A STUDY ON CARDIOVASCULAR COMPLICATIONS IN INFANTS OF DIABETIC MOTHER

DISSERTATION SUBMITTED TO

In partial fulfillment of the requirement for the degree of

(Branch VII) M. D. (PAEDIATRIC MEDICINE)

of

THE TAMIL NADU DR. M. G. R MEDICAL UNIVERSITY

CHENNAI- 600032

DEPARTMENT OF PAEDIATRIC MEDICINE

TIRUNELVELI MEDICAL COLLEGE

TIRUNELVELI- 11

MAY 2019

BONAFIDE CERTIFICATE

This is to certify that the dissertation entitled **"A STUDY ON CARDIOVASCULAR COMPLICATIONS IN INFANTS OF DIABETIC MOTHER"** submitted by **Dr. KIRUTHIGA. K** to the Tamilnadu Dr. M.G.R Medical University, Chennai, in partial fulfillment of the requirement for the award of M.D. Degree Branch – VII (Pediatric Medicine) is a bonafide research work carried out by her under direct supervision & guidance.

Professor &Head of the Department,
Department of Pediatric Medicine
Tirunelveli Medical College,
Tirunelveli.

Unit chief
Department of Pediatric Medicine
Tirunelveli Medical College,
Tirunelveli.

CERTIFICATE

This is to certify that the Dissertation **"A STUDY ON CARDIOVASCULAR COMPLICATIONS IN INFANTS OF DIABETIC MOTHER"**presented herein by **Dr.KIRUTHIGA.K** is an original work done in theDepartment of Pediatric Medicine, Tirunelveli Medical College Hospital,Tirunelveli for the award of Degree of M.D. (Branch VII) Pediatric Medicine. Under my guidance and supervision during the academic period of 2016 -2019.

The DEAN
Tirunelveli Medical College,
Tirunelveli - 627011.

DECLARATION

I solemnly declare that the dissertation titled **"A STUDY ON CARDIOVASCULAR COMPLICATIONS IN INFANTS OF DIABETIC MOTHER"**is done by me at Tirunelveli Medical College Hospital,Tirunelveli Under the guidance and supervision of Associate Prof.Dr.A.S.Babu Kandhakumar M.D.,D.C.H.,DNB.,M.N.A.M.S., the dissertation is submitted to The TamilnaduDr.M.G.R.MedicalUniversity towards the partial fulfilment of requirements for the award of M.D., Degree (Branch VII) in Pediatric Medicine.

Place: Tirunelveli

Date:

Dr.KIRUTHIGA.K,
Postgraduate Student,
M.D Pediatric Medicine,
Department of Pediatric Medicine,
Tirunelveli Medical College
Tirunelveli.

CERTIFICATE – II

This is to certify that this dissertation work title **"A STUDY ON CARDIOVASCULAR COMPLICATIONS IN INFANTS OF DIABETIC MOTHER"**of the candidate **Dr. KIRUTHIGA. K** with registration Number **201617351** for the award of **M.D.,** Degree in the branch of **PAEDIATRIC MEDICINE (VII).** I personally verified the urkund.com website for the purpose of plagiarism check. I found that the uploaded thesis file contains from introduction to conclusion page and result shows 0% p**ercentage** of plagiarism in the dissertation.

Guide & Supervisor sign with Seal.

URKUND

Urkund Analysis Result

Analysed Document:	A STUDY ON CARDIOVASCULAR ABNORMALITIES IN INFANTS OF DIABETIC MOTHER.pdf (D41900958)
Submitted:	9/28/2018 3:17:00 PM
Submitted By:	kirthi6509@gmail.com
Significance:	0 %

Sources included in the report:

Instances where selected sources appear:

0

ACKNOWLEDEGMENT

I wish to express my heartfelt gratitude to our Dean Prof. Dr. S. M. Kannan M.S., M.Ch., Tirunelveli Medical College for allowing me to do the study in this institution.

I would like to express my humble thanks to our professor & Head of the Department Prof. Dr. C. Krishnamurthy M.D., Department of paediatrics.

I express my sincere thanks to my renowned teacher and my guide Dr.A.S. Babukandhakumar, MD., DCH.,DNB.,M.N.A.M.S., Associate Professor Department of paediatrics, Tirunelveli Medical College for his able guidance, valuable suggestions and constant encouragement throughout the study.

I express my sincere thanks my professors Dr. T. R. R. Ananthy Shri M.D., Dr. C. Baskar M.D.,DCH., Dr. Rukmani M.D., for their constant support, encouragement and suggestions which helped me greatly to expedite this dissertation .

I also thank cardiologist for their support for doing ECHO.

I express my sincere thanks to my PG registrar Dr. B. Naresh M.D., department of Paediatrics.

I am greatly obliged to Dr.G.Jeyanthi M.D., DCH., Dr. A. Maheswari M.D.,DCH., and Dr. M. Muthuramasubramanian M.D., Assistant Professors, Dept.of paediatrics for their valuable suggestions in preparing this dissertation.

TIRUNELVELI MEDICAL COLLEGE

INSTITUTIONAL RESEARCH ETHICS COMMITTEE
TIRUNELVELI, STATE OF TAMILNADU, SOUTH INDIA PIN 627011
91-462-2572733-EXT; 91-462-2572944; 91-462-2579785; 91-462-2572611-16
online@tvmc.ac.in, tirec@tvmc.ac.in; www.tvmc.ac.in

CERTIFICATE OF REGISTRATION & APPROVAL OF THE TIREC REF NO: 1041/PAE/2017

PROTOCOL TITLE: CARDIAVASCULAR COMPLICATION IN INFANTS OF DIABETIC MOTHER
PRINCIPAL INVESTIGATOR: Ms.K.KIRUTHIGA,
DESIGNATION OF PRINCIPAL INVESTIGATOR: I YEAR MBBS STUDENT
DEPARTMENT & INSTITUTION: TIRUNELVELI MEDICAL COLLEGE, TIRUNELVELI

Dear,Ms.K.KIRUTHIGA, The Tirunelveli Medical College Institutional Ethics Committee (TIREC) reviewed and discussed your application during the IEC meeting held on 10.03.2017.

THE FOLLOWING DOCUMENTS WERE REVIEWED AND APPROVED

1.	TIREC Application Form	☐
2.	Study Protocol	☐
3.	Department Research Committee Approval	☐
4.	Patient Information Document and Consent Form in English and Vernacular Language	☐
5.	Investigator's Brochure	☐
6.	Proposed Methods for Patient Accrual Proposed	☐
7.	Curriculum Vitae of the Principal Investigator	☐
8.	Insurance /Compensation Policy	☐
9.	Investigator's Agreement with Sponsor	☐
10.	Investigator's Undertaking	☐
11.	DCGI/DGFT approval	☐
12.	Clinical Trial Agreement (CTA)	☐
13.	Memorandum of Understanding (MOU)/Material Transfer Agreement (MTA)	☐
14.	Clinical Trials Registry-India (CTRI) Registration	☐

THE PROTOCOL IS APPROVED IN ITS PRESENTED FORM ON THE FOLLOWING CONDITIONS

1. The approval is valid for a period of 2 year/s or duration of project whichever is later
2. The date of commencement of study should be informed
3. A written request should be submitted 3weeks before for renewal / extension of the validity
4. An annual status report should be submitted.
5. The TIREC will monitor the study
6. At the time of PI's retirement/leaving the institute, the study responsibility should be transferred to a person cleared by HOD
7. The PI should report to TIREC within 7 days of the occurrence of the SAE. If the SAE is Death, the Bioethics Cell should receive the SAE reporting form within 24 hours of the occurrence.
8. In the events of any protocol amendments, TIREC must be informed and the amendments should be highlighted in clear terms as follows:
   a. The exact alteration/amendment should be specified and indicated where the amendment occurred in the original project. (Page no. Clause no. etc.)
   b. The PI must comment how proposed amendment will affect the ongoing trial. Alteration in the budgetary status, staff requirement should be clearly indicated and the revised budget form should be submitted.
   c. If the amendments require a change in the consent form, the copy of revised Consent Form should be submitted to Ethics Committee for approval. If the amendment demands a re-look at the toxicity or side effects to patients, the same should be documented.
   d. If there are any amendments in the trial design, these must be incorporated in the protocol, and other study documents. These revised documents should be submitted for approval of the IEC, only then can they be implemented.
   e. Approval for amendment changes must be obtained prior to implementation of changes.
   f. The amendment is unlikely to be approved by the IEC unless all the above information is provided.
   g. Any deviation/violation/waiver in the protocol must be informed.

STANDS APPROVED UNDER SEAL

Dr.K.ShantaramanMD
Registrar, TIREC
Tirunelveli Medical College, Tirunelveli – 627011
State of Tamilnadu, South India

Dr.J.SureshDurai, MD
Member Secretary, TIREC
Tirunelveli Medical College, Tirunelveli – 627011
State of Tamilnadu, South India

CONTENTS

LIST OF TABLES

LIST OF FIGURES

ABBREVIATIONS

1.ACHD	Acyanotic congenital heart disease
2.AGA	Appropriate for gestational age
3.ASD	Atrial septal defect
4.CCHD	Congenital cyanotic heart disease
5.CHD	Congenital heart disease
6.CNS	Central nervous system
7.CVS	Cardiovascular system
8.CXR	Chest x ray
9.GDM	Gestational diabetes mellitus
10.IDM	Infant of diabetic mother
11.L/S RATIO	Lecithin spingomyelin ratio
12.LGA	Large for gestational age
13.NICU	Neonatal intensive care unit
14.OGTT	Oral glucose tolerance test
15.PDA	Patent ductus arteriosus
16.PFO	Patent foramen ovale
17.SGA	Small for gestational age
18.TA	Tricuspid atresia
19.TGA	Transposition of great vessels
20.VSD	Ventricular septal defect

1.INTRODUCTION

Diabetes mellitus is one of the most common medical problems worldwide. The world health organization has predicted that the prevalence of diabetes will increase by 35% by 2025.[1,2] Women of Asian origin have more risk of developing diabetes. Now, gestational diabetes mellitus is increasing and amounts to 17% in Asian women but only 4% of American & European women.[3,4]

In southern India, the prevalence of GDM was 17% in urban women, 13.8% in semi urban and 9.8% in rural .[4,5] Overt Diabetes and GDM where associated with high perinatal mortality and morbidity, to add to that there was increased incidence of still birth and birth defects. When compared to babies of non-diabetic women, IDM babies showed more incidence of neonatal complications.[6] By reviewing recent data was shown that OHA were effective in control of diabetes during pregnancy, with no teratogenic effect.[7]

Birth defects were common in the order of cardiac and neural tube defects. In the offspring's of diabetic mother, the incidence of cardiac anomalies was 3 to 6% which is 5 times higher than non-diabetic pregnancy and most of the time it included complex congenital heart disease.[8,9]More frequently reported anomalies were conotruncal such as truncus arteriosus ,tricuspid atresia , TGA. The incidence of TGA, in overt

diabetes mother was 17 times more than that of non diabetic women[10]. The closure of ductus and decrease in pulmonary pressure were delayed in babies of diabetic mother compared to a normal neonate. [11,12] Good glycemic control had better outcome, lower occurrence of fetal heart disease, but did not decrease the incidence of asymmetrical septal hypertrophy.[13] Diabetic cardiomyopathy was self-limiting with no clinical consequence and was a transient phenomenon that usually regressed within first few months of life.[1o] Septal hypertrophy occurred even in mother with good glycemic control,without relation to the type of diabetes.[14] The prevalence of septal hypertrophy in type 1 diabetes was more than that of type 2 and GDM.[15] The risk of congenital malformations was 3 to 4 times higher than that of non-diabetic mother[16.] These babies were prone to be larger, because of hyperglycemia & hyperinsulinemia[2]

2.STUDY JUSTIFICATION

Diabetes complicating pregnancy is increasing in incidence. Babies born to these mothers have increased risk of complications including intrauterine, intrapartum, perinatal . These babies are more susceptible for various congenital anomalies such as cardiovascular, neural tube defects and others.

Various studies have been done in this and showed the relationship between maternal diabetes and fetal malformations / complications. This study analyses the association between maternal type of diabetes, its treatment and degree of glycemic status control with cardiac anomalies of newborn.

3.AIM OF THE STUDY

To explore the spectrum of cardiovascular complication in infants of diabetic mother and probable association between infant's heart lesion.

- Type of maternal diabetes.

- Maternal treatment regimen

- Mother's glycemic control

4. REVIEW OF LITERATURE

INFANT OF DIABETIC MOTHER

DIABETES OVERVIEW

Diabetes mellitus is one of the most common health problems in pregnancy and it has negative outcomes to the infant born to them. Mostly birth defects, most common defects involved cardiovascular and nervous system-NTD [neural tube defect].

Diabetes mellitus: etiological classification

1. Type 1 diabetes mellitus (beta cell destruction)

    a. immune mediated

    b. idiopathic

2. Type 2 diabetes mellitus (insulin resistance)

3. Other specific types

    A. Genetic defects of beta cell function

    -maturity onset diabetes of young (MODY) defects in chromosome 12,7,20,13,17,2. MODY 1 to 6

    B. Genetic defects in insulin action -insulin resistance

    C. Diseases of exocrine pancreas

        Pancreatitis

Trauma / pancreatectomy

Neoplasia

Cystic fibrosis

Calculus pancreatopathy

D. Endocrinopathies

Cushing syndrome

Acromegaly

Glucagonoma

Pheochromocytoma

Hyperthyroidism

E. Drug or chemical induced diabetes

Diabetes types – In pregnancy

1. Pregestational diabetes is the term used for women who were diagnosed as diabetic before pregnancy.
    a. Type I
    b. Type II
2. Gestational diabetes is used to women who didn't have diabetes before pregnancy or became diabetic at some point during pregnancy.

Whatever may be the type of diabetes during pregnancy there are some adverse effects to both mother and fetus health.[17]

Pregnancy and diabetes

There is altered carbohydrate metabolism during pregnancy, it is the reason for increased tendency of diabetes during pregnancy.

Pregnancy and its diabetogenic effects

1. Insulin resistance

- Placenta produces human placental lactogen
- Cortisol, estriol, and progesterone increases during pregnancy
- Kidney and placenta destroys more insulin.

2. Lipolysis increased during pregnancy

Because utilization of glucose for fetus and mother energy based on fat metabolism.

3. Changes in gluconeogenesis

The fetus preferentially utilizes alanine and other amino acids, depriving themother of a major neogluconic source.

Factors that predict the pregnant women to become diabetic in future are :

1 .Early diagnosis of gestational diabetes mellitius

2. Requiring insulin therapy

3. Preterm delivery

4. Large baby

5. Abnormal glucose tolerance test after 2months of childbirth

GDM is associated with increased risk of perinatal morbidities and mortalities[18]

TYPES OF DIABETES MELLITUS

TYPE 1 DIABETES

Type I Diabetes is an autoimmune disorder characterized by self destruction of beta cells of pancreas, involving 5 to 10% of all peoples with diabetes. In this, the body immune mechanism destroys beta cells whose main function is secreting insulin.[17]The rate of destruction varies in every individuals. [19]

Type I features are complete absence of insulin, elevated blood sugar level, catabolism of protein and fat. These people are susceptible to develop DKA, a life threatening condition if not treated quickly. The main action of insulin is to prevent lipolysis (i.e.) fat breakdown, to release free fatty acids.

In type I diabetes, due to absence of insulin, free fatty acids are released from the cells and the liver converts them into ketones causing ketoacidosis. Decreased insulin production in pancreas necessitates the use of exogenous insulin to prevent catabolic state and to achieve glucose

control & prevent ketosis [19]. Survival of these people is dependent on this exogenous insulin[17].There is genetic predisposition in some people. Susceptible genes are HLA, insulin, PTPN22, ILR2a and CTLA4. TYPE 1diabetes is also called as insulin dependent Diabetes Mellitus or Juvenile Diabetes.

TYPE II DIABETES MELLITUS.

This type accounts for 90 to 95% of all cases, characterized by a heterogeneous condition. With an absolute absence of insulin associated with the body cells failing to respond to available insulin properly. In this, pancreas produces insulin, which is inadequate or not used well by the cells.[17]The major abnormality is insulin resistance, when trying to metabolize glucose & lipids.[19]

This is due to the unresponsive state of insulin receptors present in liver,fat cells and muscle which result in hyperglycemia.[17]During the initial stages of insulin resistance; pancreas produces high insulin in response to hyperglycemia. This creates temporary hyperinsulinemia along with hyperglycemia.[19] In type II, there is an unbalanced insulin secretion & increase in liver glucose. In type 2, people can have insulin levels of high,to normal to low depending on many factors. The liver doesn't regulate the glucose release in response to changes in blood glucose. Type 2 diabetes is of gradual onset, many people being diagnosed only during routine lab testings[17].

Risk factors for type 2 Diabetes [according to International Diabetes federation]

1. Family history of diabetes
2. Obesity
3. Physical inactivity
4. High blood pressure
5. Impaired glucose tolerance
6. Increased age

GESTATIONAL DIABETES MELLITUS

Third type of diabetes occurs in pregnant women, who had never been diabetic before.GDM refers to any degree of glucose intolerance that is identified during pregnancy.[19]Most commonly it affects the women who are obese &with family history of diabetes.[17]In GDM, the developing placenta impairs the maternal insulin during pregnancy. As pregnancy advances the placenta produces more insulin blocking hormones thus increasing maternal glucose & chance to have diabetes during pregnancy.[17]GDM mothers are usually asymptomatic, but present with excessive thirst& polyuria.

Risk factors:

a. Elderly mother
b. H/o GDM in previous pregnancy
c. Family h/o diabetes

d. Obesity prepregnancy BMI >30 [21]

e. Multifetal gestation

f. Recurrent UTI

g. Infertility treatment

h. H/o large babies

i. Unexplained neonatal death

j. Pre-eclampsia

k. Asian descent women[17,18].

l. Birth of malformed infant before.[21]

m. Polyhydramnios h/o

n. Maternal birth weight >4 kg[21]

o. PCOD

TABLE 1: WHITE CLASSIFICATION OF MATERNAL DIABETES.[20]

GD	Gestational diabetes: diabetes not known to be present before pregnancy
GD diet	Euglycemia maintained by diet only
GD insulin	Diet alone insufficient required insulin
Class A	Chemical diabetes; glucose intolerance before pregnancy; treated by diet alone Prediabetes; history of large baby or unexplained still birth after 28 weeks
Class B	Insulin dependent , onset after 20 years of age Duration < 10 years
Class C	C1 onset @ 10 to 19 years C2 duration of diabetes 10 to 19 years
Class D	D1 onset before 10 years age D2 duration of diabetes 20 years D3 calcification of leg vessels D4 hypertension D5 benign retinopathy
Class F	Nephropathy with proteinuria > 500mg/day
Class R	Proliferative retinopathy / vitreal hemorrhage
Class RF	Criteria for both R&F present
Class G	Reproductive failure
Class H	Atherosclerotic heart disease
Class T	Prior to renal transplantation

PATHOPHYSIOLOGY:

Maternal hyperglycemia leads to fetal hyperglycemia and fetal hyperinsulinemia[20] which results in fetal overgrowth: Pederson's hypothesis. Insulin requirements increases as pregnancy advances because of hormones that are produced in placenta. As organogenesis occurs in early trimester, hyperglycemia in early trimester has poor outcome.

1. Maternal hyperglcemia in early trimester that is periconceptional period leads to fetal hyperglycemia will cause fetal embryopathic effects.

2. Fetal hyperglycemia leads to fetal hyperinsulinemia and cause

    a. Neonatal hypoglycaemia

    b. Surfactant deficiency

    c.Immature liver metabolism – NNH

3. Fetal hyperglycemia and hyperinsulinemia causes

    a. Fetal macrosomia – difficult labour , birth asphyxia,

    b. TTN

    c. Cardiomyopathy

    d. Polycythemia vascular events

DIAGNOSIS OF GDM

Gestational diabetes, generally, has few symptoms and it is most commonly diagnosed by screening during pregnancy. Most women are screened between 24 & 28 weeks of gestation as recommended by American college of Obstetricians and Gynaecologist. ACOG – 2 Step approach

Step 1: oral glucose challenge test[21]

Performed by giving 50grams of oral glucose irrespective of previous meal,no fasting is required. Plasma glucose is measured after 1 hour.

Cutoff value< 140mg/dl.

If plasma glucose levels > 140mg/dl the screening is positive& requires confirmative test.

STEP 2: Oral glucose tolerance test[21]

After overnight fasting of 8hours, this test done with 100 grams of glucose. Totally, 4 blood samples are taken.

a. 1st sample: FBS
b. 2nd sample: 1st hour PP sample
c. 3rd sample: 2nd hour PP sample
d. 4th sample: 3rd hour PP sample

Criteria for diagnosing gestational diabetes mellitus:

TABLE 2: CARPENTER AND COUSTAN CRITERIA[22]

Samples	Blood glucose (mg/dl)	Blood glucose (mmol)
Fasting	95	5.3
1 hour	180	10.0
2 hour	155	8.6
3 hour	140	7.8

If, out of these, any 2 values are abnormal, it is confirmed as a case of gestational diabetes

SINGLE DIAGNOSTIC TEST [21]

It is recommended by American Diabetes Association and International Association of Diabetes and by WHO

1. Done only in females with high risk factor
2. Used for both screening & diagnostic purpose
3. Patients advised unrestricted diet for 72 hours followed by overnight fasting and then 75gram of glucose is given.
4. Three samples taken
    a. 1st – fasting sample
    b. 2nd sample after 1 hour of 75gram glucose
    c. 3rd sample after 2 hours of 75 gram glucose

TABLE 3: SINGLE DIAGNOSTIC TEST - VALUES

Samples	Blood glucose (mg/dl)	Blood glucose (mmol)
Fasting	92	5.1
1 hour	180	10.1
2 hour	153	8.5

Out of these 3 values if any one value is abnormal, then the patient is considered as having gestational diabetes mellitus[21]

Women diagnosed with GDM have 60% lifetime risk of developing overt type 2 diabetes[20].

COMPLICATIONS OF MATERNAL DIABETES

MATERNAL COMPLICATIONS:[22]

TYPE 1 &2:

a. Ketoacidosis

b.Macrovascular:nephropathy, hypertension

c. Microvascular: retinopathy, neuropathy

d. Diabetic cardiomyopathy

e. Uteroplacental insufficiency

f. Polyhydramnios

Gestational diabetes mellitus:

1. Pregnancy induced hypertension
2. Preterm labour[22]
3. Still birth[23]
4. Antepartum hemorrhage
5. Premature rupture of membranes
6. Instrumental delivery
7. Pre-eclampsia
8. Caesarean section
9. Chorioamnionitis
10. Urinary tract infections.

FETAL COMPLICATIONS:

PREGESTATIONAL DIABETES:

1. **Diabetic embryopathy**[20] **–Periconceptional hyperglycemia** and poor glycemic control has been associated with a significantly increased risk of congenital malformations. Risk increases with increasing maternal HbA1c level in and around period of conception and 1st trimester. women with >7.5% level of HbA1c in 1st trimester had a 9 fold increased risk of congential malformations. Increased levels of glucose and increased formation of ketone bodies found to be teratogenic in animal models. Increased blood glucose leads to more free radical production which will

cause membrane damage and mitochondrial dysfunction leads to embryopathy.

2. **Diabetic fetopathy**[20] - due to hyperglycemia in 2nd&3rd trimester. These are any congenital malformations anticipated to be lethal or require surgical repair. There is no anomalies pathognomic for maternal diabetes. But pregestational diabetes mellitus confers 26 fold increased risk for caudal regression syndrome.

3. **Congenital malformations**[24]: most common cardiac anomalies comprise 40% to 50% of malformations encountered in IDM.

BIRTH DEFECTS:

When comparing pregestational diabetes & gestational diabetes more birth defects occur in pregestational diabetes. Of these, neural tube defects and cardiac defects are most common. The risk factor for congenital malformations is hyperglycemia during early Trimester. Pregestational diabetes is a risk for NTD& cardiac defects. When compared with non diabetic mothers, the pre-existing diabetic mothers have 6 times high risk to have babies with congential malformations.[25] The birth defectsthat occurs in newborn of diabetic mother with good glycemic control was similar to that of non diabetic mother.[26]

The birth defects in order of prevalence[20]

a. Congenital heart disease

b. Central nervous system defect

c. Urogenital defects

d. Limb defects

e.Orofacial defects

f. Sacral agenesis / caudal regression syndrome

There were many associations noted between pre-gestational diabetes & congenital malformations of non cardiac defects – hydrocephalus, anotia,microtia,anencephaly, craniorachischis, cleft lip, renal agenesis, anorectal malformations , longitudinal limb abnormalities.

16 cardiac defects are identified. Of them 11 are associated with pregestational diabetic mother. These includes TOF, TGA,VSD,ASD,TAPVC,aortic stenosis, left & right ventricular outflow tract obstruction. Complete AV canal defect and non-isolated AVseptal defects are the leading cause of death in congenital anomaly- related deaths. More risk for malformation is obese women, pregestational diabetes mother. Because of this, congenital heart defects incidence in newborn of diabetic mother was 5 times more than that of general population.[27]Maternal diabetes modifies the gene expression involved in heart development, which results from maternal hyperglycemia being toxic to embryo.[28]

Most negative outcomes such as miscarriage& still births, congenital anomalies are associated with pregnancy with poor glycemic control[29]. Maternal hyperglycemia is an important factor affecting maternal well

being and neonatal morbidity and mortality.[30] Pregestational diabetes with good control and intense treatment had decreased malformations.

NEONATAL COMPLICATIONS: [20]

1. **Macrosomic babies or large babies** : maternal diabetes is associated with abnormal growth patterns more commonly large for gestational age . It is defined as birth weight greater than 90th percentile for gestational age. This is due to maternal hyperglycemia and fetal hyperinsulinemia as stated by Pederson hypothesis.fetal hyperinsulinemia and increased insulin like growth factor results in fetal over growth as evidenced by increased fat deposition and visceral enlargement.

2**. Shoulder dystocia**[31]: leads to difficult vaginal delivery , birth injuries and increased risk of perinatal asphyxia.

3. **IUGR:** Pregestational diabetes mellitus mother with microvascular complications or associated chronic hypertension have an increased risk of fetal growth restriction.

4. **Respiratory distress**:RDS is more common in IDM as compared with newborn of non diabetic mother with same gestational age. It may also involve late preterm IDMs. Respiratory distress in IDM is due to delayed pulmonary maturation and surfactant production. Insulin regulates fetal lung maturation, excess insulin levels have an

inhibitory effect on phosphatidyl glycerol synthesis and choline incorporation in phosphatidyl choline.

5.**Hypoglycemia**[32]: it is one of the most common neonatal morbidities in IDM. blood glucose <47mg/dl is defined as hypoglycaemia. It is due to fetal hyperinsulinism. Symptoms of hypoglycaemia may include tremor, jitteriness, irritability,lethary, hyotonia. Convulsion, apnoea occurs in severe hypoglycaemia. Recurrent or persistent hypoglycaemia may cause adverse neurological outcome.

6. **Hypocalcemia**/ **Hypomagnesimia**: serum calcium <7mg/dl or ionized calcium <4.4mg/dl defined as hypoglycaemia in newborns. Hypomagnesemia defined as serum magnesium <1.5 mmg/dl. Symptoms of hypocalcemia and hypomagnesemia are similar to those of hypoglycaemia. Throughout the gestation , Calcium and magnesium are transferred transplacentally. At delivery, these supply terminated. In IDM increased risk of hypocalcemia is due to delayed transition from fetal to neonatal parathyroid action.

7. **Polycythemia:** venous hematocrit above 65% or haemoglobin > 20gm /dl. Polycythemic newborns have increased complications resulting from hyperviscosity. These complications are ischemia and infarction within kidneys – renal vein thrombosis, CNS- stroke. Clinically polycythemic babies are plethoric, lethargic.

8**. Hyperbilirubinemia** : this is due to polycythemia and ineffective erythropoiesis with increased RBCs turnover, immaturity of hepatobiliary system.

9. **Renal vein thrombosis** : due to polycythemia and increased viscosity and sluggish circulation.

10. Small left colon syndrome

11. Poor feeding:

12. Premature baby

Management of diabetes in pregnancy.

Monitoring:

1. Testing for type 1 & 2 diabetes (first trimester)
   a. By Measuring of glycosylated hemoglobin in the 1st trimester can assess the risk for congenital anomalies by reflecting glucose concentration in organogenesis period.
   b. Accurate dating of pregnancy by USG
   c. Ophthalmic examination is mandatory. Retinopathy is common in type 1 &2. Mother with retinopathy needs periodic ophthalmic visit & they are the candidate for laser photocoagulation.
   d. Renal function: assessed by spot protein creatinine ratio or urine for microalbuminuria, 24hr urine protein estimation , serum creatinine. Baseline proteinuria impacts the diagnosis of pre-eclampsia later.
   e. Thyroid function test: essential

f. Nuchal translucency and serum screening for aneuploidy.

2. Testingfor type 1 & 2 diabetes in 2nd trimester

a. Maternal serum screening for neural tube defects between 15 to 19 weeks . women with diabetes have 10 fold increased risk for NTD .

b. All mothers should undergo anomaly scan including detailed fetal ECHO.[20]

3. Testing in third trimester – type 1 &2 & GDM:

a. USG examination monthly

b. Fetal surveillance weekly or twice weekly

D. Fetal surveillance

Non stress test

Biophysical profile

Fetal heart rate monitoring.

Treatment of diabetes in 1st trimester

Maternal diabetes, through its adverse effects on maternal metabolism, is the responsible factor for increase in malformations in the offsprings[33], so adequate control of maternal blood sugar is essential.

INSULIN TREATMENT:

The treatment choice for overtly and pregestational diabetic mother is insulin.[34] Oral hypoglycemic agents are not effective in these

mothers. By regularizing diet and multiple insulin injections daily we can achieve glycemic control.

TABLE 4: INSULIN TREATMENT

Insulin type	Onset	Peak	Duration
SHORT ACTING			
Lispro	<15mins	0.5 -1.5	3-4
Glulisine	<15mins	0.5 -1.5	3-4
Aspart	<15mins	o.5-1.5	3-4
Regular	30 to 60mins	2-3	4-6
LONG ACTING			
Detemir	1to 4hr	Minimal	Upto 24
Glargine	1to 4hr	Minimal	Up to 24
NPH	1to 4hr	6 – 10	10 – 16

Monitoring: self monitoring of capillary blood glucose using glucometer is essential for insulin titration.

TABLE 5: CBG GOALS

Samples	**Blood sugar (mg/dl)**
Fasting	<95
Premeal	<100
1hr postprandial	<140
2hr postprandial	<120
Mean	100

DIET: carbohydrate, protein, fat is adjusted according to individual patient's preference. Minimum carbohydrate diet of 175mg should be provided as small to moderate meal size with snacks.

TREATMENT FOR ALL TYPE OF GLUCOSE TOLERANCE:

Strict diabetic controlachieved by diet modification,exercise, medications.

Goals: Fasting <95mg/dl

Postprandial < 140mg/dl

By insulin therapy with more perinatal safety.

Recently oral hypoglycemic agents such as glyburide, metformin are as effective as insulin therapy.

MANAGEMENT OF LABOR & DELIVERY

Iatrogenic preterm labour is common in diabetic mother[20] because of fetal complications such as non reassuring fetal heart tracing, microvascular disease, uteroplacental insufficiency & pregnancy induced hypertension. Antenatal steroids are necessary for fetal lung maturity. Labour after 39 weeks doesn't require Antenatal steroids for lung maturity.

Lung maturity is assessed by measuring amniotic fluid lecithin spingomyelin ratio. Depending on fetal weight , maternal complications and previous obstetric history route of delivery is decided. At an emergency situation, L/S measurement is not necessary

Management of IDM babies.

After the infant is born initial assessment is done by APGAR score. Based on that, resuscitation required or not is decided. Airway is cleared of secretion. Initial screening examination has to be done to rule out major congenital anomalies. Basic supportive care, initial blood glucose monitoring, and continuousevaluation of newborn should be given in newborn nursery . Newborn should be evaluated for hemodynamic stability, respiratory distress features, and cyanosis. Thorough clinical examination should be done. Blood glucose monitoring @ 1,2,3,6,12,24,48 hours of life. Hematocrit, calcium blood levels should be checked.

All newborns should be thoroughly examined for cariac abnormalities (ECG, CXR, ECHO).

5.MATERIALS AND METHOD

STUDY DESIGN

Its is CROSS SECTIONAL DESCRIPTIVE STUDY

STUDY CENTRE

Our study was conducted in the sick new born ward, department of paediatrics Tirunelveli medical college hospital, a tertiary care centre.

STUDY PERIOD

One year April 2017 to march 2018

STUDY POPULATION

Infants of diabetic mothers born at Tirunelveli medical college hospital, a tertiary care centre.

INCLUSION CRITERIA

All babies born to diabetic mothers both pre gestational and gestational diabetic mother.

EXCLUSION CRITERIA

1. Babies born as still born
2. Babies born with features suggestive of chromosomal abnormality

3. Babies born to diabetic mothers with other co- morbidities like hypothyroidism, hypertension, anemia complicating pregnancy, and seizure disorder on AEDs.

4. Babies born to diabetic mother with family history of heart disease.

5. Babies whose parents didn't give consent to undergo study.

6.METHODOLOGY

A prestructured proforma was used to obtain informations. The following parameters at the time of admission were considered in the study

1. Maternal diabetes type,

2. Maternal treatment regimen,

3. Maternal glycemic status,

4. Antenatal ultra sonogram

5. Baby's sex

6. Maturity,

7. Birth weight,

8. Clinical manifestation murmur/ cyanosis {SaO2},

9. Chest x-ray finding,

10. ECG finding

11. ECHO finding

All babies satisfying inclusion criteria were enrolled for study after obtaining consent. At first, a data sheet was completed for each newborn with the informations obtained.

All infants will undergo thorough physical examination with special attention to cardiovascular system and following investigations done. Oxygen saturation – by pulse oximetry low spo2 (95%), Electrocardiogram (chamber enlargement changes in ECG),Chest X- ray (cardiomegaly),Echo cardiogram (structural abnormality of heart- septal defect, shunt lesions.)

STATISTICAL ANALYSIS

Data collected and recorded in the Proforma during the whole study period were entered in Microsoft excel sheet and analysed to identify the relation between maternal diabetes &cardiovascular abnormality in the newborn born to them. The Software used in this study was SPSS software version 21.0. Tests used were or comparing mean is T test and ANOVA. For comparing the relation between groups, Chisquare and kruskal Wallis test were used.

7.OBSERVATION AND RESULTS

TOTAL CASES 100

TABLE 6: TYPES OF MATERNAL DIABETES

TYPE	NO OF PATIENTS(N=100)	PERCENTAGE (%)
PREGESTATIONAL	17	17
GESTATIONAL	83	83

Among **100 mothers**, 17 had pre gestational diabetes and 83 had gestational diabetes.

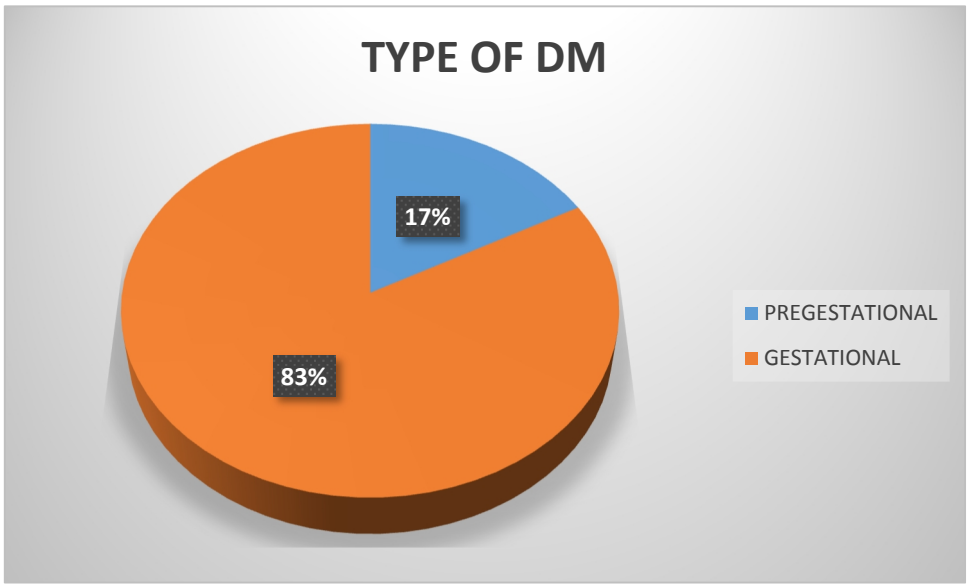

FIG 1: TYPES OF MATERNAL DIABETES

TABLE 7: TYPES OF DIABETES AND ECHO ABNORMALITY

TYPE	TOTAL NO. OF BABIES EXAMINED (N)	ECHO ABNORMALITY	
		PRESENT (N) (%)	ABSENT (N) (%)
PREGESTATIONAL	17	16 (94)	1 (6)
GESTATIONAL	83	12 (14)	71 (85.5)
CHI SQUARE TEST			
P VALUE - 0.001			

There was significant influence of the type of maternal diabetes present on the outcome of these babies born to them. (i.e.)The ECHO abnormality was significantly more common in pre gestational diabetes group.

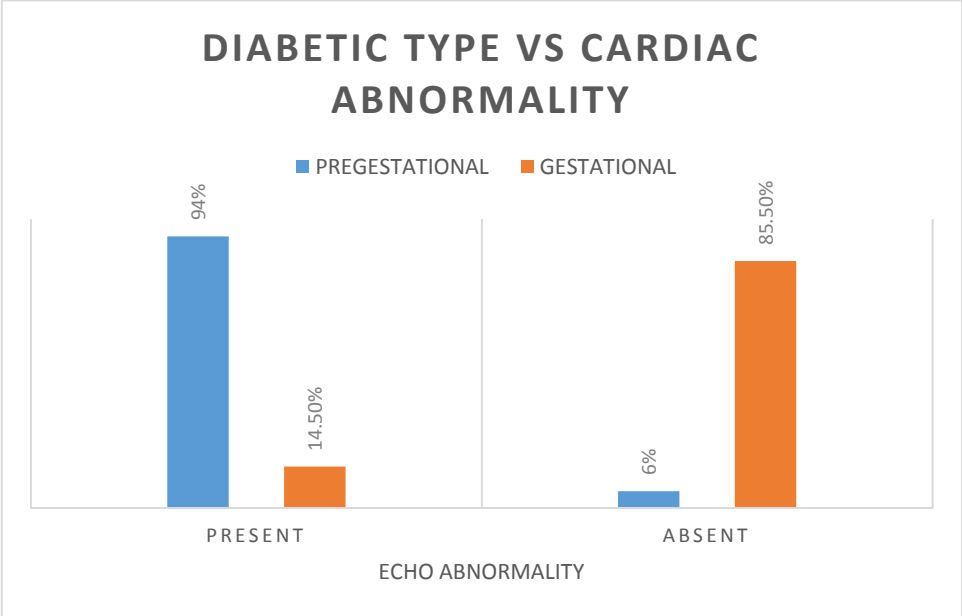

FIG 2: TYPES OF DIABETES AND ECHO ABNORMALITY

TABLE 8: MATERNAL DIABETES AND TYPE OF ECHO ABNORMALITY

TYPE	TOTAL NO. ECHO ABNORMALITY (N)	TYPE OF ECHO ABNORMALITY	
		ACYANOTIC (N)(%)	CYANOTIC (N)(%)
PREGESTATIONAL	16	11 (68.7)	5 (31.3)
GESTATIONAL	12	12 (100)	0 (0)
CHI SQUARE TEST			
P VALUE - 0.033 SIGNIFICANT			

ECHO abnormalities were more common pre gestational diabetes group, more of cyanotic heart disease.

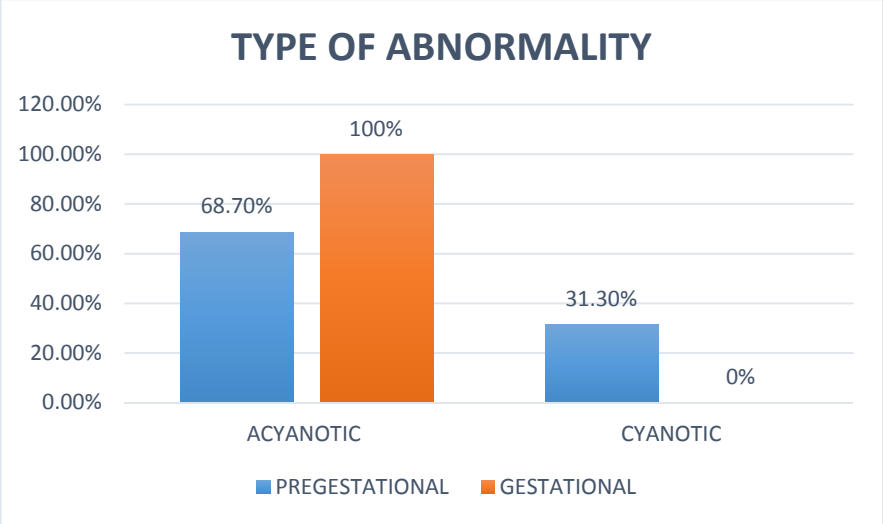

FIG 3: MATERNAL DIABETES AND TYPE OF ECHO ABNORMALITY

In our study, all babies with cyanotic heart disease were born to mother with pre gestational diabetes.

TABLE 9: DISTRIBUTION OF TREATMENT REGIMEN OF MOTHERS

TREATMENT REGIMEN	NO OF PATIENTS(N)	PERCENTAGE (%)
MEAL PLAN	66	66
OHA METFORMIN	21	21
INSULIN	13	13

Out OF 100 diabeticmothers, 66 were on meal plan, 21 were on OHA metformin , 13 were on insulin therapy.

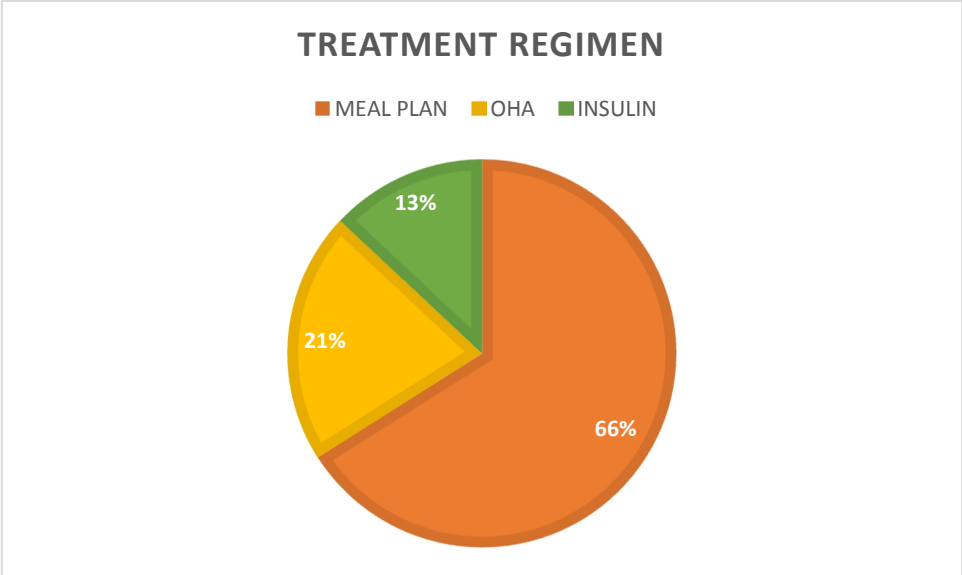

FIG 4: DISTRIBUTION OF TREATMENT REGIMEN OF MOTHERS

TABLE 10: ECHO ABNORMALITY AND MOTHERS TREATMENT REGIMEN

TREATMENT REGIMEN	TOTAL NO. OF EXAMINED (N=100)	ECHO ABNORMALITY	
		PRESENT (N)(%)	ABSENT (N) (%)
MEAL PLAN	66	8 (12)	58(88)
OHA	21	7 (33)	14(67)
INSULIN	13	13 (100)	0 (0)
KRUSKAL WALLIS TEST			
P VALUE - 0.001			

There was significant influence of maternal diabetes treatment regimen in the outcome of babies born to them. Among 66 babies whose mother was on meal plan, 8 had ECHO abnormality. Of the 21 OHA mothers, 7 had ECHO abnormality and of 13 INSULIN taking mothers, 13 had ECHO abnormality significantly.

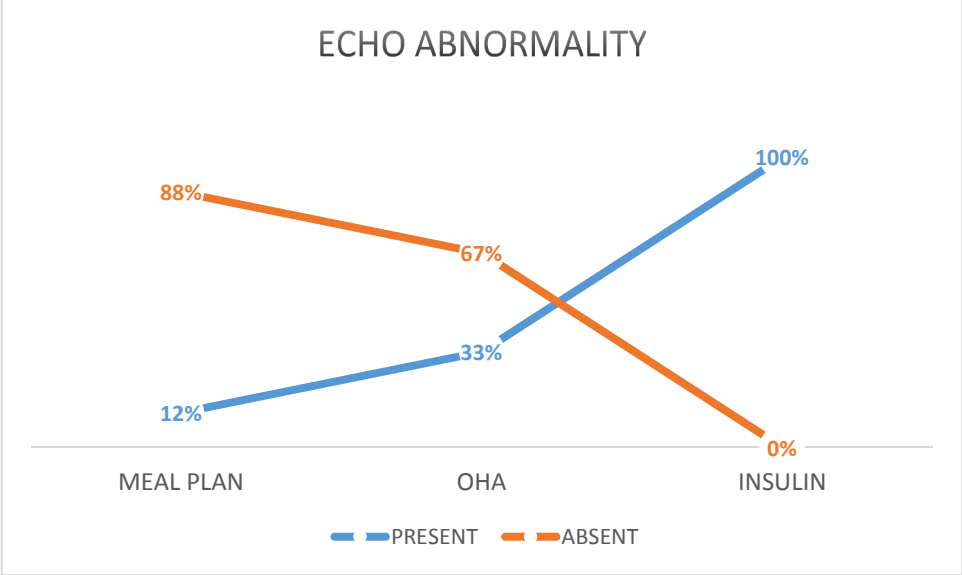

FIG 5: ECHO ABNORMALITY AND MOTHERS TREATMENT REGIMEN

This representation showed that ECHO abnormality was found in 12% of babies born to mother on meal plan, 33% babies born to mother on metformin , 100% of babies born to mother on insulin.

TABLE 11: TYPE OF ECHO ABNORMALITY AND VARIOUS MATERNAL TREATMENTREGIMENS

TREATMENT REGIMEN	TOTAL NO OF ECHO ABNORMALITY (N)	TYPE OF ECHO ABNORMALITY	
		ACYANOTIC (N)(%)	CYANOTIC (N)(%)
MEAL PLAN	8	8 (100)	0(0)
OHA	7	7(100)	0 (0)
INSULIN	13	8 (61.53)	5 (38)
KRUSKAL WALLIS TEST			
P VALUE - 0.030			
SIGNIFICANT			

Significant influence of maternal diabetes treatment regimen was present on newborn heart disease. Mother on insulin treatment more number of CHD.

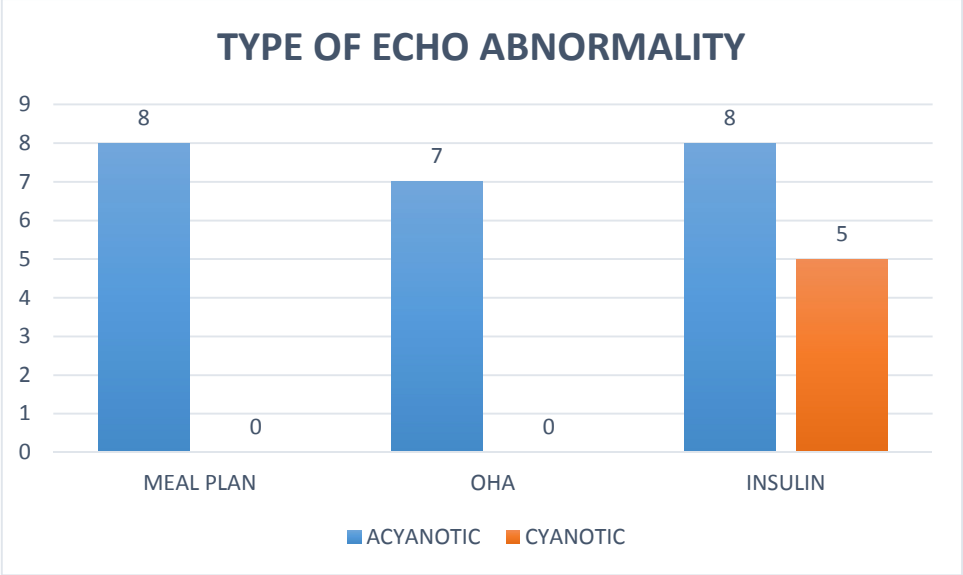

FIG 6: TYPE OF ECHO ABNORMALITY AND VARIOUS MATERNAL TREATMENTREGIMENS

Of these, cyanotic heart disease was more common on mothers taking insulin treatment, who had pre gestational diabetes mellitus.

TABLE 12: GLYCEMIC STATUS AMONG MOTHERS

GLYCEMIC CONTROL HbA1c	NO OF PATIENTS (N)	PERCENTAGE (%)
WELL (<6)	83	83
POOR(>7)	7	7
UNKNOWN	10	10

Of 100 mothers, 83 had good glycemic control , 7 had poor glycemic control,10 had their glycemic status unknown.

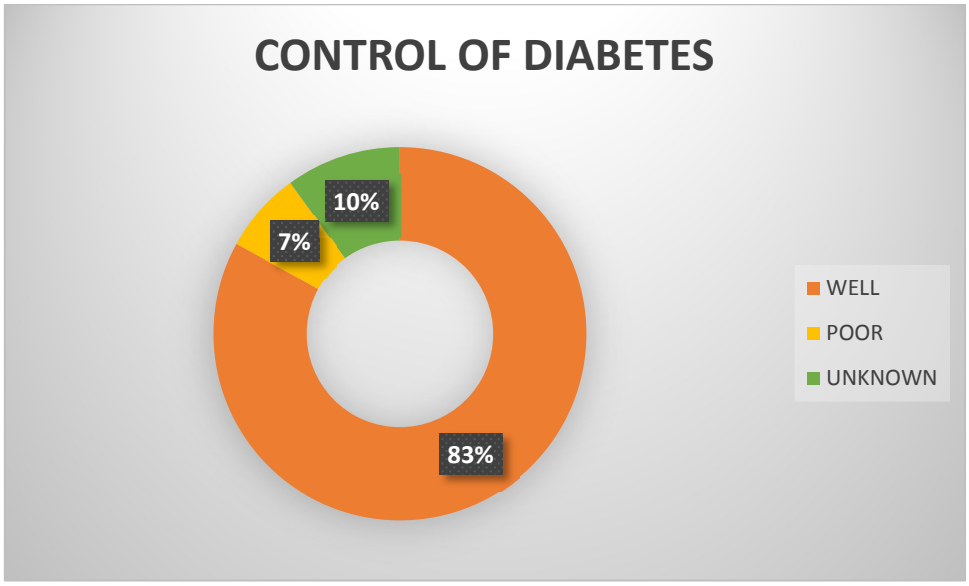

FIG 7: GLYCEMIC STATUS AMONG MOTHERS

TABLE 13: MATERNAL GLYCEMIC STATUS AND ECHO ABNORMALITIES AMONG THEIR IDM

GLYCAEMIC CONTROL	TOTAL NO. BABIES OF EXAMINED (N)	ECHO ABNORMALITY	
		PRESENT (N) (%)	ABSENT (N) (%)
WELL (HbA1c <6)	83	18 (21.6)	65 (78.3)
POOR (HbA1c >6)	7	7 (100)	0 (0)
UNKNOWN	10	3(30)	7(70)
KRUSKAL WALLIS TEST			
P VALUE - 0.001			

Mother's glycemic status had highly significant influence over the cardiovascular abnormalities in infants born to them.(< 0.001)

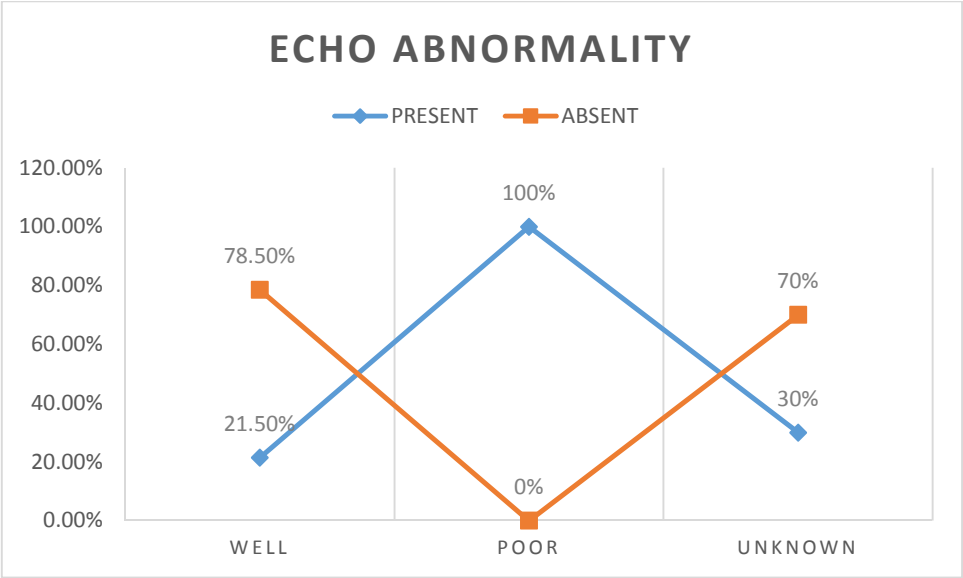

Fig 8:MATERNAL GLYCEMIC STATUS AND ECHO ABNORMALITIES AMONG THEIR IDM

This representation showed that 21% of babies born to well controlled mother and 100% of babies born to poorly controlled mother had ECHO abnormality .

TABLE 14: MATERNAL GLYCEMIC STATUS & TYPE OF ECHO NORMALITY

GLYCEMIC CONTROL	TOTAL NO OF ECHO ABNORMALITY (N)	TYPE OF ECHO ABNORMALITY	
		ACYANOTIC (N) (%)	CYANOTIC (N) (%)
WELL (HbA1c<6)	18	18 (100)	0 (0)
POOR (HbA1c>6)	7	2(28.57)	5 (71.42)
UNKNOWN	3	3(100)	0(0)
KRUSKAL WALLIS TEST			
P VALUE - 0.01			

Babies born to mothers with poor glycemic control had significantly more cyanotic heart disease than those born to mothers with good glycemic control.

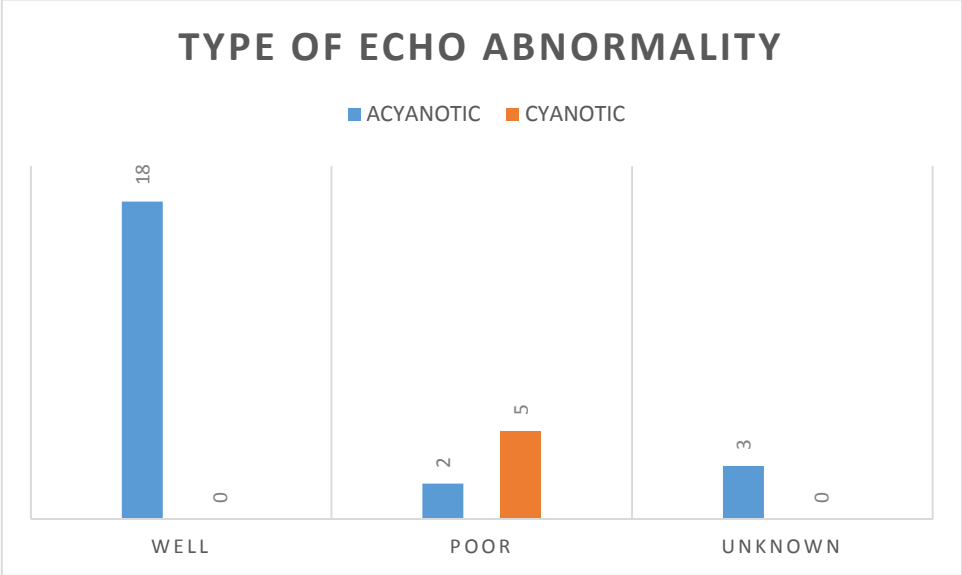

FIG 9: MATERNAL GLYCEMIC STATUS & TYPE OF ECHO ABNORMALITY

TABLE 15: AN – USG ABNORMALITIES AMONG IDM

AN- USG FINDING	NO OF PATIENTS (N)	PERCENTAGE (%)
PRESENT	6	6
ABSENT	94	94

Among 100 babies, 6 had abnormal findings in antenatal USG.

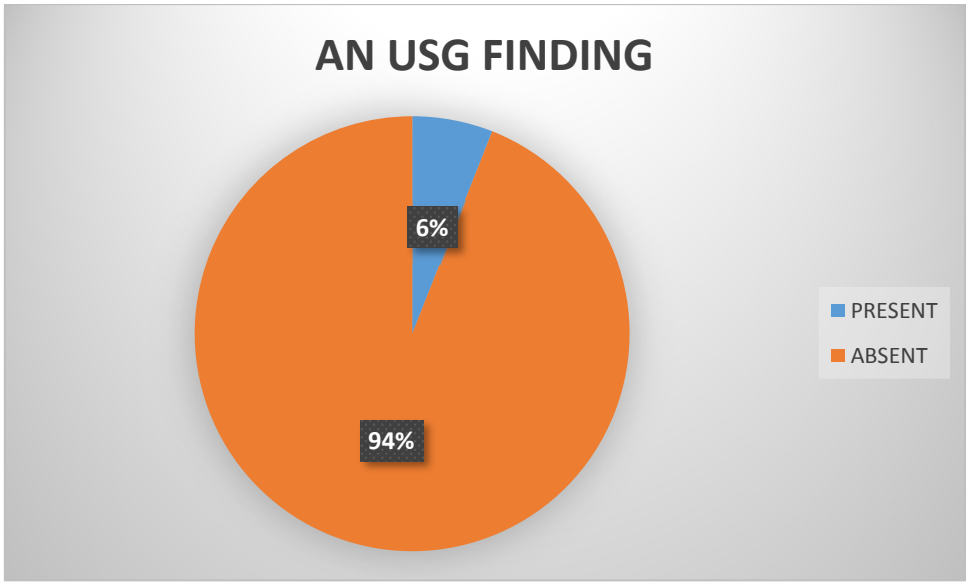

FIG 10: AN – USG ABNORMALITIES AMONG IDM

TABLE 16: AN – USG FINDINGS AND POSTNATAL ECHO ABNORMALITY IN IDM

AN- USG FINDINGS	TOTAL NO. BABIES EXAMINED (N)	ECHO ABNORMALITY	
		PRESENT (N) (%)	ABSENT (N) (%)
PRESENT	6	5 (83.33)	1 (16.6)
ABSENT	94	23 (24.46)	71 (75.53)

Out of 100IDM babies 6 had abnormal AN-USG findings, post natal ECHO shows abnormality in 5 IDM.

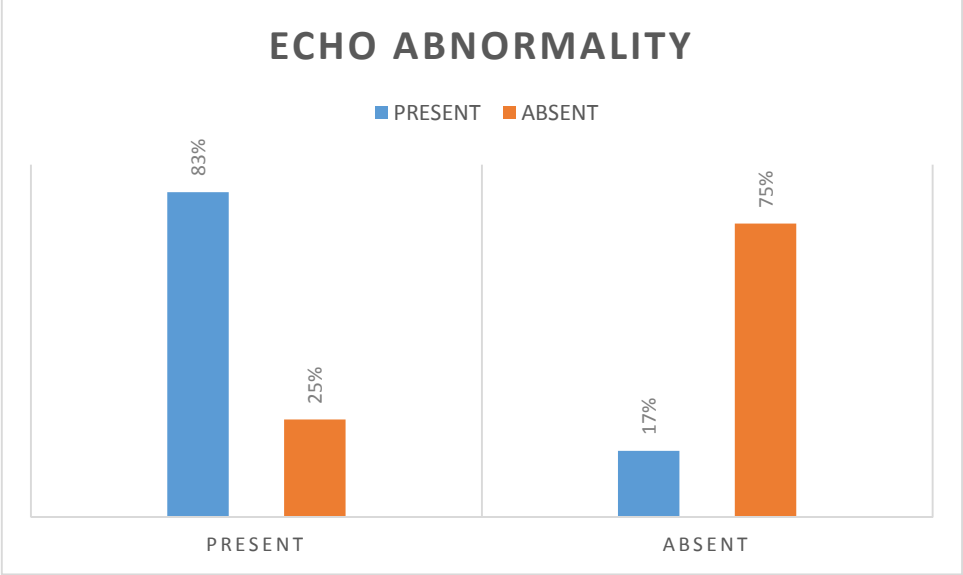

Fig 11: AN – USG FINDINGS AND POSTNATAL ECHO ABNORMALITY IN IDM

Among 6 babies with antenatal ultra-sonogram findings, 5 babies had ECHO abnormality signifying the importance of antenatal ultra-sonogram.

TABLE 17: GESTATIONAL AGE & ECHO ABNORMALITY

GESTATIONAL AGE	TOTAL NO. OF BABIES EXAMINED (N)	ECHO ABNORMALITY	
		PRESENT (N)	PERCENTAGE (%)
TERM	73	17	20
PRETERM	27	11	40

Among 100 babies 73 were term and 27 were preterm. Of 73 term babies 17 (20%) babies and 11 (40%) out of 27 preterm babies had ECHO abnormality.

TABLE 18: GENDER DISTRIBUTION AMONG IDM BABIES

GENDER	TOTAL NO. OF BABIES EXAMINED (N)	ECHO ABNORMALITY	
		PRESENT (N)	PERCENTAGE(%)
MALE	38	12	31
FEMALE	62	16	34

Among 100 babies, 38 were boys of which 12 had abnormal ECHO finding and 62 were girl babies out of which 16 babies had abnormality in ECHO. There was no difference in the incidence of ECHO abnormality with respect to sex.

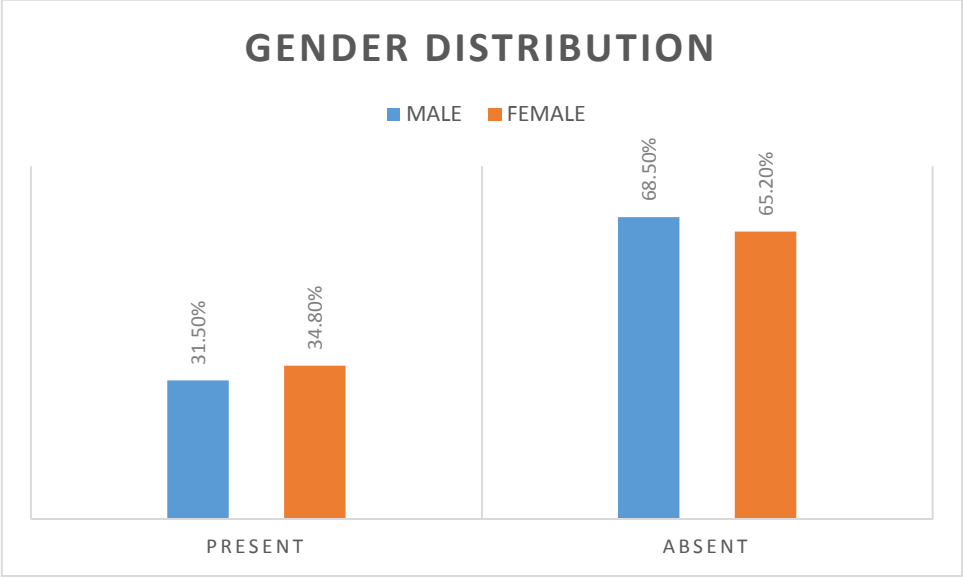

FIG 12: GENDER DISTRIBUTION AMONG IDM BABIES

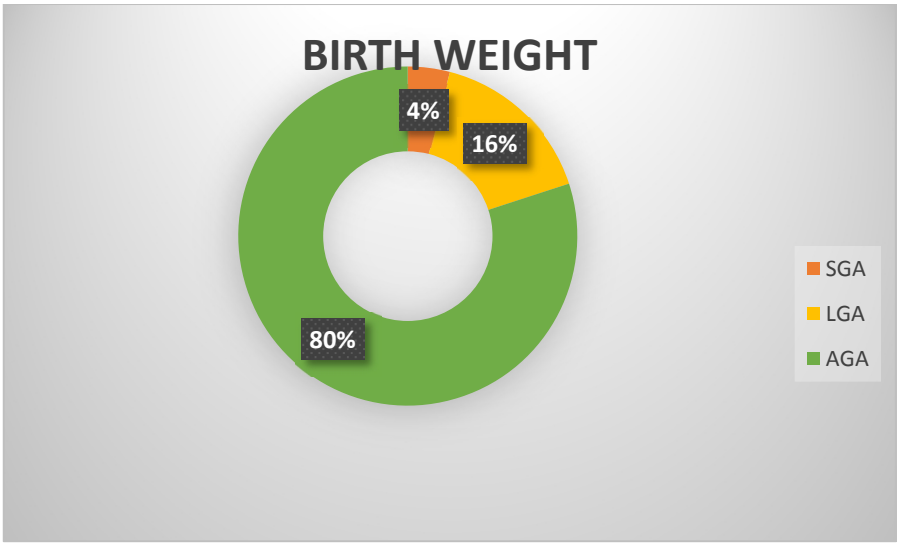

FIG 13: INTRAUTERINE GROWTH STATUS IN IDM

TABLE 19: INTRAUTERINE GROWTH STATUS & ECHO ABNORMALITY IN IDM

BIRTH WEIGHT	TOTAL NO. OF BABIES EXAMINED (N)	ECHO ABNORMALITY	
		PRESENT (N)	PERCENTAGE (%)
SGA	4	4	100
AGA	80	16	20
LGA	16	8	50

Among 100 babies studied 4 are SGA, 80 are AGA, 16 LGA. ECHO abnormality present in all4 SGA, 16 (20%) AGA, 8(50%) LGA.

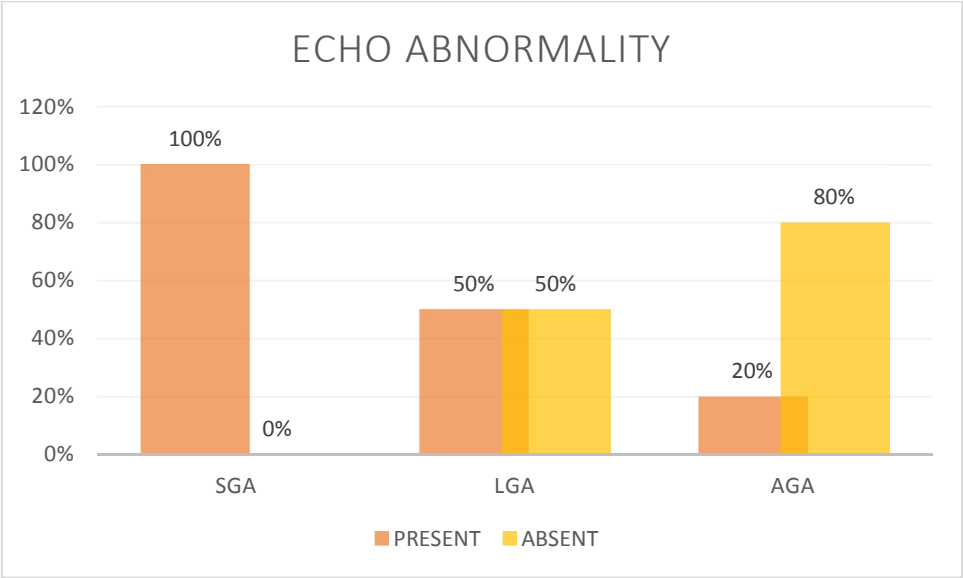

FIG 14: INTRAUTERINE GROWTH STATUS & ECHO ABNORMALITY IN IDM

Babies'birth weight had significant influence on ECHO abnormality. SGA babies showed 100% ECHO abnormality which reflected maternal diabetes complications .next comes LGA with 50% ECHO abnormality reflects maternal hyperglycemia & fetal hyperinsulinemia.

TABLE 20: PRESENTATION OF DISEASE

PRESENTATION	NO OF PATIENTS (N))	PERCENTAGE (%)
SYMPTOMATIC	15	15
ASYMPTOMATIC	85	85

Out of 100 babies admitted in NICU, 15 were with symptoms and 85 were asymptomatic.

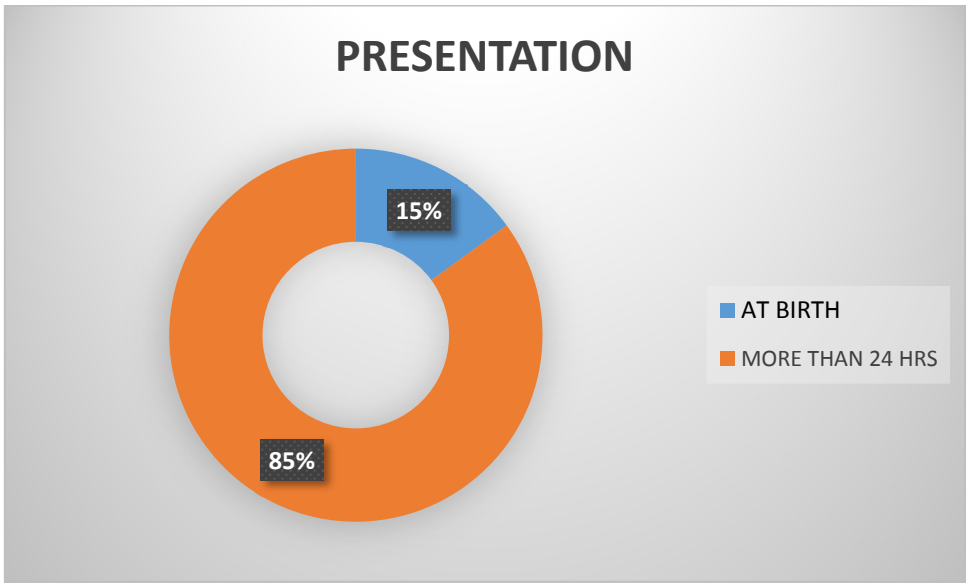

FIG 15: PRESENTATION OF DISEASE

TABLE 21: PRESENTATION OF DISEASE & EXTENT OF ECHO ABNORMALITY

PRESENTATION	TOTAL NO. OF BABIES EXAMINED (N)	ECHO ABNORMALITY	
		PRESENT (N) (%)	ABSENT (N)(%)
SYMPTOMATIC	15	13 (86.6)	2(13.33)
ASYMPTOMATIC	85	15 (17.64)	70 (82.35)

Among symptomatic babies, 13(86.6%) had ECHO abnormality and 15 (17.64%) asymptomatic babies had ECHO abnormality.

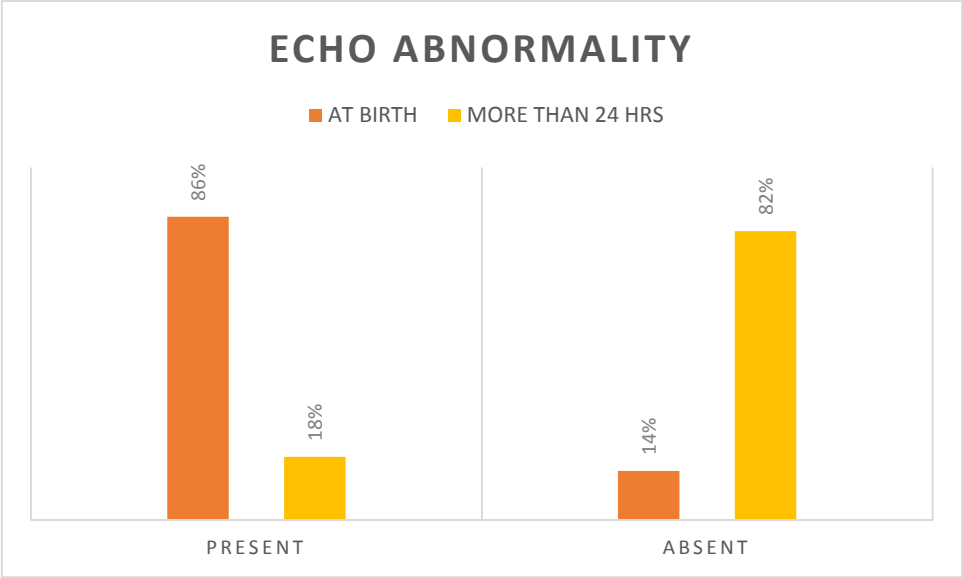

FIG 16: PRESENTATION OF DISEASE & EXTENT OF ECHO ABNORMALITY

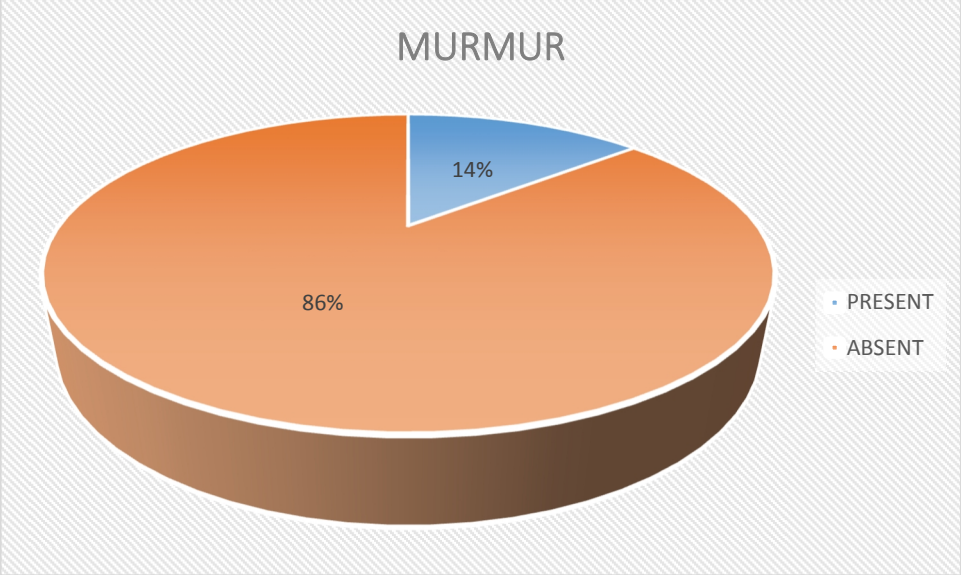

FIG 17: MURMUR & IDM

Out of 100 newborns taken in the study, 14 newborn had murmur, 86 newborn had no murmur

TABLE 22: MURMUR & ECHO ABNORMALITY IN IDM

MURMUR	TOTAL NO.OF BABIES EXAMINED(N)	ECHO ABNORMALITY	
		PRESENT(N)	ABSENT(N)
BABIES WITH MURMUR	14	11	3
BABIES WITHOUT MURMUR	86	17	69

11(78%) out of 14 newborn babies with murmur had ECHO abnormality. 17 (21%) out of 86 newborn babies without murmur had ECHO abnormality.

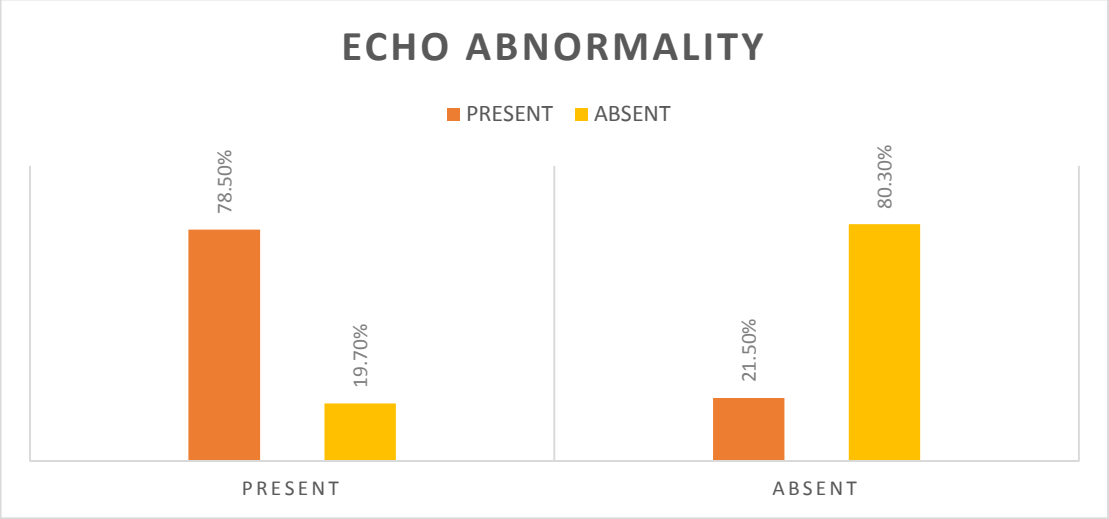

Fig 18: MURMUR & ECHO ABNORMALITY IN IDM

Echo abnormality was present in 21% babies presented without murmur, it signifying that every IDM babies should be screened for cardiovascular anomalies.

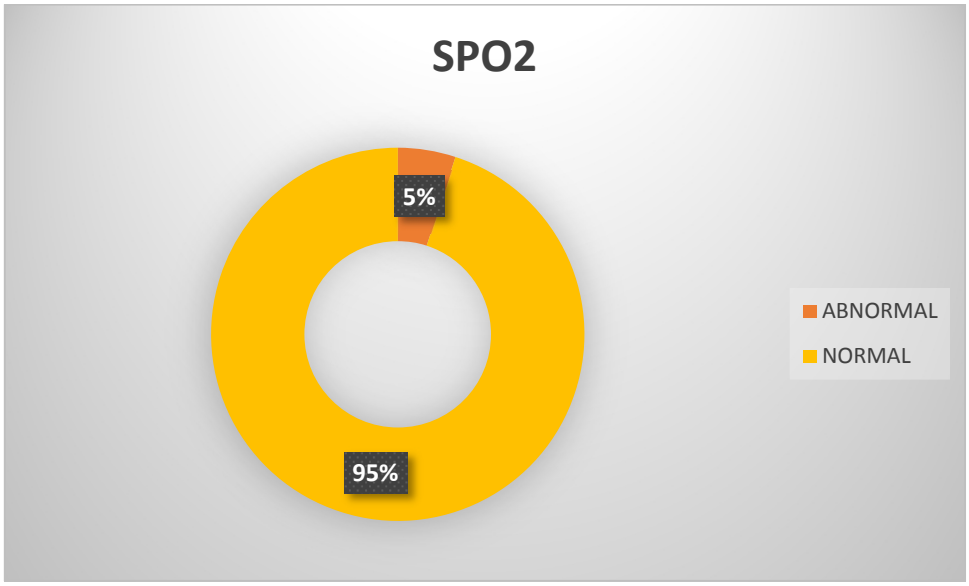

FIG 19: SPO2 LEVEL IN IDM

Out of 100 babies studied 5 babies presented with abnormal spo2 (spo2 <95%).

TABLE 23: SPO2 ABNORMALITY & ECHO ABNORMALITY IN IDM

SPO2	TOTAL NO. OF BABIES(N)	ECHO ABNORMALITY	
		PRESENT(N)(%)	ABSENT(N)(%)
SPO2 < 95%	5	5 (100)	0 (0)
SPO2 > 95%	95	23(24.21)	72 (75.7)

All newborns with low spo2 had abnormal echo findings.

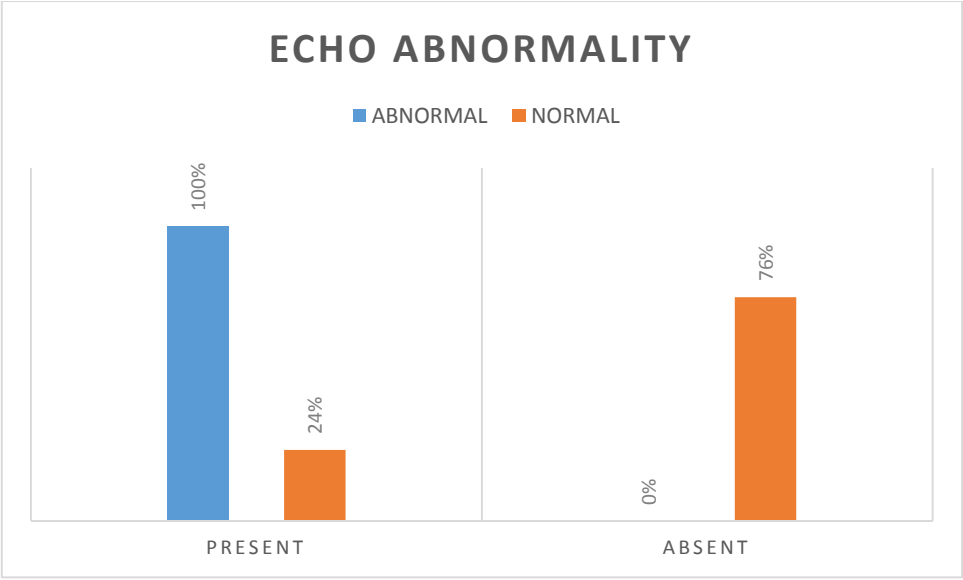

FIG 20: SPO2 ABNORMALITY & ECHO ABNORMALITY IN IDM

All (100%) newborns with abnormal spo_2 (<95%) had ECHO abnormality signifying the importance of spo_2 monitoring.

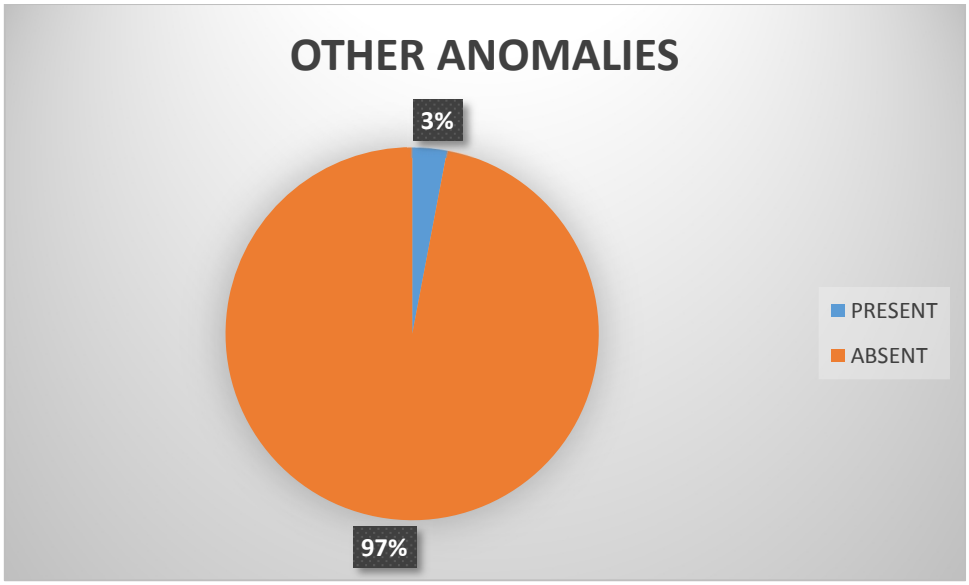

FIG 21: ASSOCIATED ANOMALIES IN IDM

Out of 100 babies 3 babies had other congenital external anomalies such as cleft lip, polydactyly.

TABLE 24: ASSOCIATED ANOMALIES & ECHO ABNORMALITY IN IDM

OTHER ANOMALIES	TOTAL NO. OF BABIES EXAMINED (N)	ECHO ABNORMALITY (N=28)	
		PRESENT (%)	ABSENT (N) (%)
PRESENT	3	3 (100)	0 (0)
ABSENT	97	25 (25.77)	72 (74.42)

Of 3 babies with other anomalies, all had ECHO abnormality.

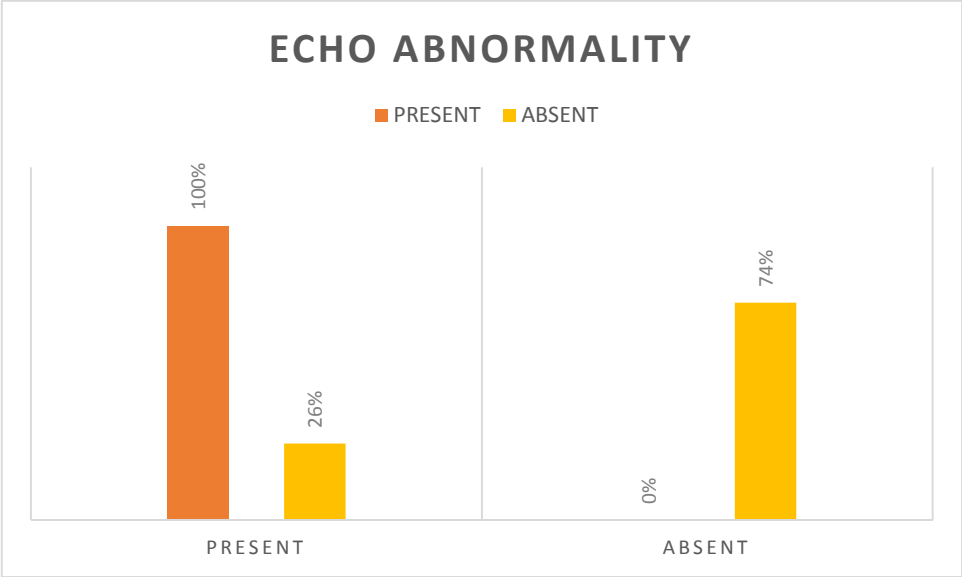

FIG 22: ASSOCIATED ANOMALIES & ECHO ABNORMALITY IN IDM

TABLE 25: CARDIOMEGALY IN IDM

CARDIOMEGALY	NO. OF PATIENTS (N)	PERCENTAGE (%)
PRESENT	9	9
ABSENT	91	91

Out of 100 babies studied 9 babies presented with cardiomegaly.(CXR findings.)

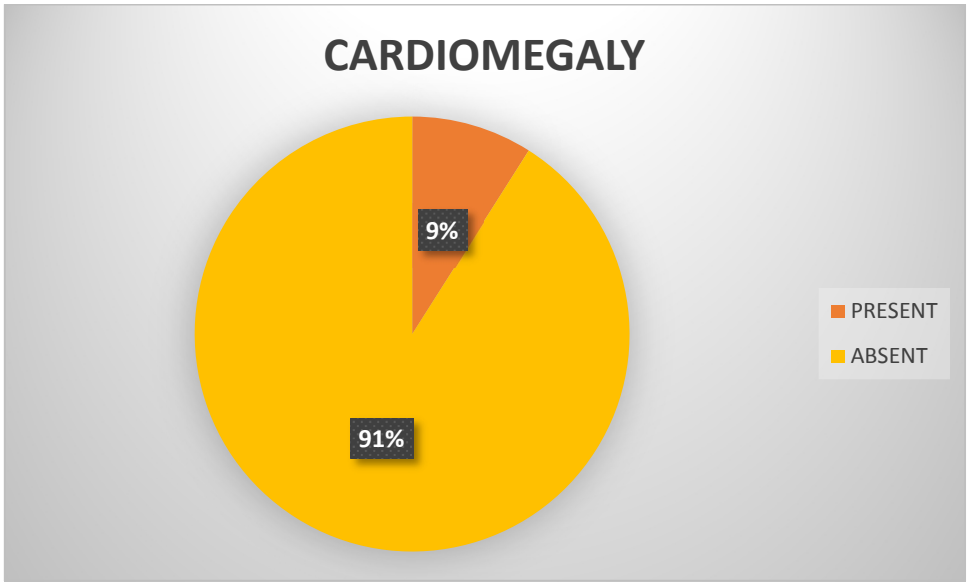

FIG 23: CARDIOMEGALY IN IDM

TABLE 26 : CARDIOMEGALY & ECHO ABNORMALITY IN IDM

CARDIOMEGALY	TOTAL NO. OF BABIES WITH CARDIOMEGALY (N)	ECHO ABNORMALITY	
		PRESENT (N) (%)	ABSENT (N) (%)
PRESENT	9	9 (100)	0 (0)
ABSENT	91	19(20.8)	72 (79.12)

All 9 babies with cardiomegaly all had ECHO abnormality.

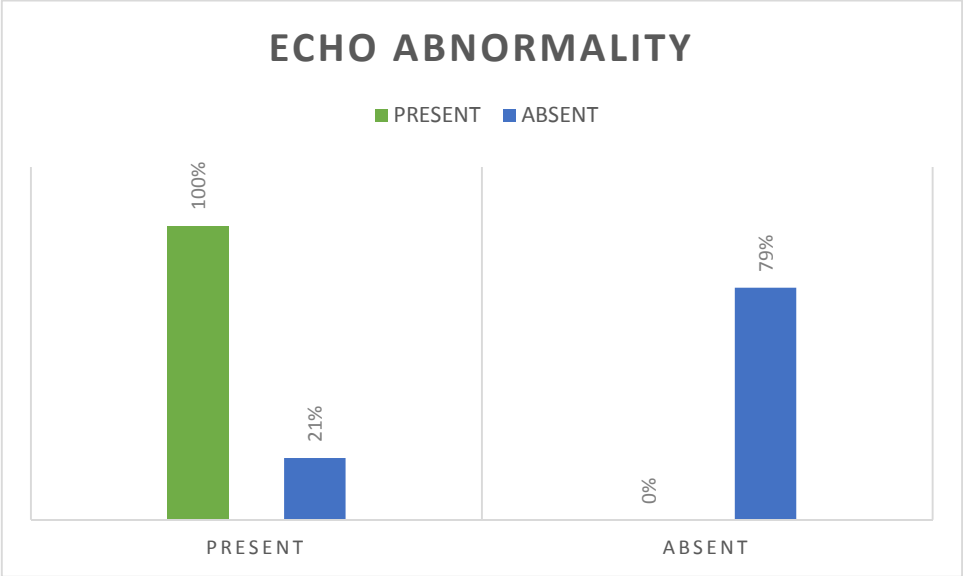

FIG 24: CARDIOMEGALY & ECHO ABNORMALITY IN IDM

Of the babies with cardiomegaly 100% had ECHO abnormality

TABLE 27: ECG ABNORMALITY IN IDM

ECG ABNORMALITY	NO OF PATIENTS (N)	PERCENTAGE (%)
PRESENT	8	8
ABSENT	92	92

Out of 100 IDM examined 8 babies had abnormal ECG findings.

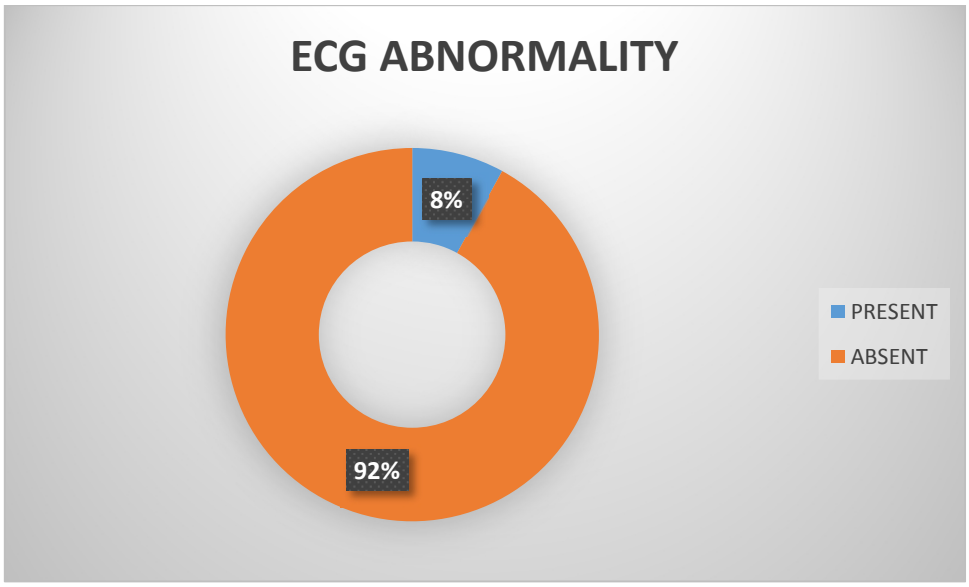

FIG 25: ECG ABNORMALITY IN IDM

TABLE 28: ECG ABNORMALITY & ECHO ABNORMALITY IN IDM

ECG ABNORMALITY	TOTAL NO. OF BABIES (N)	ECHO ABNORMALITY	
		PRESENT (N) (%)	ABSENT (N)(%)
PRESENT	8	8 (100)	0 (0)
ABSENT	92	20 (21.73)	72 (78.2)

ECG abnormality was present in 8 babies all of which (100%) were associated with abnormal ECHO findings

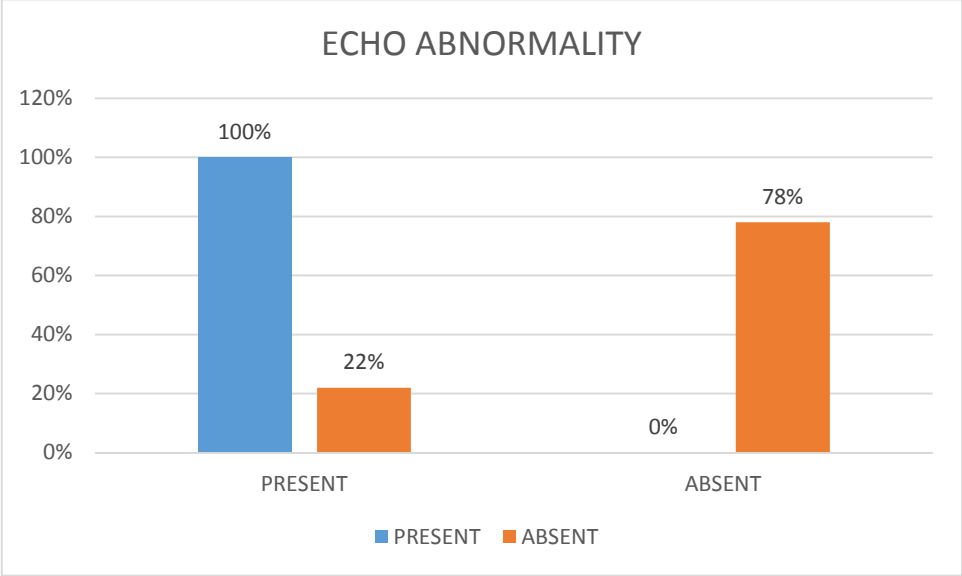

FIG 26: ECG ABNORMALITY & ECHO ABNORMALITY IN IDM

TABLE 29: DISTRIBUTION OF ECHOABNORMALITY IN IDM

ECHO ABNORMALITY	NO OF PATIENTS(N)	PERCENTAGE (%)
PRESENT	28	28
ABSENT	72	72

Out of 100 babies examined , 28 (28%)babies had ECHO abnormality.72 babies had no ECHO abnormality

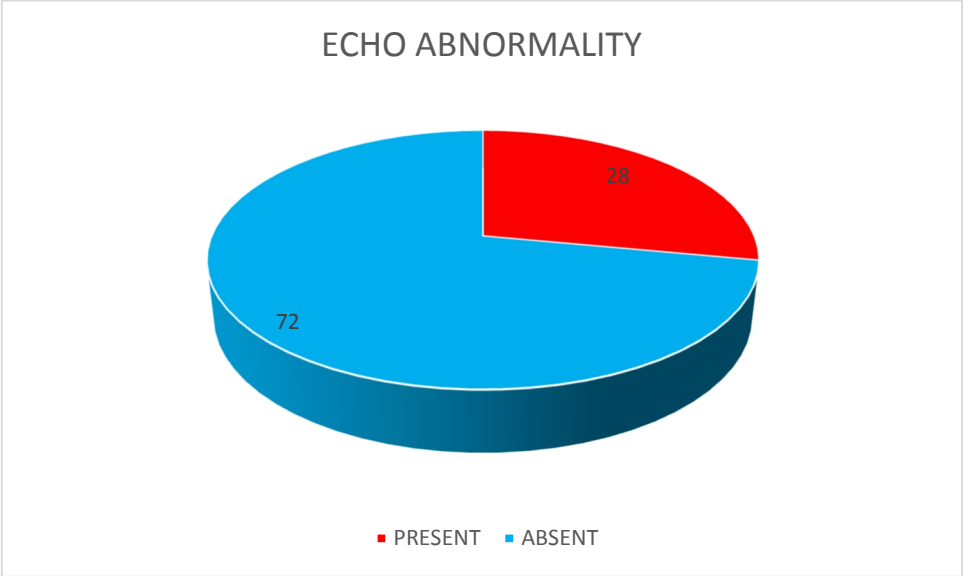

FIG 27: DISTRIBUTION OF ECHO ABNORMALITY IN IDM

TABLE 30: DISTRIBUTION OF ACYANOTIC & CYANOTIC HEART DISEASE IN IDM

TYPE OF CONGENITAL HEART DISEASE	NO.OF PATIENTS (N=28)	PERCENTAGE (%)
ACYANOTIC	23	82
CYANOTIC	5	18

Out of total 28 babies with ECHO abnormality, 23(82%) had acyanotic heart disease,and 5(18%) had cyanotic heart disease.

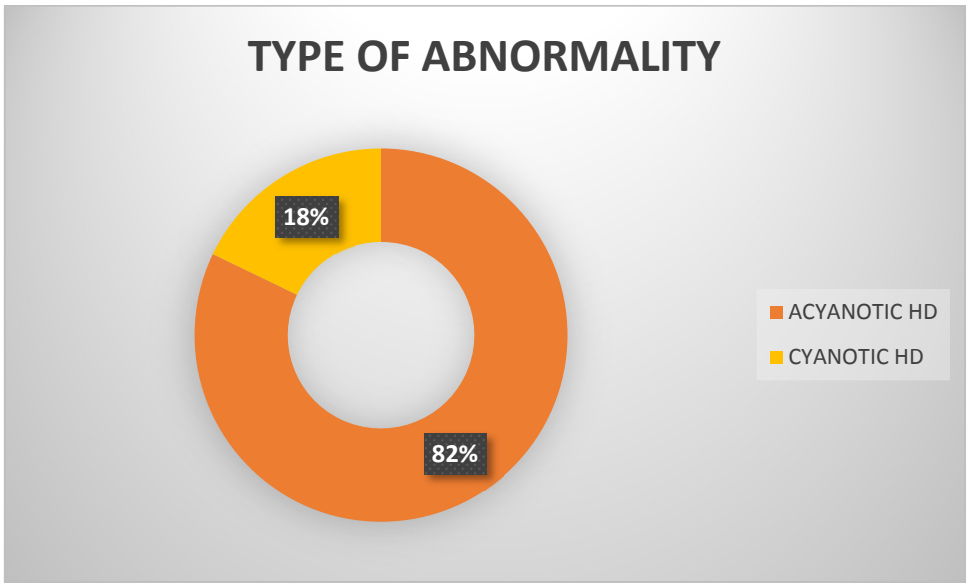

FIG 28: DISTRIBUTION OF ACYANOTIC& CYANOTIC HEART DISEASE IN IDM

TABLE 31: DISTRIBUTION OF ACYANOTIC HEART DISEASE IN IDM

ACYANOTIC HEART DISEASE	NO. OF PATIENTS (N=23)	PERCENTAGE (%)
ATRIAL SEPTAL DEFECT	6	24
ASYMMETRICAL SEPTAL HYPERTROPHY	8	32
OSTIUM PRIMUM ASD	1	4
PATENT DUCTUS ARTERIOSUS	3	12
PATENT FORAMEN OVALE	4	16
VENTRICULAR SEPTAL DEFECT	4	16

Of acyanotic heart diseases, septal hypertrophy had more incidence.

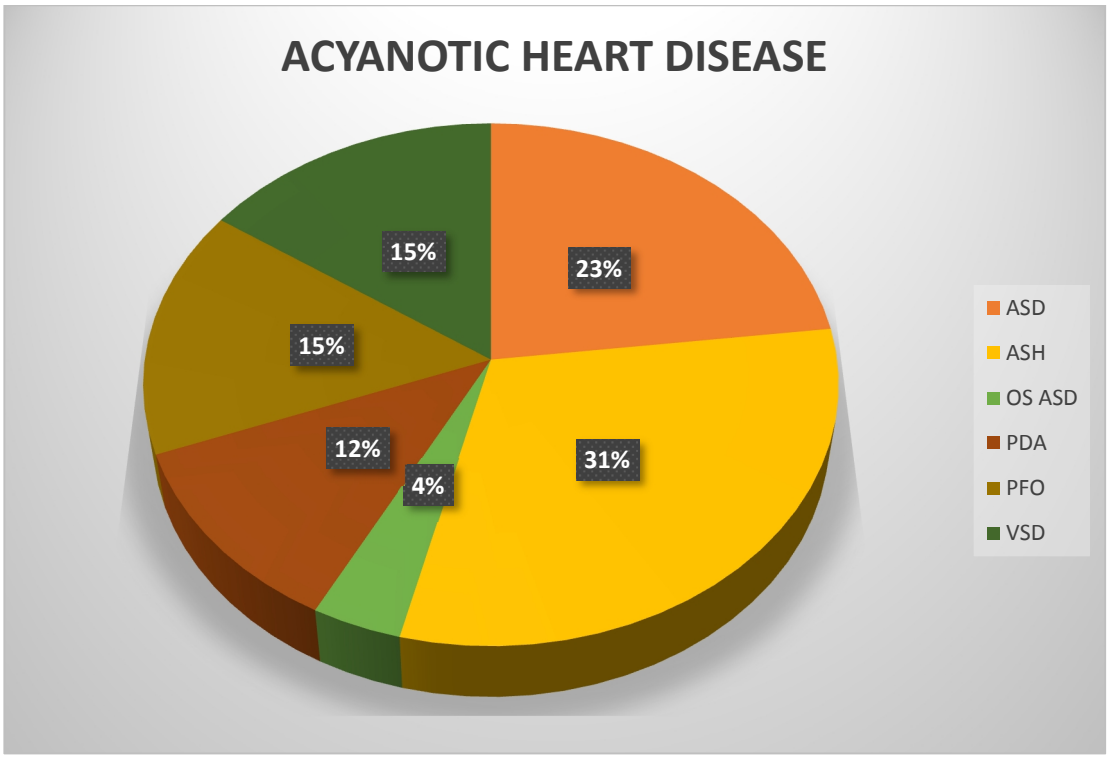

Fig 29 : DISTRIBUTION OF ACYANOTIC HEART DISEASE in IDM

In acyanotic congenital heart disease , asymmetrical septal hypertrophy was more common than others. Followed by ASD, PFO, VSD, PDA..

TABLE 32 : DISTRIBUTION OF CYANOTIC HEART DISEASE IN IDM

CYANOTIC HEART DISEASE	NO OF PATIENTS(N=5)	PERCENTAGE(%)
HYPOPLASTIC LEFT HEART SYNDROME	1	20
TRICUSPID ATRESIA	1	20
TRANSPOSITION OF GREAT VESSELS	2	40
TRUNCUS ARTERIOSUS	1	20

Of 18% cyanotic heart disease TGA had 40% incidence

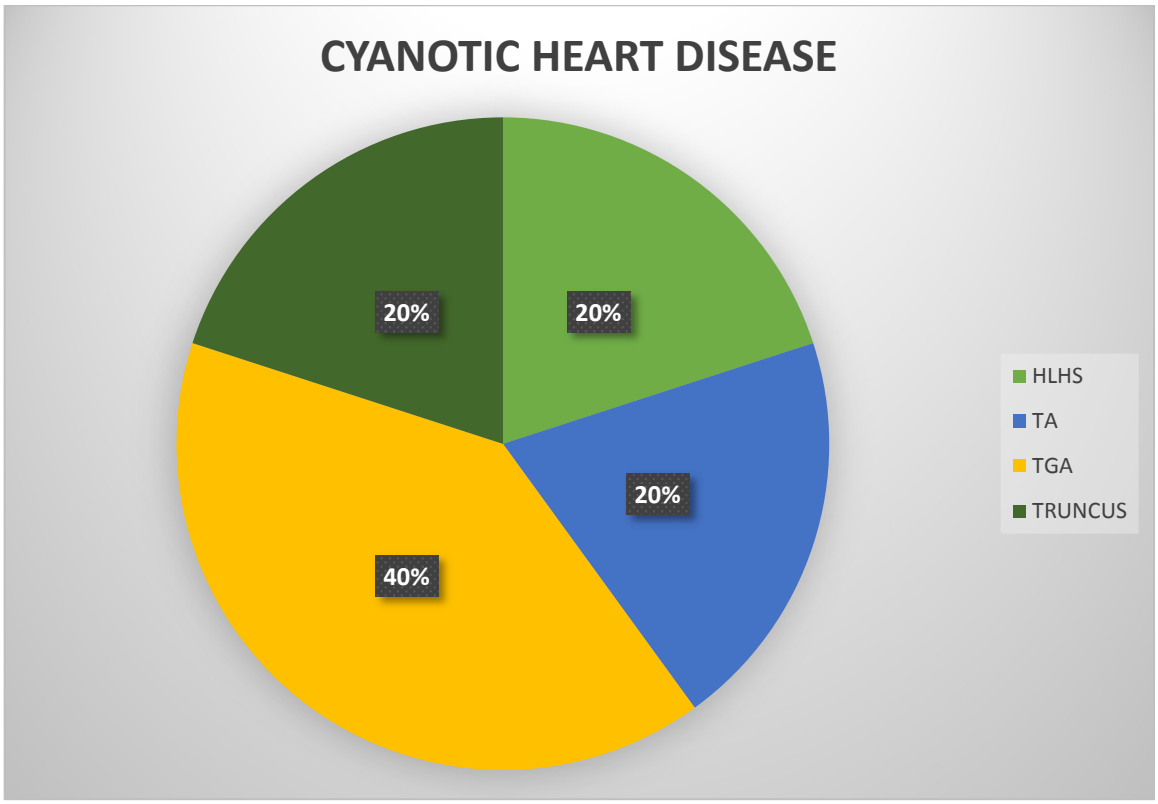

FIG 30: DISTRIBUTION OF CYANOTIC HEART DISEASE IN IDM

Out of 5 cyanotic congenital heart disease babies , TGA 2(40%) was most common. Followed by others.

TABLE 33: RELATION BETWEEN TYPE OF MATERNAL DIABETES AND HEART LESION

TYPE OF CARDIAC LESION	PREGESTATIONAL (N)	GDM (N)	P VALUE
ATRIAL SEPTAL DEFECT	3	4	0.662
ASYMMETRICAL SEPTAL HYPERTROPHY	7	1	0.01
PATENT FORAMEN OVALE	0	4	0.05
VENTRICULAR SEPTAL DEFECT	2	2	0.823
PATENT DUCTUS ARTERIOSUS	1	2	0.791
HYPOPLASTIC LEFT HEART SYNDROME	1	0	0.887
TRANSPOSITION OF GREAT VESSELS	2	0	0.596
TRICUSPID ATRESIA	1	0	0.887
TRUNCUS ARTERIOSUS	1	0	0.887

On studying the relation between the type of heart lesion and maternal diabetes, septal hypertrophy was more present in pregestational diabetic mother[1] (7 out of 17).

TABLE 34: RELATION BETWEEN MATERNAL TREATMENT REGIMEN AND HEART LESION

TYPE OF CARDIAC LESION	MEAL PLAN (N)	OHA (N)	INSULIN (N)	P VALUE
ATRIAL SEPTAL DEFECT	4	1	2	0.729
ASYMMETRICAL SEPTAL HYPERTROPHY	0	2	6	0.92
PATENT FORAMEN OVALE	3	1	0	0.931
VENTRICULAR SEPTAL DEFECT	0	2	2	0.887
PATENT DUCTUS ARTERIOSUS	2	1	0	0.512
HYPOPLASTIC LEFT HEART SYNDROME	0	0	**1**	**0.05**
TRANSPOSITION OF GREAT VESSELS	0	0	2	0.173
TRICUSPID ATRESIA	0	0	**1**	**0.05**
TRUNCUS ARTERIOSUS	0	0	**1**	**0.05**

On comparing the relationship between maternal diabetes and treatment plan , babies whose mother were on insulin had more number of echo abnormality – cyanotic heart disease HLHS (1), TA(1), TRUNCUS(1) TGA(2). More asymmetrical septal hypertrophy ASD(2), VSD(2).

TABLE 35: RELATION BETWEEN MATERNAL GLYCEMIC CONTROL AND HEART LESION

TYPE OF CARDIAC LESION	WELL CONTROL (N)	POOR CONTROL (N)	NOT KNOWN (N)	P VALUE
ATRIAL SEPTAL DEFECT	4	1	1	0.862
ASYMMETRICAL SEPTAL HYPERTROPHY	7	1	0	0.537
PATENT FORAMEN OVALE	3	0	1	0.741
VENTRICULAR SEPTAL DEFECT	3	1	0	0.596
PATENT DUCTUS ARTERIOSUS	2	0	1	0.891
HYPOPLASTIC LEFT HEART SYNDROME	0	1	0	0.718
TRANSPOSITION OF GREAT VESSELS	0	2	0	0.152
TRICUSPID ATRESIA	0	1	0	0.718
TRUNCUS	0	1	0	0.718

Based on diabetic control, babies born to mothers with poor glycemic control had significant ECHO abnormality with more incidence of cyanotic heart disease. [TGA (2) TA (1) HLHS (1), truncus(1)] than acyanotic heart disease {ASH(1), ASD (1)}.

8.DISCUSSION

In our study, 100 infants of diabetic mother were subjected to echocardiogram, of this 28 (28%) babies had ECHO abnormality. Previous similar study done on cardiovascular malformations in IDM reported by meyer et al, showed incidence of 3.2 to 6.9[36]and Avisa tabib et al showed incidence of 8.8%[35]

Of the 100 IDM babies studied 17% mother was found to have pre-gestational diabetes and 83% was found to have gestational diabetes. In our study ECHO showed 94% of babies born to pre- gestational diabetes had abnormality. Which is significant when compared to14%babies born to gestational diabetes with significant P Value of 0.03.

Out of 28 babies who showed ECHO abnormality, 16 babies (57%) were born to pre gestational diabetic mother and 12 babies (42%) were born to gestational diabetic mother.

TABLE 36: Comparison between Type of Maternal Diabetes and Malformation In Related Studies

TYPE OF DIABETES	Avisa tabib et al[35]	Present study
Pre gestational	68	17
Gestational	102	83
MalformationIn pregestational	7	16
MalformationIn gestational	8	12

Of the 16 babies born to pre gestational diabetic mother, 11 babies (68.7) had ACHD and 5 babies (31.3) had CCHD.

Out of 100 IDM babies included in this study , 66% mothers were on meal plan , 21% on OHA, 13% on insulin.

TABLE 37 : Comparison Between Maternal Treatment Regimen And Malformation In Related Studies

TREATMENT REGIMEN	Avisa tabib et al[35]	Present study
Meal plan	53	66
Insulin	117	13
Malformations in meal plan group	4	8
Malformations in insulin group	11	13

Previous study done by Avisa tabib et al[35], showed mother who was on insulin had increased risk of major cardiovascular malformation which is comparable to our study. (P value 0.03)

In our study taken 100 IDM babies, 83% mothers had well controlled glycemic status, 7% had poorly controlled, 10% had unknown glycemic status. Of which echo abnormality was found to be more significant in babies born to mother with poor controlled diabetes with preponderance of cyanotic heart disease. It showed that mother's glycemic status had significant influence on CVS malformations compatible withthe studiesdone by khan et al[38] and mohan mekwana et al[43]. Babies of poorly controlled glycemic status mother had poorer cardiac function.[39,40]

Of the 100 IDM study, 6 babies (6%) had AN- USG abnormality and 94 babies normal AN – USG. On further evaluation using postnatal echo 5 out of 6 AN – USG positivity cases showed abnormality, which is similar to the previous studies done by avisa tabib et al[35] and meyer et al,[36]. Where AN- USG screening with fetal ECHO had sensitivity of 90% and specificity of 99[35,36]

Based on birth weight , 50% of LGA babies and all SGA babies had significant cardiovascular abnormality in ECHO.

In our study with 100 IDM babies, 14 babies presented with murmur, 5 babies with low spo2 and 3 babies with external congenital anomalies like polydactyl and cleft palate.

Of the 14 babies with ,11 babies (78%) had ECHO abnormality and all babies with low spo2 and external congenital anomalies had ECHO abnormality.

On investigation basis cardiomegaly was present in 9% babies and all had ECHO abnormality more of septal hypertrophy.

Abnormal ECG was found in 8 babies out of 100 examined and all had ECHO findings consistent with more of septal hypertrophy and ASD , TGA.

In our study, of 100 IDM babies , 28 had echo abnormality, out of this 23{82%} had acyanotic & 5 [18%] had cyanotic heart disease. In acyanotic heart diseases septal hypertrophy is more common than others which was similar to that of previous studiesdone by avisa tabib et al and zielinsky P et al .[35,42]

Parvin Akbariasbagh[44] et al conducted a study on cardiovascular malformations in infant of diabetic mother which showed significant cardiovascular in infant of diabetic mother with p value of 0.018 which is comparable to our study.(p= 0.01)

In cyanotic heart disease, increased incidence was seen with TGA(2)(40%).HLHS 1, Truncus arteriosus 1, tricuspid atresia 1. These types were more prevalent in diabetic pregnancy.[10]

On studying the relation between type of heart lesion and maternal diabetes, septal hypertrophy was more in pregestational diabetic mother[35] (7 out of 17) concordance to previous study done by avisa tabib et al, followed by ASD (3), VSD(2), PDA(1).

Babies born to pre gestational diabetic mother showed more cyanotic heart disease. {TGA(2), TA(1) Truncus(1) HLHS(1).} and babies born to GDM mothers had more acyanotic heart disease.

On studying the relationship between maternal diabetes and treatment plan , babies whose mother were on insulin had more number of echo abnormality. Of which 5 babies had cyanotic heart diseaseTGA (2) (40%) HLHS (1) (20%), TA (1) (20%), TRUNCUS ARTERIOSUS (1) (20%).

Babies whose mother was on meal plan also had cardiac abnormality but of acyanotic heart disease with less significant ASD(4) PFO(3), PDA (2). Mother with OHA treatment had echo abnormality of ASD(1), VSD(2), PFO(1),PDA(1), ASH (2)

Based on diabetic control, babies born to mothers with poor glycemic control, had significant ECHO abnormality with more incidence of

cyanotic heart disease. TGA(2) TA(1) HLHS(1), truncus(1). Acyanotic heart disease ASH(1), ASD (1).

Most of the babies born to well controlled mother had more of acyanotic heart disease , with increased ASH(6) PFO(3), VSD(3), PDA(2), ASD (5).

9..LIMITATIONS.

1. HbA1c level could not be measured in 10 of diabetic mothers studied.

2. In our study HbA1c level was measured only once during the pregnancy period randomly. So its effect on organogenesis specifically could not be interpreted .

.

10. CONCLUSION

1. In present study, acyanotic heart diseases were more common than cyanotic heart disease in IDM.
2. Babies born to mother with pre gestational diabetic mellitus had complex cyanotic heart disease than GDM mothers.
3. Babies whose mother had poor glycemic control had more incidence of congenital heart disease.
4. Among acyanotic heart disease asymmetrical septal hypertrophy was more common than other types.
5. Among cyanotic heart disease, Transposition of great arteries was the most common cardiac anomaly in IDM babies.

BIBLIOGRAPHY

1. AngC, HoweD, Lumsden M in james DK, steer Pj, Weiner CP and Gonikhigh risk pregnancy management options ,third edition. Philadelpia.saunders.2005:986 – 1004
2. perinatal outcome of infants born to diabetic mother in a developing country.niranjan Thomas, anniejothirmayichinta, santhanam Sridhar, manishkumar, kurienanilkuruvila and atanukumarjana. Research paper
3. Ferrara A increasing prevalence of gestational diabetes mellitus:a public health perspective diabetes care.2007:302;S141
4. seshiah V , Balaji V, BalajiMS,paneerselvam, arthi, tamilarasi et al prevalence of gestational mellitus in south india. A community based study.J A assoc physicians india 2008,56:329
5. Ramachandran A, Snehalatha C, Shymala p, vijayV ,vishwanathan M prevalence of diabetes in southindia . diabetes Res clinpract 1994;25;71-4
6. Nasrat HA , Salleh M, ArdawiM,Ghafouri H. Outcome of pregnancy in diabetic mothers. IntJGynecol Obstet1993:43:29 – 34
7. Dhulkotia JS, Ola B, Fraser R, Farrel T. Oral hypoglycaemicagentsvs insulin in management of gestational diabetes. A systematic review and meta analysis.Am J Obstet Gynecol. 2010;203:457e1-9

8. Ullmo S, Vial Y, Di bernardo S, Roth Kleiner M, Mivaelaz Y, Sekarski N et al ventricular hypertrophy in the offspring of diabetic mother, a retrospective study.eur heartJ2007;228
9. LiwokiLA,verheijen pm, copel,keilmancs, wassinksvisser et al, congenital heart disease in pregnancies complicated by diabetes. An international collaboration
10. C wren G birrell ,G hawthorne et al conducted a study on cardiovascular malformations in infant of diabetic mothers heart 2003;89
11. vela-hurta m, Aguilera lopez A, Alarcon santos S, Amador N, Aldana C, cardiopulmonary adaptation in large for gestational age infants of diabetic mother and non diabeticmothe
12. seppanenmarko p, ojaneraouti, kaapa , keropentti, delayed postnatal adaptation of pulmonary hemodynamics in infant of diabetic mothers J paediatrics.1997
13. jensenDm, Damm P, moelsted –pedersen l, ovesen p, westergaardjg,etal outcomes in type 1 diabetic pregnancies a nationwide, population study diabetes care.
14. Oberhofer[1], hogrl j stozfkohneflang D conducted a study in cardiac and extracardiac complications ininfant of diabetic mother and their relations to parameters of carbohydrate metabolism
15. pildes RS inants of diabetic mother

16. versani BR, gilbert barness, Giuliani lR, peres LC, pinaneto caudal dysplasia sequence. Severe phenotype presenting in infants of diabetic mother
17. michel, B (2011) diabetes mellitus in k.geen&J horn. Medical surgical nursing st.louis,Missouri
18. KHAN, R.,Ali, K & khan maternal and fetal outcome of gestational diabetes. Gomal journal of medical sciences.
19. guven ,S., Matfin G, &kuenzi . diabetes mellitus and metabolic syndrome. In H.surrena&H.Kotgut pathophysiology concepts of altered health states.
20. Aviva lee- parritz and john P.Cloherty. manual of neonatal care , 7th edition, diabetes mellitus, chapter 2,11-21.
21. Gargeepandit and kaizadR.Damania , arias high risk pregnancy and delivery 4th edition, diabetes in pregnancy. Chapter 15 ;256-257.
22. Lakshmi seshadri , essentials of obstetrics, pregestational and gestational diabetes.chapter 48;725-730.
23. Zhu Liping, Nakabayashi masao, statistical analysis of perinatal outcome in pregnancies complicated with diabetes mellitus
24. Dunne FP, Brydon PA, Proffit M, smith T, fetal and neonatal outcomes in indo-Asian compared to Caucasian women with diabetes in pregnancy.
25. Oakley , G.P. failing to prevent birth defects caused by maternal diabetes mellitus. American journal of obstetrics &gynaecology.

26. Correa, A., Gilbosa, S.M., Besser, L. M., Botto, L.D., Moore, diabetes mellitus and birth defects. American Journal of Obstetrics and Gynecology.
27. Tabib, A., Shirzad, N., S., Mohammadi,S.,Qorbani,M.,Haghpanah, V., cardiac malformations in foetuses of gestational and pregestational diabetic mothers. Iranian Journal of Paediatrics.
28. Lale,S., Yu,S., &Ahmed,A.complex congenital heart defects in association with maternal diabetes and partial deletion of the gene.Fetal and paediatric pathology
29. Dunne, F., Brydon,P.,Smith, K.,&Gee,H. Pregnancy in women with type 2 diabetes:12 years outcome data 1990to 2002. A Journal of British Diabetic Association.
30. Abdelgadir M, Elbagir M, Eltom M, Berne Factors affecting perinatal morbidity and mortality in pregnancies complicated by diabetes mellitus in sudan.
31. Jaffe.R. identification of fetal growth abnormalities in diabetes mellitus.
32. Negrato CA, Mattar R, Gomes MB. Adverse pregnancy outcome. Diabetology and metabolic syndromes.
33. Chung CS,Myrianthopoulos. Factors affecting risks of congenital malformations. Effect of maternal diabetes on congenital malformations.

34. Iqbal R, rafique G, Baddurudin S increased body fat percentage and physical inactivity are independent predictors of GDM in south asian women

35.cardiac malformations in fetuses of gestational and pregestational diabetic mothers. Avisatabib, nooshinshirzad, sarasheikhbahai, saramohammadi, mostafQorbani, vahidhaghpanah, arzanehabbasishirinhasaniranjibar

36.meyer-wittkopfm, simpsonJM,sharland GK. Incidence of congenital heart disease in fetuses of diabetic mother ultrasound obstet gynecol.

37.Becerra JE, Khoury MJ, Cordero JF Diabetes mellitus during pregnancy and risk of speciic birthdefects:population based study.

38.Khan SE, Hull RL, Utzschneider KM. Mechanism linking obesity to insulin resistance and type 2 diabetes.

39.bruyerehj, jrkargas SA. The causes and underlying mechanism in congenital cardiac malformations.a critical review.

40.cedegrenMIselbing AJ, kallen BA risk factor for cardiac malformations a study based on prospectively collected data.

41.echocardiographic abnormalities in infants of diabetic mother MaceS, Hirschield SS, Riggs, fanaroff

42.zielinsky P, piccoli A, diseases o myocardium, endocardium and pericardium during fetal life. Yagelsilverman fetal cardiology

43.Mohan Makwana*, Jagdish Chand Dabi, Jai PrakashSoni, RatanLalBhati, Harish Kumar Mourya, YogeshGarg conducted a study the clinical evaluvation of the infant of diabetic mother born in tertiary hospital.

44.Parvin Akbariasbagh1, Mamak Shariat2, Naseredin Akbariasbagh3, and BitaEbrahim cardiovascular malformations in infant of diabetic mother retrospective case control study.

CASE PROFORMA

1. NAME :

2. AGE ON ADMISSION :

3. GENDER : FCH /MCH

4. MATERNAL DIABETES TYPE : PREGESTATIONAL / GESTATIONAL

5. MATERNAL TREATMENT REGIMEN: MEAL PLAN/ OHA/INSULIN

6. MATERNAL GLYCEMIC CONTROL : WELL CONTROLLED / POORLY CONTROLLED/ UNKNOWN

7. AN-USG : NORMAL / ABNORMAL

8. MATURITY : TERM/ PRETERM

9. BIRTH WEIGHT : SGA/AGA/LGA

10. PRESENTATION : SYMPTOMATIC / ASYMPTOMATIC

11. MURMUR : PRESENT / ABSENT

12. SPO2 ABNORMALITY : PRESENT / ABSENT

13. OTHER EXTERNAL ANOMALIES : PRESENT / ABSENT

14. CARDIOMEGALY : PRESENT / ABSENT

15. ECG ABNORMALITY : PRESENT / ABSENT

16. ECHO ABNORMALITY : PRESENT / ABSENT

17. TYPE OF ECHO ABNORMALITY : ACHD/CCHD

18. ACHD : ASD/VSD/ PDA/ PFO ASYMMETRICAL SEPTAL HYPERTROPHY

19. CCHD : TGA/ TA/ HLHS/ TRUNCUS ATERIOSUS

20. OUTCOME : ALIVE / DEATH

S.NO	NAME	SEX	AGE PRESENTATION		MATERNAL TYPE OF DIABETES		MATERNAL TREATMENT REGIMEN			MATERNAL DIABETIC CONTROL			ANUSG	TERM / PRE TERM	SGA/ AGA/ LGA	CYANOSIS	MURMUR	SPO2 ABNORMALITY	OTHER ANOMALIES	CARDIOMEGALY	ECG ABNORMALITY	ECHO FINDINGS	ECHO ABNORMALITIY	CHD	ACHD	OUTCOME
			at Birth	>24 hrs	PREGESTATIONAL	GESTATIONAL	MEAL PLAN	ORAL HYPOGLYCEMICS	INSULIN	WELL CONTROLLED	POORLY CONTROLLED	STATUS UNKNOWN														
1	B/o Anitha	Fch	2		G		M			WC			A	T	AGA	A	A	A	A	A	A	A				a
2	B/o Mangai	Fch	2		G		M			WC			A	T	AGA	A	A	A	A	A	A	A				a
3	B/o Meena	Mch	2		G		M			WC			A	LPT	AGA	A	A	A	A	A	A	A				a
4	B/o Madathy	Mch	2		G		M			WC			A	T	AGA	A	A	A	A	A	A	A				a
5	B/o Umarani	Mch	1		G		H			WC			A	T	LGA	A	A	A	A	A	P	P	ACHD		ASH	a
6	B/o Muthulakshmi	Fch	2		G		M			WC			A	T	AGA	A	A	A	A	A	A	A				a
7	B/o Mahadevi	Fch	2		G		M			WC			A	T	AGA	A	A	A	A	A	A	A				a
8	B/o Prema	Fch	2		G		M			WC			A	LPT	AGA	A	A	A	A	A	A	P	ACHD		PDA	a
9	B/o Stella	Mch	2		G		H			WC			A	T	AGA	A	P	A	A	A	A	A				a
10	B/o Sudha malathi	Mch	1		G		M			WC			A	LPT	AGA	A	A	A	A	A	A	P	ACHD		PFO	a
11	B/o Selvi	Mch	2		G		M			WC			A	T	AGA	A	A	A	A	A	A	A				a
12	B/o Veeralakshmi	Mch	2		G		M			WC			A	T	AGA	A	A	A	A	A	A	A				a
13	B/o Muthuselvi	Mch	1		PG		I			PC			P	T	SGA	P	P	P	A	P	A	P	CCHD	TGA		D
14	B/o Antony ammal	Fch	2		G		M			WC			A	T	AGA	A	A	A	A	A	A	A				a
15	B/o Vellakanni	Fch	2		G		M			WC			A	T	AGA	A	A	A	A	A	A	A				a
16	B/o Lakshmi	Fch	2		G		M			UN			A	T	AGA	A	P	A	A	A	A	P	ACHD		PFO	a
17	B/o Poomari	Fch	2		PG		H			WC			A	PT	LGA	A	A	A	A	A	A	A				a
18	B/o Ulagammal	Fch	1		G		M			WC			A	LPT	AGA	A	A	A	A	A	A	A				a
19	B/o Muthumari	Fch	2		G		M			WC			A	LPT	LGA	A	A	A	A	A	A	A				a

20	B/o Tamil veni	Fch	2	G	H	WC	A	T	A	A	A	A	A	A	A	A				a
21	B/o Muthu petchi	Fch	2	G	M	WC	A	LPT	A	A	A	A	A	A	A	A				a
22	B/o Rajeshwari	Mch	2	G	M	WC	A	T	A	A	A	A	A	A	A	A				a
23	B/o Jancy Rani	Fch	2	G	M	WC	A	T	A	P	A	A	A	A	A	A				a
24	B/o Meenakshi	Fch	2	G	H	UN	A	T	A	P	A	A	A	A	A	P	ACHD		ASD	a
25	B/o Sudha	Mch	1	G	H	UN	A	T	A	P	A	P	P	A	A	P	ACHD		PDA PFO	a
26	B/o Daisy	Mch	1	G	M	WC	A	T	A	A	A	A	A	A	A	A				a
27	B/o Kani	Mch	1	PG	I	PC	P	LPT	P	A	P	A	A	P	P	P	CCHD	TGA		a
28	B/o Ayeesha	Fch	2	G	M	WC	P	LPT	A	A	A	A	A	A	A	A				a
29	B/o Saratha	Fch	2	G	M	WC	A	T	A	A	A	A	A	A	A	A				a
30	B/o Rathika	Fch	2	G	M	WC	A	T	A	A	A	A	A	A	A	A				a
31	B/o Isai priya	Fch	1	PG	I	WC	A	T	A	A	A	A	A	A	P	P	ACHD		ASH	a
32	B/o Aruna	Fch	2	G	M	WC	A	T	A	A	A	A	A	A	A	A				a
33	B/o Valarmathi	Mch	2	G	M	WC	A	T	A	A	A	A	A	A	A	A				a
34	B/o Uma	Mch	2	G	M	WC	A	LPT	A	A	A	A	A	A	A	A				a
35	B/o Parvathy (Twin 1)	Fch	2	G	H	WC	A	T	A	A	A	A	A	A	A	A				a
36	B/o Parvathy (Twin 2)	Fch	2	G	H	WC	A	T	A	A	A	A	A	A	A	A				a
37	B/o Mariammal	Fch	2	PG	I	PC	A	T	A	P	A	A	A	P	P	P	ACHD		ACH	d
38	B/o Sasikala	Fch	2	G	H	WC	A	T	A	A	A	A	A	A	A	A				a
39	B/o Selvarani	Fch	2	G	M	WC	A	T	A	A	A	A	A	A	A	A				a
40	B/o Amutha	Fch	2	G	M	WC	A	LPT	A	A	P	A	A	A	A	P	ACHD		OSASP	a
41	B/o Kollsu Fathima	Fch	2	G	M	WC	A	LPT	A	A	A	A	A	A	A	P	ACHD		ASD	a
42	B/o Nivetha	Mch	2	G	M	WC	A	T	A	A	A	A	A	A	A	A				a
43	B/o Esakkiammal	Mch	2	G	H	WC	A	T	AGA	A	A	A	A	A	A	A				a

44	B/o Mari	Mch	2	G	M	WC	A	T	AGA	A	A	A	A	A	A	A				a
45	B/o Ramalakshmi	Fch	2	G	M	WC	A	T	LGA	A	A	A	A	A	A	A				a
46	B/o Chitra	Mch	2	G	H	WC	A	LPT	LGA	A	A	A	A	A	A	A				a
47	B/o Banu	Mch	1	PG	I	WC	A	T	AGA	A	P	A	P	A	A	P	ACHD		VSD ASD	a
48	B/o Velsumathi	Fch	2	G	M	WC	A	T	AGA	A	A	A	A	A	A	A				a
49	B/o Jeya lakshmi	Fch	1	PG	I	PC	P	T	SGA	P	A	P	A	A	P	P	CCHD	TA		a
50	B/o Radha	Fch	2	G	M	WC	A	T	AGA	A	A	A	A	A	A	A				a
51	B/o Sagayarani	Mch	2	G	M	WC	A	T	AGA	A	A	A	A	A	A	A				a
52	B/o Mallika	Mch	2	G	M	UN	A	PT	AGA	A	A	A	A	A	A	A				a
53	B/o Muthukani	Fch	2	G	M	WC	A	T	AGA	A	A	A	A	A	A	A				a
54	B/o Ranjitha	Mch	1	PG	I	PC	A	PT	AGA	A	P	A	P	A	A	I	ACHD		ASD VSD	a
55	B/o Mary	Fch	2	G	M	UN	A	T	AGA	A	A	A	A	A	A	A				a
56	B/o Vanitha	Fch	2	G	H	WC	A	T	AGA	A	A	A	A	A	A	A				a
57	B/o Thangalakshmi	Mch	2	G	H	WC	A	T	AGA	A	A	A	A	A	A	A				a
58	B/o Vani	Mch	2	G	M	WC	A	T	AGA	A	A	A	A	A	A	A				a
59	B/o Nathiya	Fch	2	PG	I	WC	A	T	LGA	A	A	A	A	P	P	P	ACHD		ASM	a
60	B/o Mookambikai	Fch	2		M	UN	A	PT	AGA	A	A	A	A	A	A	A				a
61	B/o Mariyam	Fch	2	G	H	WC	A	T	AGA	A	A	A	A	A	A	A				a
62	B/o Akila	Fch	2	G	M	WC	A	T	AGA	A	A	A	A	A	A	A				a
63	B/o Shibana	Fch	2	G	M	WC	A	LPT	AGA	A	A	A	A	A	A	A				a
64	B/o Swarnam	Fch	2	G	M	WC	A	T	LGA	A	A	A	A	A	A	A				a
65	B/o Kasthuri	Mch	1	PG	H	WC	A	T	LGA	A	A	A	A	P	A	P	ACHD		ASH	a
66	B/o Parameshwari	Fch	2	G	M	WC	A	T	AGA	A	A	A	A	A	A	A				a
67	B/o Mari Selvi	Mch	2	G	M	WC	A	PT	AGA	A	A	A	A	A	A	A				a

68	B/o Arumugathai	Fch	2	G	M	UN	A	T	LGA	A	P	A	A	A	A	A			a
69	B/o Jesmin	Fch	2	PG	INSULIN	IC	P	PT	SGA	P	A	P	A	P	A	P	CCHD	TRUNCUS		a
70	B/o Jeya lakshmi	Fch	2	G	H	WC	A	PT	AGA	A	A	A	A	A	A	A				a
71	B/o Dowlath	Mch	2	PG	M	WC	A	LPT	AGA	A	A	A	A	A	A	A	ACHD		ASD	a
72	B/o Radha	Mch	2	G	M	WC	A	T	AGA	A	A	A	A	A	A	A				a
73	B/o Syed Beevi	Fch	2	G	H	WC	A	T	AGA	A	A	A	A	A	A	A				a
74	B/o Punitha	Fch	2	G	M	UN	A	T	AGA	A	A	A	A	A	A	A				a
75	B/o Kumari	Mch	2	G	M	WC	A	T	AGA	A	A	A	A	A	A	A				a
76	B/o Rathinam	Fch	2	G	H	WC	A	T	LGA	A	P	A	A	A	A	P	ACHD		VSD	a
77	B/o Uma	Fch	2	G	M	WC	A	T	AGA	A	A	A	A	A	A	A				a
78	B/o Sermakani	Mch	1	PG	I	PC	A	T	SGA	P	A	P	A	P	P	P	CCHD	HLHS		d
79	B/o Swarnam	Fch	2	G	M	WC	A	T	AGA	A	A	A	A	A	A	A				a
80	B/o Sankaraeshwari	Fch	2	G	M	WC	A	T	AGA	A	A	A	A	A	A	A				a
81	B/o Nagaveni	Mch	2	G	H	WC	A	T	AGA	A	A	A	A	A	A	A				a
82	B/o Divan Meeral	Mch	2	G	M	WC	A	T	AGA	A	A	A	A	A	A	A				a
83	B/o Kalliammal	Fch	1	PG	I	WC	A	T	AGA	A	A	A	A	P	A	P	ACHD		ASYM SEP.HY	a
84	B/o Sangavi	Fch	2	G	M	WC	A	T	AGA	A	A	A	A	A	A	A				a
85	B/o Kalaivani	Fch	2	G	M	WC	A	T	AGA	A	P	A	A	A	A	P	ACHD		ASD	a
86	B/o Muthumalai	Mch	2	G	M	WC	A	LPT	LGA	A	P	A	A	A	A	A				a
87	B/o Krishnammal	Fch	2	G	M	WC	A	T	AGA	A	A	A	A	A	A	A				a
88	B/o Lakshmi	Fch	2	G	M	WC	A	PT	AGA	A	P	A	A	A	A	P	ACHD		PFO	a
89	B/o Subbu	Fch	2	G	M	WC	A	T	AGA	A	A	A	A	A	A	A				a
90	B/o Vellammal	Mch	2	PG	I	WC	P	T	LGA	A	A	A	A	A	P	P	ACHD		ASYMP SEP.HY	a
91	B/o Dhanam	Fch	2	G	M	WC	A	T	AGA	A	A	A	A	A	A	A				a

92	B/o Jeya Roslin	Mch	2	G	M	UN	A	LPT	AGA	A	A	A	A	A	A	A			a
93	B/o Maha	Mch	2	G	M	WC	A	T	AGA	A	A	A	A	A	A	A			a
94	B/o Selvi	Fch	2	G	M	WC	A	T	AGA	A	A	A	A	A	A	A			a
95	B/o Chandra	Fch	2	PG	H	WC	A	PT	LGA	A	P	A	A	A	A	P	ACHD		PDA	a
96	B/o Suganya	Fch	2	G	M	UN	A	T	AGA	A	A	A	A	A	A	A			a
97	B/o Ponmani	Mch	2	G	M	WC	A	LPT	LGA	A	A	A	A	A	A	A			a
98	B/o Subha	Fch	2	G	M	WC	A	T	AGA	A	A	A	A	A	A	A			a
99	B/o Valli	Fch	1	PG	I	WC	A	LPT	LGA	A	A	A	A	P	A	P	ACHD		ASYM.SEP.HY	a
100	B/o Sangeetha	Mch	2	G	H	WC	A	T	LGA	A	P	A	A	A	A	P	ACHD		VSD	a