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" Study of Fetal Echocardiography in Pregnant Women at Risk of Having a baby with Congenital Heart Anomalies in Port Said, Egypt "

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ABSTRACT:

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Introduction: congenital heart abnormalities are 30% of all cardiac conditions with significant infant death rate. Fetal echocardiography (FE) is effective in detecting congenital heart disease (CHD) and essential part of routine prenatal care

Objectives: To determine the prevalence of congenital heart anomalies by FE among high-risk pregnant women in Port Saied Governorate, Egypt

Materials and methods: The study included 298 pregnant women with CHD risk at 18 weeks of gestation or more referred for FE. Risk factors for CHD included a previous child with CHD, maternal insulin-dependent diabetes, or a CHD family history, increased nuchal translucency greater than 2.5 mm between 11 and 14 weeks of gestation, the presence of another congenital anomaly and exposure to teratogenic medications in early pregnancy.

Results: The study revealed that the prevalence of fetal CHD was 14.43%: atrial septal defect was 32.56%, tetralogy of Fallot was 18.60%, pulmonary stenosis was 13.95%, atrioventricular septal defect was 6.98%, double outlet right ventricle was 4.65%, hypoplastic left heart syndrome 4.65%, transposition of the great arteries was 4.65%, and single ventricle was 2.33%. A statistically significant higher percentage of fetuses with nuchal translucency greater than 2.5 mm was observed in the CHD group compared with the normal group ($p=.047$).

Conclusion: The study reported a CHD prevalence was 14.43%, with atrial septal defect and tetralogy of Fallot being the most common. Follow-up imaging between 18–22 weeks of gestation is essential for optimal detection. Early diagnosis of severe malformations can reduce postnatal complications, improve survival, and neurodevelopmental outcomes.

Keywords: Congenital Heart Defects - Fetal Echocardiography

Running title: Study of Fetal Echocardiography in Pregnant Women at Risk of Having a baby with Congenital Heart Anomalies in Port Said, Egypt

INTRODUCTION

Congenital heart disease (CHD) generally arises from abnormal formation of the fetal heart during the early stages of embryonic development.⁽¹⁾ Some abnormalities emerge when the heart divides into four chambers or when the cardiac valves are formed, with issues such as conotruncal malformations or faulty valve development often originating in these phases.⁽²⁾ These structural anomalies may not necessarily cause complications during fetal life or affect circulatory dynamics. Among birth defects, congenital vascular malformations are the most prevalent, occurring in approximately 6–8 out of every 1,000 live births.⁽³⁾

CHD encompasses diverse structural defects classified by anatomy, blood flow patterns, familial recurrence, and genetic susceptibility. Key categories include right-sided lesions (e.g., Hypoplastic Right Heart Syndrome [HRHS], Ebstein's anomaly), left-sided lesions (e.g., Bicuspid Aortic Valve [BAV], Coarctation of the Aorta [CoA], Hypoplastic Left Heart Syndrome [HLHS]), conotruncal defects (e.g., Tetralogy of Fallot [TOF], Double Outlet Right Ventricle [DORV]), laterality defects (e.g., heterotaxy, Transposition of the Great Arteries [TGA]), and isolated septal defects (Atrial Septal Defect [ASD], Ventricular Septal Defect [VSD]). Mild forms (Atrial Septal Defect [ASD], Ventricular Septal Defect [VSD], Patent Ductus Arteriosus [PDA]) make up approximately 58% of cases. Since 1970, the prevalence of CHD has risen by approximately 10% every five years. Chromosomal-related CHD accounts for 8–10% of cases, often due to de novo protein-truncating or missense mutations, differing from non-syndromic CHD.⁽⁴⁾

One essential method for prenatal screening is fetal echocardiography (FE), which makes it possible to identify structural cardiac abnormalities and arrhythmias early on. It is currently regarded as a regular component of prenatal care and has good

sensitivity and specificity for detecting CHD in both low- and high-risk pregnancies.⁽⁵⁻⁷⁾

Procedures such as fetal cardiac magnetic resonance imaging and fetal electrocardiography are available in only a limited number of centers due to their high cost and limited accessibility, and they are primarily used within research settings. These methods serve as acceptable alternatives for diagnosing fetal atrial and ventricular arrhythmias.⁽⁸⁾

Fetal echocardiography should be recommended for fetuses exhibiting anomalies on obstetric anatomical ultrasonography or exhibiting risk factors for heart disease.⁽⁹⁾ CHD risk factors are classified as maternal or fetal, though many cases have no identified cause. Genetics play a key role, with chromosomal abnormalities (e.g., trisomy 21 linked to atrioventricular septal defects) and gene mutations contributing to specific malformations. Familial cases further highlight inherited influences. CHD is broadly categorized into three main types. Maternal risk factors include insulin-dependent diabetes, phenylketonuria, stress, obesity, connective tissue diseases, certain medications, substance use, and teratogen exposure. Teratogen effects depend on timing, genetic susceptibility, dose, and mechanism, with the highest fetal sensitivity between weeks 2–8 of the first trimester.^(10,11)

Fetal cardiology has progressed beyond merely diagnosing CHD whether simple or complex prenatally, as These days, FE makes it easier to evaluate dynamic fetal heart physiology and possible prenatal or postnatal treatments. Its greatest benefit lies in detecting critical CHD before birth, enabling prompt cardiac management immediately after delivery to reduce neonatal morbidity and mortality. Evaluating the severity of abnormal cardiac physiology in different CHD types prior to birth helps fetal cardiologists predict fetal outcomes, anticipate postnatal hemodynamic challenges, plan delivery through multidisciplinary coordination, and assess the potential neonatal impact after birth.⁽⁹⁾

Prenatal detection of CHDs through fetal echocardiography improves outcomes by allowing early intervention planning, reducing perinatal morbidity in severe cases like HLHS, and enhancing cognitive outcomes in TGA. It also provides families with vital prognostic information and the option of pregnancy termination. Early diagnosis further enables fetal cardiac interventions, including transplacental drug therapy, which is particularly effective for managing fetal tachyarrhythmias.^(5,12) Therefore, this study

aimed to evaluate the prevalence of CHDs detectable through FE in high-risk pregnant women—those with diabetes or a CHD family history—in Port Said Governorate, providing a baseline for a structured and effective awareness campaign highlighting the importance of early diagnosis of birth defects in children.

PATIENTS AND METHODS:

This was a prospective cross-sectional study conducted at the Ultrasonography Special Care Unit, Port Said Insurance Hospitals, between January and December 2024. The study was performed as part of the Master's thesis.

Participants: A total of 298 pregnant women with a gestational age of 18 weeks or more and one or more risk factors for congenital heart disease (CHD) were recruited.

Inclusion criteria: previous child with CHD, maternal insulin-dependent diabetes mellitus, family history of CHD, increased nuchal translucency (>2.5 mm at 11–14 weeks), detection of other congenital anomalies on detailed ultrasound, or maternal exposure to teratogenic medications during early pregnancy.

Exclusion criteria: first-trimester pregnancies, multiple gestations, and women at low risk for CHD.

Recruitment was consecutive, and all participants gave informed consent after being informed of the study objectives.

Data Collection:

A structured questionnaire was administered privately to each participant, covering maternal demographic data, body mass index (BMI), obstetric and medical history, drug exposure, and family history of CHD. Pregnancy characteristics, including plurality and gestational age at ultrasound, were also recorded.

Fetal Echocardiography⁽¹³⁾: All examinations were performed using a Voluson E8 ultrasound system (GE Healthcare, USA) equipped with a 2–5 MHz curvilinear transducer. The protocol adhered to the American Society of Echocardiography (ASE) guidelines for fetal echocardiography. Each scan included:

- Two-dimensional echocardiography, M-mode, color Doppler, and pulsed-wave Doppler assessment.
- Standard imaging planes: four-chamber, five-chamber, left and right ventricular outflow tracts, long-axis, three-vessel view, ductal arch, and aortic arch.
- All studies were conducted by experienced obstetric sonographers trained in FE, with quality assurance and review by senior faculty specialists.

Statistical analysis

The gathered data underwent statistical analysis using version 25 of the Statistical Package for the Social Sciences (SPSS) program⁽¹⁰⁾. The data were described using descriptors such as minimum, maximum, mean, standard deviation, and a 95% confidence interval (CI) for the mean⁽¹⁴⁾. When establishing the sample size, a 20% margin of error (beta error) was considered acceptable to maintain a study power of 80%. The significance level (alpha) was set at 5%, representing a 95% confidence level. Statistical significance was determined with a *p*-value less than .05⁽¹⁵⁾.

RESULTS (data are presented as mean $\pm$ SD)

Indications for FE (Table 1, Figure 1 (a&b))

Fetal echocardiography indications were: 26.51% for family history of CHD, 19.13% for history of previous child with CHD; 25.17% for diabetic mother; 21.14% for teratogenic medication use; 21.14% for Nuchal Translucency > 2.5 mm and 10.59% for other congenital anomalies findings during obstetric US.

Prevalence and type of CHDs detected (Table 1)

CHD was detected in 14.43%; out of them 32.56% had ASD, 18.60% had TOF, 13.95% had PS, 6.98% had AVSD, 4.65% had DORV, 4.65% had HLHS, 4.65% had TGA and 2.33% had single ventricle.

Maternal data (Table 2 & 3- Figure 2&3)

Maternal Biodata

The maternal age ranged from 18.00 to 42.00 years, with a mean age of 28.59 ± 6.54 years. For normal group age ranged from 18.00 to 42.00 years, with a mean age of 28.18 ± 6.45 years, while for CHD Group it ranged from 18.00 to 40.00 years, with a mean age of 31.02 ± 6.60 years. The age was statistically significant higher in the CHD group compared with the Normal Group ($p=.008$). Moreover, there was a statistically marked association with maternal age > 35 years and fetal CHD occurrence ($p=.042$). There was no statistically notable difference in BMI distribution or in gestational age (weeks) between the two studied groups.

Maternal obstetric, medical and familial history

Gravidity, parity, abortion history, and gestational age at FE showed no notable differences between the groups. (Table 2)

There was no statistically marked difference in the DM distribution between the two studied groups ($p=.113$). However, Type 1 DM is a risk factor for CHD occurrence

($p=.003$). Having Type 1 DM increases the risk of CHD occurrence by nine folds [OR: 9.4875; 95% CI: 1.5372 to 58.5580; $p=.0154$]

No notable differences were found between the two groups regarding family history or previous offspring with CHD. However, the CHD Group showed a statistically significant higher percentage in the history of Teratogenic medication administration compared with the Normal Group ($p=.017$). There was no notable variation between the two groups in the types of teratogenic drugs used, apart from lithium. Lithium use was notably higher in the CHD group ($p=.0139$) (Table 3)

Fetal data (Table 4, Figure 4)

There was a statistically notable higher percentage of fetus with > 2.5 mm NT in the CHD group compared with Normal group ($p=.047$). Moreover, there was no statistically significant difference in the presence of other anomalies between the two studied groups ($p=.718$)

DISCUSSION

Congenital heart defects are among the most prevalent congenital anomalies, representing about 28% of all birth-related malformations.⁽¹⁶⁾ The global prevalence of CHD is estimated at 8–12 in 1,000 live births, with a median of 9 in 1,000, resulting in nearly 1.35 million newborns each year.⁽¹⁷⁾ When minor defects are included, the incidence may rise to approximately 75 per 1,000 live births.⁽¹⁸⁾ CHD remains a leading contributor to morbidity and mortality, particularly within the 1st year of life. While many cases present as isolated cardiac malformations, more than 30% are associated with additional organ or systemic defects.⁽¹⁹⁾ Improved prenatal screening and advancements in echocardiography have enhanced detection, particularly of minor defects such as small ASDs and VSDs, many of which resolve spontaneously, contributing to higher reported prevalence.^(20,21)

Beyond its medical implications, CHD profoundly affects patients' quality of life, imposes considerable financial burdens on families and healthcare systems, and often necessitates ongoing follow-up and multiple interventions.⁽²²⁾ Although progress in evidence-based medicine and early intervention has markedly improved survival and outcomes in high-income countries, disparities persist in low- and middle-income regions where access to advanced care remains limited.⁽¹⁷⁾

The present study revealed that the prevalence of FE detected fetal CHD in the studied population in Port-Said is 14.43% which is aligning with **Khorshid et al.**

(2019)⁽²³⁾ study carried in Zagazig University, among the 60 examined cases, fetal echocardiography identified 8 (13.33%) with CHD. Moreover, **Alipour et al. (2022)**,⁽²⁴⁾ revealed a fetal CHD prevalence of 13.1%. Furthermore, the prevalence in the present study is close to that identified by **Chitra and Vijayalakshmi (2013)**⁽²⁵⁾, as CHD was identified in 18.2% of cases.

However, the present study prevalence findings is far lower than that found in Mansoura governorate in a **Rakha and El Marsafawy (2019)**⁽²⁶⁾ that identified that the fetal CHD was 28.60%. Moreover, **Al-Fahham et al. (2021)**,⁽²⁷⁾ examined 101 singleton pregnant women with indications for prenatal CHD risk factors over a period of one year. Fetal cardiac abnormalities were identified in 46.5%. Among these, CHDs were found in 34.6%. In **Ghiasi et al. (2019)**⁽²⁸⁾ study, fetal cardiac abnormalities were detected in 33.22% of patients.

In the current study, the indications for performing FE included a family history of CHD which was the highest percent (26.51%), followed by maternal diabetes (25.17%), exposure to teratogenic medication and increased NT measurements greater than 2.5 mm (21.14% for each), as well as the presence previous child affected by CHD (19.13%) the presence of other congenital anomalies detected during routine screening (10.59%).

Khorshid et al. (2019)⁽²³⁾ reported that the third the leading indication for referral was maternal diabetes (25%). However, the abnormal obstetric ultrasound findings were not considered a relevant reason for referral to FE in their study. While in the present study it ranked the last (10.59%) The result could point to gaps in obstetric screening services in Egypt, especially within Port Said and rural settings. Similar trends have been noted in studies from multiple nations.⁽²⁹⁻³¹⁾ Furthermore, the significance of intrauterine diagnosis, proper management, and their impact on fetal and neonatal outcomes may not be completely understood by all obstetricians.

However, **Al-Fahham et al. (2021)**,⁽²⁷⁾ study, demonstrated that the leading indication for FE referral was suspicious routine obstetric scans in 52.5%, maternal diabetes was 10.9%, and positive CHD family history in (10.9%). In **Barati et al. (2022)**,⁽³²⁾ study, 18.2% of the pregnant women were diagnosed with gestational diabetes mellitus. The most frequent referral reason for FE was abnormal ultrasound results, which accounted for 57.6% of referrals. A history of abortion was in second (36.5%), followed by elevated NT. IVF pregnancies, high-risk screening results, and a

history of a prior child with an intellectual handicap were the least common explanations, accounting for 1% of instances. Similar findings have been reported by **Nair et al. (2016)**.⁽³³⁾

Moreover, in **Rakha and El Marsafawy (2019)**⁽²⁶⁾ study, the suspected abnormalities on obstetric ultrasound ranked as the third most common referral indication, while the leading indication for performing FE was a family history of CHD (34.1%), followed by the presence of nonimmune hydrops (21.2%) and abnormal findings on routine obstetric ultrasound (14%). **Kovavisarach and Mitinunwong (2011)**⁽³⁴⁾ reported maternal diabetes (25.6%) as the most frequent indication for FE. **Papazoglou et al. (2022)**⁽³⁵⁾ showed that a significantly increased risk of coronary heart disease (CHD) is associated with both pregestational diabetes mellitus (PGDM) and gestational diabetes mellitus (GDM) when compared to the general population. However, PGDM shows a stronger correlation, being linked to a 3.5-fold greater risk of fetal malformations. Therefore, effective management of diabetes both before conception and throughout pregnancy is critical to reducing the harmful effects of hyperglycemia on fetal cardiac development. **Al Subhi et al.**⁽³⁶⁾, who found that type 1 diabetes has a larger risk than type 2 diabetes, and that maternal diabetes can affect fetal heart development and raise the risk of CHD. The most demonstrated that the most frequent reasons for referral in **Hamar et al. (2006)**,⁽³⁷⁾ **Wright et al. (2014)**⁽³⁸⁾, and **Komisar et al. (2017)**⁽³⁹⁾ comprised probable fetal heart disease (9%–41%), maternal diabetes (12%–21%), extracardiac anomalies (9%–21%), and a family history of structural CHD (13%–37%). Fetal arrhythmias (5%–14%), suspected or confirmed genetic abnormalities (3%–16%), and maternal drug exposure (2%–7%) were additional referral grounds. A greater percentage of instances with elevated NT, cystic hygroma, or a two-vessel cord are probably the cause of the higher referral rate for extracardiac anomalies. **Clur et al (2012)**⁽⁴⁰⁾, which highlights increased NT, incidental anomalies detected on obstetric ultrasound, and a family history of CHD as the most frequent referral causes.

By contrast, **Cha et al. (2012)**,⁽⁴¹⁾ identified suspected CHD on obstetric ultrasound as the most frequent referral cause. They demonstrated that referral indication was (62.4%), increased NT at the first trimester (5.7%), maternal DM (3.9%), previous child with CHD diagnosis (6.1%), familial risk (3.6%). **Chiatra et al. (2017)**,⁽⁴²⁾ reported The referral reasons included abnormal fetal cardiac scans in 26.8%,

a history of CHD in a previous sibling 20.7%, maternal indications in 18.2%. **Gill et al. (2003)**⁽⁴³⁾ reported 56% of the reason for referral was a family history of CHD. The incidence of CHD in pregnancies referred due to a sibling with CHD was 2.7% aligns with findings from several large population-based studies^(44,45).

Jacobson et al., (1992)⁽⁴⁶⁾ study followed 138 pregnancies with first-trimester lithium exposure and found no overall increase in birth defects, though one case of Ebstein anomaly was detected via antenatal fetal echocardiography. Although lithium exposure does increase the risk of congenital heart defects, this risk is frequently overestimated; the true incidence is roughly 1 in 2,000.⁽⁴⁷⁾

There is conflicting evidence on the teratogenic potential of ACE inhibitors (ACE-Is) in the early stages of pregnancy. Some studies (**Cooper et al., (2006)**⁽⁴⁸⁾; **Malm et al., (2008)**⁽⁴⁹⁾) raise the possibility of heart and central nervous system abnormalities, while maternal diabetes may be a contributing factor. Other research (**Lennestål et al., (2009)**⁽⁵⁰⁾; **Diav-Citrin et al., (2011)**⁽⁵¹⁾) reports no significant difference in malformation rates compared to other antihypertensives or no increased risk overall. Aligning with our findings concerning no statistical significance in CHD occurrence between the two groups regarding ACE-Is, **Bateman et al (2017)**⁽⁵²⁾ demonstrated that First-trimester exposure to ACE-Is was not found to increase the risk of major congenital malformations. In contrast, **van der Zande et al. (2024)**,⁽⁵³⁾ reported that ACE inhibitors should be avoided during pregnancy, including the first trimester, due to their potential teratogenic effects. Warfarin-associated embryotoxicity typically occurs between six and nine weeks of gestation^(54,55), although **Schaefer et al. (2006)**⁽⁵⁶⁾ reported no warfarin-related effects when exposure occurred before 8 weeks of gestation.

Early pregnancy exposure to paroxetine has been associated with an increased risk of congenital anomalies. **Bakker et al. (2010)**⁽⁵⁷⁾ reported a higher incidence of atrial septal defects, while **Bérard et al. (2016)**⁽⁵⁸⁾ discovered a 28% increased risk of significant cardiac abnormalities and a 23% increased risk of major congenital deformities, which were consistent across studies and populations.

Maternal vitamin A status significantly influences fetal outcomes. Deficiency increases adverse risks, while excessive intake—particularly of preformed vitamin A—can be teratogenic, especially within the first 60 days after conception. Teratogenicity is linked to elevated retinoic acid metabolites disrupting gene activity during

organogenesis. **Rothman et al. (1995)**⁽⁵⁹⁾ reported higher risks of neural crest-related defects in women consuming over 15,000 IU per day from diet or 10,000 IU per day from supplements. High intake levels are more common in high-income countries due to supplement use and vitamin A-rich diets. As a developing country, this can explain the finding of the present study concerning Vitamin A as although 17.46% of our studied women used Vitamin A but here was only one case with CHD.

Dathe et al. (2022)⁽⁶⁰⁾ determined that the use of NSAIDs for a few days during the second trimester does not seem to present a significant danger. However, prolonged use in the advanced second trimester may result in constriction of the ductus arteriosus and oligohydramnios, which are comparable to the consequences seen after third trimester use.

van Gelder et al. (2011)⁽⁶¹⁾ showed that there was no significant correlation between the incidence of some birth abnormalities and the use of NSAIDs during the early stages of pregnancy, but that exposure to several NSAIDs increased the odds ratio for septal malformations.

In the present study, there was a statistically significant higher percentage of fetus with increased NT in the CHD group compared with Normal group ($p=.047$)

Morga et al. (2015),⁽⁶²⁾ shown that NT values above the 99th centile (>3.5 mm) are associated with a significantly higher risk of major cardiac defects (positive likelihood ratio 26.1), whereas NT measurements between the 95th and 99th centiles (2.5–3.5 mm) are linked to a moderate increase in risk (positive likelihood ratio 3.5). Chromosome abnormalities, structural malformations, genetic disorders, and an increased risk of CHD are all closely linked to elevated fetal NT as indicated by **Souka et al. (2001)**,⁽⁶³⁾ and **Arjmandnia et al. (2021)**⁽⁶⁴⁾

Therefore, a comprehensive series of prenatal evaluations—such as fetal karyotyping, detailed ultrasound assessment, fetal echocardiography, genetic analysis, and infection screening (ideally completed by the 20th week of gestation)—can help differentiate high-risk from low-risk pregnancies. **Müller et al (2007)**⁽⁶⁵⁾ found that among 481 fetuses with confirmed CHDs and normal chromosomes, 47% had increased nuchal translucency ($NT \geq 2.5$ mm). Atrioventricular septal defect showed the highest prevalence of abnormal NT (62%). Overall, nearly half of CHD cases presented with elevated NT in early scans. The study supports recommending fetal

echocardiography for all fetuses with NT greater than 95th or 99th percentile due to its strong association with major cardiac anomalies.

The findings of the present study revealed that maternal age was notably greater in the CHD group than in the normal group ($p = .008$), with more mothers aged above 35 years ($p=.042$). CHD was observed more often in fetuses born to mothers over 35 years., consistent with **Owens et al.**⁽⁶⁶⁾ who linked advanced maternal age to increased CHD risk and stressed the importance of early prenatal detection.

Eltohamy et al. (2023),⁽⁶⁷⁾ reported a significantly higher CHD incidence with advanced maternal age (>35 years) being the most common contributing factor.

The most commonly detected CHDs in the current study was ASD (32.56%), followed by TOF (18.60%), VSD (13.95%), AVSD (6.98%), PS (11.63%), DORV (4.65%), HLHS (4.65%), TGA (4.65%), and single ventricle anomalies (2.33%).

In **Rakha and El Marsafawy (2019)**⁽²⁶⁾ study, VSD was the most frequently identified anomaly (29.01%), which is in line with the results of **Trivedi et al. (2012)**⁽⁶⁸⁾ However, other investigations reported HLHS as the most common diagnosis on fetal echocardiography.^(69,70) **Cha et al. (2012)**,⁽⁴¹⁾ reported that the was the most frequently identified anomaly was TOF (15.9%), followed by VSD (13.1%), AVSD (6.2%), DORV (6.2%), HLHS (5.5%), ASD (2.1%), PS (6.9%) **Eltohamy et al. (2023)**,⁽⁶⁷⁾ results revealed that the most common CHD were VSD) in 20% and TOF 13.3%. Additionally, single cases (6.6% each) were identified for hypoplastic left heart syndrome, DORV, AS, cardiomegaly, ASD, single ventricle, and transposition of the great vessels. In **Alipour et al. (2022)**,⁽²⁴⁾ study, VSD was the most frequently detected CHD, accounting for 4.4% of cases. **Sharma et al. (2017)**⁽¹⁰⁾ study reported VSD in 44.4% of cases, while **Nayak et al. (2016)**⁽⁷¹⁾ study noted endocardial cushion defects in 19.2%.

Chiatra et al. (2017),⁽⁴²⁾ reported the most fetal CHD found was VSD with 18.4% followed with 14.9% HLHS, 11.5 HRHS, 9.2% AVSD, 4.6% TOF, 4.6% single ventricle, 3.5% DORV. **Ghiasi et al. (2019)**⁽²⁸⁾ observed intra-cardiac echogenic focus was estimated at 25.6% in 25.6% of cases, while complex CHD and VSD were detected in 21.76% and 8.05% of cases, respectively. The complex CHD was identified as the most prevalent type, 3.79% with TGA, 1.4% with Ebstein, 0.94% with TOF and 0.47% with HLHS, while patent ductus arteriosus (PDA) and atrial septal defect (ASD) were not detected. In contrast, **Wu et al. (2010)**⁽⁷²⁾ reported higher rates of pulmonary

stenosis and TOF in a Taiwanese cohort, where ASD and PDA were frequently identified, likely due to the extensive use of echocardiography. Additionally, research shows that Asian populations are more likely than Caucasians to have right-sided obstructive lesions, while left-sided obstructive lesions are less common in these ethnicities.^(73,74)

Singh et al. (2024)⁽⁷⁵⁾ determined the CHDs incidence in 850 cases in a tertiary care setting in normal population. They demonstrated that VSD was identified in 0.35% of cases, ASD in 0.7%, Ebstein's anomaly in 0.35, hypoplastic left heart syndrome in 0.7, TOF in 0.7 cases, TGA in 0.7, and severe fetal hydrops with bradycardia 0.35%. this indicates that a normal pregnancy does not rule out the presence of a major heart defect, moreover the ability of FE to detect CHD even in normal population which encourages its use for routine screening.

CONCLUSIONS

The study concludes that CHD had a prevalence of 14.43%, with ASD and TOF being most common. Early fetal cardiac imaging by skilled specialists allows timely diagnosis, parental reassurance, and proper prenatal planning. Follow-up echocardiography at 18–22 weeks is advised to confirm early findings. Early detection of severe cardiac malformations can reduce postnatal complications, improve preoperative care, and enhance survival and neurodevelopmental outcomes.

RECOMMENDATIONS

The study recommends further research to validate the benefits of fetal echocardiography on survival and neurodevelopmental outcomes. Continuous training for practitioners is vital to improve diagnostic accuracy and early recognition of congenital heart defects. A multidisciplinary approach involving obstetricians, pediatric cardiologists, sonographers, geneticists, psychologists, and cardiac surgeons is encouraged for comprehensive family support. Routine prenatal ultrasounds should include a four-chamber heart view, with abnormalities referred for specialist assessment. First-trimester echocardiography is advised for early detection, though some anomalies may appear later. Finally, incorporating detailed echocardiography as a standard screening tool, even in low-risk populations, is recommended to enhance detection and prenatal care.

LIMITATIONS

Availability of patients with high risk factor for CHD. Availability and high cost of the fetal echocardiography.

Author Contributions:

the first author conceived the research idea, designed the study protocol, collected and analyzed the data, and wrote the first draft of the manuscript. other authors supervised the study design, provided methodological guidance, contributed to statistical analysis and interpretation of results and revised the manuscript critically for intellectual content.

All authors approved the final manuscript and agree to be accountable for its content.

Conflict of Interest: The authors declare no conflicts of interest.

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Tables Legends:

Table (1): Indication for FE type of CHDs findings of the studied group

Indication for FE (n=298)	n	%
• Family history of CHD	79	26.51
• History of previous child with CHD	57	19.13
• Diabetes	75	25.17
• Teratogenic medication use	63	21.14
• Nuchal Translucency > 2.5 mm	63	21.14
• Other Congenital anomalies findings	27	10.59
Congenital heart defects findings (n=298)	n	%
Detected CHD		
No	255	85.57
Yes	43	14.43
Type CHD (n=43)		
ASD	14	32.56
TOF	8	18.60
VSD	6	13.95
PS	5	11.63
AVSD	3	6.98
DORV	2	4.65
HLHS	2	4.65
TGA	2	4.65
Single ventricle	1	2.33

n: Number of patients

Table (2): The Maternal Demographic data by group

Demographic data	Total (n=298)	Group		Test of significance p-value
		Normal (n=255)	CHD (n=43)	
Age (years)				
- Min. – Max.	18.00-42.00	18.00-42.00	18.00-40.00	$t_{(df=296)}=2.667$ $p=.008^*$
- Mean $\pm$ S.D.	28.59 $\pm$ 6.54	28.18 $\pm$ 6.45	31.02 $\pm$ 6.60	
- 95% CI of the Mean	27.84-29.33	27.38-28.97	28.99-33.05	
- 25 th - 75 th Percentile	24.00-34.00	23.00-34.00	27.00-36.00	
BMI (kg/m²)				
- Min. – Max.	17.30-37.80	17.30-37.80	18.30 $\pm$ 37.00	$t_{(df=296)}=1.168$ $p=.244$ NS
- Mean $\pm$ S.D.	27.17 $\pm$ 4.00	27.06 $\pm$ 3.90	27.83 $\pm$ 4.57	
- 95% CI of the Mean	26.71-27.62	26.58-27.54	26.42-29.23	
- 25 th - 75 th Percentile	24.40-29.90	24.20-29.80	25.10-30.90	
The maternal age groups				
≤ 35 years	247 (82.89%)	216(84.71%)	31 (72.09%)	$\chi^2_{(df=1)}=4.127$ $p=.042^*$
> 35 years	51 (17.11%)	39 (15.29%)	12 (27.91%)	

n: Number of patients, Min-Max: Minimum – Maximum, S.D.: Standard Deviation

CI: Confidence interval, t=independent t-test, χ^2 =Pearson Chi-Square, df= degree of freedom

NS: statistically not significant ($p \geq .05$), * Statistically significant ($p < .05$)

Table (3): Maternal obstetric, familial, and drug history by group

Maternal data	Total (n=298)		Group				Test of significance <i>p</i> -value
			Normal (n=255)(85.57%)		CHD (n=43) (14.43%)		
	n	%	n	%	n	%	
Gravidity							
- One	12	4.03	8	3.14	4	9.30	<i>Z</i> =1.9023, <i>p</i> =.0574
- Two	76	25.5	66	25.88	10	23.26	NS
- Three	121	0	103	40.39	18	41.86	<i>Z</i> =0.3655, <i>p</i> =.7113
- Four	89	40.6	78	30.59	11	25.58	NS
		0					<i>Z</i> =0.1814, <i>p</i> =.8572
		29.8					NS
		7					<i>Z</i> =0.6636, <i>p</i> =.5093
							NS
Parity							
- Zero	39	13.0	30	11.76	9	20.93	<i>Z</i> =1.6485, <i>p</i> =.0989
- One	143	9	128	50.20	15	34.88	NS
- Two	89	47.9	77	30.20	12	27.91	<i>Z</i> =1.8592, <i>p</i> =.0629
- Three	27	9	20	7.84	7	16.28	NS
		29.8					<i>Z</i> =0.3034, <i>p</i> =.7642
		7					NS
		9.06					<i>Z</i> =1.7827, <i>p</i> =.0751
							NS
Abortion							
- No abortion	166	55.7	139	54.51	27	62.79	<i>Z</i> =1.2963, <i>p</i> =.1936
- One	87	0	74	29.02	13	30.23	NS
- Two	39	29.1	36	14.12	3	6.98	<i>Z</i> =0.1618, <i>p</i> =.8729
- Three	6	9	6	2.35	0	0.00	NS
		13.0					<i>Z</i> =1.2844, <i>p</i> =.2005
		9					NS
		2.01					<i>Z</i> =1.0161, <i>p</i> =.3077
							NS
Family history of CHD							□ ² (df=1)=0.050
-No	219	73.4	188	73.73	31	72.09	<i>p</i> =.822 NS
-Yes	79	9	67	26.27	12	27.91	
		26.5					
		1					
History of previous baby with CHD							□ ² (df=1)=2.054
-No	241	80.8	210	82.35	31	72.09	<i>p</i> =0.114 NS
	57	7	45	17.65	12	27.91	

-Yes		19.1 3					
History of Teratogenic medication use	235 63	78.8 6 21.1 4	207 63	81.18 18.82	28 15	65.22 34.88	$\chi^2_{(df=1)}=5.693$ $p=.017^*$
-No							
-Yes							
Teratogenic Medication administered	15	23.8	12	25.53	3	18.75	$Z=0.5501, p=.5823$
- ACE Inhibitor	12	1	11	23.40	1	6.25	NS
- Ibuprofen	2	19.0	0	0.00	2	12.50	$Z=1.5093, p=.1310$
- Lithium	10	5	7	14.89	3	18.75	NS
- Paroxetine	12	3.17	7	14.89	5	31.25	$Z=2.4633, p=.0139^*$
- Valproate	11	15.8	10	21.28	1	6.25	$Z=0.3646, p=.71884$
- Vitamin A	1	7	0	0.00	1	6.25	NS
- Warfarin		19.0 5 17.4 6 1.59					$Z=1.4391, p=.1499$ NS $Z=1.3675, p=.1707$ NS $Z=1.7277, p=.0836$ NS
Gestational age (weeks) at FE		18.00- 22.86		18.00- 22.86		18.00- 22.86	$t_{(df=296)}=1.319$ $p=.188$ NS
- Min. – Max.		20.33±1. 70		20.38±1.7 2		20.01±1. 60	
- Mean ± S.D.							
Diabetic women	223 75	74.8 3 25.1 7	195 60	76.47 23.53	28 15	65.12 34.88	$Z=1.587, p=.112$ NS
- No							
- Yes							
Type of Diabetes (n=75)	29	9.73	26	10.20	3	6.98	$Z=0.659, p=.509$ NS
- Gestational	5	1.68	2	0.78	3	6.98	$Z=2.925, p=.003^*$
- Type 1 DM	41	13.7 6	32	12.55	9	20.93	$Z=1.4759, p=.139$ NS
Type 2 DM							
Test of significance p -value			$\chi^2_{(MC)(df=2)}=11.314,$ $p=.013^*$				

n: Number of patients, S.D.: Standard Deviation, χ^2 =Pearson Chi-Square, df= degree of freedom

NS: statistically not significant ($p \geq .05$), * Statistically significant ($p < .05$)

Table (4): Fetal data of the studied group

Fetal data		Group	
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	Total (n=298)		Normal (n=255)		CHD (n=43)		Test of significance <i>p</i> -value
	n	%	n	%	n	%	
Nuchal Translucency	235	78.86	206	80.7	29	67.44	$\chi^2_{(df=1)}=3.929$ <i>p</i> =.047*
< 2.5 mm	63	21.14	49	8	14	32.56	
> 2.5 mm				19.2			
				2			
Other congenital anomalies	273	91.3	233	91.3	40	93.02	$\chi^2_{(df=1)}=0.130$ <i>p</i> =.718 NS
- No	25	7	22	7	3	6.98	
- Yes		8.39		8.63			
Other anomaly present	2	8.00	2	9.09	0	0.00	$\chi^2_{(MC)(df=4)}=8.597$ <i>p</i> =.072 NS
Caudal	8	32.0	7	31.8	1	33.33	
regression	1	0	0	2	1	33.33	
syndrome	7	4.00	6	0.00	1	33.33	
Cleft palate	7	28.0	7	27.2	0	0.00	
Hypoplastic nasal bone		0		7			
		28.00		31.8			
Renal agenesis				2			
Spina bifida							

n: Number of patients 638 , χ^2 =Pearson Chi-Square 639 ,df= degree of freedom 640
NS: statistically not significant (*p*≥.05)

Figure Legends:

Figure (1): Bar chart of (a) Indications for FE of the studied group 645 (b) distribution of CHD types (n=43)

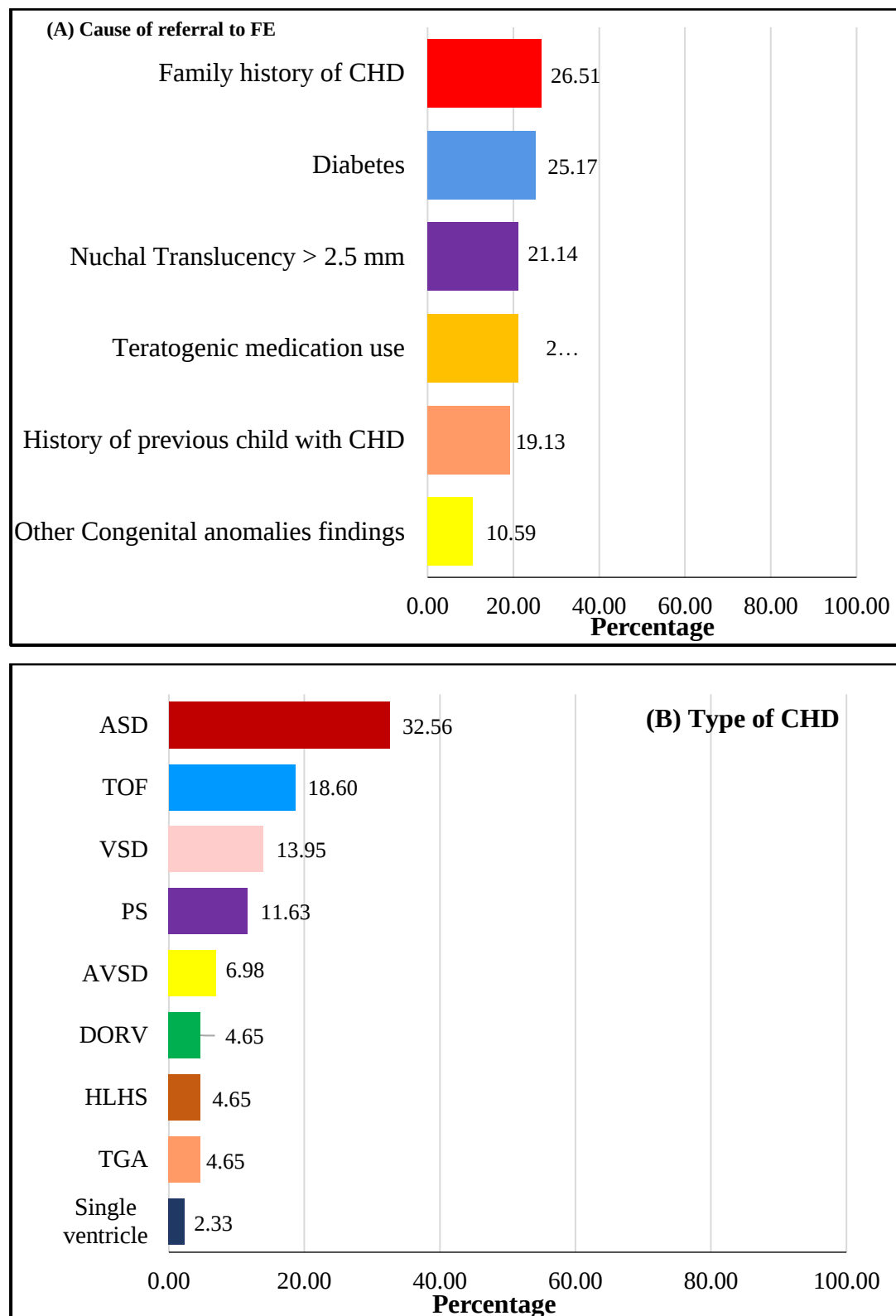
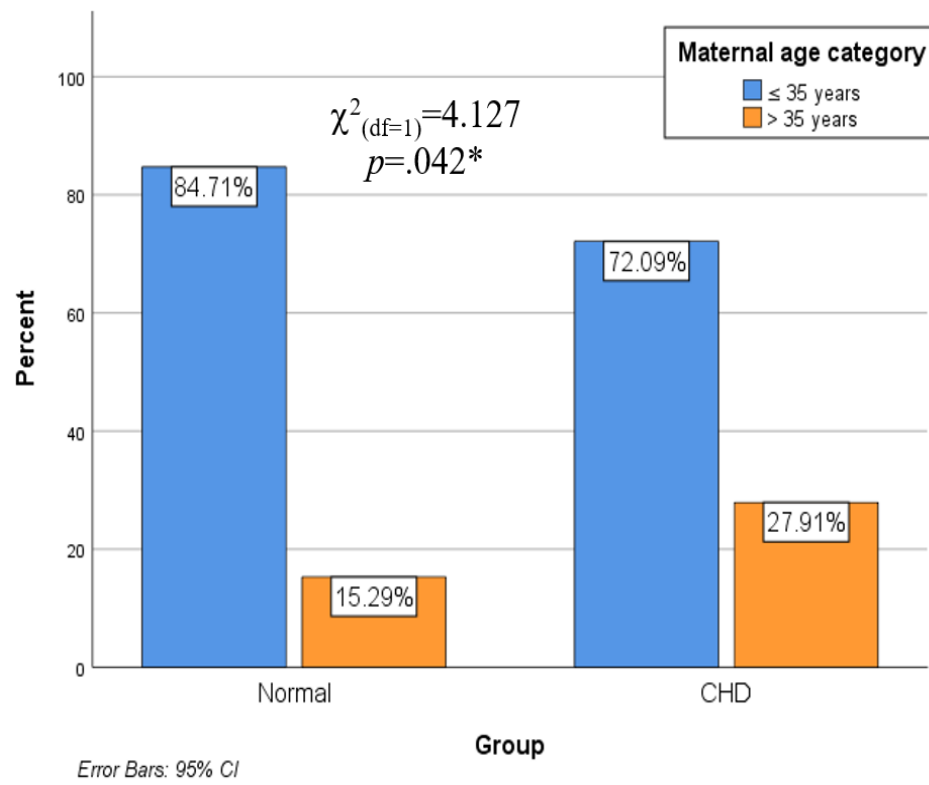


Figure (2): Clustered bar chart of the maternal age group by CHD



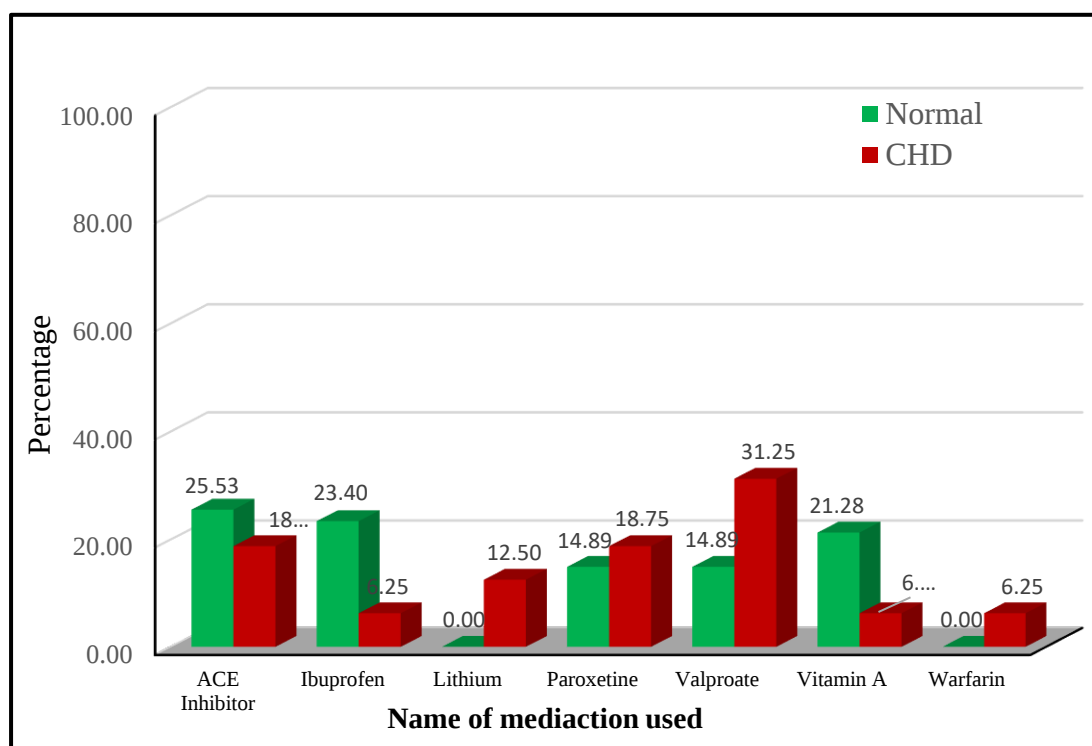
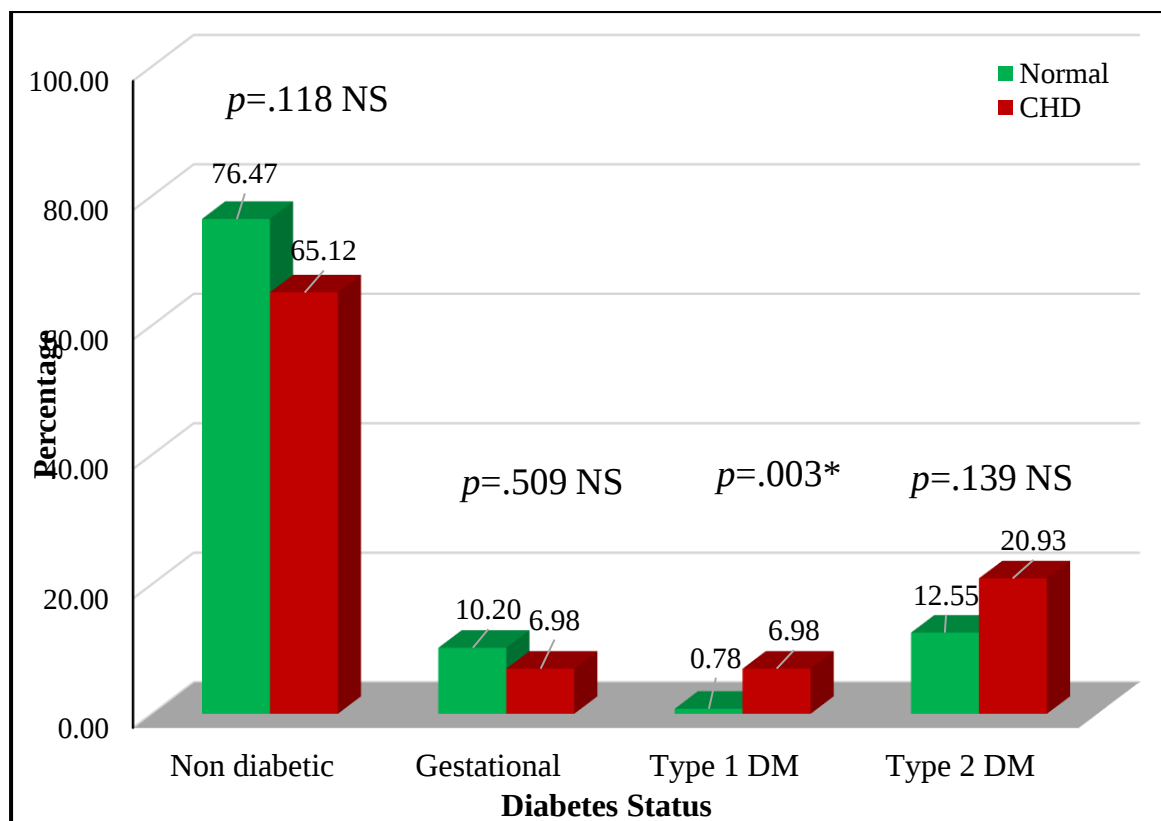


Figure (3): Cluster bar chart of (a) the Diabetes status by Group (b) distribution of teratogenic medication in women who used them (n=63) by Group

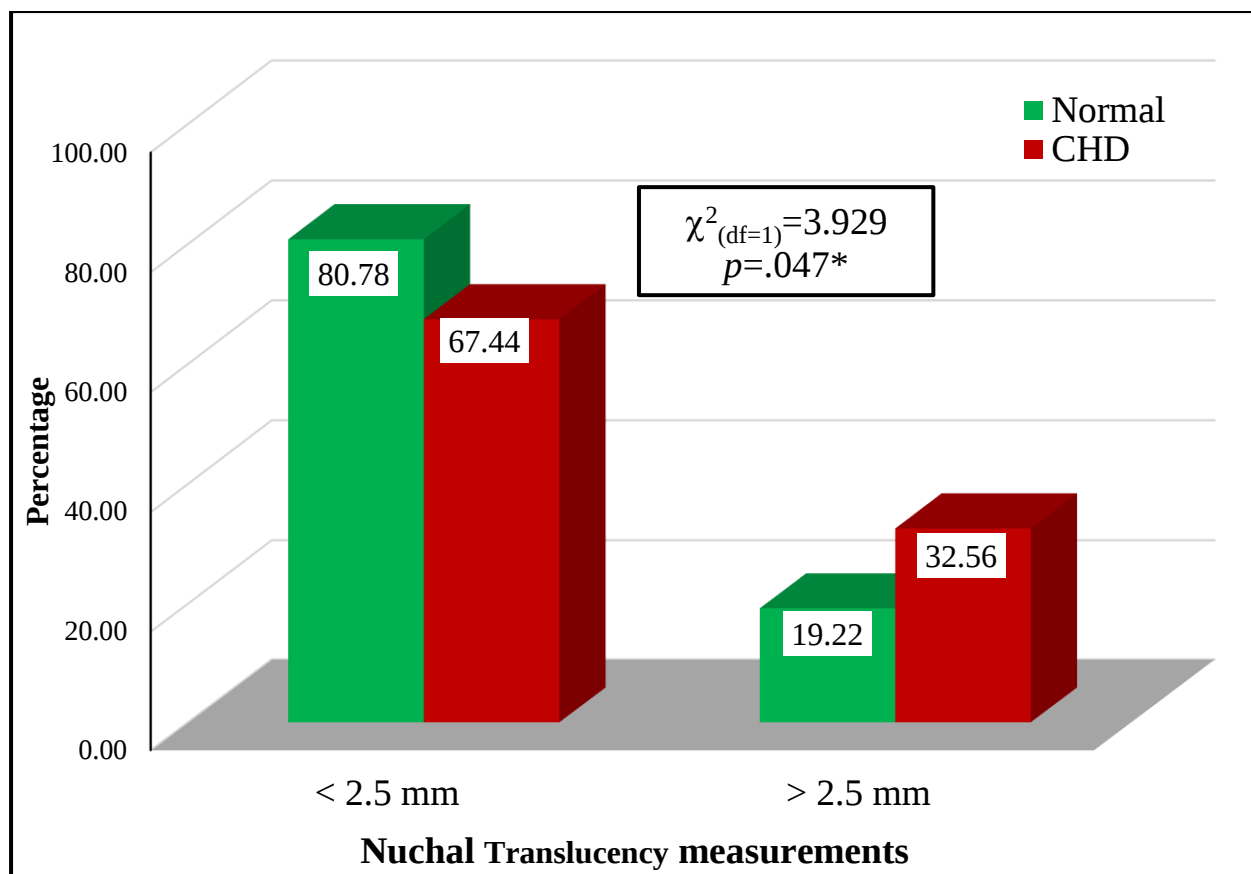


Figure (4): Cluster bar chart of NT measurements in the studied women by the Group

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