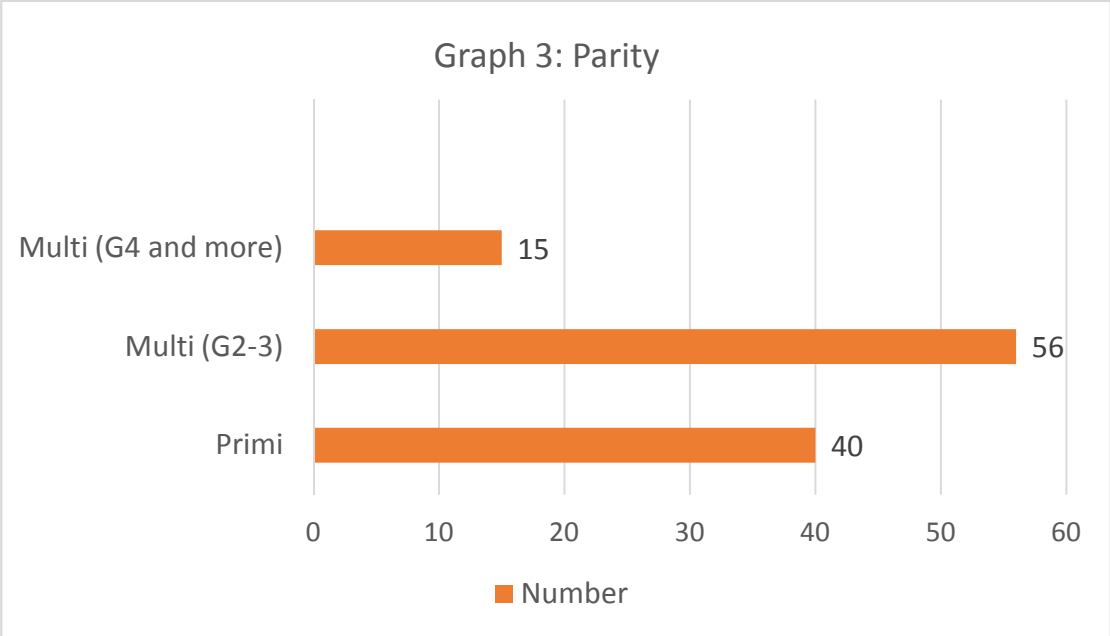
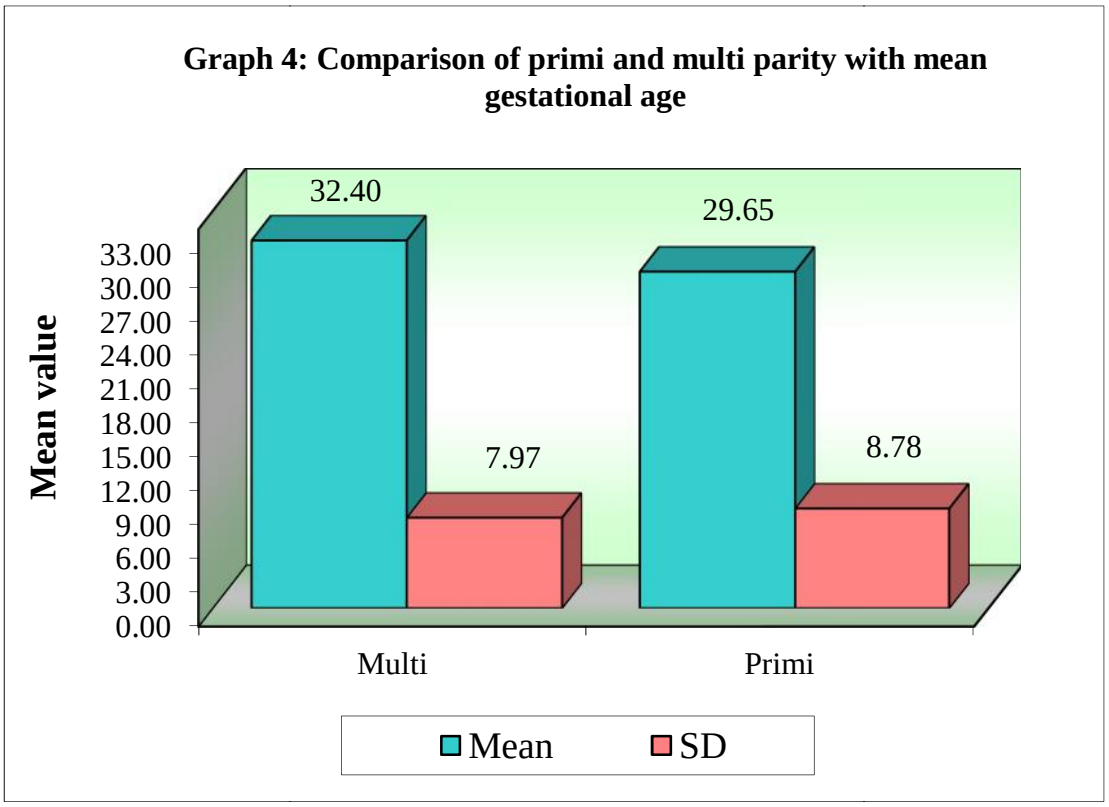


Table 4: Comparison of primi and multi parity with mean gestational age by t test.

Parity	n	Mean	SD	SE	t-value	P-value
Multi	40	32.40	7.97	1.26	1.6373	0.1045
Primi	71	29.65	8.78	1.04		



System wise distribution of the anomalies

Table 5: System wise distribution of the anomalies

System	Number of cases	Percentage
CNS	28	25.2%
Genitourinary	21	18.9%
Cardiovascular	18	16.2%
Musculoskeletal	14	12.6%
GIT	10	9.0%
Respiratory system	7	6.3%
Multiple system affected	7	6.3%
Others	6	5.4%
TOTAL	**111**	**100%**

CNS anomalies were 25.2%, followed by genitourinary (18.9%) and CVS anomalies (16.2%)

Graph 5: System Wise Distribution

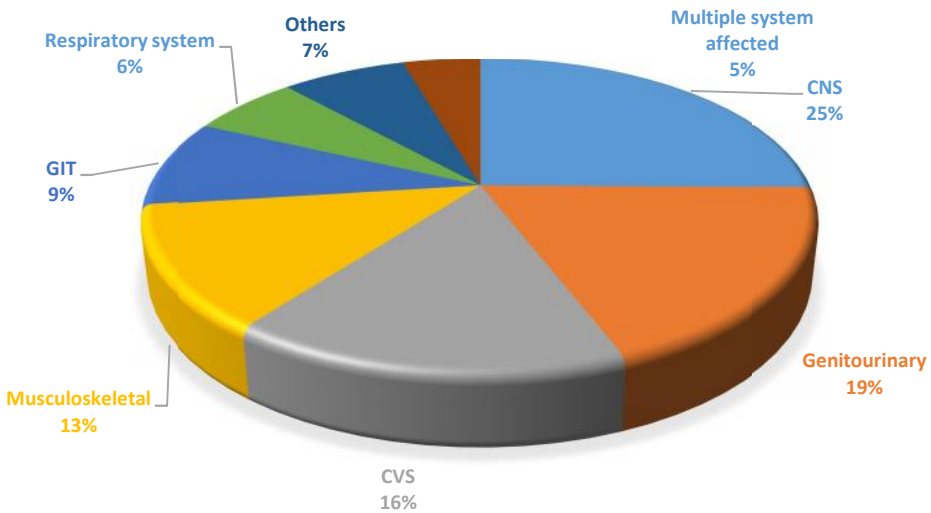


Table 6: Anomalies under Central nervous system

CNS	n = 28 (25.2%)	% to system	% to total
Open spina bifida	8	28.5%	7.2%
Anencephaly	5	17.8%	4.5%
Encephalocele	5	17.8%	4.5%
Ventriculomegaly	5	17.8%	4.5%
Dandy Walker malformation	3	10.7%	2.7%
Multiple CNS anomaly	1	3.5%	0.9%
Holoprocencephaly	1	3.5%	0.9%

Table 7: Anomalies under Genitourinary system

Genitourinary	N = 21 (18.9%)	% to system	% to total
Hydronephrosis	13	61.9%	11.7%
Renal agenesis	3	14.2%	2.7%
Dysplastic kidney	2	9.5%	1.8%
Bladder outlet obstruction	2	9.5%	1.8%
Polycystic kidney	1	4.7%	0.9%

Table 8: Anomalies under Cardio vascular system

CVS	N= 18 (16.2%)	% to system	% to total
VSD	5	27.7%	4.5%
Complex heart disease	3	16.6%	2.7%
Cardiomegaly	2	11.1%	1.8%
TOF	2	11.1%	1.8%
Single umbilical artery	2	11.1%	1.8%
Congenital heart disease	1	5.5%	0.9%
Dextrocardia	1	5.5%	0.9%
Hypoplastic left heart syndrome	1	5.5%	0.9%
Echogenic mass in left ventricle	1	5.5%	0.9%

Table 9: Anomalies under Musculoskeletal system

Musculoskeletal	N= 14 (12.6%)	% to system	% to total
Skeletal dysplasia	4	28.5%	3.6%
CTEV	3	21.4%	2.7%
Syndactyly, polydactyly	3	21.4%	2.7%
KyphoScoliosis	2	14.2%	1.8%
Amelia	1	7.1%	0.9%
Vertebral anomaly	1	7.1%	0.9%

Table 10: Anomalies under GIT

GIT	N = 10 (9.0%)	% to system	% to total
Duodenal atresia	2	20%	1.8%
Omphalocele	2	20%	1.8%
Cleft lip, cleft palate	2	20%	1.8%
Esophageal atresia	1	10%	0.9%
Congenital pyloric stenosis	1	10%	0.9%
TEF	1	10%	0.9%
Gastroschisis	1	10%	0.9%

Table 11: Anomalies under respiratory system

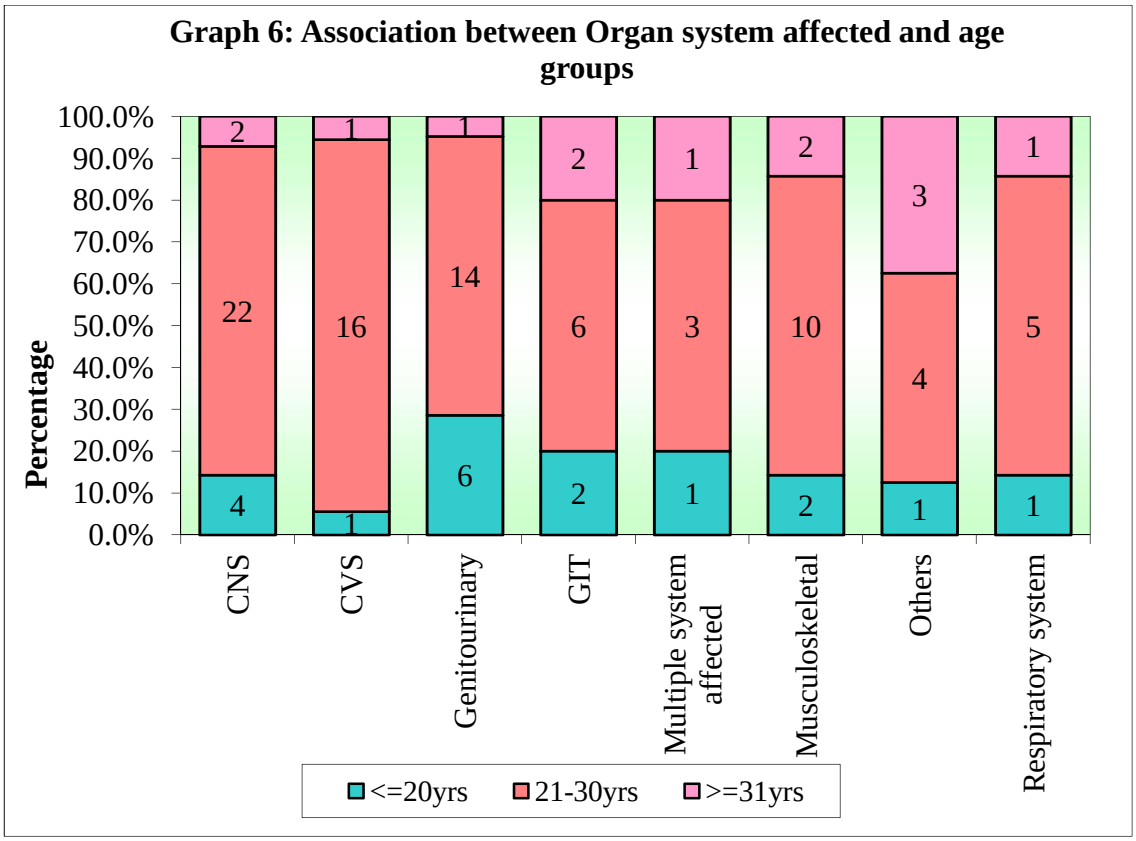
Respiratory system	N= 7 (6.3%)	% to system	% to total
Congenital diaphragmatic hernia	6	85.7%	5.4%
Right lung hypoplasia	1	14.2%	0.9%

Table 12: Anomalies not falling under any specified system or with multiple systems affected

Others	N= 6 (5.4%)	% to system	% to total
Cystic hygroma	3	50.0%	2.7%
Hydrops	3	50.0%	2.7%
Multiple system affected	**N= 7 (6.3%)**		**6.3%**

Table 13: Association between Organ system affected with age groups

Organ system affected	<=20yrs	21-30yrs	>=31yrs	Total
CNS	4	22	2	28
CVS	1	16	1	18
Genitourinary	6	14	1	21
GIT	2	6	2	10
Multiple system affected	1	3	1	5
Musculoskeletal	2	10	2	14
Others	1	4	3	8
Respiratory system	1	5	1	7
Total	18	80	13	111



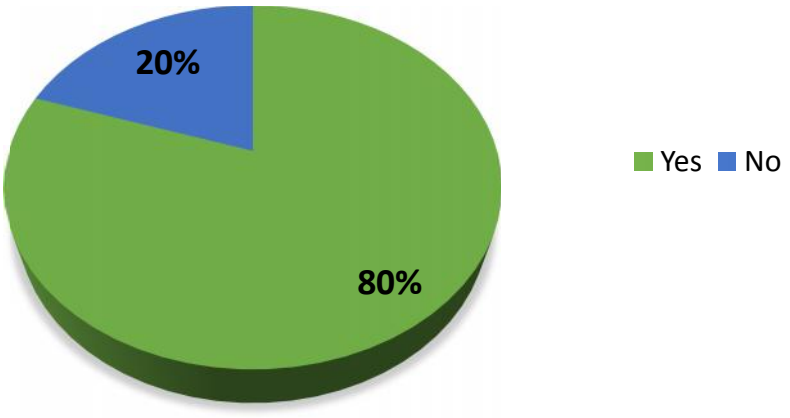
Non intake of folic acid

Table 14: Folic acid intake

History of folic acid intake	Number	Percentage
Yes	89	80.2%
No	22	19.8%
Total	111	100.0%

No patient received pre-conceptional folic acid

Graph 7: History of folic acid intake



Non intake of folic acid was associated in 20% cases (n=23)

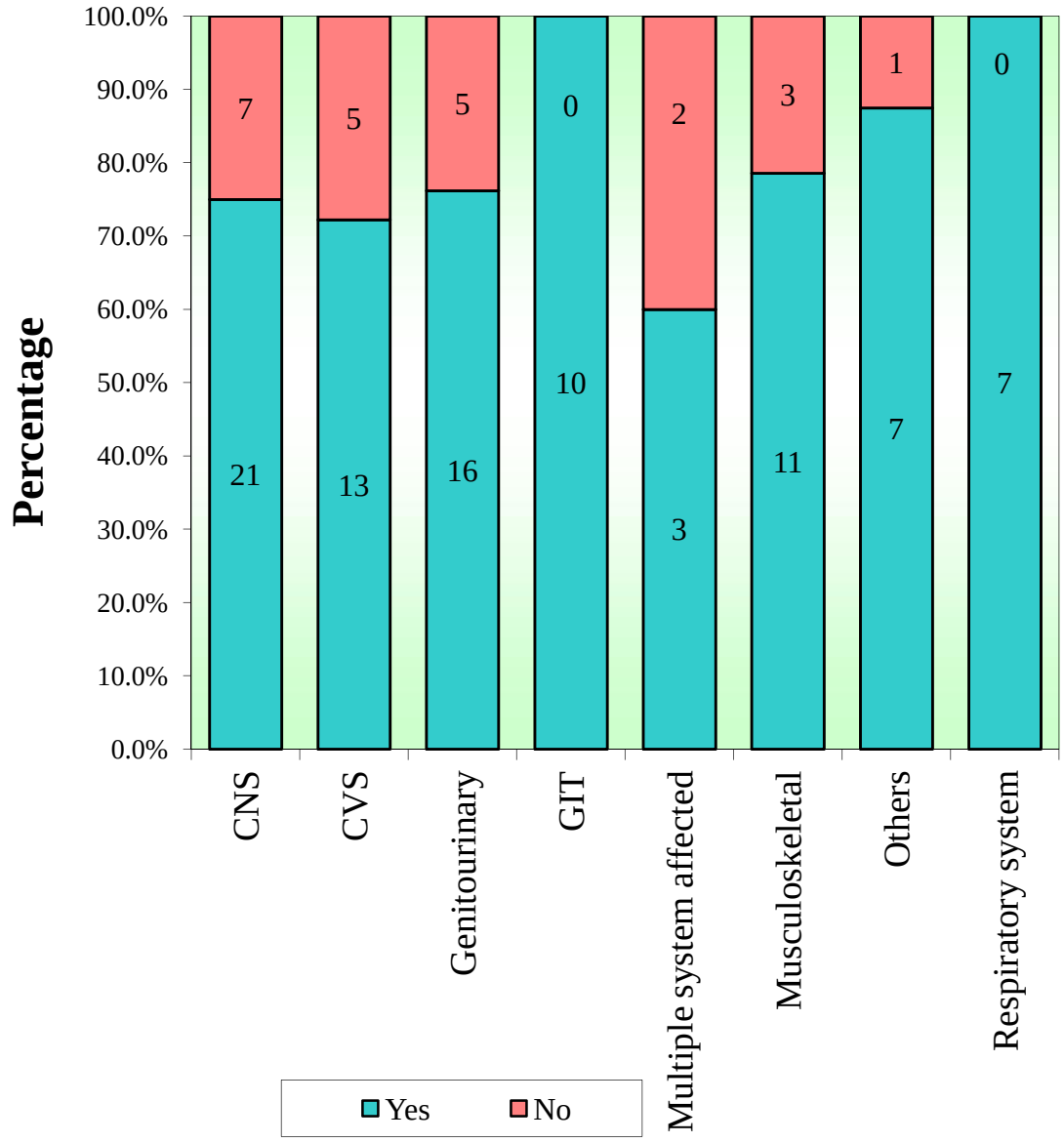
It was more commonly associated with CNS anomalies (n=7, 30.4%)

Table 15: Association between Organ system affected and History of intake of Folic acid

Organ system affected	History Folic acid intake					
	Yes	**%**	**No**	**%**	**Total**	**%**
CNS	21	75.00	7	25.00	28	25.23
CVS	13	72.22	5	27.78	18	16.22
Genitourinary	16	76.19	5	23.81	21	18.92
Musculoskeletal	11	78.57	3	21.43	14	12.61
Multiple system affected	3	60.00	2	40.00	5	4.50
GIT	10	100.00	0	0.00	10	9.01
Respiratory system	7	100.00	0	0.00	7	6.31
Others	7	87.50	1	12.50	8	7.21
Total	88	79.28	23	20.72	111	100.00
Chi-square= 6.8883 P = 0.4411						

The results showed p value of 0.4411, with no statistical significance.

Graph 8 : Association between Organ system affected and intake of folic acid



Consanguinity

Table 16: History of consanguinity

History of consanguinity	Number	Percentage
Yes	18	16.2%
No	93	83.7%
Total	**111**	100.0%

Consanguinity was associated in 16.2 % of cases (n= 18).

Graph 9 : History of consanguinity wise distribution

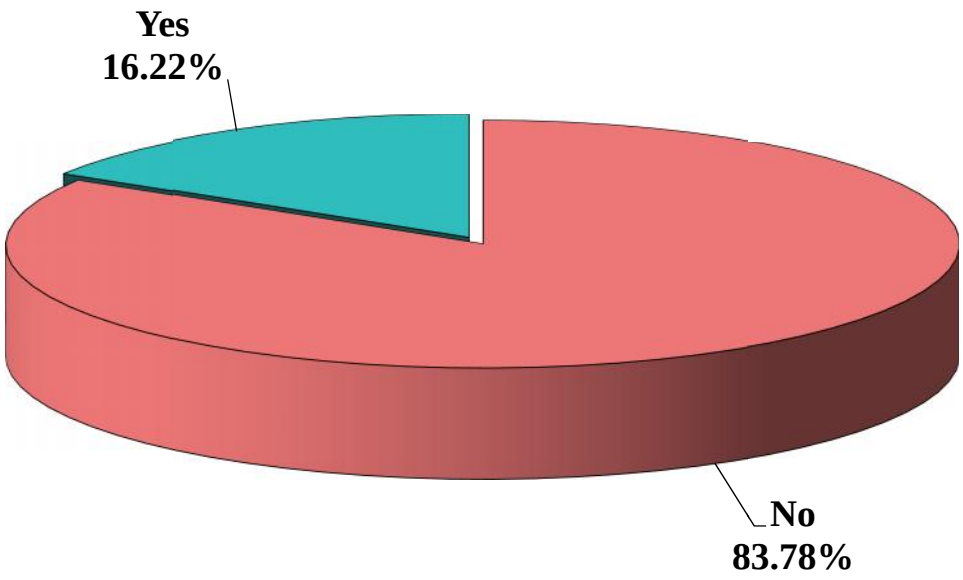


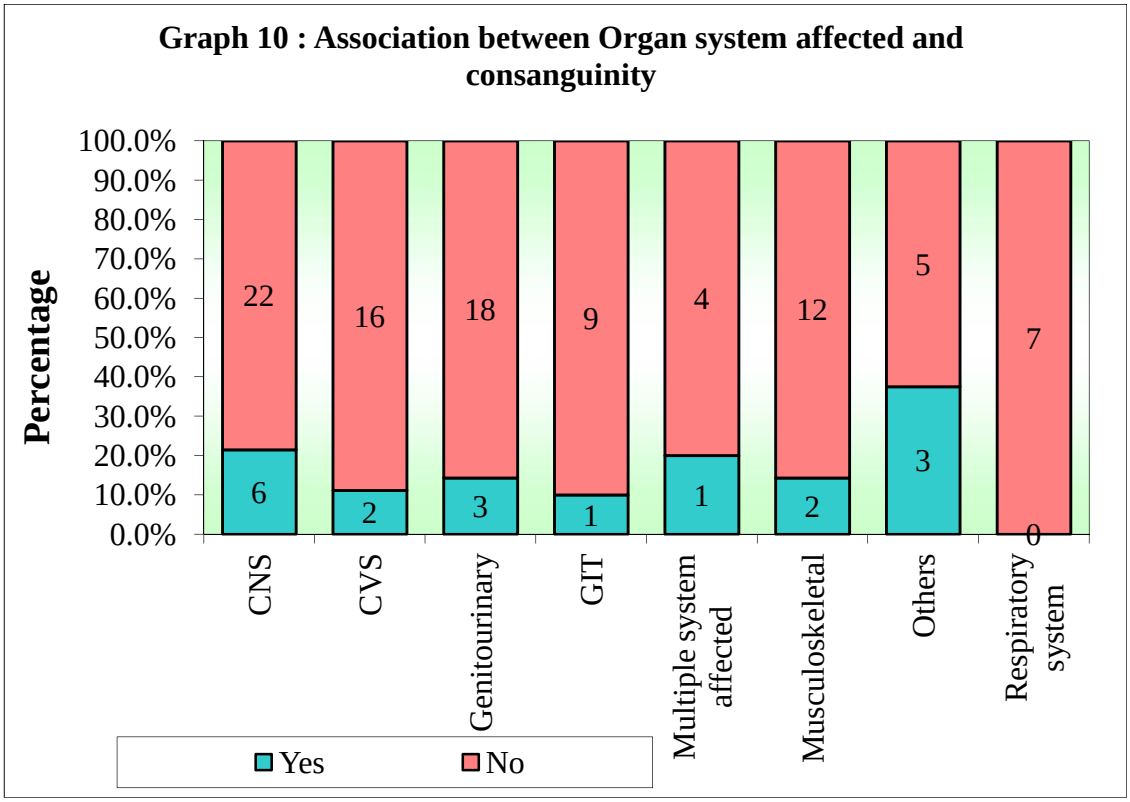
Table 17: Degree of consanguinity

Degree of consanguinity	Number of cases
2nd degree	0
3rd degree	17
4th degree	1
total	**N= 18**

Most of the cases of consanguinity were of 3rd degree consanguinity. One case had a 4th degree consanguinity.

Table 18: Association between Organ system affected with consanguinity

Organ system affected	Consanguinity					
	Yes	**%**	**No**	**%**	**Total**	**%**
CNS	6	21.43	22	78.57	28	25.23
Genitourinary	3	14.29	18	85.71	21	18.92
CVS	2	11.11	16	88.89	18	16.22
Musculoskeletal	2	14.29	12	85.71	14	12.61
GIT	1	10.00	9	90.00	10	9.01
Multiple system affected	1	20.00	4	80.00	5	4.50
Respiratory system	0	0.00	7	100.00	7	6.31
Others	3	37.50	5	62.50	8	7.21
Grand Total	**18**	**16.22**	**93**	**83.78**	**111**	**100.0**
Chi-square= 5.3601 P = 0.6162						



P- value calculated between the history of consanguinity and organ system involved showed no statistical significance, p=0.616

Table 19: Association between parity and History of consanguinity

History of consanguinity	Multi	%	Primi	%	Total	%
No	58	62.37	35	37.63	93	83.78
Yes	13	72.22	5	27.78	18	16.22
Total	**71**	**63.96**	**40**	**36.04**	**111**	**100.00**
Chi-square= 0.6362 P = 0.425						

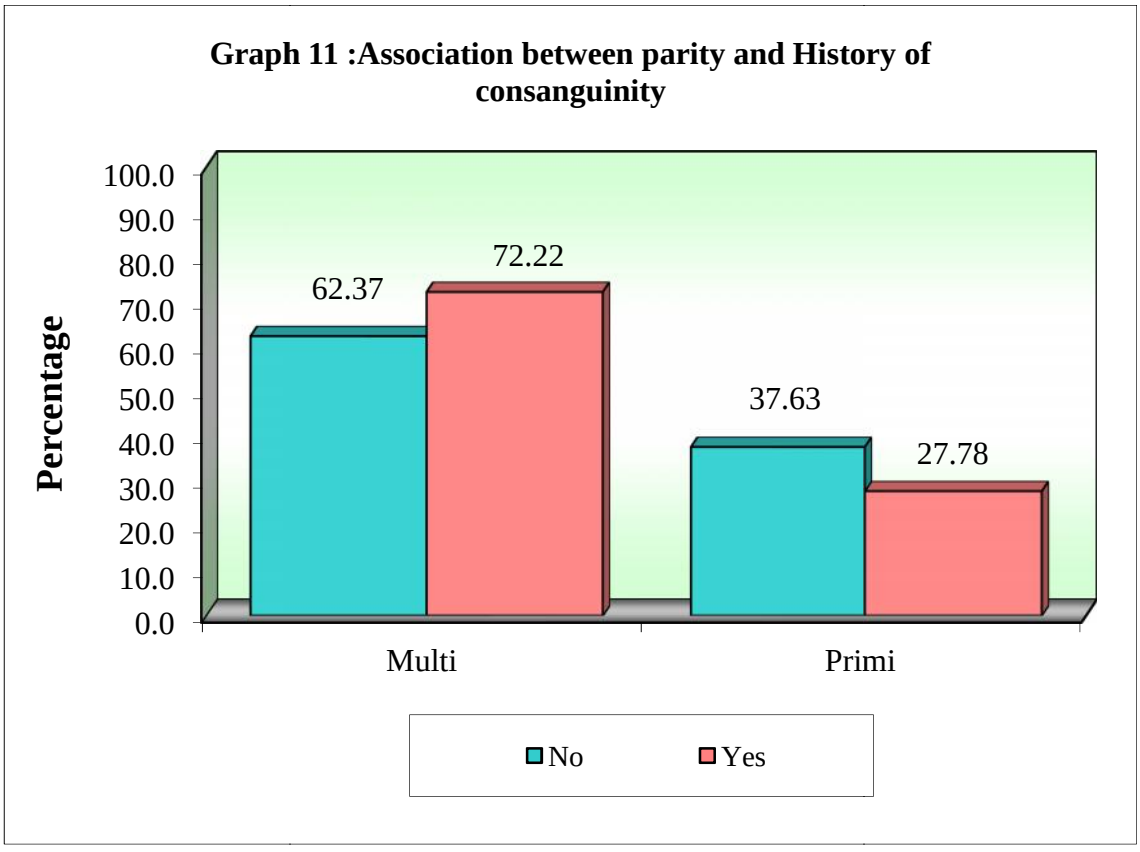
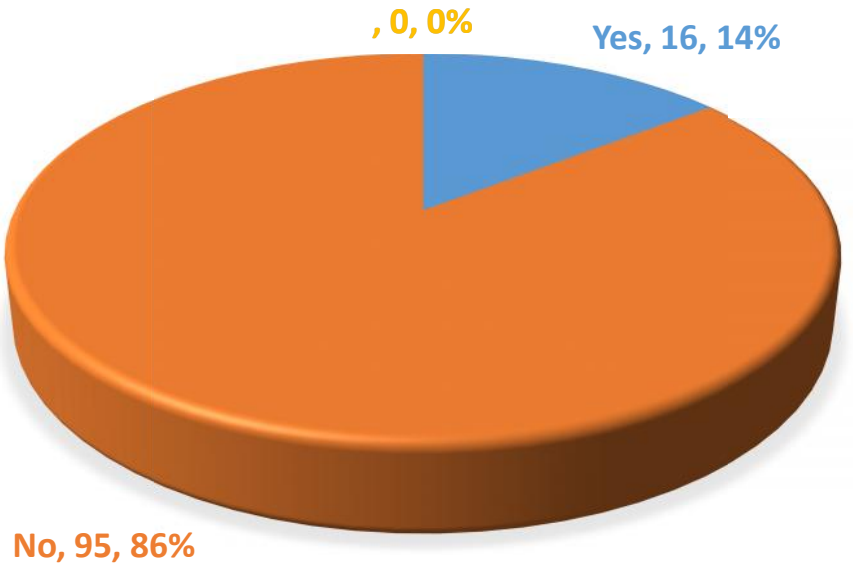


Table 20: History of previous anomalous baby

History of previous anomalous baby	Number	Percentage
Yes	16	14.41%
No	95	85.58%
Total	**111**	100%

Graph 12: History of previous congenital anomalies

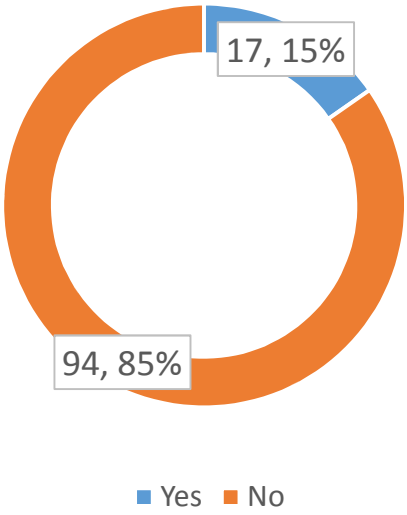


History of previous congenital anomalous baby was present in 14.41% cases (n=16)

Table 21: History of previous abortions

History of previous abortions	Number	Percentage
Yes	17	15.3%
No	94	84.7%
Total	111	100%

Graph 13: History of previous abortions



History of previous abortions was present in 17 cases (15.3%)

Table 22: History of Diabetes

History of Diabetes	Number	Percentage
Yes	6	5.4%
Overt -2 GDM - 4		
No	105	94.6%
Total	**111**	

Graph 14 : Diabetes distribution

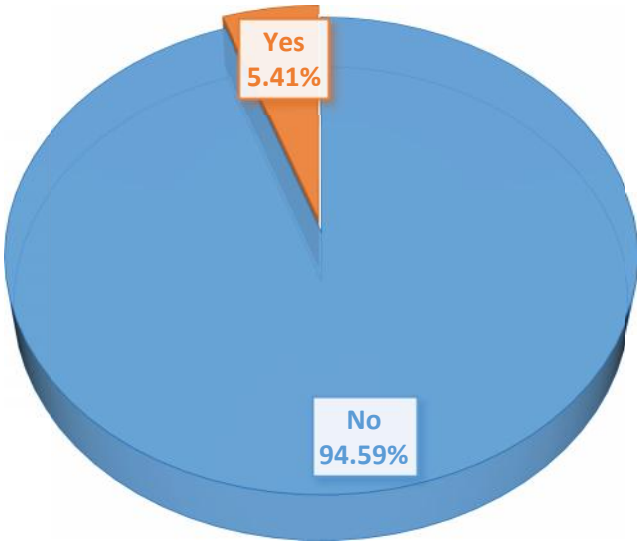
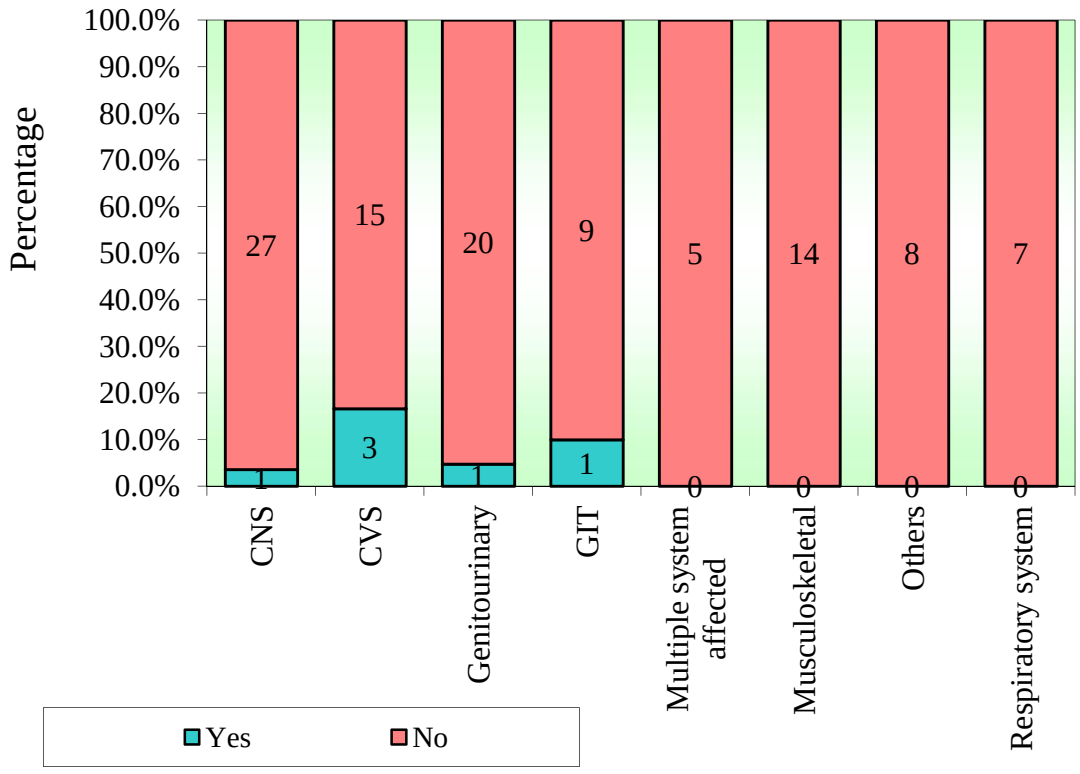


Table 23: Association between Organ system affected with Diabetes

Organ system affected	Diabetes					
	Yes	**%**	**No**	**%**	**Total**	**%**
CVS	3	16.67	15	83.33	18	16.22
CNS	1	3.57	27	96.43	28	25.23
Genitourinary	1	4.76	20	95.24	21	18.92
GIT	1	10.00	9	90.00	10	9.01
Multiple system affected	0	0.00	5	100.00	5	4.50
Musculoskeletal	0	0.00	14	100.00	14	12.61
Respiratory system	0	0.00	7	100.00	7	6.31
Others	0	0.00	8	100.00	8	7.21
Total	**6**	**5.41**	**105**	**94.59**	**111**	**100.00**

The p- value calculated for >0.05. Hence there is no statistical significance between the present of diabetes history and the organ system involved.

Graph 15 : Association between Organ system affected and Diabetes

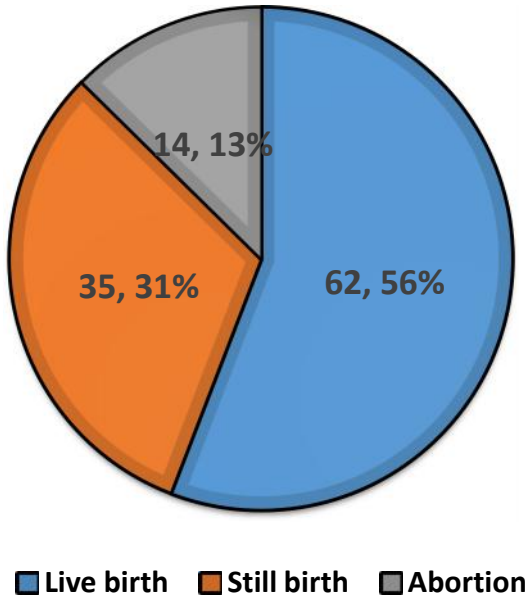


Fetal outcome

Table 24: Fetal outcome

Fetal outcome	Number	Percentage
Live birth	62	55.8%
Still birth	35	31.5%
	FSB: 32 MSB: 3	
Abortion	14	12.6%
Total	**111**	**100%**

Graph 16: Fetal outcome

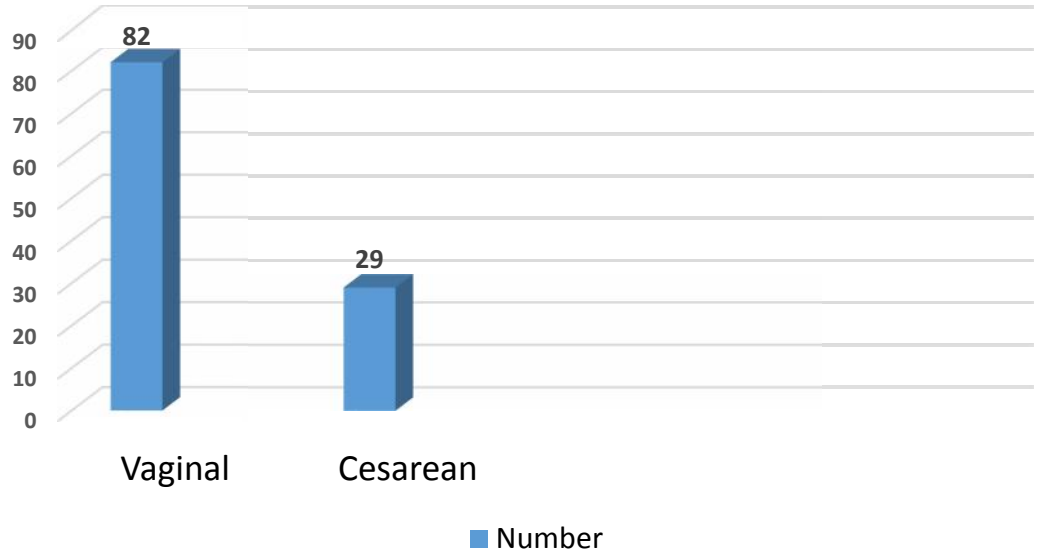


55.8 % had live births (n=62). 31.5% had stillbirths. 12.6% had abortions.

Table 25: Mode of termination

Mode	Number	Percentage
Vaginal	82	73.9%
Cesarean	29	26.1%
Total	**111**	**100%**

Graph 17: Mode of termination



Birth weight

Table 26: Birth weight distribution

Birth weight	Number	Percentage
<500 g	25	22.52%
500 g- 1 kg	14	12.61%
1 kg- 1.5 kg	10	9.00%
1.5 – 2 kg	15	13.51%
2 -2.5 kg	16	14.41 %
2.5 kg – 3 kg	18	16.21 %
3 – 3.5 kg	9	8.10 %
3.5 – 4 kg	3	2.7 %
> 4 kg	1	0.9%
Total	**111**	**100%**

Table 27: Sex of baby

Sex of baby	Number	Percentage
Female	48	43.24%
Male	59	53.15%
NA	4	3.60%
Total	**111**	**100.00%**

Graph 18 : Sex Of Baby

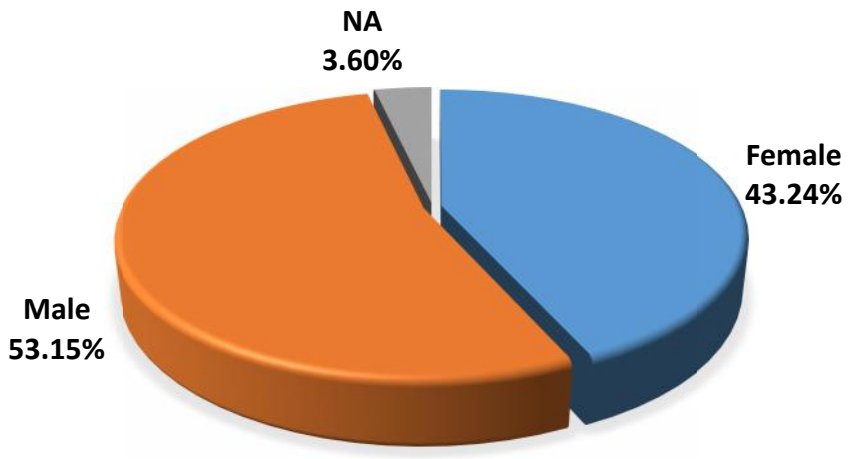


Table 28: Number of NICU admissions

NICU admission	Number	Percentage
Yes	42	37.8%
No	69	62.6%
Total	**111**	**100%**

Table 29: Neonatal follow up of live babies

Management	Number of cases
Conservative	19
On Follow up	15
No complication	2
Operated	10
Lost to follow up	12
Death	1
Total	62

DISCUSSION

The present study sought to determine the prevalence of congenital anomalies delivering at the tertiary hospital and assess the risk factors. The one- year hospital based study was conducted between the period of January 2017 and December 2017 in the labour rooms of Dr Prabhakar Kore Hospital Belagavi. A total of 111 cases met the inclusion criteria. The cases were studied and analysed.

Out of the total 5755 deliveries, total number of babies with congenital malformations was 111 (1.92%). The prevalence rate was 1.92% in our hospital during the study period. This is nearly same as the global estimate of 2-3% of congenital anomalies of all births. [33] A similar study done by Vinodh S in South India in 2016 showed an incidence of congenital anomalies to be 2.48%, which is comparable with our data. [10]

Maternal characteristics:

Majority of the congenital anomalies were present in maternal age group of 21-30 years**,** approximately 72.0%. 16.22% belonged to less than 20 years age group. 11.71% were of more than 30 years. This is comparable with the results of a study done in Central India between 2005-2007 wherein the mothers of the babies with congenital anomalies were mostly between 20 and 30 years (90.49%)[4]. The majority being in 21-30 years corresponds to the maximum reproductive function of the women during the period. Most of the women had BMI between 21-30 kg/m^2. Only 1 case had BMI of more than 30 kg/m^2. 56 cases (50%) were of multigravida (G2-3). 36% were primigravida. 13.5% were fourth gravida or more. A study conducted in Eastern India in 2011-2012 by Sarkar S, Patra C showed similar distribution of

congenital anomalies being more common in multiparas in comparison with primiparas.[5]

System wise distribution:

All anomalies were categorized based on the ICD-10 classification. System wise distribution of anomalies showed Central Nervous system anomalies contributed the maximum, a total of 28 cases accounting to 25.2%. Genitourinary system anomalies were 18.9%. Those belonging to cardiovascular system were 16.2%. Musculoskeletal system anomalies were 12.6%. 9.0% anomalies belonged to Gastrointestinal system. Remaining 20 cases were of respiratory system, multiple system affected and others.

Singh A and Sinha S conducted a similar study in North India in 2008 and found the most common system involved to be Cardiovascular system (27.1%), followed by GIT (23.1%), CNS (12.8%) and Genitourinary system (9.8%). [6]

A hospital based study conducted in Central India in 2014 by Swapnil R, Jayashree D showed prevalence rate of 2.69% . The predominant system was Cardiovascular system (29.6%).[3] Similar study conducted in Eastern India in 2011-2012 revealed a prevalence of 2.22%. Musculoskeletal system malformations were highest (33.2%) followed by gastrointestinal system (15%) . [5]

In the present study, among the CNS anomalies, open spina bifida were 8 cases, accounting to 28.5% of the CNS anomalies. Next in occurrence were Anencephaly (5 cases), Encephalocele (5 cases), Ventriculomegaly (5 cases). There were 3 cases of Dandy Walker malformations. 1 case of Holoprocencephaly was seen.

In the Genitourinary system, Hydronephrosis cases were 13 (61.9%). 3 cases of renal agenesis were present. 2 cases of dysplastic kidney and 1 case of polycystic kidney was present.Cardiovascular system anomalies were varied. VSD accounted for 27.7% cases (5 cases). 3 had complex heart disease. 2 cases of TOF were seen.

In the musculoskeletal system, 4 cases of skeletal dysplasia were present (28.5%). CTEV (3 cases) were 21.4%. Syndactyly/ polydactyly were present in 3 cases (21.4%). 2 cases of kyphoscoliosis, and 1 case of amelia were documented. A total of 10 GIT anomalies were studied. 2 cases of duodenal atresia (20%) , 2 cases of Omphalocele (20%), 1 case of esophageal atresia (10%) were noted. Cleft lip and cleft palate cases were 2 in number , 20%. One case of Tracheoesophageal fistula (10%) and one case each of Gastroschisis (10%) and Congenital pyloric stenosis (10%) were included. Under respiratory system, Congenital diaphragmatic hernia cases were 6 (85.7%). One case of Right lung hypoplasia was noted. Other anomalies studies were belonging to multiple system (6.3%).

History of Folic acid intake was taken in all cases. None of the patients had history of pre-conceptional folic acid intake. 22 cases (19.8%) did not receive folic acid antenatally. It was more commonly associated with CNS anomalies, 7 cases (30.4%). 5 cases belonging to CVS had non intake of folic acid (22.7%). The p-value calculated was 0.4411. Hence, there is no association noted between non intake of folate and the systems involved in the present study.

This is in contrast to other studies including a study conducted in North India in 2008 that found that non intake of folic acid by mother during pregnancy was found to be significantly associated with congenital malformations [6] Blencowe et al concluded that folic acid supplementation and fortification are effective in reducing

neonatal mortality from NTDs. [38] Mao B, Qiu J conducted a cohort study in 2010-2012 in China, and concluded that folic acid supplementation before pregnancy was associated with a reduced risk of congenital heart diseases. A lower dietary folate intake during pregnancy had an increased risk for the same.[37]

History of consanguinity was present in 18 cases (16.2%). Most common was third degree consanguinity (17 cases). 1 case had history of 4th degree consanguinity. Among the cases with history of consanguinity, 6 cases were of CNS system (33.3%). Genitourinary system anomalies were 3 in number (16.6%). 2 cases each of CVS and musculoskeletal system were noted. The cases which had history of third degree consanguinity had the following anomalies noted: Anencephaly, meningocele, cystic hygroma, Dandy Walker malformations, hydronephrosis, Bladder outlet obstruction, VSD, Gastroschisis, Omphalocele, Skeletal dysplasia, CTEV, hydrops.

The p-value calculated was 0.6162. The association of history of consanguinity with the system involvement was not significant in the present study. A study conducted in South India between 2011-2014 wherein consanguinity was found to be a predisposing factor for congenital anomalies in high risk pregnancies with previous Bad obstetric history. [7] In South India, consanguineous marriage is practiced as an important social culture.

History of previous congenital anomalies was present in 16 cases (14.41%). History of previous abortions was present in 17 cases (15.3%). A prospective study conducted by Ronya et al found higher incidence of congenital malformations with previous history of congenital malformations.[35]

History of Diabetes was studied in all cases. Overt DM was present in 2 cases, and GDM in 4 cases, a total of 6 cases (5.4%). Out of the 6 cases, 3 cases belonged to

CVS (50%), one case each in CNS, Genitourinary and GIT. There was no statistical significance.

A metanalysis conducted in Canada by Ray et al stated that out patient preconception care probably reduces risk of major congenital anomalies among women with pregestational diabetes mellitus. [26]

In our study, fetal outcome was measured in terms of live birth, still birth and abortions. There were a total of 62 live births (55.8%), 35 still births (31.5%) and 14 abortions (12.6%). Out of the 35 still births, 32 were Fresh stillbirths and 3 were macerated still births.

A study conducted in Central India between 2005-2007 tabulated that congenital anomalies were significantly in stillbirths ($p<0.01$) as compared to live births.[4] The present study noted that the mode of termination in 82 cases (73.9%) was by vaginal route and 29 patients underwent Cesarean section (26.1%).

The birth weight distribution of all the anomalous babies showed maximum cases being less than 500 grams (25 cases, 22.52%), mainly relating to the abortions and early terminations. 18 cases (16.21%) fell between 2.5-3kg range. 16 cases (14.41%) were of 2-2.5 kg. One case was of birth weight more than 4 kg. Among the live borns, 42 babies (37.8%) had admission in NICU.

The limitations of the study are to be mentioned. Since this is a hospital based study, it not represent the true community prevalence of the magnitude or pattern of congenital anomalies. There was no compulsory blood test or other investigation done for every newborn. However, neonatal screening by physical examination was done in all cases.

CONCLUSIONS

Out of the total 5755 deliveries, total number of babies with congenital malformations was 111 (1.92%). CNS (25.2%) and genitourinary (18.9%) abnormality constitutes the majority of cases. There was no significant risk factor associated with the anomalies in the present study.

The prevalence rate in our study is comparable with the national and global estimate. By improvement in antenatal, postnatal diagnosis and early referral to tertiary hospital, most of the morbidity can be prevented.

A well-documented birth defects registry at every level must be initiated and maintained.

Data on magnitude of the birth defects is needed not only because some of these conditions can be prevented through primary care interventions targeted towards women in the preconception, intra-conception and antenatal period, but also because strategies that target the prevention of births affected by congenital anomalies, also target the shared risk factors for other adverse pregnancy outcomes, effectively aiming at reduction of reproductive wastage and improving pregnancy outcome.

There is a need for a well defined national programme with components of prevention, care and surveillance.

SUMMARY

Congenital anomalies have been a major cause of neonatal and infant morbidity and mortality.

The one – year hospital based study was conducted in the study period of January 2017- December 2017 at Prabhakar Kore Hospital Belagavi. The objectives of the study was to determine the prevalence of congenital anomalies among total births. Secondary objective was to assess the associated risk factors.

During the one year period, total of 5755 births occurred. Out of this, 111 were detected to have congenital anomalies, either detected antenatally or postnatally. The data was collected using a data collection instrument and analysed. The prevalence of congenital anomalies was calculated to be 1.92%. System wise categorization of the anomalies was done using ICD-10 classification. CNS anomalies were 25.2%, followed by genitourinary (18.9%) and CVS anomalies (16.2%).

A considerable proportion of women did not have antenatal folic acid supplementation (20%). None had taken pre-conceptional folic acid. However no significant association was obtained between the non-intake of folic acid and the organ system affected in the present study.

History of consanguinity was present in as much as 16.2% of the women. 33% of the cases belonged to Central nervous system. History of previous congenital anomalous baby was present in 14.41% cases. 15.3% had history of previous abortions. 31.5% of the cases were stillbirths. 55.8% were live born. Out of these, 37.8% required NICU admission.

BIBLIOGRAPHY

1) WHO Fact sheet http://www.who.int/topics/congenital_anomalies
2) Christianson AL, Howson CP, Modell B. White plains, New York, USA: March of Dimes birth defects foundation; 2006. Last accessed on 2011 Feb 24. March of Dimes global report on birth defects: the hidden toll of dying and disabled children. http://www.marchofdimes.com
3) Swapnil R, Jayashree D . Pattern of congenital malformations in newborn: a hospital-based study. International Journal of Research in Medical Sciences , Feb 2016; 4(2): 524-528
4) Taksande A, Vilhekar K. Congenital malformations at birth in Central India: A rural medical college hospital based data. Indian Journal of Human Genetics, Sep-Dec 2010; 16(3):159-163
5) Sarkar S, Patra C. Prevalence of Congenital anomalies in neonates and associated risk factors in a tertiary care hospital in Eastern India. Journal of Clinical Neonatology, July-Sep 2013; 2(3):131-134
6) Singh A, Sinha S. Risk factors of Congenital malformations in North India: A case control study. J Postgraduate Med Edu Res 2016; 50(1):22-27
7) Sunitha T, Prasoona K. Risk factors for congenital anomalies in high risk pregnant women: a large study from South India. Egypt J Med Hum Genet (2016)
8) Kokate P, Bang R. Study of congenital malformations in tertiary care centre, Mumbai. International Journal of Reproduction, Contraception, Obstetrics and Gynecology. January 2017; 6(1): 89-93

9) Gandhi M H, Chaudhari U. A study on incidence of congenital anomalies in newborn and their association with fetal factors : a prospective study; April 2016; 4(4):1200-3

10) Vinodh S, Balakrishnan D: Pattern of congenital anomalies in a tertiary care centre. Journal of Medical Science and Clinical research; Jan 2017; 5(1):15826-31

11) Doddabasappa P, Adarsh E, Divya N. Prevalence of congenital anomalies: a hospital based study. International Jounral of Contemporary Pediatrics. Jan-Feb 2018; 5(1):119-23

12) Wills V, Abraha, J. Congenital anomalies: the spectrum of distribution and associated maternal risk factors in a tertiary teaching hospital Int J Reprod Contracept Obstet Gynecol; April 2017 ; 6(4):1555-60

13) Lavanya S, Seethalakshmi V. A two- year study of patterns and prevalence of congenital malformations; Int J Reprod Contracept Obstet Gynecol ; January 2018; 7(1): 114-18

14) Jayasree S, Smitha D'Couth. Prevalence of congenital anomalies in a tertiary care centre in North Kerala, India. Int J Reprod Contracept Obstet Gynecol 2018; 7:864-9

15) Om Kumari, Singh V. Prevalence and Pattern of Congenital musckuloskeletal Anomalies: a single centre study. Journal of clinical and diagnostic research, Jan 2018, 12(1); QC16-QC19

16) Bhide P, Gund P. Prevalence of Congenital anomalies in an Indian Maternal Cohort: Healthcare, Prevention, and Surveillance Implications. 2016 . PLos ONE 11(11): e0166408.

17) Bhide P, Kar A. A national estimate of the birth prevalence of congenital anomalies in India: systematic review and meta-analysis. BMC Pediatrics (2018) 18:175

18) Querieshi M, Querieshi U. Prevalence and pattern of birth defects in a tertiary care hospital in Kashmir; Global journal of Medicine and public health; 2016; 5(1)

19) ICD 10 (CDC):version 2010 http://apps.who.int/classifications/icd10/browse/2016/en#/XVII

20) Marcia L, John C. Etiology and clinical presentation of birth defects: a population based study; BMJ 2017; 357: j2249

21) Ahmed W, Dipika D. Prevalence and pattern of Congenital anomalies and its outcome at Chattargram Maa-O_Shishu General Hospital. January 2017; 16(1):22-25

22) Francine R, Pascale S. Congenital anomalies: Prevalence and risk factors. Universal journal of Public health, 2014; 2(2): 58-63

23) Seba B, Shubhankar M. Congenital anomalies in neonates and Associated risk factors in a tertiary care hospital: a single center study from India. Indian Journal of applied research, November 2017; 7(11): 173-6

24) Mohamed A, Ehab A, Ibrahim L. Pattern of congenital anomalies in newborn: a hospital- based study. Pediatric Reports 2013, 5(5):20-23

25) Shamnas M, Arya PS. Congenital anomalies: a major public health issue in India. International journal of pharmaceutical, chemical and biological sciences 2013, 3(3), 577-585

26) J G Ray, O'Brien T E. Preconception care and risk of congenital anomalies in the offspring of women with diabetes mellitus: a meta-analysis. 2001; 94: 435-444

27) Allagh KP, Shamanna BR. Birth prevalence of neural tube defects and orofacial cleft in India: A systematic review and meta-analysis. 2015. PloS ONE 10(3): e0118961

28) Suresh S, Thangavel G. Methodical issues in setting up a surveillance system for birth defects in India. National Medical Journal of India. 2005; 18(5):259-62

29) Bhat BV, Ravikumar M. Perinatal mortality in India – Need for introspection. Indian Journal of Maternal Child Health 1996; 7:31-3

30) Agarwal SS, Singh U, Singh PS, Singh SS. Prevalence and spectrum of congenital malformations in a prospective study at a teaching hospital. Indian Journal Med Res 1991; 94:413-9

31) Slavotinev A. Dysmorphology. In: Kliegman, Stanton, St. Nelson textbook of Pediatrics. Vol I. 1st South Asia Edition. 899-909

32) Liu L, Johnson HL , Cousens S, Perin J. Global, regional and national causes of child mortality: an updated systematic analysis for 2010 with time trends since 2000. Lancet 2012; 379 (9832): 2151-61

33) Dolk H, Loane M, Garne E. The prevalence of congenital anomalies in Europe. Adv Exp Med Bio;. 2010; 686:349-64

34) Capacity Building for Birth Defect Surveillance in South East Asia. Report of the regional workshop on Birth Defects surveillance. August 2015.

35) Ronya R, Gupta D. Spectrum of congenital malformations in newborns. Journal of the Indian Medical Association, 2002, 100(9): 565-566

36) Viegi C, Bertini M. Folic acid in the prevention of neural tube defects. 2018, J Birth Defects. Vol 1 No1:5

37) Mao B, Qui J, 1027. Maternal folic acid supplementation and dietary folate intake and congenital herat defects. PLoS ONE 12(11): e0187996

38) Hannah B, Simon C. Folic acid to reduce neonatal mortality from neural tube disorders. International Journal of Epidemiology. 2010; 39: i110-i121

39) Laura Mitchell. Epidemiology of Neural tube defects (2005). American journal of Medical Genetics Part C 135C:88-94

40) Correa A, Gilbosa SM, Besser LM. Diabetes mellitus and birth defects. Am J Obstet Gynecol 2008; 199:237,e1-9

41) Centres for disease control and prevention (CDC). Update on overall prevalence of major birth defects. Atlanta Georgia, 1978-2005. MMWR Morb Mortal Wkly Rep 2008;57:1-5

ANNEXURE I – ETHICAL CLEARANCE CERTIFICATE

K.L.E.UNIVERSITY'S
JAWAHARLAL NEHRU MEDICAL COLLEGE,
NEHRU NAGAR, BELAGAVI-590010 (KARNATAKA-INDIA)
(Accredited 'A' Grade by NAAC)

Website: http//www.jnmc.edu
E-Mail : dome@jnmc.edu

Phone: (+ 91-(0)831 Office : 2471350
Principal: 2471701
Fax No. +91 (0)831 – 2470759

Ref: MDC/DOME/ 24 **Date: 17/10/2016**

To,

Dr.
PG student in Obstetrics and Gynecology,
J.N.Medical College,
BELAGAVI.

Sub: Institutional Ethical Clearance for the study.

With reference to the above, we wish to inform you that your proposed research project titled **"PREVALENCE OF CONGENITAL ANOMALIES AND ASSESSMENT OF ASSOCIATED MATERNAL RISK FACTORS: A ONE – YEAR CROSS SECTIONAL STUDY AT KLE DR. PRABHAKAR KORE CHARITABLE HOSPITAL, BELAGAVI"**, is ethical and justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics Committee on Human Subjects Research.

(Dr. Arathi Darshan)
Member Secretary
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

(Dr. Ganga Pilli)
Chairman,
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

24

ANNEXURE I – CONSENT FORM

Consent for participation in the Research study

Information sheet for women participating in the Research.

Prevalence of Congenital anomalies and assessment of associated maternal risk factors: A One- year Cross sectional study at KLES Dr. Prabhakar Kore Charitable Hospital, Belagavi.

Names of Investigators:

Dr. _____,

Postgraduate in Department of Obstetrics and Gynaecology

Guide :

Dr. _____,

Professor and Unit head

Department of Obstetrics and Gynaecology,

KLE University's JN medical college

Belagavi.

Name of the Institution: KLE University's Jawaharlal Nehru Medical College, Women's & Children's Health Research Unit, Belagavi

Purpose:

We are conducting a research study to assess the prevalence of congenital anomalies and analyze the risk factors associated with congenital anomalies. Most of these are preventable factors, which if cautiously managed or prevented, can significantly reduce the number of congenital malformations in the newborns, and

thereby also reduce the perinatal mortality and morbidity.

Procedure:

If you agree to participate, you will be asked to sign a consent form. We will be collecting all the data regarding your socio-demographic data, antenatal and past history, results of the investigations done during your hospital stay and the details regarding your baby.

Benefits:

We want to let you know that there may be no benefits to you at present by participating in this study. By participating you will be helping to ensure that women in future get the best care and outcome.

Side Effects, Risks & Discomforts:

There are no side effects to participating in this study.

Confidentiality:

Any information you provide during the study will be kept confidential. Your full name will not appear on any study document and only staff participating in the study will have access to the information you provide.

Right to Refuse or Withdraw:

You are free to choose whether or not you wish to participate. You are also free to withdraw from the study at any time should you wish to do so for any reason. We hope you will participate and thank you if you do.

Questions:

If you have any questions you may ask now or later. If you wish to ask questions later, you may contact the responsible doctor attending you at the moment or you may contact,

1. Dr. _____

   Postgraduate in Department of Obstetrics and Gynaecology, KLE University's Jawaharlal Nehru Medical College, Belagavi.

2. Dr. _____

   Professor and unit head in Department of Obstetrics and Gynaecology, KLE University's Jawaharlal Nehru Medical College, Belagavi

If you have any queries about your rights as a study subject, you may call

3. Dr. Ganga Pilli,

   Professor of Pathology as Chairperson of J. N. Medical College Institutional Ethics Committee on Human Subjects Research, Phone No.0831 2473777 ext-1527 at J. N. Medical College, Belagavi or Phone number: 9480275601.

CONSENT STATEMENT:

Prevalence of Congenital anomalies and assessment of associated maternal risk factors: A One- year Cross sectional study at KLES Dr. Prabhakar Kore Charitable Hospital, Belagavi.

IP number:

Name:

Age:

Address:

Phone number:

I have read the information sheet concerning this study and I understand what is required of me if I take part in the study. All my questions and doubts have been answered by you. I understand that I can withdraw from the study at any time I wish without giving a reason and this will not affect the normal health care I receive.

I agree to take part in this study.

Participant's signature Or Left thumb impression	
Name and signature of the Investigator.	
Name and signature of the witness	

Date: Place:

ಸಂಶೋಧನೆಯಲ್ಲಿ ಭಾಗವಹಿಸುವ ಮಹಿಳೆಯರಿಗೆ ಮಾಹಿತಿ ಪತ್ರಿಕೆ

"ಜನ್ಮಜಾತ ವೈಪರೀತ್ಯಗಳ ಬಗ್ಗೆ ಮತ್ತು ಅದಕ್ಕೆ ಸಂಬಂಧಿಸಿದ ಅಪಾಯದ ಅಂಶಗಳ ಮೌಲ್ಯಮಾಪನ": ಕೆ.ಎಲ್.ಇ. ಪ್ರಭಾಕರ ಕೋರೆ ಆಸ್ಪತ್ರೆ ಬೆಳಗಾವಿಯಲ್ಲಿ ಕೈಕೊಂಡ ಒಂದು ವರ್ಷ ಅವಧಿಯ ವಿಭಾಗೀಯ ಅಧ್ಯಯನ.

ಸಂಶೋಧಕರ ಹೆಸರುಗಳು:

ಡಾ॥ , ಪ್ರಸೂತಿ ಮತ್ತು ಸ್ತ್ರೀರೋಗ ಶಾಸ್ತ್ರದ ಸ್ನಾತಕ್ಕೋತ್ತರ ವಿಭಾಗ,
ಮಾರ್ಗದರ್ಶಕರು: ಡಾ॥ , ಪ್ರಾಧ್ಯಾಪಕರು ಮತ್ತು ಪ್ರಸೂತಿ ಮತ್ತು ಸ್ತ್ರೀರೋಗ ಶಾಸ್ತ್ರ ವಿಭಾಗದ ಮುಖ್ಯಸ್ಥರು, ಕೆ.ಎಲ್.ಇ. ವಿಶ್ವವಿದ್ಯಾಲಯದ ಜೆ.ಎನ್. ಮೆಡಿಕಲ್ ಕಾಲೇಜು ಬೆಳಗಾವಿ.

ಉದ್ದೇಶ:

ನಾವು ಜನ್ಮಜಾತ ವೈಪರೀತ್ಯಗಳ ಬಗ್ಗೆ ಮತ್ತು ಅದಕ್ಕೆ ಸಂಬಂಧಿಸಿದ ಅಪಾಯದ ಅಂಶಗಳ ಮೌಲ್ಯಮಾಪನದ ಬಗ್ಗೆ ಒಂದು ಸಂಶೋಧನೆಯನ್ನು ಮಾಡುತ್ತಿದ್ದೇವೆ. ಇವುಗಳಲ್ಲಿ ಹೆಚ್ಚಿನವುಗಳನ್ನು ತಡೆಗಟ್ಟಬಹುದಾಗಿದೆ. ಇವುಗಳನ್ನು ಎಚ್ಚರಿಕೆಯಿಂದ ನಿರ್ವಹಿಸಿದರೆ ಅಥವಾ ತಡೆಗಟ್ಟಿದರೆ, ನವಜಾತ ಶಿಶುಗಳಲ್ಲಿ ಹೆಚ್ಚಿನ ಜನ್ಮಜಾತ ವೈಪರೀತ್ಯಗಳನ್ನು ಕಡಿಮೆ ಮಾಡಬಹುದು ಮತ್ತು ನವಜಾತ ಶಿಶುಗಳ ಮರಣ ಸಂಖ್ಯೆ ಮತ್ತು ಜನ್ಮಜಾತ ನ್ಯೂನ್ಯತೆಗಳು ಮತ್ತು ಅನಾರೋಗ್ಯಗಳನ್ನು ಕಡಿಮೆಗೊಳಸಬಹದು.

ವಿಧಾನ:

ನೀವು ಭಾಗವಹಿಸಲು ಒಪ್ಪಿದರೆ, ನೀವು ಒಪ್ಪಿಗೆ ಪತ್ರಕ್ಕೆ ಸಹಿ ಮಾಡಲು ಕೇಳಲಾಗುವುದು. ನಾವು ನಿಮ್ಮ ಸಾಮಾಜಿಕ ಸಂಖ್ಯಾಶಾಸ್ತ್ರದ ಮಾಹಿತಿ, ನೀವು ಆಸ್ಪತ್ರೆಯಲ್ಲಿ ಇರುವಾಗ ಕೈಕೊಂಡ ಸಂಶೋಧನೆಯ ಫಲಿತಾಂಶಗಳು ಮತ್ತು ನಿಮ್ಮ ಮಗುವಿನ ವಿವರಗಳನ್ನು ಸಂಗ್ರಹಿಸುತ್ತೇವೆ.

ಪ್ರಯೋಜನೆಗಳು:

ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸುವದಕ್ಕಾಗಿ ನಾವು ನಿಮಗೆ ಯಾವುದೇ ಪ್ರಯೋಜನ ಅಥವಾ ಉಪಯೋಗದ ಬಗ್ಗೆ ಆಶ್ವಾಸನೆ ಕೊಡುವದಿಲ್ಲ, ಆದರೆ ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸುವದರಿಂದ ದೊರೆಯುವ ಮಾಹಿತಿಯಿಂದಾಗಿ ಭವಿಷ್ಯದಲ್ಲಿ ಮಹಿಳೆಯರು ಉತ್ತಮ ಆರೈಕೆ ಮತ್ತು ಫಲಿತಾಂಶಗಳನ್ನು ಪಡೆಯುವರೆಂಬುದನ್ನು ಖಚಿತಪಡಿಸುತ್ತೇವೆ.

ಅಡ್ಡಪರಿಣಾಮಗಳು, ಅಪಾಯಗಳು ಮತ್ತು ತೊಂದರೆಗಳು:

ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸುವುದರಿಂದ ಯಾವುದೇ ಅಡ್ಡಪರಿಣಾಮಗಳಾಗುವುದಿಲ್ಲ.

ಗೌಪ್ಯತೆ:

ಈ ಅಧ್ಯಯನದಲ್ಲಿ ನೀವು ಒದಗಿಸುವ ಯಾವುದೇ ಮಾಹಿತಿಯನ್ನು ಗೌಪ್ಯವಾಗಿಡಲಾಗುವುದು. ಅಧ್ಯಯನದ ಯಾವುದೇ ದಾಖಲೆಯಲ್ಲಿ ನಿಮ್ಮ ಪೂರ್ಣ ಹೆಸರನ್ನು ಉಪಯೋಗಿಸ-ಲಾಗುವುದಿಲ್ಲ. ಕೇವಲ ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸುವ ಸಂಶೋಧನಾ ತಂಡದವರಿಗೆ ಮಾತ್ರ ನೀವು ಒದಗಿಸುವ ಮಾಹಿತಿಯ ವಿವರಗಳನ್ನು ಅವಲೋಕಿಸಲು ಅವಕಾಶ ಇರುತ್ತದೆ.

ಭಾಗವಹಿಸುವಿಕೆಯಿಂದ ಹಿಂದೆ ಸರಿಯುವ ಅಥವಾ ತಿರಸ್ಕರಿಸುವ ಹಕ್ಕು:

ನೀವು ಈ ಸಂಶೋಧನೆಯಿಂದ ಹಿಂದೆ ಸರಿಯಲು ಅಥವಾ ತಿರಸ್ಕರಿಸಲು ಸಂಪೂರ್ಣ ಸ್ವತಂತ್ರರಾಗಿರುತ್ತೀರಿ. ನೀವು ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಈ ಸಂಶೋಧನೆಯಲ್ಲಿ ಭಾಗವಹಿಸುವುದರಿಂದ ಯಾವುದೇ ಕಾರಣ ನೀಡದೇ ಹಿಂದೆ ಸರಿಯಬಹುದು. ಆದ್ದರಿಂದ ಈ ಸಂಶೋಧನೆಯನ್ನು ಭಾಗವಹಿಸಲು ನೀವು ಒಪ್ಪಿಕೊಳ್ಳುತ್ತೀರಿ ಎಂದು ಆಶಿಸುತ್ತೇವೆ. ನೀವು ಹಾಗೆ ಮಾಡಿದರೆ ಅದಕ್ಕಾಗಿ ನಮ್ಮ ಧನ್ಯವಾದಗಳು.

ಸಂಪರ್ಕಿಸಬೇಕಾದ ಮಾಹಿತಿ:

ಈ ಅಧ್ಯಯನಕ್ಕೆ ಸಂಬಂಧಿಸಿದಂತೆ ಯಾವುದೇ ಪ್ರಶ್ನೆಗಳಿದ್ದಲ್ಲಿ ನೀವು ಈ ವೇಳೆಯಲ್ಲಿ ಅಥವಾ ಪ್ರಸವದ ನಂತರ ಕೂಡ ಕೇಳಬಹುದು. ನೀವು ಪ್ರಶ್ನೆಗಳನ್ನು ನಂತರ ಕೇಳಲು ಬಯಸಿದರೆ, ನೀವು ಪ್ರಸವ ಸಮಯದಲ್ಲಿ ನಿಮ್ಮನ್ನು ನೋಡಿಕೊಳ್ಳುವ ವೈದ್ಯರನ್ನು ಅಥವಾ ಈ ಕೆಳಗಿನವರನ್ನು ಸಂಪರ್ಕಿಸಬಹುದು.

1. ಡಾ॥ , ಪ್ರಸೂತಿ ಮತ್ತು ಸ್ತ್ರೀರೋಗ ಶಾಸ್ತ್ರದ ಸ್ನಾತಕೋತ್ತರ ವಿಭಾಗ, ಕೆ.ಎಲ್.ಇ. ವಿಶ್ವವಿದ್ಯಾಲಯದ ಜೆ.ಎನ್. ಮೆಡಿಕಲ್ ಕಾಲೇಜು ಬೆಳಗಾವಿ. ಮೊಬೈಲ್ ನಂ.:
2. ಡಾ॥ ಪ್ರಾಧ್ಯಾಪಕರು ಮತ್ತು ಪ್ರಸೂತಿ ಮತ್ತು ಸ್ತ್ರೀರೋಗ ಶಾಸ್ತ್ರ ವಿಭಾಗದ ಮುಖ್ಯಸ್ಥರು, ಕೆ.ಎಲ್.ಇ. ವಿಶ್ವವಿದ್ಯಾಲಯದ ಜೆ.ಎನ್. ಮೆಡಿಕಲ್ ಕಾಲೇಜು ಬೆಳಗಾವಿ. ಮೊಬೈಲ್ ನಂ.:

ಪ್ರಶ್ನೆಗಳು:

ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಪ್ರಯೋಗಾರ್ಥಿಯಾಗಿ ನಿಮ್ಮ ಹಕ್ಕುಗಳ ಬಗ್ಗೆ ಯಾವುದೇ ಪ್ರಶ್ನೆಗಳಿದ್ದರೆ, ನೀವು ಡಾ॥ ಗಂಗಾ ಪಿಳ್ಳೈ, ಪ್ರಾಧ್ಯಾಪಕರು ಮತ್ತು ಪೆಥಾಲಾಜಿ ವಿಭಾಗದ ಮುಖ್ಯಸ್ಥರು, ಚೇರಮನ್ ಜೆ.ಎನ್. ಮೆಡಿಕಲ್ ಕಾಲೇಜ್ ಇನ್ಸಸ್ಟಿಟ್ಯೂಷನಲ್ ಎಥಿಕ್ಸ್ ಕಮೀಟಿ ಆನ್ ಹ್ಯೂಮನ್ ಸಬ್ಜೆಕ್ಟ್ಸ್ ರಿಸರ್ಚ, ಫೋನ್: 0831-2473777 ಎಕ್ಸಟೆನ್ಷನ್: 1527 ಅಥವಾ ಮೊಬೈಲ ನಂ.: 9480275601, ಮೂಲಕ ಸಂಪರ್ಕಿಸಬಹುದು.

ಸಮ್ಮತಿಯ ಹೇಳಿಕೆ

ಜನ್ಮಜಾತ ವೈಪರೀತ್ಯಗಳ ಬಗ್ಗೆ ಮತ್ತು ಅದಕ್ಕೆ ಸಂಬಂಧಿಸಿದ ಅಪಾಯದ ಅಂಶಗಳ ಮೌಲ್ಯಮಾಪನ: ಕೆ.ಎಲ್.ಇ. ಪ್ರಭಾಕರ ಕೋರೆ ಆಸ್ಪತ್ರೆ ಬೆಳಗಾವಿ ಕೈಕೊಂಡ ಒಂದು ವರ್ಷ ಅವಧಿಯ ವಿಭಾಗೀಯ ಅಧ್ಯಯನ.

ಒಳರೋಗಿ ಸಂಖ್ಯೆ:

ಹೆಸರು:

ವಯಸ್ಸು:

ವಿಳಾಸ:

ಫೋನ ನಂ.

ಈ ಅಧ್ಯಯನಕ್ಕೆ ಸಂಬಂಧಿಸಿದ ಮಾಹಿತಿ ಪತ್ರಿಕೆಯನ್ನು ನಾನು ಓದಿರುತ್ತೇನೆ. ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ನಾನು ಏನನ್ನು ಒದಗಿಸಬೇಕೆಂದು ನನಗೆ ತಿಳಿದಿದೆ. ನನ್ನ ಎಲ್ಲ ಪ್ರಶ್ನೆಗಳಿಗೆ ಮತ್ತು ಸಂದೇಹಗಳಿಗೆ ಸಮರ್ಪಕವಾಗಿ ಉತ್ತರ ಪಡೆದಿರುತ್ತೇನೆ. ನಾನು ಯಾವುದೇ ಸಂದರ್ಭದಲ್ಲಿ ಯಾವುದೇ ಕಾರಣ ನೀಡದೇ ಈ ಅಧ್ಯಯನದಿಂದ ನಿರ್ಗಮಿಸಬಹದು ಎಂದು ನಾನು ತಿಳದುಕೊಂಡಿರುತ್ತೇನೆ ಮತ್ತು ಅದರಿಂದಾಗಿ ನಾನು ಪಡೆದುಕೊಳ್ಳುತ್ತಿರುವ ಸಾಮಾನ್ಯ ಆರೋಗ್ಯ ರಕ್ಷಣೆಯ ಮೇಲೆ ಯಾವುದೇ ಪರಿಣಾಮ ಉಂಟಾಗುವುದಿಲ್ಲ.

ನಾನು ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ಒಪ್ಪಿಕೊಂಡಿರುತ್ತೇನೆ.

ಭಾಗವಹಿಸುವವರ ಹೆಸರು ಅಥವಾ ಎಡಗೈ ಹೆಬ್ಬೆರಳಿನ ಗುರುತು	
ಸಂಶೋಧಕರ ಹೆಸರು ಮತ್ತು ಸಹಿ	
ಸಾಕ್ಷಿಗಳ ಹೆಸರು ಮತ್ತು ಸಹಿ	

ದಿನಾಂಕ:

ಸ್ಥಳ:

संशोधनामध्ये भाग घेणाऱ्या स्त्रियांसाठी– माहिती पत्रक

मातृत्वाच्या सबंधी व मुलामंध्ये जन्मापासुन असंगत किंवा विचित्रपणा असल्यासबंधीच्या गोष्टी :– या सबंधी के. एल. ई. डॉ. प्रभाकर कोरे, चॅरिटेबल हॉस्पीटल बेळगांव येथे एक वर्षाचा सामुहिक अभ्यासक्रम .

*** संशोधन करणाऱ्यांची नांवे :–**

डॉ. स्त्रिरोग व प्रसुती विभाग प्रमुख , के. एल. ई. युनिव्हसिर्टी . जे, एन. मेडिकल कॉलेज, बेळगांव. व यांच्या हाताखाली काम करणाऱ्या डॉ. , पोष्टग्रॅज्युऐट, स्त्रिरोग व प्रस्तुती विभाग.

*** संस्थेचे नांव : –**

के . एल. ई. युनिव्हसिर्टी . जे, एन. मेडिकल कॉलेज, बेळगांव, व स्त्रिया आणि मुले आरोग्य चिकित्सा विभाग बेळगांव.

*** उद्देश : –**

आम्ही जन्म देणाऱ्या मातामध्ये प्रसुतिच्यावेळी व त्या सबंधी असंगत व विचित्र गोष्टी संशोधनाचा अभ्यासाचे वर्गीकरण करणार आहोत. या सर्व असंगत गोष्टी आम्ही प्रभावीपणे टाळु शकतो व आम्ही दक्षता घेवुन त्यांचे प्रमाण कमी करु शकतो. तसेच जन्म घेतलेल्या मुलामध्ये अपंगत्वता कमी करु शकतो. तसेच मातेच्या उदरामध्ये मुलाची मरणाची प्रक्रिया आणि विकृति कमी करु शकूतो.

*** पध्दत : –**

- तुम्ही जर या संशोधनामध्ये सहभागी झाला ते तुमच्याकडुन सहभागी झालेला सम्मतीचा नमूना सही करुन घेण्यात येईल. आम्ही तुमच्याकडुन तुमच्या सबंधीचा सामाजिक तसेच तुमच्या सबंधीता बद्दल ही माहिती मिळवु व प्राप्त झालेल्या माहितीनुसार तुमच्या जन्म दिलेल्या मुलांसबंधी माहिती प्राप्त करुन घेवु.

*** फायदे :**

या संशोधन कार्यक्रम मध्ये तुम्ही सहभागी झाल्यामुळे तुम्हास कोणतेही फायदे होणार नाहीत. तुम्ही सहभागी झाल्यामुळे स्त्रियांची भविष्यात कोणती काळजी घ्यावी हे आम्हास समजु शकेल व त्या सबंधी आम्ही निष्कर्ष काढू.

✻ *अनिष्ट परिणाम, धोके व त्रास :*

या संशोधन अभ्यासामध्ये कोणतेही तुमच्या शरीरावर कोणतेही परिणाम होणार नाहीत.

✻ **गुप्तता :**

कोणतीही प्राप्त झालेली माहिती या संशोधनामध्ये गुप्त राखली जाईल. तुमचे पुर्ण नांव कोणत्याही ठिकाणी या अभ्यासामध्ये नमुद केले जाणार नाही. आणि या संशोधन अभ्यासामध्ये सहभागी झालेल्या संशोधन करणाऱ्या व्यक्तीना ही माहीती फक्त उपलब्ध होईल.

✻ **माहिती न देणे किंवा गुप्तता :**

या अभ्यासामध्ये सहभागी होणे किंवा न होणे हे तुमच्या इच्छेवर आहे. जर का तुम्ही या अभ्यासामध्ये सहभागी झाला असाल तर तुम्हास कोणतेही कारण न सांगता या मधुन आपले नांव कमी करण्याचा अधिकार आहे. या संशोधन अभ्यास क्रमामध्ये तुम्ही सहभागी व्हाल अशी आशा आहे व त्याबद्दल तुम्हास धन्यवाद.

✻ **याबाबत कुणाशी संपर्क साधावा :**

तुम्हास या बाबत प्रश्न विचारण्याचा अधिकार आहे. व ते तुम्ही प्रसुतिच्या वेळी सुध्दा विचारु शकता. या सबंधी जर तुम्हास प्रश्न विचारवयाचे असतील तर ते प्रश्न तुम्ही खालील व्यक्तीशी संपर्क साधुन विचारावे.

१) डॉ. ३ स्त्रिरोग प्रसुती पोष्ट ग्रॅज्युएट विभाग के. एल. ई युनिव्हसिर्टी जे. एन. मेडिकल कॉलेज बेळगांव, टेलिफोन नं.

२) डॉ. - स्त्रिरोग व प्रस्तुती विभाग, प्रोफेसर के. एल. ई युनिव्हसिर्टी जे. एन. मेडिकल कॉलेज बेळगांव, टेलिफोन नं.

✻ **प्रश्न :-**

जर का तुमच्या कांही तुमच्या अधिकारासंबंधी शंका असल्यास या संबंधी तुम्ही डॉ. गंगा पिली, प्रोफेसर व पॅथॉलॉजी विभाग प्रमुख, ज्या जे. एन. मेडिकल कॉलेज, इथिक्स कमिटी व मानवीय अधिकार संस्था संबंधीच्या चेअरमन आहेत व यांचा फोन नं. 0831-2473777, इक्टेंशन 1527 व मोबाईल फोन नं. 9480275601 असा आहे, त्यांच्याशी शंका समाधान संबंधी संपर्क साधु शकता.

.....

मातृत्वाच्या सबंधी व मुलामध्ये जन्मापासुन असंगत किंवा विचित्र असण्यासबंधीच्या गोष्टी : या संबंधी के. एल. ई . डॉक्टर प्रभाकर कोरे चॅरीटेबल हॉस्पीटल, बेळगावी येथे एक वर्षाचा सामूहीक अभ्यासक्रम.

आप पी. नं.

नांव :

पत्ता :

फोन नंबर :

मी वरील नमुनातील माहीती संशोधनासबंधी वाचली असून मी ती पुर्णपणे समजावून घेऊन या अभ्यासक्रमात भाग घेत आहे. माझ्या प्रश्नाचे व शंकाचे समाधानपूर्वक उत्तर तुम्ही दिलेला आहात. मी या अभ्यास संशोसधनातून कोणत्याही वेळी मुक्त घेऊ शकते. व त्यासाठी मला कोणतेही कारण सागण्याची जरुरी नाही. या कार्यक्रमामुळे मला स्वास्थ सेवे विषयी मिळणाऱ्या सुविधांवर कोणताही परीणाम होणार नाही .

मी या क्रार्यक्रमात सहभागी होऊ इच्छिते.

सही किंवा डाव्या हाताचा अंगठा	
संशोधन करणाऱ्यांचे नाव व सही	
साक्षिदाराचे नाव व सही	

तारीख

स्थळ :

संशोधन में सम्मिलित होनेवाले औरतों के लिए – जानकारि पत्र

मातृत्व के संबंध में और उनके जन्मे हुए बच्चों में असंगतता और विकृति संबंधी के. एल. ई. डॉ. प्रभाकर कोरे चॅरिटेबल हॉस्पीटल बेलगाम में एक सल का सामुहिक अभ्यासक्रम .

*** सं शोधन करनेवालों के नाम :–**

डॉ. प्रोफेसर स्त्रिरोग व प्रसुती विभाग प्रमुख , के. एल. ई. युनिव्हसिर्टी . जे, एन. मेडिकल कॉलेज, बेळगांम. और उनके साथ काम करनेवाले डॉ. , पोष्टग्रॅज्युऐट, स्त्रिरोग व प्रस्तुती विभाग.

*** संस्था का नाम –**

के . एल. ई. युनिव्हसिर्टी . जे, एन. मेडिकल कॉलेज, बेळगांम, औरस्त्रिया और बच्चे आरोग्य चिकित्सा विभाग बेळगांम.

*** उद्देश :–**

हम बच्चे को जन्म देनेवाले माता में प्रसुती के समय और उस सबंध में असंगती और विकृती का इस संशोधन अभ्यासक्रम में वर्गिकरण करनेवाले है । यह सब असगतता और विकृती हम उपचार पध्दती द्धारा नष्ट कर सकते है । और दक्षता लेकर हम उनको कम कर सकते है । जन्म लेने वाले बच्चो में हम इसके व्दारा अपंगत्वता कम कर सकते है । तथा माता के उदर में जन्म लेने वाले बच्चे की मरने संख्या और विकृती कम कर सकते है ।

*** पध्दत :**

यदि आप संशोधन में सम्मिलित हुए गए तो आपकेव्दारा सम्मिती का सम्मती नमुना सही कर लिया जाएगे। हम आपकी इस सबंध में सामाजिक जानकारी तथा आपके सबंधीतो सबंधी जानकारी प्राप्त करेंगे और प्राप्त किए गए जानकारी के व्दारा आपको जन्म होने वाले बच्चे सबंधी जानकारी ले सकते है ।

*** मुनाफा :–**

इस संशोधन अभ्यासक्रम में यदि आप सम्मिलित होते है तो कोई फायदे आपको नही मिल सकते । लेकिन यदि आप सम्मिलित हो गये तो भविष्य में स्त्रियों की कौनसी हिफाजत लेनी चाहिए यह हमें समज में आ जाऐगा और हम इस सबंध में हमारे निष्कर्ष निकाल सकते है ।

✻ *अनिष्ट परिणाम, धोका तथा धक्का :-*

इस संशोधन अभ्यासक्रम व्दारा आपको कोई भी शारिरिक इजा या परिणाम नही होनेवाला है ।

✻ **गोपनियता :**

आपके व्दारा प्राप्त की गई जानकारी इस सशोधनमें गोपनिय रखी जाऐगी । आपको पुरा नाम किसी भी जगह इस अभ्यास में नमुद नही किया जाऐगा और इस संशोधन में सम्मिलित होनेवाले व्यक्तियोंकी जानकारी सिर्फ संशोधन करनेवाले व्यक्ति को ही उपलब्ध होगी ।

✻ **जानकारी न देना और गोपनियता :-**

इस अभ्यासक्रम में सम्मिलित होना या न होना यह आपके ऊपर निर्भर करता है । यदि आप इस अभ्यासक्रम में सम्मिलित हो गये है तो, आपको आप का नाम, कोई भी कारण न बताते हुए, नाम कम करने का अधिकार है । इस संशोधन अभ्यासक्रम में आप सम्मिलित होंगे ऐसी हमें उम्मीद है और इसके लिए धन्यवाद !

✻**इस सबंध में आप किसके साथ संपर्क कर सकते है :-**

आपको इस सबंध में सवाल पुछने का अधिकार है और वे सवाल आप प्रसुती के समय पुछ सकते है । इस सबंध में आपको कोई जानकारी या प्रश्न है तो आप निम्नलिखित व्यक्तियों से संपर्क कर सकते है ।

१) डॉ. स्त्रिरोग प्रसुती पोष्ट ग्रॅज्युएट विभाग के. एल. ई युनिव्हसिर्टी जे. एन. मेडिकल कॉलेज बेळगाम, टेलिफोन नं.

२) डॉ. - स्त्रिरोग व प्रस्तुती विभाग, प्रोफेसर के. एल. ई युनिव्हसिर्टी जे. एन. मेडिकल कॉलेज बेळगाम, टेलिफोन नं.

✻ **प्रश्न :-**

यदि आपको आप के अधिकार सबंधी इस सबंध में जानकारी हासिल करनी है तो आप डॉ. गंगा पिली, प्रोफेसर व पॅथॉलॉजी विभाग प्रमुख, जो जे.एन. मेडिकल कॉलेज, इथिक्स कमिटी और मानवीय अधिकार संस्था के चेअरमन है उनके साथ उनके फोन नं. 0831-2473777, इक्टेंशन 1527 और मोबाईल फोन नं. 9480275601 पर संपर्क कर सकते है ।

✻✻✻✻✻✻✻✻✻✻✻✻

संशोधन में सम्मिलित होनेवाले औरतों के लिए – जानकारि पत्र

मातृत्व के संबंध में और उनके जन्मे हुए बच्चों में असंगतता और विकृति संबंधी के. एल. ई. डॉ. प्रभाकर कोरे चॅरिटेबल हॉस्पीटल बेलगाम में एक सल का सामुहिक अभ्यासक्रम .

आय पी नं.

नाम :

पत्ता :

फोन नं.

मैने ऊपर लिखित फॉम में दि हुई जानकारी जो संशोधन सबंधीत है वह पढी है । और मैने उसीको संपुर्णता समजा लिया है और इसके बाद मै इस अभ्यासक्रम शामिल हो रही हुँ । मैरे प्रश्न और शंकासमाधान का आपके व्दारा उत्तर दिया हुआ है मै इस संशोधन अभ्यास में अपना नाम कभी भी कम कर सकती हुँ और इसके लिए मुझे कोई भी कारण बताने की जरुरत नही है । इस कार्यक्रम की वजह से मुझे मिलनेवाली आपके व्दारा स्वास्थ सेवा की सुविधा पर कोई भी परिणाम नही होनेवाला हैं ।

मै इस कार्यक्रम में सम्मिलित होना चाहती हुँ ।

सही और बाये हात का अंगुठा	
संशोधन करणेवाले का नाम और सही	
साक्षिदार का नाम और सही	

दिनांक :

स्थळ :

NNEXURE III – PROFORMA

1. Patient details :

   a. Name:

   b. IP number :

   c. Age :

   d. Full address:

   e. Contact number:

   f. Occupation:

   g. Height :

   h. Weight :

   i. BMI:

2. Parity :

1	2	3	>= 4

3. LMP _____

4. Gestational age:

5. Mode of conception

Spontaneous	Assisted

   Married life : _____ years

6. Registered at : _____

7. History of previous congenital anomalous baby :

Yes	No

8. History of abortions :

Spontaneous			Induced		
0	1	>=2	0	1	>=2

Cause: _____

9. History of drug use

Yes	No

10. If yes, specify _____at __ month

11. Folic acid taken

Yes	No

From _____ month

12. GDM/ overt diabetes:

Yes	No

13. Chronic hypertension :

Yes	No

14. PIH:

Yes	No

15. Significant past history _____

16. History of fever with rash:

Yes	No

_____ months of amenorrhea

17. History of vaccination of mother

Yes	No

Specify: _____

18. Congenital anomaly diagnosed on Antenatal scan

Yes	No

19. Age at marriage: _____

20. History of consanguinity :

Yes	No

21. History of contraception use

Yes	No

22. If yes, specify _____

23. Alcohol use:

Yes	No

24. Tobacco use:

Yes	No

25. Gestational age at birth _____

Term	Preterm

26. Mode of delivery:

Vaginal	LSCS

Indication for LSCS _____

27. Delivery :

Spontaneous	Induced

28. Live birth / IUD

Live birth	IUD

29. Sex of baby:

Male	Female

30. Birth weight :

31. Congenital anomaly:

32. Investigation reports, if any:

33. NICU admission:

Yes	No

34. Newborn complication:

Yes	No

35. Specify: _____

36. Duration of hospital stay: _____

Sl. No	IP No	Age	BMI (kg/m2)	Parity	Gestational age wks	Mode of conception	H/o drug use	Drug taken	Folic acid intake	FA from month	H/o fever with rash	H/o consanguinity	H/o Previous congeintal anomalous baby	H/o abortions	GDM	Mode of delivery	Indication for LSCS	Date of delivery	Live/IUD	FSB/MSB	Sex of baby	Birth weight	Congenital anomaly	Organ system affected	NICU admission
1	781416	22	19.7	G2A1	20	Spontaneous	No	No	Yes	3rd	No	No	No	Yes	No	Vaginal		6/1/2017	IUD	FSB	Male	530g	Cervical meningocele	CNS	No
2	781445	26	21.9	G4P3L1	39	Spontaneous	No	No	Yes	2nd	No	No	Not known	No	No	Vaginal		5/1/2017	Live	_	Male	2.9kg	Congenital diaphragmatic hernia	Respiratory system	Yes
3	780897	26	23.8	G2P1L1	38	Spontaneous	No	No	Yes	3rd	No	No	No	No	No	LSCS	Prev CS with scar tenderness	6/1/2017	Live	_	Female	2.2kg	Duodenal atresia	GIT	Yes
4	781579	20	24.1	Primi	20	Spontaneous	No	No	Yes	3rd	No	Yes,3rd degree	No	No	No	Vaginal		8/1/2017	IUD	FSB	Female	400g	Cystic hygroma	Others	No
5	781566	25	29.6	Primi	37	Spontaneous	No	No	Yes	2nd	No	No	No	No	No	LSCS	poliomyelitis	10/1/2017	Live	_	Female	4.05kg	open spina bifida, Hydrocephalus, Chiari II malformations	CNS	Yes
6	782336	23	22.8	Primi	20	Spontaneous	No	No	Yes	3rd	No	No	No	No	No	Vaginal		11/1/2017	IUD	FSB	Female	540 g	Open neural tube defect in lumbo-sacral region	CNS	No
7	784797	25	24	G2P1L1	30	Spontaneous	No	No	Yes	2nd	No	No	No	No	No	Vaginal		24/1/17	IUD	FSB	Female	1 kg	Anencephaly	CNS	No
8	782825	25	21.4	G7P6L2D4	30	Spontaneous	No	No	Yes	2nd	No	Yes,3rd degree	Yes, CHD	No	No	Vaginal		13/1/17	IUD	FSB	Male	900 g	Skeletal dysplasia	Musculoskeletal	No
9	783033	29	23.1	G3P2L3	38	Spontaneous	No	No	Yes	3rd	No	No	No	No	No	LSCS		16/1/17	Live	_	Male	3.2 kg	Left hydronephrosis	Genitourinary	Yes
10	785289	21	22.5	Primi	39	Spontaneous	No	No	Yes	3rd	No	No	No	No	No	Vaginal		27/1/17	Live	_	Female	2.6 kg	Small perimembranous VSD	CVS	Yes
11	786588	22	21.8	G3P2L2	39	Spontaneous	No	No	No	_	No	Yes,3rd degree	Yes, ataxia	No	No	Vaginal	_	3/2/2017	Live	_	Male	3.2 kg	Dandy Walker malformation	CNS	No
12	786600	25	18.7	G5P3L2A1	11	Spontaneous	No	No	Yes	1.5	No	Yes,3rd degree	Yes, fetal hydrops	Yes, 1	No	Vaginal	_	4/2/2017	Abortion	_	Not detected	_	Cystic hygroma	Others	No
13	787878	24	24.4	G3P2L2	34	Spontaneous	No	No	Yes	2	No	No	No	No	No	Vaginal	_	12/2/2017	Live	_	Male	1.8 kg	B/l renal agenesis	Genitourinary	No
14	787924	21	22.1	Primi	40	Spontaneous	No	No	Yes	2	No	No	No	No	No	Vaginal	_	10/2/2017	Live	_	Female	3 kg	Rt ventriculomegaly	CNS	No
15	788160	28	21.7	G6P5L3	31	Spontaneous	No	No	Yes	3	No	No	Not known	No	No	LSCS	Non progress	12/2/2017	IUD	MSB	Male	2.2 kg	Hydrops fetalis	Others	No
16	789496	21	22	Primi	34	Spontaneous	No	No	No	_	No	No	No	No	No	Vaginal	_	21/2/17	Live	_	Male	1.8 kg	Ventriculomegaly, corpus callosal agenesis, B/l grade I hydronephrosis	CNS	Yes
17	785751	20	21.6	Primi	38	Spontaneous	No	No	Yes	2	No	No	No	No	No	Vaginal	_	1/2/2017	Live	_	Male	2.8 kg	Ventriculomegaly	CNS	No
18	78634	22	22.8	G3P1L1A1	38	Spontaneous	No	No	Yes	2	No	No	No	Yes, 1	No	Vaginal	_	1/2/2017	Live	_	Male	3.3 kg	Left renal pelviectasis	Genitourinary	No
19	786118	26	21.2	Primi	40	Spontaneous	No	No	Yes	2	No	No	No	No	No	LSCS	Non progress	1/2/2017	Live	_	Male	3.12 kg	Left ventricular echogenic focus	CVS	No
20	788561	28	23.2	G5P2L1A2	38	Spontaneous	No	No	Yes	2	No	Yes, 3rd degree	Yes	Yes,2	No	LSCS	Prev 2 lscs	14/2/17	Live	_	Male	3.14 kg	B/l hydronephrosis	Genitourinary	No
21	789835	25	21.9	G2A1	37	Spontaneous	No	No	Yes	2	No	No	No	Yes, 1	No	LSCS	Fetal distress	20/2/17	Live	_	Male	2.3 kg	Ventriculomegaly	CNS	No
22	789918	27	23.1	Primi	37	Spontaneous	No	No	Yes	2	No	No	No	No	No	LSCS	Fetal distress	22/2/17	Live	_	Female	2.92 kg	Right renal pelviectasis	Genitourinary	No
23	790474	24	22.1	G2P1L1	34	Spontaneous	No	No	Yes	3	No	No	No	No	No	LSCS	Prev LSCS with MCDA twins	28/2/17	Live	_	Male	1.5 kg	Omphalocele	GIT	Yes
24	787276	27	24.8	Primi	38	Spontaneous	No	No	Yes	3	Yes	No	No	No	No	Vaginal	_	7/2/2017	Live	_	Male	2.3 kg	Hypoplastic rigth heart, VSD	CVS	Yes
25	791309	20	22.8	Primi	37	Spontaneous	No	No	Yes	3	No	No	No	No	No	Vaginal	_	28/2/17	Live	_	Male	2.6 kg	Hydronephrosis	Genitourinary	No
26	789615	26	23.7	G2P1L0	20	Spontaneous	No	No	No	_	No	No	Yes, dextrocardia, PKD	No	No	Vaginal	_	19/2/17	IUD	FSB	Female	500g	Right multicystic dysplastic kidney	Genitourinary	No
27	789640	27	24.4	G2P1L1	20	Spontaneous	No	No	Yes	3	No	No	No	No	No	Vaginal	_	20/2/17	IUD	FSB	Female	470g	esophageal atresia, Arnold Chiari malformation	GIT	No
28	791739	23	23.2	GP1L1A1	25	Spontaneous	NO	No	Yes	2	No	Yes,3rd degree	No	nduced at 5th mo	No	Vaginal	_	3/3/2017	IUD	FSB	Female	1 kg	Encephalocele	CNS	No
29	791885	23	25.7	GP2L1D1	25	Spontaneous	No	No	No	_	No	No	Yes, chd, cardiac anomaly	No	No	Vaginal	_	3/3/2017	IUD	FSB	Male	940 g	VSD	CVS	No
30	793533	22	24.2	G2P1L1	28	Spontaneous	No	No	Yes	2	No	No	No	No	No	Vaginal	_	11/3/2017	IUD	FSB	Male	1.2 kg	Congenital Diaphragmatic hernia	Respiratory system	No
31	795744	19	18.7	Primi	40	Spontaneous	No	No	Yes		No	No	No	No	No	Vaginal	_	23/3/17	Live	_	MAle	2.8kg	Conginatal pyroric stenosis	GIT	Yes
32	790474	24	20.9	g2P1L1	34	Spontaneous	No	No	Yes	3	No	No	No	No	No	LSCS	Abruption with prev LSCS	28/3/17	Live	_	Male	1.5 kg	Omphalocele	GIT	Yes

Sl. No	IP No	Age	BMI (kg/m2)	Parity	Gestational age wks	Mode of conception	H/o drug use	Drug taken	Folic acid intake	FA from month	H/o fever with rash	H/o consanguinity	H/o Previous congeintal anomalous baby	H/o abortions	GDM	Mode of delivery	Indication for LSCS	Date of delivery	Live/IUD	FSB/MSB	Sex of baby	Birth weight	Congenital anomaly	Organ system affected	NICU admission
33	794755	27	20.2	G2A1	25	Spontaneous	No	No	Yes	2	No	Yes,4th degree	No	Yes, 1 , spont	No	Vaginal	_	19/3/17	IUD	FSB	Female	540 g	Hypplastic left heart syndrome	CVS	_
34	796065	35	20	G7P5A1	23	Spontaneous	No	No	Yes	2	No	Yes, 3rd degree	Yes, PCK	Yes,1 , spont	No	Vaginal	_	25/3/17	IUD	MSB	Male	250 g	Hydrops	Others	_
35	799953	24	25.5	G2P1L1	42	Spontaneous	No	No	Yes	3rd	No	No	No	No	No	Vaginal	_	16/4/17	Live	_	Male	2.4 kg	Congenital diaghramatic hernia	Respiratory system	Yes
36	800465	19	21.6	Primigravida	36	Spontaneous	No	No	Yes	3rd	No	Yes, 3rd degree	No	No	No	Vaginal	_	18/4/17	Live	_	Female	1.8 kg	Gastroschisis	GIT	yes
37	800976	22	23.7	Primigravida	36	Spontaneous	No	No	Yes	2nd	No	No	No	No	s, GD	Vaginal	_	24/4/17	Live	_	Male	1.7 kg	TOF+ Cardiomegaly	CVS	Yes
38	802488	24	24.8	G2P1L1	37	Spontaneous	No	No	Yes	3rd	No	No	No	No	No	Vaginal	_	29/4/17	Live	_	Female	2.3 kg	CTEV	Musculoskeletal	Yes
39	796987	36	25.1	G5P4L4	23	Spontaneous	No	No	Yes	2nd	No	No	No	No	s,T2D	Vaginal	_	1/4/2017	IUD	FSB	Male	730 g	Occipital encephalocele	CNS	_
40	801692	25	23.8	G2P1L1	27	Spontaneous	No	No	Yes	2nd	No	No	No	No	No	Vaginal	_	25/4/17	IUD	FSB	Female	900 g	Right multicystic dysplastic kidney	Genitourinary	_
41	804502	33	28.4	Primi	19	Assisted	No	No	Yes	2	No	No	No	No	No	Vaginal	_	10/5/2017	Abortion	_	Male	880 g	Skeletal dysplasia, ?Dandy Walker variant, ?Absent cerebellar vermis	Musculoskeletal	_
42	804632	37	32.8	G3P1L1A1	21	Spontaneous	No	No	Yes	2	No	No	No	Induced at 4th m	DM(8	Vaginal	_	13/5/17	IUD	FSB	Male	680 g	TGA with ASD with small right ventricle	CVS	_
43	805078	25	20.8	G2P1L1	18	Spontaneous	No	No	No	_	No	No	No	No	No	Vaginal	_	15/5/17	IUD	FSB	Male	200 g	Open spina bifida, Sacral agenesis	CNS	_
44	804546	24	17.5	G2P1L1	20	Spontaneous	No	No	Yes	2	No	Yes,3rd degree	No	No	No	Vaginal	_	16/5/17	IUD	FSB	Female	480 g	Left renal grade III Hydronephrosis with ventriculomegaly	Genitourinary	_
45	806917	21	23.1	Primi	17	Spontaneous	No	No	No	_	No	No	No	No	No	Abortion	_	25/5/17	Abortion	_	Female	369g	Cerebrovertebral anomaly	Musculoskeletal	_
46	807450	20	22.2	G2P1L1	24	Spontaneous	No	No	No	_	No	Yes,3rd degree	No	No	No	Vaginal	_	27/5/17	IUD	FSB	Female	690 g	Occipital encephalocele with Dandy Walker variant	CNS	_
47	807149	2	22.2	G3P2L1	20	Spontaneous	No	No	No	_	No	No	Yes, CNS anomaly	No	No	Vaginal	_	25/5/17	IUD	FSB	Female	260 g	Occipital encephalocele	CNS	_
48	808230	23	24.3	Primi	31	Spontaneous	No	_	Yes	2	No	No	No	No	No	Vaginal	_	1/6/2017	IUD	MSB	Male	2.4 kg	Hydrops fetalis	Others	_
49	809064	20	23.1	Primi	29	Spontaneous	No	_	Yes	3	No	No	No	No	No	Vaginal	_	3/6/2017	IUD	FSB	Male	1kg	Left kidney hydronephrosis with right kideny pelviectasis	Genitourinary	_
50	809538	30	24.8	G4P3L3	30	Spontaneous	No	_	Yes	3	No	No	No	No	No	Vaginal	_	11/6/2017	IUD	FSB	Male	2.2 kg	Hydrocephalus	CNS	_
51	809313	21	22.5	G2A1	35	Spontaneous	No	_	Yes	3	No	No	No	es, 1, spontaneou	No	Vaginal	_	5/6/2017	Live	_	Male	2.2 kg	Right renal pelviectasis	Genitourinary	Yes
52	809801	22	22.8	G3P1L1A1	34	Spontaneous	No	_	Yes	3	No	No	No	Yes, 1, induced	No	Vaginal	_	7/6/2017	Live	_	Male	1.9 kg	Hydrocephalus, Single umbilical artery, epispadias, CTEV	Multiple system affected	No (AMA)
53	810245	20	23.1	Primi	41	Spontaneous	No	_	Yes	2	No	Yes, 3rd degree	No	No	No	LSCS	CPD	9/6/2017	Live	_	Female	2.8 kg	VSD	CVS	Yes
54	810711	20	22.9	Primi	40	Spontaneous	No	_	Yes	3	No	No	No	No	No	Vaginal	_	13/6/17	Live	_	Male	2.5 kg	Left diaphragmatic hernia with hypoplastic lungs	Respiratory system	Yes
55	810323	40	25	G2P1L1	32	Spontaneous	No	_	Yes	2	No	No	No	No	Yes	LSCS	Previous LSCS with fetal distress	14/6/17	Live	_	Male	2.4 kg	Duodenal atresia	GIT	Yes
56	811345	25	23.4	Primi	39	Spontaneous	No	_	Yes	2	No	No	No	No	No	Vaginal	_	16/6/17	Live	_	Female	2.76 kg	Cardiomegaly, TR, DA stenosis	CVS	Yes
57	811549	22	23.7	G2A1	17	Spontaneous	No	_	Yes	2	No	No	No	es, 1 , spontaneou	No	Abortion	_	18/6/17	Abortion	_	_	_	Hydrocephalus, renal agensis, echogenic small bowel	Multiple system affected	_
58	814625	24	26.2	Primi	28	Spontaneous	No	_	Yes	2	No	No	No	No	No	Vaginal	_	4/7/2017	Live	_	Female	1.07 kg	Cleft lip, cleft palate	GIT	Yes
59	814721	26	23.2	G2P1L1	19	Spontaneous	No	_	No	_	No	No	No	No	No	Abortion	_	5/7/2017	Abortion	_	Male	350g	Trisomy 13	Others	_
60	814845	35	26.4	G2P1L1	19	Spontaneous	No	_	Yes	2	No	No	No	No	No	Abortion	_	5/7/2017	Abortion	_	Female	180 g	B/L renal agensis	Genitourinary	_
61	815501	25	21.8	Primi	13	Spontaneous	No	_	Yes	2	No	Yes, 3rd degree	No	No	No	D&E	_	8/7/2017	Abortion	_	_	_	Anencephaly	CNS	_
62	815076	24	25.2	Primi	24	Spontaneous	No	_	Yes	3rd	,3rd m	Yes	No	No	No	Vaginal	_	9/7/2017	IUD	FSB	Female	690 g	Meningomyolocele	CNS	_
63	815191	27	23.1	G2P1L1	19	Spontaneous	No	_	Yes	3	No	No	No	No	No	Abortion	_	8/7/2017	Abortion	_	Female	400 g	Kyphoscoliosis	Musculoskeletal	_
64	816647	22	26.8	G2P1L1	41	Spontaneous	No	_	Yes	3	No	No	No	No	No	Vaginal	_	15/7/17	Live	_	Female	3.16 kg	Left kidney hydronephrosis	Genitourinary	No

Sl. No	IP No	Age	BMI (kg/m2)	Parity	Gestational age wks	Mode of conception	H/o drug use	Drug taken	Folic acid intake	FA from month	H/o fever with rash	H/o consanguinity	H/o Previous congeintal anomalous baby	H/o abortions	GDM	Mode of delivery	Indication for LSCS	Date of delivery	Live/IUD	FSB/MSB	Sex of baby	Birth weight	Congenital anomaly	Organ system affected	NICU admission
65	815451	22	25.5	G4P3L1D2	19	Spontaneous	No	_	Yes	3	No	No	No	No	No	Abortion	_	11/7/2017	Abortion	_	Female	280g	Polycystic kidney	Genitourinary	_
66	817534	22	19.8	G2P1L0	40	Spontaneous	No	_	No	_	No	No	Yes, hydrocephalus	No	No	Vaginal	_	18/7/17	Live	_	Female	2.2 kg	Large VSD, Single umb artery, absent radius	CVS	Yes
67	815755	34	20.3	G2P1L1	36	Spontaneous	No	_	Yes	2	No	No	No	No	No	LSCS	Imminenent eclampsia	10/7/2017	Live	_	Female	1.86 kg	Trisomy 21	Others	Yes
68	817102	2527	27.5	Primi	31	Spontaneous	No	_	No	_	No	No	No	No	No	Vaginal	_	16/7/17	Live	_	Female	920 g	Gr I Hydronephrosis, shortening of long bones	Genitourinary	Yes
69	816016	22	24	G2P1L1	35	Spontaneous	No	_	Yes	2	No	No	No	No	No	LSCS	Prev LSCS	11/7/2017	Live	_	Male	1.9 kg	Congenital heart block	CVS	Yes
70	815689	26	21.3	G2P1L1	39	Spontaneous	No	_	Yes	3	No	No	No	No	No	LSCS	Breech	8/7/2017	Live	_	Male	1.7 kg	Amelia (absence of one lower limb)	Musculoskeletal	No
71	818216	20	30	G5PL1A1	36	Spontaneous	No	_	Yes	2	No	No	Yes, anencephaly (2)	Yes,1	Yes	LSCS	Fetal distress	25/7/17	Live	_	Female	2.7 kg	Renal pelviectasis	Genitourinary	Yes
72	818981	22	19.5	Primi	41	Spontaneous	No	_	Yes	2	No	No	No	No	No	LSCS	Fetal distress	26//17	Live	_	Male	1.9 kg	Bladder outlet obstruction, Echogenic small bowel	Genitourinary	Yes
73	819132	19	24	G6A5	28	Spontaneous	No	_	No	_	No	Yes, 3rd degree	No	Yes, 4 (Missed)	No	Vaginal	_	27/7/17	IUD	FSB	Male	1.3 kg	B/L hydronephrosis, bladde outlet obstruction	Genitourinary	No
74	819083	28	23.5	Primi	20	Spontaneous	No	_	Yes	1.5	No	No	No	No	No	Vaginal	_	30/7/17	IUD	FSB	Male	350 g	Multiple CNS anomaly	CNS	_
75	819011	28	26.1	G2E1	34	Spontaneous	No	_	No	_	No	No	No	No	No	Vaginal	_	29/7/17	Live	_	Female	2.8 kg	Cardiomegaly	CVS	No
76	817440	22	22.6	G2P1L1	38	Spontaneous	No	_	Yes	3	No	No	No	No	No	LSCS	Prev LSCS	26/7/17	Live	_	Female	2.7 kg	Small VSD with MVP	CVS	Yes
77	819164	19	20.8	Primi	37	Spontaneous	No	_	Yes	2	No	No	No	No	No	Vaginal	_	26/7/17	Live	_	Male	2.6 kg	Gr I Hydronephrosis	Genitourinary	No
78	815915	25	18.7	G5PL1A1D2	35	Spontaneous	No	_	No	_	No	No	Yes,?cardiac anomaly	Yes, 1, spontaneou	No	Vaginal	_	28/7/17	Live	_	Female	2.47 kg	Right renal pelviectasis	Genitourinary	No
79	820919	20	24.8	Primi	36	Spontaneous	No	_	Yes	2	No	No	No	No	No	LSCS	Fetal distress	5/8/2017	Live	_	Female	1.3 kg	B/l CTEV, polydactyly, syndactyly, choanal atresia, Intracardiac mass,	Musculoskeletal	Yes
80	823149	23	24.9	G3P2L2	41	Spontaneous	No	_	Yes	3	No	Yes, 3rd degree	No	No	No	Vaginal	_	17/8/17	Live	_	Female	3 kg	Right CTEV, B/l hydronephrosis	Musculoskeletal	No
81	823477	25	23.8	G3P2L2	21	Spontaneous	No	_	No	_	No	Yes, 3rd degree	No	No	No	Vaginal	_	19/8/17	IUD	FSB	Female	s	Omphalocele, hypoplastic lungs, kyphoscoliosis	Multiple system affected	_
82	824238	26	24.3	G3P2L2	33	Spontaneous	No	_	Yes	3	No	No	No	No	No	Vaginal	_	23/8/17	Live	_	Male	1.7kg	TEF	GIT	Yes
83	823858	21	24	Primi	36	Spontaneous	No	_	Yes	3	No	No	No	No	No	Vaginal	_	24/8/17	IUD	FSB	Male	1.9 kg	Dandy walker syndrome	CNS	_
84	824713	26	20.7	Primi	30	Spontaneous	No	_	Yes	2	No	No	No	No	No	Vaginal	_	28/8/17	IUD	FSB	Female	1.6 kg	Right lung hypoplasia with pleural effusion	Respiratory system	_
85	825029	23	22.6	Primi	19	Spontaneous	No	_	Yes	3	No	No	No	No	No	Vaginal	_	27/8/17	Abortion	_	Male	240 g	Anencephaly	CNS	_
86	822585	21	20.5	Primi	37	Spontaneous	No	_	Yes	3	No	No	No	No	No	Vaginal	_	14/8/17	Live	_	Male	3.296 kg	B/l CTEV, abnormal foot, claw hand	Musculoskeletal	Yes
87	822351	29	20.7	G3P2L1	22	Spontaneous	No	_	Yes	3	No	No	Yes,congenital heart disease	No	No	Vaginal	_	13/8/17	IUD	FSB	Female	300 g	Holoprocencephaly, cyclops	CNS	_
88	819955	23	22.6	G2P1L1	40	Spontaneous	No	_	Yes	3	No	No	No	No	No	LSCS	Prev LSCS with maternal tachycardia	12/8/2017	Live	_	Male		Diaphragmatic hernia with hypoplastic lungs	Respiratory system	Yes
89	820103	26	25.4	G2P1L1	37	Spontaneous	No	_	No	_	No	No	No	No	No	LSCS	Prev LSCS	2/8/2017	Live	_	Female	3 kg	Dandy Walker variant	CNS	Yes
90	828261	25	25.1	G2P1L1	18	Spontaneous	No	_	No	_	No	No	No	No	No	Abortion	_	14/9/17	Abortion	_	Female	350 g	upper throacic scoliosis	Musculoskeletal	_
91	831862	29	24.8	G2A1	14	Spontaneous	No	_	No	_	No	No	No	Yes,1, spontaneou	No	Abortion	_	29/9/17	Abortion	_	_	_	Anencephaly	CNS	_
92	930786	23	22	G2A1	40	Spontaneous	No	_	yes	2	No	No	No	Yes,1, spontaneou	No	Vaginal	_	23/9/17	Live	_	Female	2 kg	TOF	CVS	Yes
93	832556	20	24.3	Primi	29	Spontaneous	No	_	No	_	No	No	No	No	No	Vaginal	_	4/10/2017	IUD	FSB	Male	1.10 kg	B/l renal agenesis, VSD	Genitourinary	_
94	831953	23	20.54	G3P2L1	41	Spontaneous	No	_	Yes	3	No	No	No	No	No	LSCS	Previous LSCS	4/10/2017	Live	_	Female	3.59 kg	Lateral encephalocele	CNS	Yes
95	833391	33	25.7	G4P2L2A1	37	Spontaneous	No	_	Yes	2	No	No	No	Yes,1, induced	No	LSCS	Prev 2 LSCS in labour	7/10/2017	Live	_	Male	3 kg	Diaphragmatic hernia	Respiratory system	Yes

Sl. No	IP No	Age	BMI (kg/m2)	Parity	Gestational age wks	Mode of conception	H/o drug use	Drug taken	Folic acid intake	FA from month	H/o fever with rash	H/o consanguinity	H/o Previous congeintal anomalous baby	H/o abortions	GDM	Mode of delivery	Indication for LSCS	Date of delivery	Live/IUD	FSB/MSB	Sex of baby	Birth weight	Congenital anomaly	Organ system affected	NICU admission
96	833058	24	24.6	G3P2L2	38	Spontaneous	No	_	Yes	3	No	Yes, 3rd degree	No	No	No	Vaginal	_	7/10/2017	Live	_	Female	3.8 kg	B/l ventriculomegaly, sacral meningocele	CNS	Yes
97	835669	28	20.8	G2P1L1	39	Spontaneous	No	_	Yes	1.5	No	No	No	No	Yes	Vaginal	_	18/10/17	Live	_	Male	2.9 kg	Singleumbilical artery	CVS	No
98	835967	33	20.5	G7P3L2D1A1	36	Spontaneous	No	_	No	_	No	No	No	Yes,3, spontaneou	No	LSCS	Previous 2 LSCS with scar tenderness	20/10/17	IUD	FSB	Female	3.79 kg	Multiple deformities	Multiple system affected	_
99	842629	32	23.3	G2P1L1	15	Spontaneous	No	_	Yes	2	No	No	No	No	No	Vaginal	_	23/11/17	Abortion	_	Male	80 g	Anencephaly	CNS	_
100	842755	32	19.1	G2P1L1	11	Spontaneous	No	_	Yes	2	No	No	No	No	No	Vaginal	_	25/11/17	Abortion	_	_	_	Cystic hygroma	Others	_
101	838517	22	24.7	G3P1L1A1	33	Spontaneous	No	_	No	_	No	No	Yes,?anomaly	Yes	No	LSCS	Severe oligohydramnios	2/11/2017	Live	_	Male	1.8 kg	Dextrocardia	CVS	Yes
102	839800	30	20.8	G5P4L2D2	20	Spontaneous	No	_	No	_	No	No	No	No	No	Vaginal	_	9/11/2017	IUD	FSB	Male	400 g	Skeletal dysplasia	Musculoskeletal	_
103	840026	36	24.1	G2P1L1	34	Spontaneous	No	_	Yes	3	No	No	No	No	No	Vaginal	_	12/11/2017	Live	_	Male	1.4 kg	Cleft lip	GIT	Yes
104	840266	19	25.1	Primigravida	34	Spontaneous	No	_	Yes	3	No	No	No	No	No	LSCS	Breech	9/11/2017	Live	_	Male	1.82 kg	Syndactyly	Musculoskeletal	Yes
105	844691	22	22.6	Primi	38	Spontaneous	No	No	Yes	2	No	No	No	No	No	Vaginal	_	5/12/2017	Live	_	Female	2.43 kg	Syndactyly of left fingers and toes	Musculoskeletal	No
106	845062	31	21.3	G3P2L2	40	Spontaneous	No	No	Yes	3	No	No	No	No	No	LSCS	Obstructed labour	7/12/2017	IUD	FSB	Male	2.94 kg	Skeletal dysplasia	Musculoskeletal	_
107	844089	25	22.8	G2A1	20	Spontaneous	No	No	Yes	1.5	No	No	Yes, Anencephaly	Yes,1,induced	No	Abortion	_	4/12/2017	Abortion	_	Male	520 g	lumbar meningomyelocele	CNS	_
108	845428	25	20.5	Primi	40	Spontaneous	No	No	Yes	2	No	No	No	No	No	LSCS	Fetal distress	7/12/2017	Live	_	Female	3.2 kg	single umbilical artery, short neck	CVS	Yes
109	844680	22	23.8	Primi	22	Spontaneous	No	No	No	_	No	No	No	No	No	Vaginal	_	3/12/2017	IUD	FSB	Female	500g	complex heart disease	CVS	_
110	844625	19	24.2	Primi	36	Spontaneous	No	No	Yes	2	No	No	No	No	No	LSCS	Macrosomia	2/12/2017	Live	_	Male	3.19 kg	Meningocele	CNS	Yes
111	846880	20	24.2	Primi	37	Spontaneous	No	No	Yes	2	No	No	No	No	No	Vaginal	_	14/12/17	Live	_	Male	2.4 kg	Spina bifida occulta, fixed flexion deformity, absent fibula of rigth Lower limb, single umbilical artery, b/l short femur	Multiple system affected	Yes