European Journal of Clinical Pharmacy
Print ISSN: 2385-409X **Online ISSN**: 3105-0409
Website: https://farmclin.com/

Original Research Article

Correlation Between Electrocardiographic Parameters (pwd,rsr, R/Sratio) With Echocardiographic Parameters (Qp/Qs,FAC,TAPSE) In ASD Patients With Hemodynamic Effect And Right Side Remodelling

Article History:

Received: 12-05-2025
Revised: 25-05-2025
Accepted: 02-06-2025
Published: 15-06-2025

Name of Author:
[1]Zahraa Hammoody Saleh Albaldawi,
[2]Prof. Muhannad Kudhair Al-Ghanimi

Affiliation: *[1,2]University of Babylon/ College of Medicine*

Abstract: ***Background:*** Atrial septal defect (ASD) is among the most prevalent congenital heart defects and may remain clinically silent until significance hemodynamic consequences develop. left unchecked, significant interatrial shunts precipitate chronic right-heart remodeling and pulmonary vascular pathology. ***Objective:*** correlation between electrocardiographic parameters (pwd,r/s ratio in v1,ibbb/cbbb) and echocardiographic parameters in asd pediatrics and adults patients with hemodynamic effect. ***Methods:*** A cross-sectional study was conducted with 73 patients with ASD (mean age 28.93 ± 21.38 years; range 1–70 years; 78.1% were female). All patients underwent full transthoracic and electrocardiographic and echocardiographic studies that included type and size of ASD, pulmonary-to-systemic flow ratio (Qp/Qs), indexed right ventricular (RV) dimensions, tricuspid annular plane systolic excursion (TAPSE), fractional area change (FAC), and indexed right atrial area (RAA). P-wave dispersion (PWD), frontal QRS axis, incomplete/complete right bundle branch block (iRBBB/cRBBB), crochet sign, R/S ratio in V1, T-wave inversion or age-adjusted positive RV strain pattern and qR pattern were evaluated by standard 12-lead ECG analysis. Echocardiographic and ECG parameters were correlated with major hemodynamic results such as pulmonary hypertension (PH), right-sided volume overload, and Eisenmenger syndrome. Analyses were stratified into pediatric (≤14 years) and adult groups. ***Results:*** The most common anatomical subtype was secundum ASD (93.2%) and large defects constituted 65.8%. Among the patients, 39.7% had a large left-to-right shunt (Qp/Qs >2.0) and 13.7% had right-to-left shunting. Right-sided volume overload was found in 86.3%, pulmonary hypertension in 42.5% (severe PH in 13.7%), and Eisenmenger syndrome in 11.0% of the cohort. Increased indexed RV dimensions, presence of iRBBB/cRBBB and older age were significantly associated with larger ASD size. In children, severe PH was mainly characterized by a significant increase in indexed RAA (26.23 ± 7.61 vs. 10.73 ± 3.15 cm^2/m^2; $p<0.001$). Severe PH was associated with both increased RAA ($p=0.017$) decreased TAPSE (0.92 ± 0.30 cm/m^2; $p=0.025$) in adults. Eseinmenger syndrome is associated, with increased RAA (16.63 ± 8.32 cm^2/m^2; $p=0.002$) and decreased FAC (36.25 ± 8.11%; $p=0.037$). Right sided volume overload in adults was also significantly associated with increased RAA ($p=0.005$). Also, Qp/Qs was significantly related to pulmonary hypertension, right volume overload and Eisenmenger syndrome (all $p<0.001$). For the ECG variables, R/S ratio in V1, TWI/positive RV strain pattern and qR pattern were significantly associated with severe PH and Eisenmenger physiology. PWD, frontal QRS axis, iRBBB/cRBBB and crochet sign had limited discriminatory value for individual hemodynamic outcomes. ***Conclusions:*** Echocardiography is the cornerstone in the assessment of the severity of ASD anatomically and

the hemodynamic burden. Indexed right atrial area was the most consistent marker of right-heart remodeling across large hemodynamic subgroups, while reduced TAPSE and FAC identified advanced RV dysfunction in severe pulmonary hypertension and Eisenmenger syndrome. ECG parameters were less sensitive in detecting early volume overload, but specific markers of RV pressure overload (R/S ratio in V1, RV strain pattern, and qR morphology) provided valuable complementary information in advanced disease. The combination of echocardiographic and selected ECG parameters may allow for better risk stratification and timely intervention before development of irreversible cardiopulmonary remodeling.

Keywords: Atrial septal defect; Echocardiography; Electrocardiography; Pulmonary hypertension; Eisenmenger syndrome; Right ventricular function; Right atrial area; Qp/Qs ratio.

INTRODUCTION

ASD is the second common congenital defect in the world but it is the most common defect in adult represents about 40% of congenital heart defects diagnosed in adults above age 40 years old [7][8]. It is a well-defined communication defect in the IAS between the LT atrium and RT strium, type one is Seccundum ASD which lies within fossa ovalis (75%) consists the most common one ,ASD primum(15-20%) is an inferior defect considered as a part of endocardial cushion defect ,sinus venosus ASD defect(5-10%) lies near the SVC or IVC entry,and coronary sinus defect is very rare (<1%). Patients were categorized according to their ASD size into 3 subgroups: small (<5mm in infants and toddlers) and up to 10 mm in older adolescent and adults, moderate (5-8 mm in infants and toddlers)and up to 10-20mm in adolescents and adults, large(8-10mm in early infancy)and >20 mm in older children and adults [1][2][8][10]. Large ASD leads to large left-to-right shunting and dilatation of the right sided with right-sided cardiac failure. But patients exhibiting large ASD may remain without clinical symptoms until age of 3 or 4 years despite significant RV dilatation because ASD has insidious onset with lack of obvious clinical symptoms in pediatrics and young adults(it is a disease with late diagnosis) so true assessment by successive echocardiography for measurement of RV dimensions and other indicators of a clinically significant left-to-right shunt is mandatory [1][5] . The overall prevalence of Atrial septal defect is 3.89 per 1000 children to 0.88 per 1000 adults which may be underestimated to silent and not discovered cases of ASD [8]. In Iraq, A study organized at Fallujah general hospital by Dagash et al.(2008-2011)detected atrial septal defects as the most dominant anomaly ,accounting for 72% of all isolated and combined cardiac defects [6]: Neonates (1–28 days): 54%, Infants (29 days–1 year): 34%, Toddlers and Young Children (13 months–5 years): 3%, Older Children (greater than 5 years): 3%, On the contrary, comparative data from surveillance registries made in Ramadi ,Baghdad and Basra exhibited that VSD was the most frequently discovered anomaly in those localities[6]. This study aims to Correlation between electrocardiographic parameters (Qp/Qs,FAC,TAPSE) and echocardiographic parameters in and pediatrics and adults patients with hemodynamic effect.

MATERIALS AND METHODS

Study Design

This is a cross sectional analytical study designed to evaluate the correlation between electrocardiographic (ECG) changes and echocardiographic findings in patients with atrial septal defect (ASD) divided into 4 clinical groups according to its hemodynamic effect:1) right-side volume overload ,2)pulmonary hypertension 3) Eseinmenger complex 4)ASD associated with severe PS in young children.

2.2 Study population

As a cross sectional study carried out in Merjan medical city and Babylon maternity and ediatrics hospital, Babil Health Directorate, Babylon ,Iraq, over 5 months from 1st December 2025 to 1st May 2026,73 ASD cases classified according to the new WHO classification 2025 into:

1- (1-17y) Underage children were (27) ASD mixed male and female cases.
2-(18-65y) Young adult were (44) ASD mixed male and female cases
3-(66-79y) Middle age was two case.
4-(80-99y) Elderly age was zero negative.
5-(100+years) long lived was zero negative.

All patients were recruited from Merjan Teaching Hospital after obtaining verbal consent and institutional approval.

2.3 Inclusion criteria

1-Children age (1-17y), adolescent (18-65y),old adult(66-79)and old age(80-99) diagnosed with ASD defect by Echocardiography divided into 4 groups according to its hemodynamic effect:

1) with right side volume overload(62 patients).
2) with PHT and with (31 patients).
3) Eseinmenger complex(6 patients).
4) Pediatric patients with ASD and severe PS (3 patients).

2-Patients with ASD Seccundum type ,Primum type and sinus venosus types.
3-Adults Patients and pediatrics patients who have ASD with regular rhythm.
4-Patients with atrial septal defect associated with partial anomalous pulmonary venous drainage which is common with sinus venosus ASD defect or seccundum type ASD near SVC type.

2.4 Exclusion criteria

1-Complex congenital heart diseases(TOF,Tricuspid atresia,VSD).
2-Patient with primary PHT.
3-Patient with left heart diseases like DCM.
4-Patient with previous cardiac surgery or device closure used for closing the defect.
5-Patients with known primary conduction abnormalities unrelated to ASD.
6-Poor quality images.
7-Patient with cardiac arrhythmia such as AF and atrial flutter.

2.5 Ethical Approval and Consent

The University of Babylon College of Medicine Ethics Committee gave this experiment ethical permission, and participant verbal and written agreement was acquired.

2.6 Questionnaire

The researcher and supervisor developed a self-constructed questionnaire form to gather information directly from the patients. The questionnaire focused on specific variables, such as sex (male and female) and age in years. Additionally, participants were assessed for weight and height measurements, and the body surface area (BSA) was calculated using the Mosteller formula. This data collection approach aims to gather essential information for the study and ensure accuracy in the assessment of relevant parameters.

$$BSA = \sqrt{(height * weight)} / 3600$$

.7 The apparatus:

2.7.1 Electrocardiogram (ECG)

Electrocardiographic Analysis ECG Acquisition and Standardization Resting 12-lead electrocardiograms (ECGs) were performed using a standard paper speed of 25 mm/s and a voltage calibration of 10 mm/mV. Under these settings, each small square (1 mm) represented 0.04 seconds (40 ms) horizontally and 0.1 mV vertically. Consequently, a large square (5 mm) corresponded to 0.2 seconds and 0.5 mV. Standardization of 10 mm in height and 5 mm in width, ensuring a consistent 1 mV/25 mm/s reference.

P wave duration was defined as the interval from the initial deflection (either positive or negative from the isoelectric line) to the point where the wave returned to the isoelectric line. Calculation of P-wave Dispersion: Maximum and Minimum P-wave Durations, (1) P maxi- mum defined as the longest P-wave duration among the 12 leads ;(2) Minimum P duration as the shortest –P- wave duration among the 12 ECG leads.

The final PWD value for each patient was determined by calculating the mean from five consecutive measurements(PWD=maximum P duration-minimum P duration). Furthermore, at the time of the ECG recording, all participants were confirmed to be free of medications known to influence intro-atrial conduction. [60].

Normal P duration(0.08-0.12 sec)
Normal QRS duration(0.06-0.10 sec) [38]
Sokolow criteria for RVH in adult is the sum of R amplitude in v1 and S amplitude in v6 is about 35mm.[38]

2.7.2 Pediatric ECG in ASD

Pediatric ECG can be obtained on the knowledge of the patient s clinical condition .ECG is a lab test. The evidence

of abnormality depends on normal value according to the age .Abnormal ECG doesn't mean that the patient really has a disease but may be due to pitfalls due to chest deformity (pectus excavatum/scoliosis),pediatric ECG is much complex than aduts because normal values vary significantly with age,heart rate and physical state [39].

Ventricular depolarization: is Prolonged(with prolonged QRS duration >80 ms for infants till age 8y children/ >90 ms for children and early adolescence)producing iRBBB (the most common in ASD),or cRBBB.

RBBB: Widened QRS duration with rapid initial deflection followed by slurred slower portion of QRS(it means rapid depolarization of LT ventricle followed by slower delayed contraction of RT ventricle)[39].

Table (2-1) Difference between iRBBB and cRBBB

iRBBB	cRBBB
rSr pattern	rSR or RSr pattern
Normal QRS<120 ms according to the age as in table 19.2	>=120 msec(above the upper limit for age associated with normal intial forces and terminal conduction delay directed anteriorly ,to the right and superior(wide slurred S in I,v5,v6,II,III,AVF)slurred R in v1,v2,avR,).
Occurs in normal children	Only in patients with right side volume overload(mostly with ASD seccundum)
Major conduction delay	Minor conduction delay

Table (2-2) Axis deviation [38]

Axis	Degree	QRS in lead I	QRS in lead AVF
normal	0 to +110(<40y) -30 to +90(>40y)	↑	↑
left	-30 to -90	↑	↓
right	+110 to +180	↓	Not necessary

2.8 Right ventricular Hypertrophy

Criteria for RVH include voltage and repolarization criteria ,there is normal right ventricular predominance in neonates, so in children:

1) an R wave amplitude in v1 is greater than the 98th percentile for age is very specific finding for RVH after the neonatal period in children.
2) The RV systolic pressure in children patients >2y with isolated pulmonary stenosis=(R wave amplitude in v1 mm*5).
3) Criteria for RVH: R wave >7 mm in v1,but when R wave in v1>20 mm it means that RV systolic pressure is at least equals to systemic pressure(specific for Eseinmenger Syndrome).
4) High R/S ratio in v1 according to the age in 98 percentile / specific for RVH ,but should occur with other indicators of RVH ,normal ranges according to the age in 98 precentile as in this table:

Table (2-3) R/S ratio in pediatric ECG according to the age

Age	R/S in v1 (amplitude)	R amplitude in v1	S amplitude in v1
1-3y	0.1-4.3	2-18	1-21
3-5y	0.03-2.7	1-18	2-22
5-8y	0.02-2	1-14	3-23
8-12y	0.02-1.9	1-12	3-25
12-16y	0.02-1.8	1-10	3-22

5) Sokolow criteria:Total voltage >10.5(upstroke R amplitude in v1+down deep S amplitude in v6).
6) T wave orientation changes with age. Normally T wave becomes (upstroke after birth till 7days age,(between 1week -adolescence 8y)it is inverted ,then it becomes upstroke in adolescent and adulthood after 8y.

a) Mild RVH: upright T wave after 7days age but normal amplitude.
b) Moderate RVH: increase R amplitude +upright T wave after 7 days till 8years.
c)Severe RVH: increase R amplitude+T inversion.
7)qR +tall R >10mm.
8)When there is RSR` ,R ` amplitude is large.
9)Right axis deviation as associated criteria that ensures the diagnosis of RVH [39].

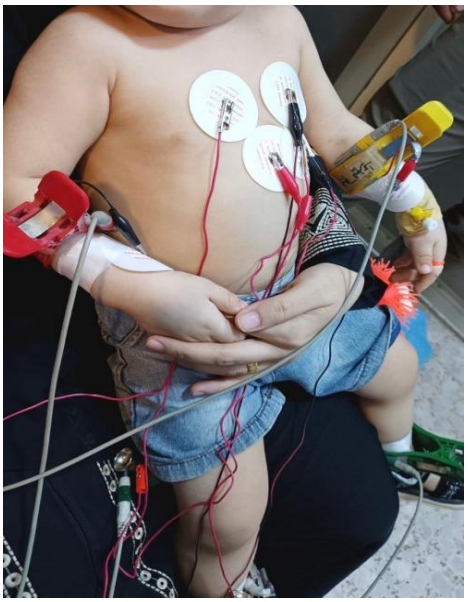


TABLE 19.2 Normal ECG Standards for Children by Age

	0–1 d	1–3 d	3–7 d	7–30 d	1–3 mo	3–6 mo	6–12 mo	1–3 y	3–5 y	5–8 y	8–12 y	12–16 y
Heart rate per minute	94–155 (122)	91–158 (122)	90–166 (128)	06–182 1 (149)	120–179 (149)	105–185 (141)	108–169 (131)	89–152 (119)	73–137 (109)	65–133 (100)	62–130 (91)	60–120 (80)
Frontal plane QRS axis (degrees)	59–189 (135)	64–197 (134)	76–191 (133)	0–160 7 (109)	30–115 (75)	7–105 (60)	6–98 (55)	7–102 (55)	6–104 (56)	10–139 (65)	6–116 (60)	9–128 (59)
PR lead II (s)	0.08–0.16 (0.107)	0.08–0.14 (0.108)	0.07–0.15 (0.102)	0.07–0.14 (0.100)	0.07–0.13 (0.098)	0.07–0.15 (0.105)	0.07–0.16 (0.106)	0.08–0.15 (0.113)	0.08–0.16 (0.119)	0.09–0.16 (0.123)	0.09–0.17 (0.128)	0.09–0.18 (0.135)
QRS duration, V5 (s)	0.02–0.07 (0.05)	0.02–0.07 (0.05)	0.02–0.07 (0.05)	0.02–0.08 (0.05)	0.02–0.08 (0.05)	0.02–0.08 (0.05)	0.03–0.08 (0.05)	0.03–0.08 (0.06)	0.03–0.07 (0.06)	0.03–0.08 (0.06)	0.04–0.09 (0.06)	0.04–0.09 (0.07)
P-wave amplitude, lead II	0.5–2.8 (1.6)	0.3–2.8 (1.6)	0.7–2.9 (1.7)	0.7–3.0 (1.9)	0.7–2.6 (1.5)	0.4–2.7 (1.6)	0.6–2.5 (1.6)	0.7–2.5 (1.5)	0.3–2.5 (1.4)	0.4–2.5 (1.4)	0.3–2.5 (1.4)	0.3–2.5 (1.4)
Q-wave amplitude, aVF	0.1–3.4 (1.0)	0.1–3.3 (1.0)	0.1–3.5 (1.1)	0.1–3.5 (1.2)	0.1–3.4 (0.9)	0–3.2 (0.9)	0–3.3 (1.0)	0–3.2 (0.9)	0–2.9 (0.6)	0–2.5 (00.6)	0–2.7 (0.5)	0–2.4 (0.4)
Q-wave amplitude, V6	0–1.7 (0.1)	0–2.2 (0.1)	0–2.8 (0.1)	0–2.8 (0.4)	0–2.6 (0.3)	0–2.6 (0.3)	0–3.0 (0.4)	0–2.8 (0.6)	0.1–3.3 (0.8)	0.1–4.6 (0.8)	0.1–2.8 (0.6)	0–2.9 (0.4)
R amplitude, V1	5–26 (13)	5–27 (15)	3–25	3–12 (12)	3–19 (10)	3–20 (10)	2–20 (9) (10)	2–18	1–18 (8) (8)	1–14 (7)	1–12 (5)	1–10 (4)
S amplitude, V1	1–23 (8)	1–20 (9)	1–17 (7)	0–11 (4)	0–13 (5)	0–17 (6)	1–18 (7)	1–21 (8)	2–22 (10)	3–23 (12)	3–25 (12)	3–22 (11)
R amplitude, V6	0–12 (4)	0–12 (5)	1–12 (5)	3–16 (8)	5–21 (12)	6–22 (13)	6–23 (13)	6–23 (13)	8–25 (15)	8–26 (16)	9–25 (16)	7–23 (14)
S amplitude, V6	0–10 (4)	0–9 (3)	0–10 (4)	0–10 (3)	0–7 (3)	0–10 (3)	0–8 (2)	0–7 (2)	0–6 (2)	0–4 (1)	0–4 (1)	0–4 (1)
R/S ratio, V1	0.1–9.9 (2.2)	0.1–6 (2.0)	0.1–9.8 (2.8)	1.0–7.0 (2.9)	0.3–7.4 (2.2)	0.1–6.0 (2.3)	0.1–4.0 (1.8)	0.1–4.3 (1.4)	0.03–2.7 (0.9)	0.02–2.0 (0.8)	0.02–1.9 (0.6)	0.02–1.8 (0.5)
R/S ratio, V6	0.1–9 (2)	0.1–12 (3)	0.1–10 (2)	0.1–12 (4)	0.2–14 (5)	0.2–18 (7)	0.2–22 (8)	0.3–27 (10)	0.6–30 (11)	0.9–30 (12)	1.5–33 (14)	1.4–39 (15)

All values are 2nd percentile to 98th percentile (mean). All amplitudes of waves are given in millimeters at full standardization, that is, 1 mm = 10 mV.
Derived from percentile charts in Davignon A, Rautaharju P, Boisselle E, et al. Normal ECG standards for infants and children. Pediatr Cardiol. 1979;1:123–152.

Figure (2-1) ECG machine and ECG connection in children
Figure (2-2) Normal ECG standards for children by age [39].

2.9 ECG cases of ASD patients :

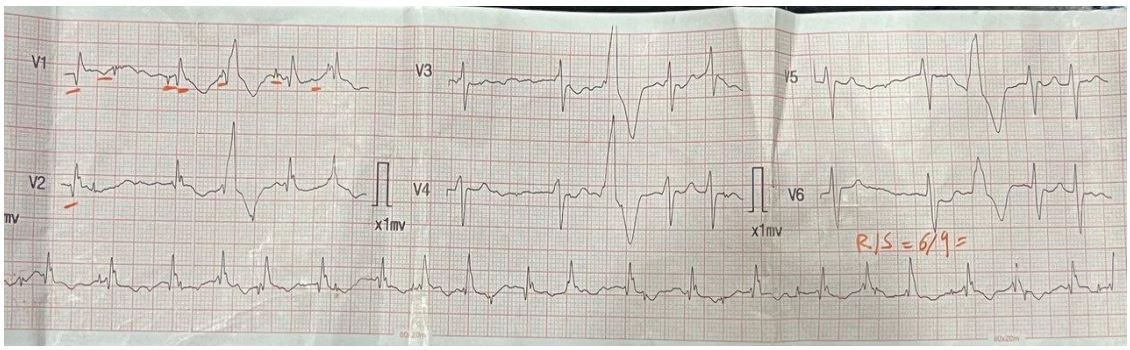
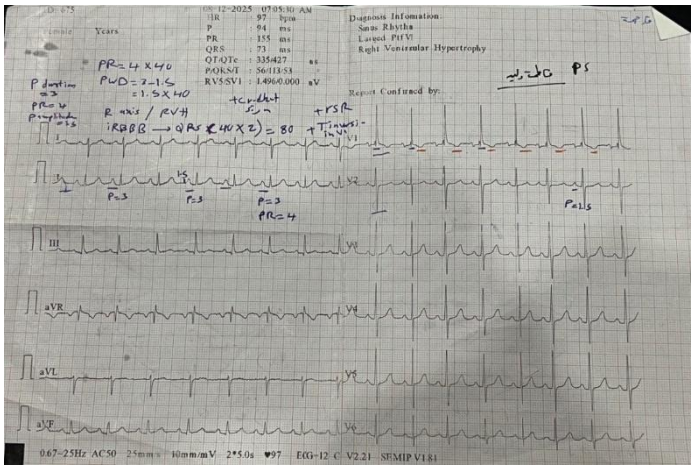


Figure (2-3) Case 1: ECG in adult patient age 60y with ASD seccundum with Eseinmenger complex showing (R amplitude =15mm,R/S>1 in v1,qR pattern in v1,cRBBB,T inversion in v1-v3).



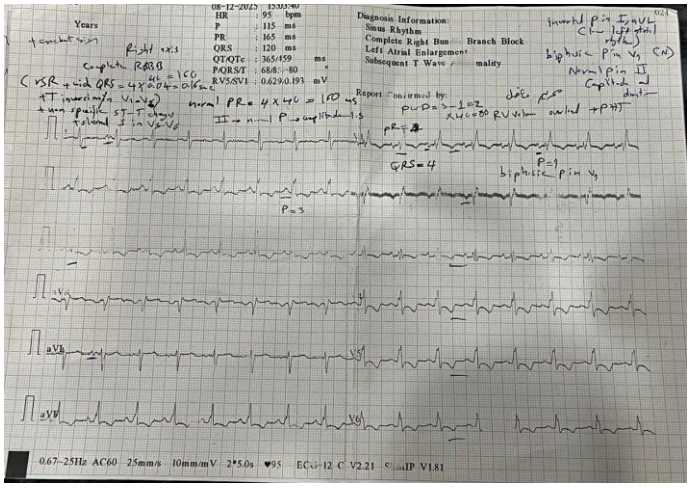


Figure (2-4) Case 2: ECG of a 38y old female with ASD seccundum with partial anomaloud pulmonary venous drainage and mild PHT,(cRBBB,RVH with R`amplitude in v1 =7mm).

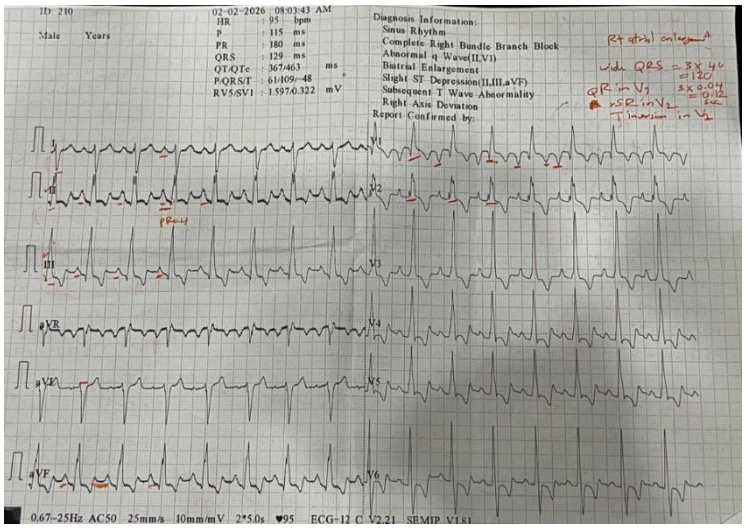


Figure (2-5) case 3: ECG of 10y old female with ASD seccundum and severe PS showing (cRBBB,RVH by qR , R`=7mm,R/S ratio in v1=5.5 and T inversion,also there is cRBBB in v1)

Figure (2-6) Case 4: ECG of 6y old female child with small ASD seccundum,severe PS showing (R in v1=18mm,Irbbb,T inversion)

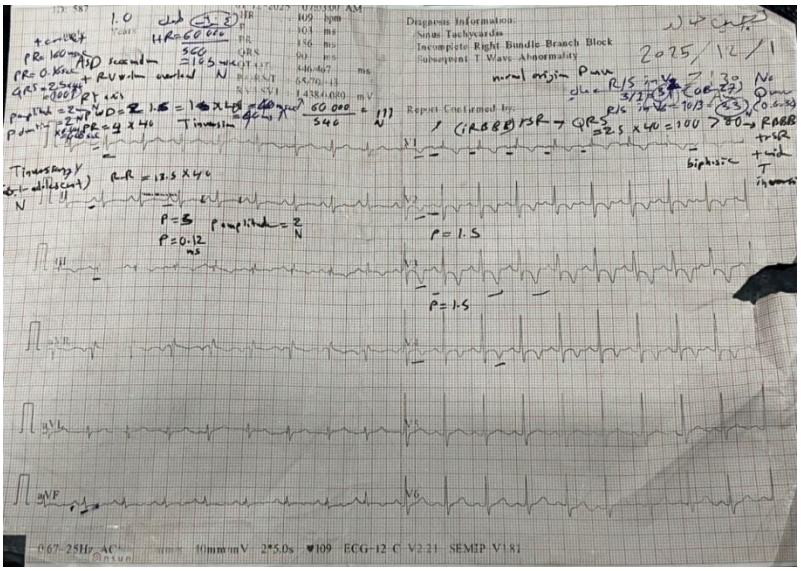


Figure (2-7) Case 5:
ECG of 4y female child with large ASD seccundum and right side volume overload, showing iRBBB and crochet sign.

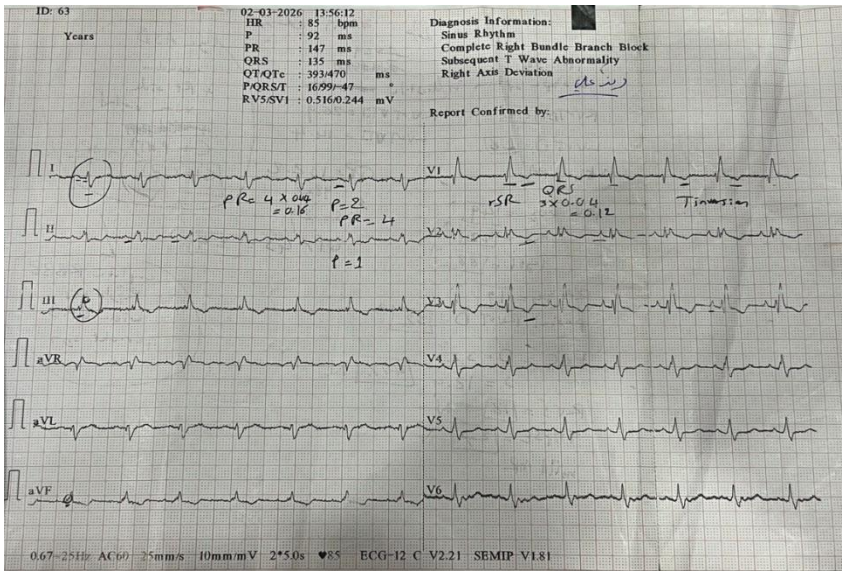


Figure (2-8) Case 6: ECG of 29y old female with large ASD seccundum with right side volume overload showing cRBBB and crochet sign.

2.10 Echocardiography

Subcostal window is the preferred view(showing superior SVC and inferior IVC rims) because the IAS is perpendicular to the echo signal, it is suitable for Visualization of ASD, adjustment of size of ASD, direction of flow. By echocardiography we identify associated abnormalities(MVP with Seccundum/cleft MV with primum / partial anomalous right pulmonary veins drainage with sinus venosus ASD and can be associated with ASD seccundum, hypermobile interatrial septum). The apical 4chamber may be useful for visualization of ASD showing (atrioventricular rim and posterior inferior rim) but the parallel alignment of atrial septum to echo signal may lead to artifact dropout which is misleading .The short axis can be complementary showing retroaortic rim . Colour Doppler can confirm ASD and detect the direction of the shunt ,it can adjust the ASD size as the width of colour doppler across IAS can be measured (larger width of colour doppler means large ASD).Pulse wave doppler can be used for assessment of lower velocity flow across the IAS and can be the base in QP/QS calculation .CWD can be used in the assessment of high velocity to assess the gradient across the IAS determining restrictive ASD.

D shape of septum during diastole and D shape septum during systole and diastole in moderate to severe PHT,D shape during systole in severe pulmonary stenosis and PA enlargement.

The right sided heart dimensions are typically obtained using focused apical 4chamber without foreshortening (The apex of RV is visible). for adult measurements:(RV basal D >=42mm,Mid diameter >=35mm,longitudinal length >=82mm,RA area >18 cm2)at end-diastole is considered dilated, according to ASE guidelines for adult(2025).The distal RVOT(at the level where the PV inserts) at end diastole >27mm is considered dilated.RV wall thickness can be measured during diastole in subcostal view by M mode echocardiography (>5mm) is considered RV hypertrophy occurs in conditions with pressure overload(pulmonary stenosis ,severe pulmonary hypertension) [40] [41].

Table (2-5) Guidelines for the Echocardiographic assessment of the Right Heart in Adults and Special Considerations in Pulmonary hypertension: Recommendations from the American Society of Echocardiography(2025)

Variable	Normal Reference Value	Mild	Moderate	Severe
RA area(cm2)	<19	19-22	22-24	>24
RV basal D(cm)	<4.1	4.1-4.4	>4.4,<=4.9	>4.9
RV mid D(cm)	<3.5	3.5-3.8	>3.8,<=4.2	>4.2
RV length(cm)	<8.2	8.2-8.9	>8.9,<=9.6	>9.6
RV plax D(cm)	<3.3	3.3-3.5	>3.5,<=3.9	>3.9
RVOT (psax)distal D(cm)	<2.9	2.9-3.0	>3,<=3.3	>3.3
RVWT(cm)	<0.5	0.5-0.7	>0.7,<=0.9	>0.9
RV end systolic area(cm2)	<14	14-16	>16,<=19	>19
RV end systolic area(cm2/m2)	<8	8-9	>9,<=11	>11
RV end diastolic area(cm2)	<25	25-28	>28,<=32	>32
RV end diastolic area(cm2/m2)	<14	14-15	>15,<=17	>17
Tapse(cm)	>17	<=17,>=13	<13,>10	<=10
RVS`	>9.5	<=9.5,>=7.2	<7.2,>5	<=5
Fac	>35	<=35,>29	<=29,>22	<=22
TRV max,m/s	<2.8	>=2.8,<=3.1	>=3.2,<=3.5	>=3.6
RVSP	<=34	>=35,<=49	>=50,<=69	>=70
RAP	>=0,<5	>=5,<10	>=10,<15	>=15

Table 1. Normal RV dimensions[23,24]

BSA (m^2)	RVOTd (cm)	RVOTs (cm)	RVEDb (cm)	RVESb (cm)	RA length (cm)	Length (cm)	RVEDL (cm)	RVESL (cm)	RVEDb (basal RV, cm)	RVEDm (mid-RV, cm)	RVEDa (cm^2)	RVESa (cm^2)
0.1[24]	0.53 (0.19-0.88)	0.28 (0.00-0.61)	0.91 (0.36-1.46)	0.91 (0.38-1.44)	1.00 (0.62-1.38)	50[23]	2.3 (1.3-3.3)	1.7 (1.2-2.3)	1.3 (0.7-1.9)	1.2 (0.6-1.7)	2.5 (1.5-4.0)	1.4 (0.8-2.4)
0.15[24]	0.62 (0.28-0.97)	0.39 (0.05-0.72)	1.06 (0.51-1.61)	1.00 (0.47-1.53)	1.15 (0.77-1.53)	55[23]	2.5 (1.5-3.5)	1.8 (1.3-2.4)	1.4 (0.8-2.0)	1.2 (0.7-1.8)	2.9 (1.8-4.7)	1.6 (1.0-2.8)
0.2[24]	0.71 (0.37-1.06)	0.49 (0.16-0.82)	1.20 (0.65-1.75)	1.09 (0.56-1.62)	1.29 (0.91-1.67)	60[23]	2.6 (1.6-3.7)	1.9 (1.4-2.6)	1.5 (0.9-2.1)	1.3 (0.7-1.9)	3.3 (2.1-5.4)	1.9 (1.1-3.2)
0.25[24]	0.80 (0.45-1.14)	0.58 (0.25-0.92)	1.33 (0.78-1.88)	1.17 (0.64-1.70)	1.43 (1.05-1.80)	65[23]	2.8 (1.8-3.9)	2.0 (1.5-2.7)	1.6 (1.0-2.2)	1.4 (0.8-2.0)	3.8 (2.4-6.1)	2.2 (1.3-3.6)
0.3[24]	0.87 (0.53-1.22)	0.67 (0.34-1.01)	1.45 (0.90-2.00)	1.25 (0.72-1.78)	1.55 (1.17-1.93)	70[23]	3.0 (1.9-4.0)	2.1 (1.6-2.9)	1.7 (1.0-2.3)	1.5 (0.9-2.1)	4.3 (2.7-6.8)	2.4 (1.5-4.1)
0.35[24]	0.95 (0.60-1.29)	0.76 (0.42-1.09)	1.56 (1.01-2.11)	1.33 (0.80-1.86)	1.66 (1.29-2.04)	75[23]	3.2 (2.1-4.2)	2.2 (1.7-3.0)	1.8 (1.1-2.4)	1.6 (1.0-2.2)	4.8 (3.0-7.6)	2.7 (1.6-4.6)
0.4[24]	1.02 (0.67-1.36)	0.84 (0.50-1.17)	1.67 (1.12-2.22)	1.40 (0.87-1.93)	1.77 (1.39-2.15)	80[23]	3.3 (2.3-4.4)	2.4 (1.7-3.2)	1.9 (1.2-2.5)	1.7 (1.0-2.3)	5.3 (3.4-8.4)	3.1 (1.8-5.1)
0.45[24]	1.08 (0.74-1.43)	0.91 (0.58-1.24)	1.77 (1.22-2.32)	1.47 (0.94-2.00)	1.87 (1.49-2.25)	85[23]	3.5 (2.4-4.6)	2.5 (1.8-3.3)	2.0 (1.3-2.6)	1.7 (1.1-2.4)	5.9 (3.7-9.2)	3.4 (2.0-5.6)
0.5[24]	1.14 (0.80-1.49)	0.98 (0.64-1.31)	1.86 (1.31-2.41)	1.54(1.01-2.07)	1.97 (1.59-2.34)	90[23]	3.7 (2.6-4.8)	2.6 (1.9-3.5)	2.1 (1.4-2.8)	1.8 (1.2-2.5)	6.4 (4.1-10.1)	3.7 (2.2-6.1)
0.6[24]	1.25 (0.91-1.60)	1.10 (0.76-1.43)	2.02 (1.47-2.57)	1.66 (1.13-2.19)	2.13 (1.75-2.51)	95[23]	3.9 (2.8-5.0)	2.7 (2.0-3.7)	2.1 (1.4-2.9)	1.9 (1.2-2.5)	7.0 (4.5-11.0)	4.1 (2.5-6.7)
0.7[24]	1.35 (1.00-1.69)	1.20 (0.87-1.53)	2.16 (1.61-2.71)	1.78 (1.24-2.31)	2.27 (1.89-2.65)	100[23]	4.0 (2.9-5.2)	2.8 (2.1-3.8)	2.2 (1.5-3.0)	2.0 (1.3-2.6)	7.7 (4.9-11.9)	4.4 (2.7-7.3)
0.8[24]	1.43 (1.08-1.77)	1.29 (0.95-1.62)	2.28 (1.72-2.83)	1.88 (1.35-2.41)	2.39 (2.01-2.77)	105[23]	4.2 (3.1-5.4)	3.0 (2.2-4.0)	2.3 (1.6-3.1)	2.1 (1.4-2.7)	8.3 (5.4-12.8)	4.8 (2.9-7.9)
0.9[24]	1.50 (1.15-1.85)	1.36 (1.02-1.69)	2.38 (1.82-2.93)	1.97 (1.44-2.50)	2.48 (2.10-2.86)	110[23]	4.4 (3.3-5.5)	3.1 (2.3-4.2)	2.4 (1.7-3.2)	2.1 (1.5-2.8)	8.9 (5.8-13.8)	5.2 (3.2-8.5)
1[24]	1.56 (1.21-1.91)	1.41 (1.08-1.75)	2.46 (1.91-3.01)	2.06 (1.53-2.59)	2.56 (2.18-2.94)	115[23]	4.6 (3.4-5.7)	3.2 (2.4-4.4)	2.5 (1.7-3.3)	2.2 (1.5-2.9)	9.6 (6.3-14.8)	5.6 (3.4-9.1)
1.1[24]	1.61 (1.27-1.96)	1.46 (1.13-1.79)	2.53 (1.98-3.08)	2.13 (1.60-2.67)	2.62 (2.25-3.00)	120[23]	4.8 (3.6-5.9)	3.4 (2.5-4.5)	2.6 (1.8-3.4)	2.3 (1.6-3.0)	10.3 (6.7-15.8)	6.0 (3.7-9.8)
1.2[24]	1.65(1.31-2.00)	1.49 (1.16-1.83)	2.59 (2.04-3.14)	2.20 (1.67-2.74)	2.67 (2.30-3.05)	125[23]	4.9 (3.7-6.1)	3.5 (2.6-4.7)	2.7 (1.9-3.5)	2.4 (1.7-3.1)	11.0 (7.2-16.8)	6.4 (3.9-10.4)
1.3[10]	1.69 (1.35-2.04)	1.52 (1.19-1.85)	2.64 (2.09-3.19)	2.27 (1.74-2.80)	2.71 (2.33-3.09)	130[23]	5.1 (3.9-6.3)	3.7 (2.7-4.9)	2.8 (2.0-3.6)	2.5 (1.8-3.2)	11.7 (7.7-17.9)	6.8 (4.2-11.1)
1.4[10]	1.72 (1.38-2.07)	1.54 (1.21-1.87)	2.69 (2.13-3.24)	2.32 (1.79-2.86)	2.74 (2.36-3.12)	140[23]	5.5 (4.2-6.7)	4.0 (3.0-5.3)	3.0 (2.2-3.8)	2.6 (1.9-3.4)	13.3 (8.8-20.1)	7.7 (4.8-12.5)
1.5[10]	1.75 (1.41-2.10)	1.55 (1.22-1.89)	2.73 (2.18-3.28)	2.38 (1.85-2.91_	2.77 (2.39-3.15)	150[23]	5.8 (4.6-7.0)	4.3 (3.2-5.7)	3.1 (2.3-4.0)	2.8 (2.0-3.5)	14.8 (9.8-22.3)	8.7 (5.4-14.0)
1.6[10]	1.78 (1.44-2.13)	1.56 (1.23-1.89)	2.77 (2.22-3.33)	2.43 (1.89-2.96)	2.79 (2.41-3.17)	160[24]	6.2 (4.9-7.4)	4.6 (3.5-6.1)	3.3 (2.5-4.2)	3.0 (2.2-3.7)	16.5 (11.0-24.7)	9.7 (60-15.5)
1.7[10]	1.81 (1.46-2.15)	1.57 (1.24-1.90)	2.82 (2.27-3.37)	2.47 (1.94-3.00)	2.81 (2.43-3.19)	170[24]	6.5 (5.2-7.8)	4.9 (3.7-6.5)	3.5 (2.6-4.4)	3.1 (2.3-3.9)	18.2 (12.2-27.1)	10.7 (6.7-17.1)

RVOTd: Parasternal long axis, m-mode echocardiographic measurement at end diastole
RVOTs: Parasternal long axis, m-mode echocardiographic measurement at end systole
RVEDb: Apical 4-chamber, distance between the RV free wall and septum at end diastole, distal to the TV annulus
RVESb: Apical 4-chamber, distance between the RV free wall and septum at end systole, distal to the TV annulus
RA: Apical 4-chamber, max left to right dimensions during systole
RVEDL: Apical 4-chamber, RV mid-cavity length at end diastole, from the midpoint of the TV annulus to apex
RVESL: Apical 4-chamber, RV mid-cavity length at end systole, from the midpoint of the TV annulus to apex
RVEDb: Apical 4-chamber, distance between the RV free wall and septum at end diastole, distal to the TV annulus
RVEDm: Apical 4-chamber, distance between the RV free wall and septum at end diastole, mid-third of RV at the level of the LV papillary muscle
RVEDa: Apical 4-chamber, RV end-diastolic area
RVESa: Apical 4-chamber, RV end-systolic area

Values: mean (±2 SD range).

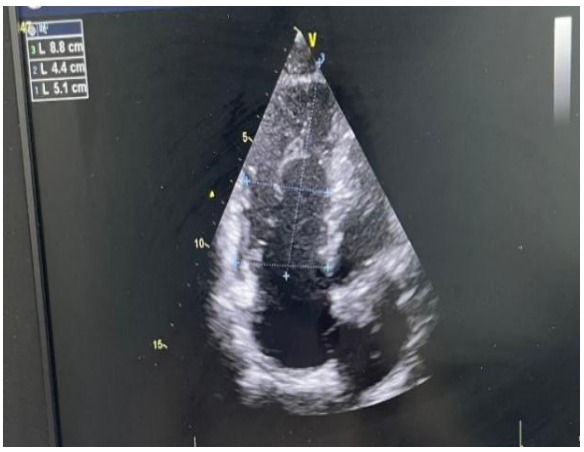
BSA, body surface area; RA, right atrium; RV, right ventricle; RVED, right ventricular end diastolic; RVEDa, right ventricular end-diastolic mid-ventricular area; RVEDb, right ventricular end-diastolic basal width; RVEDL, right ventricular end-diastolic length; RVEDm, right ventricular end-diastolic mid-ventricular width; RVES, right ventricular end systolic; RVESa, right ventricular end-systolic mid-ventricular area; RVESb, right ventricular end-systolic basal width; RVESL, right ventricular end-systolic length; RVOT, right ventricular outflow tract; RVOTd, RV outflow tract (diastole); RVOTs, RV outflow tract (systole).

Figure (2-9) RV and RA dimensions according to BSA in children (1-15y) [40].

Figure (2-10) VIVID9 GE Probe with a 3.5 MHz transducer. Made in Norway by GE Vingmed Ultrasound, 2012.

Figure (2-11) GE healthcare vivid E9 with 6MHz or 12MHz cardiac sector transducers using EchoPac software

Figure (2-12) RV dimensions at end diastole:1-RV base 51 cm D.2-RV mid D 44 cm.3-RV length 88 cm.



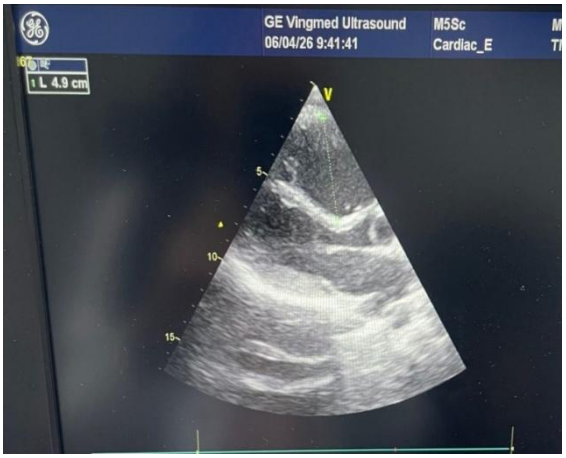


Figure (2-13) Proximal RVOT measurement at end-diastole (PLAX)view: Proximal RVOT D=49mm.

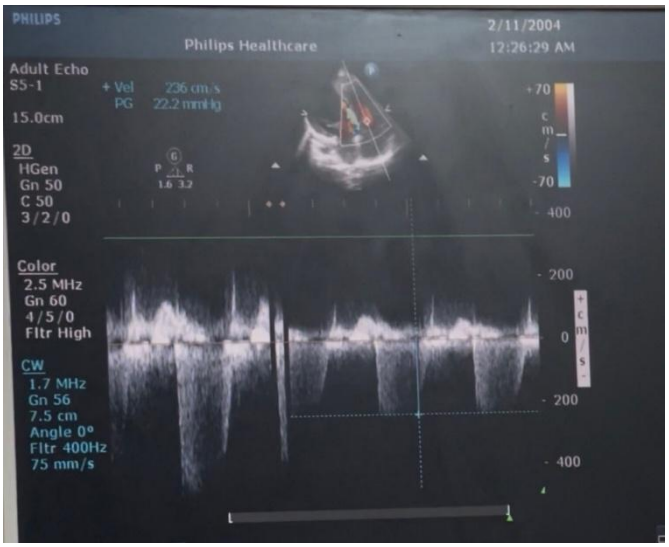


Figure (2-14) TRPG=23 mmHg(normal).
PASP can be estimated by Bernoulli equation(4*(TR velocity)2+RTatrial pressure.TR velocity can be measured by Doppler interrogation of TR velocity ,RAP can be calculated according to dilatation and collapsibility of IVC.

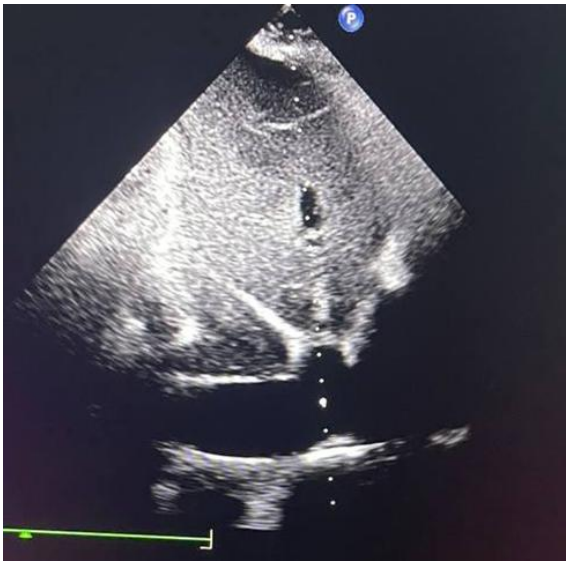


Figure (2-15) IVC dilated and poorly collapsible (RA pressure=15mmHg).

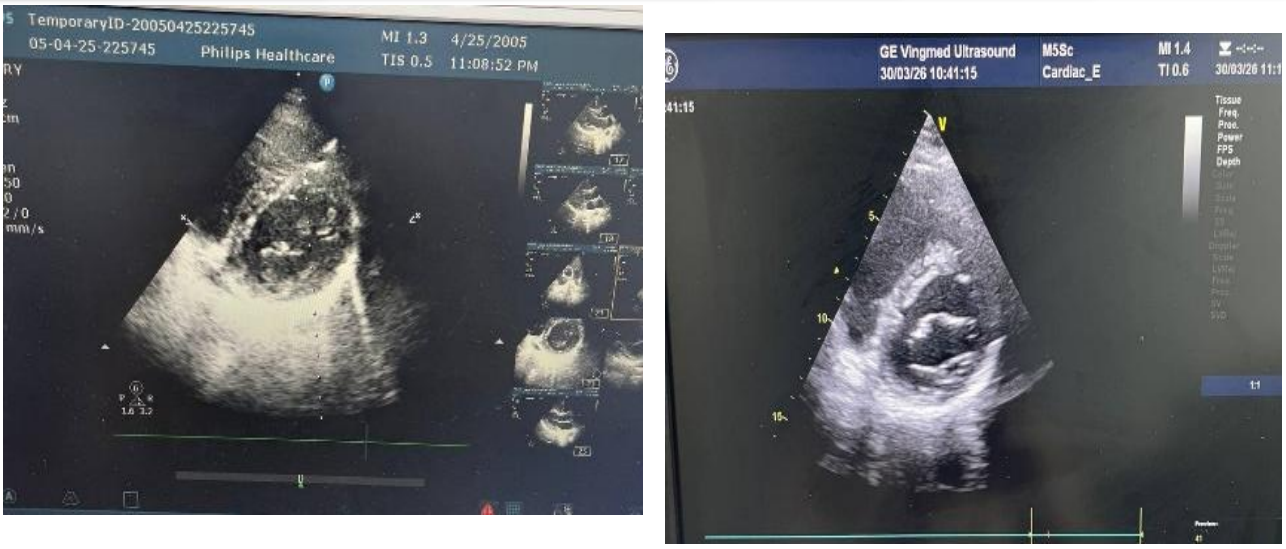


Figure (2-16) a) D shape in diastole , b) D shape in systole.

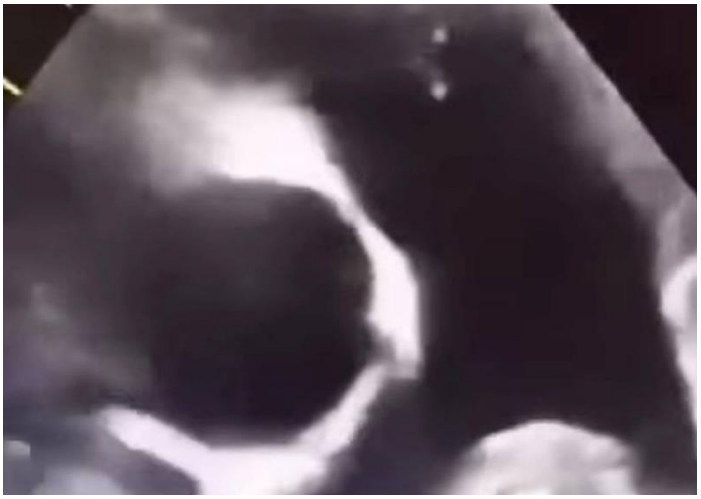


Figure (2-17) Dilated main PA and its branches (PSAX),PA d=29mm.

TAPSE

Tricuspid annular excursion and rvs` are measure of rv longitudinal function .it means the systolic function of deep fibers of rv and correlate with rvef. TAPSE can be measured by aligning the cursor parallel to rv annular plane systolic excursion .m mode then can be activated to measure the displacement of annular plane.(tapse<1.7 cm/abnormal in adults), in pediatrics tapse can be measured according to the body surface area. [40] [41].

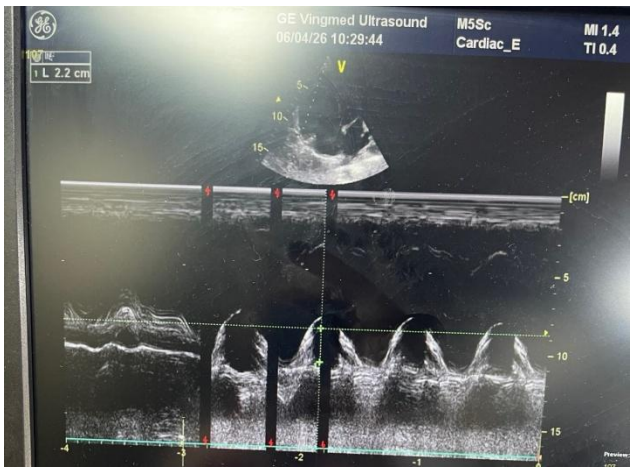


Figure (2-18) TAPSE=22 mm by M mode on lateral annulus of lateral wall of RV.

FAC(fractional area change):is the determinant of global RV systolic function ,it gives approximately results that are similar to the results of RVEF measured by cardiac MRI.FAC=(RVEDA-RVESA)/EVEDA*100%,RVEDA and RVESA can be measured by tracing the endocardial border and calculating the RV area in diastole(the largest) and in systole(the smallest) in focused apical 4 chamber view. A value of less than 35% is considered abnormal.

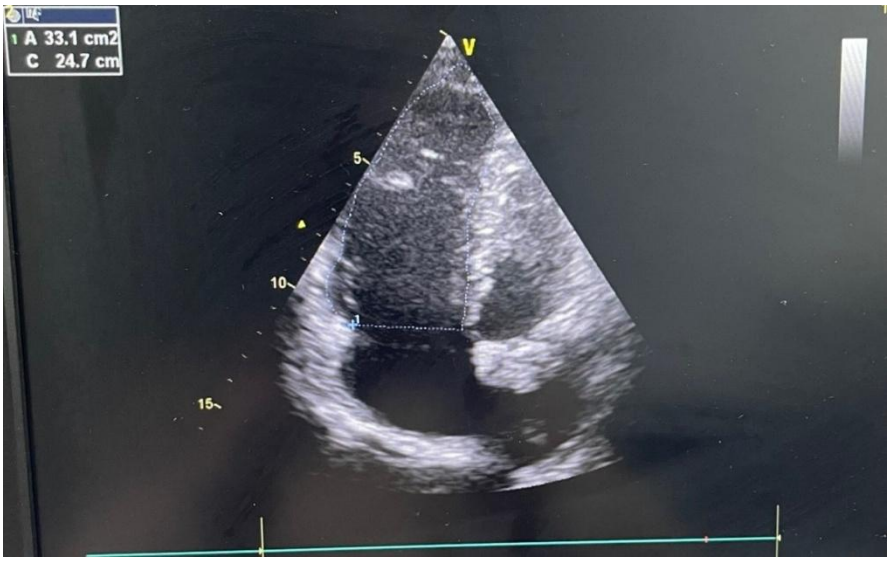


Figure (2-19) RVEDA=33.1 cm2 at end-diastole.

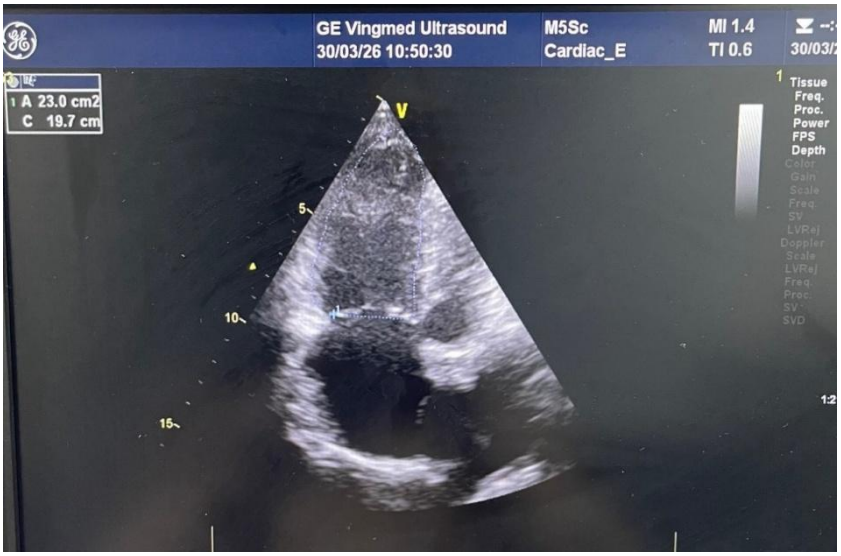


Figure (2-20) RVESA =23cm2 at end systole.

Another non invasive Doppler measurement is the assessment of QP/QS which can be estimated according to the equation:

- Systemic Stroke Volume (SV):

SV systemic=0.785× (LVOT diameter) 2×AV VTI

- Pulmonary Stroke Volume (SV):

SV pulmonary=0.785× (RVOT diameter) 2×PV VTI

- Shunt Fraction:

Qp /Qs=SV pulmonary / SV systemic

Pulmonary flow can be assessed by TTE (PSAX) by measuring RVOT diameter and then assessing VTI by using pulse wave above the pulmonary valve,aortic flow can be assessed by TTE by measurement of LVOT diameter in PLAX ,1/2 cm distance from the aortic annulus ,and VTI calculation by pulse wave on LVOT in apical 5 chamber view.

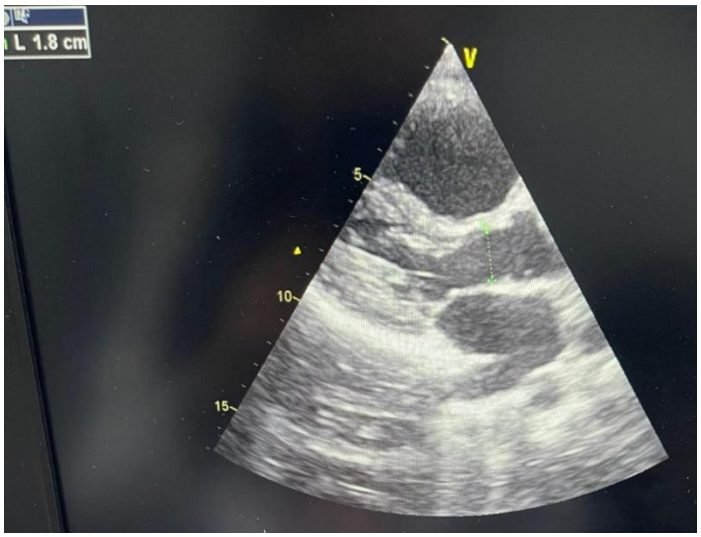


Figure (2-21) Measurement of LVOT diameter =1.8cm at mid systole(PLAX).

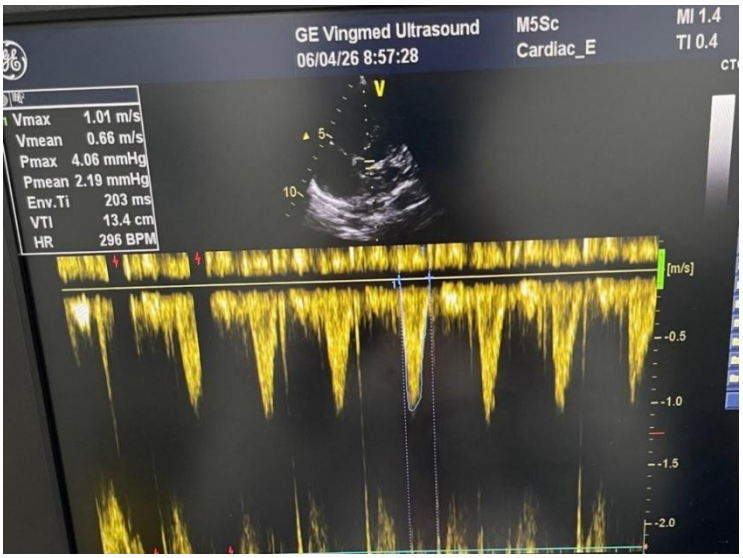


Figure (2-22) Measurement of LVOT VTI=13.4 cm (apical 5 chamber) .

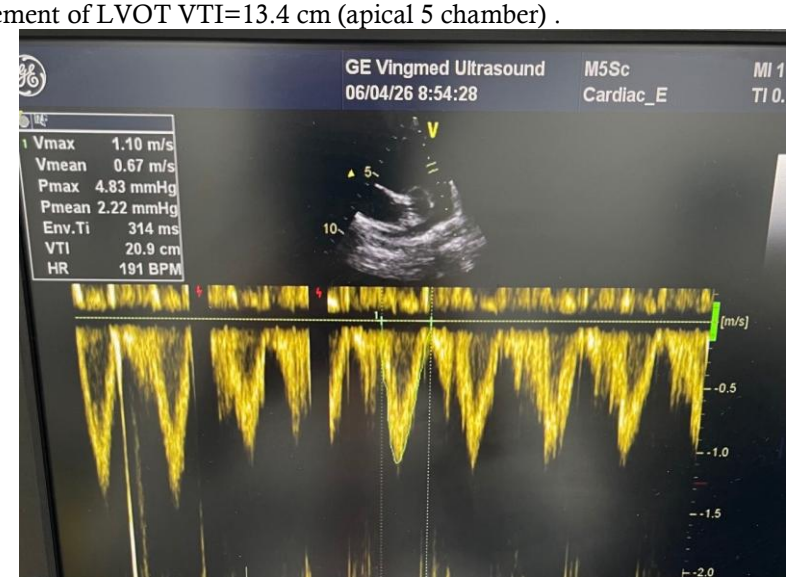


Figure (2-23) Measurement of RVOT VTI =20.9cm (PSAX).

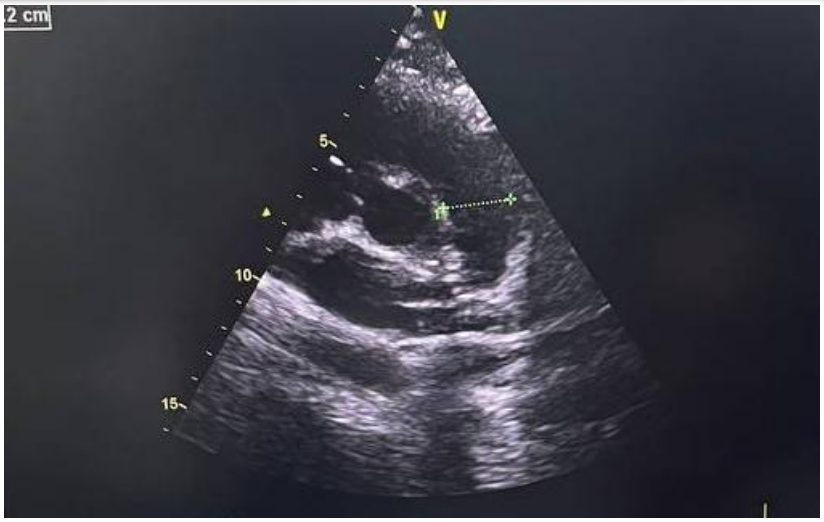


Figure (2-24) Measurement of RVOT diameter=2.2cm at mid systole(PSAX).

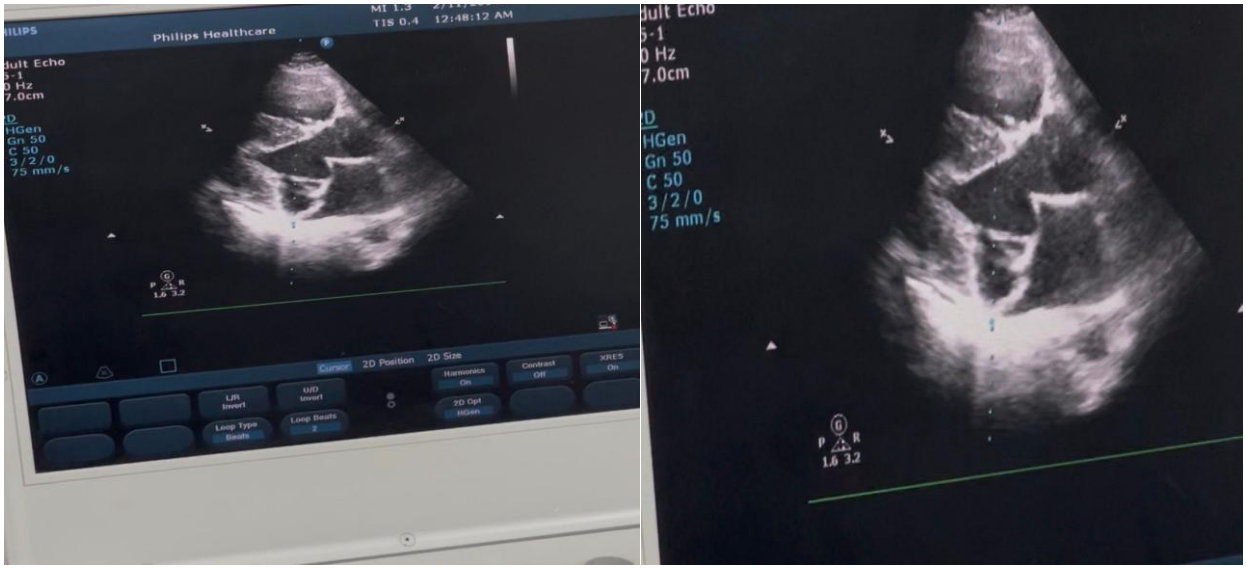


Figure (2-25) MVP can be associated with ASD seccundum, some of them have MR (MVP of bileaflet valve).

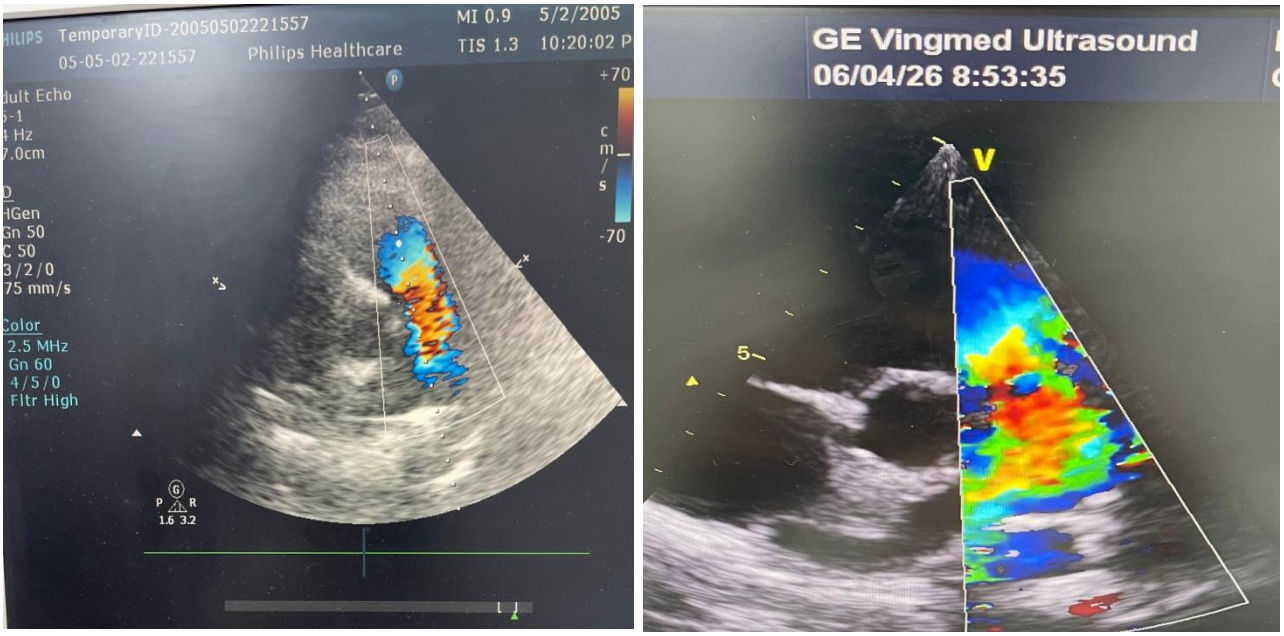


Figure (2-26) ASD especially Seccundum type can be associated with mild to moderate pulmonary stenosis a-mild pulmonary stenosis (peak gradient=20mmHg) b-moderate pulmonary stenosis(peak gradient =40mmHg)Due to overflow of ASD, it is not true pulmonary stenosis.

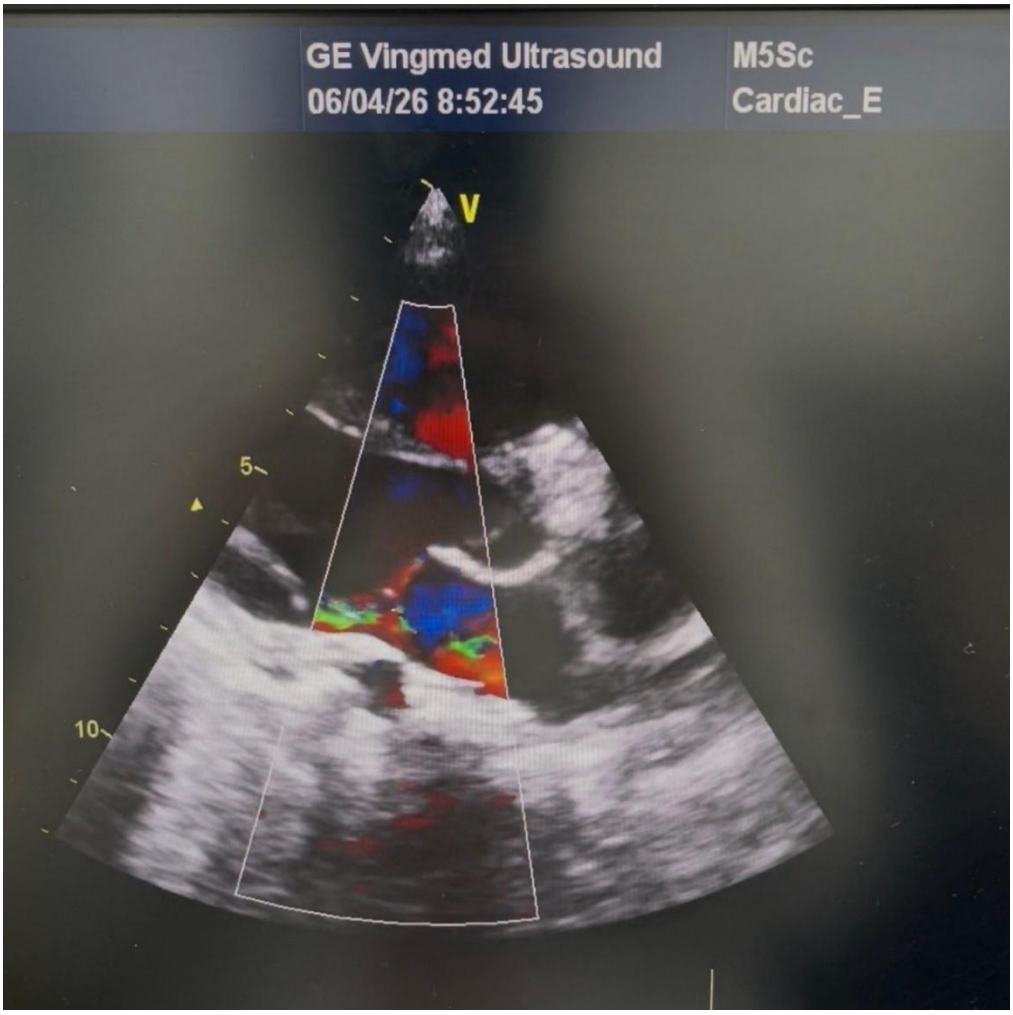


Figure (2-27) ASD seccundum associated with MVP and moderate eccenteric MR posteriorly.

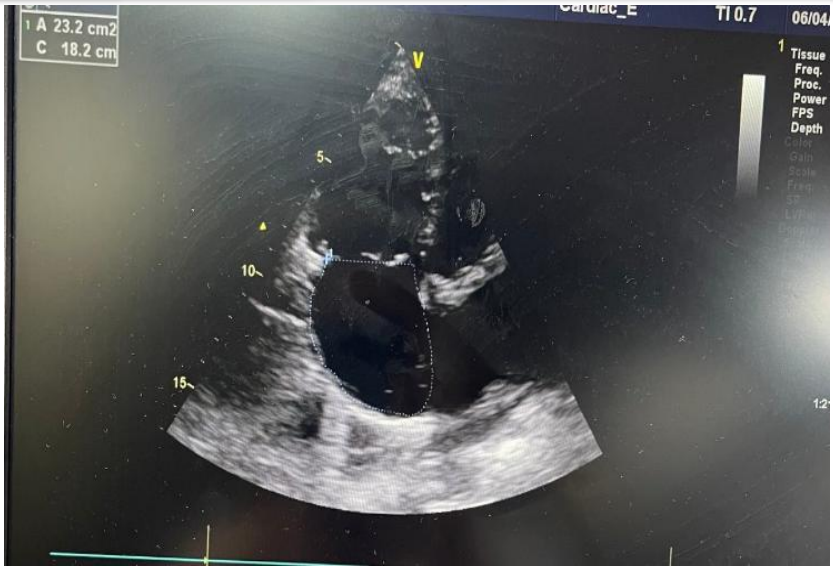


Figure (2-28) Measurement of RA area at end systole (RA area=23 cm2/dilated).

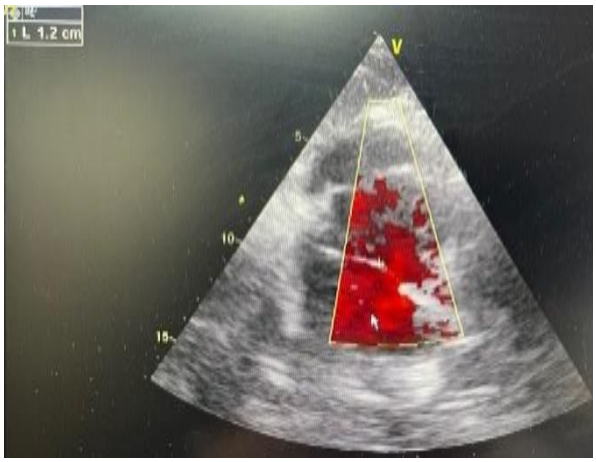
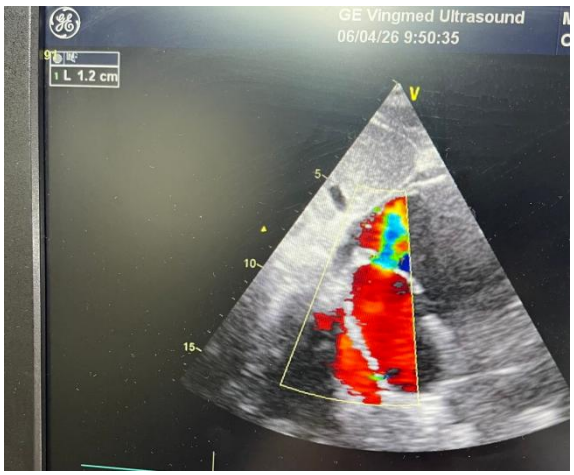


Figure (2-29) Subcostal bicaval view with SVC and IVC rims: A)large central ASD seccundum (12mm) B)large 1st ASD seccundum near IVC (12mm),2nd ASD seccundum Is central .C)large ASD seccundum near IVC (18mm).



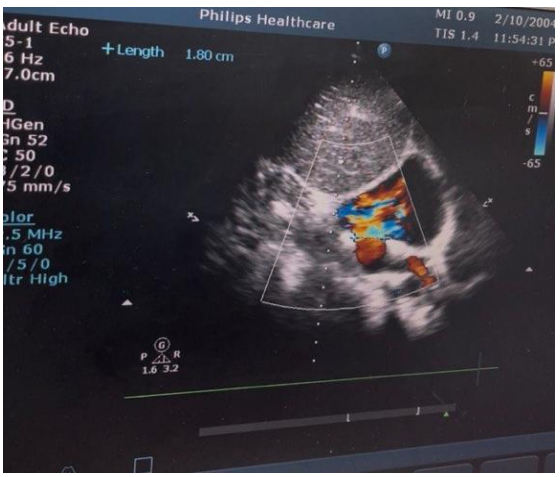


Figure (2-30) ASD seccundum moderate in size near IVC (10mm*9mm). ASD seccundum is central.

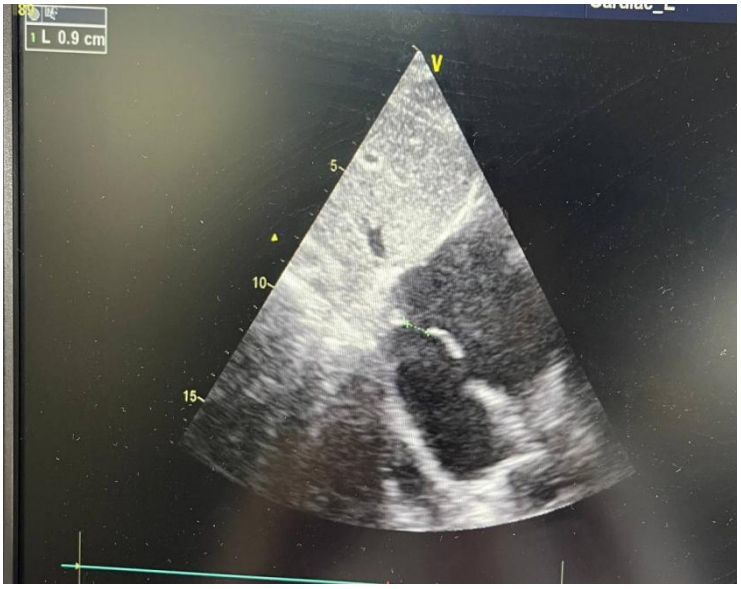


Figure (2-31) Large ASD seccundum size in apical 4 chamber (20mm*18mm).

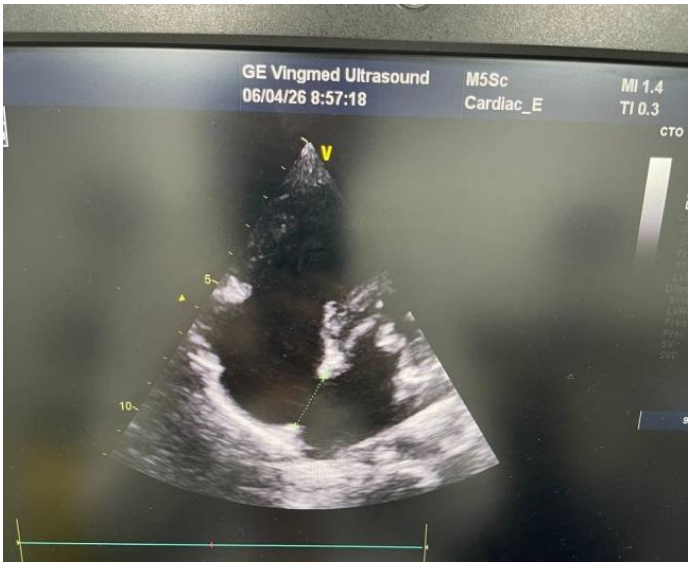
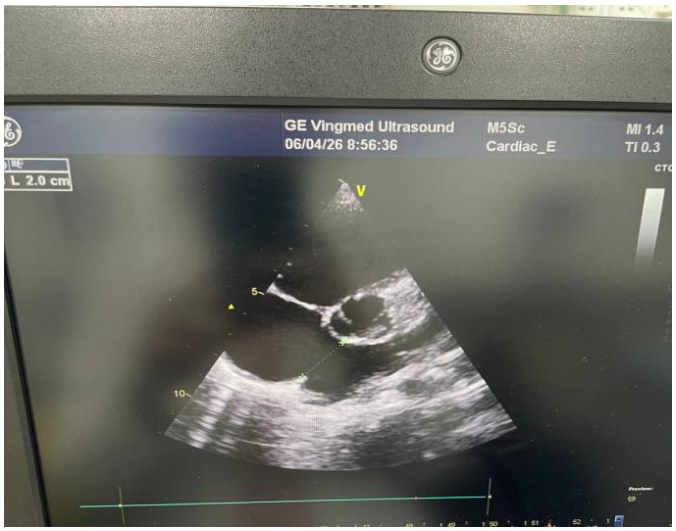


Figure (2-32) Very large ASD seccundum (20mm*18mm) at short axis aortic level with retroaortic rim.



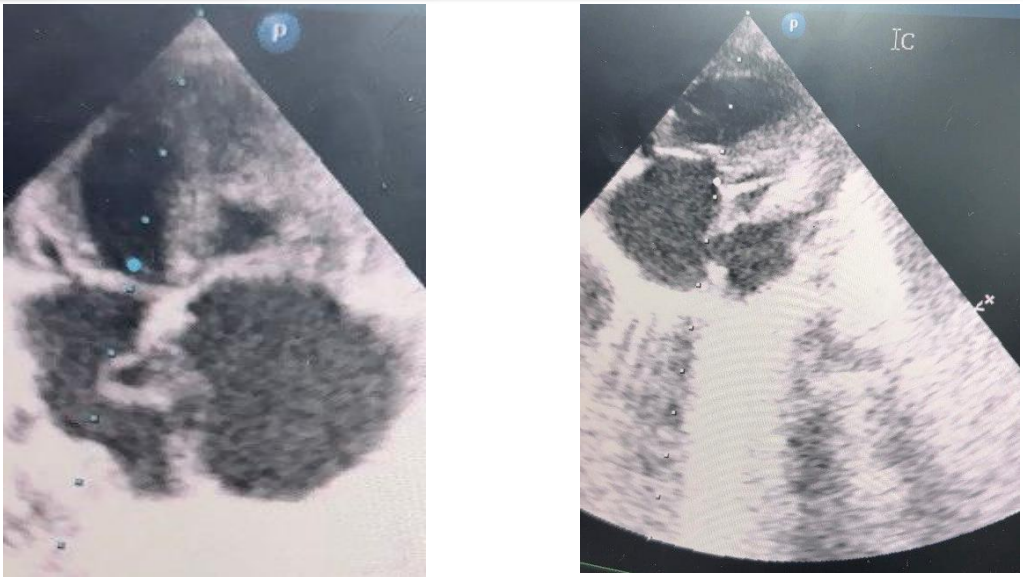


Figure (2-33) IAS aneurysm type2 with moderate size ASD seccundum (9*8).

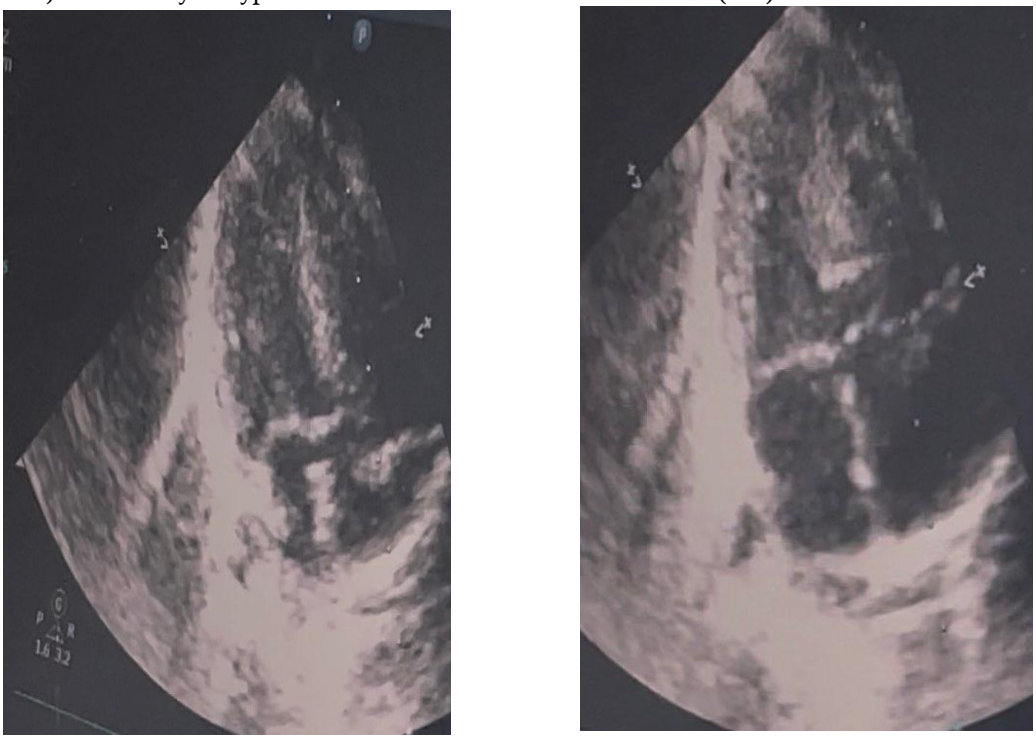


Figure (2-34) Complete AV canal, Large ASD primum with large inlet VSD.

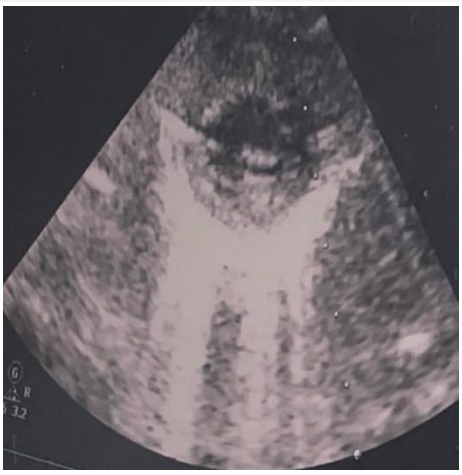


Figure (2-35) Cleft MV associated with ASD primum.

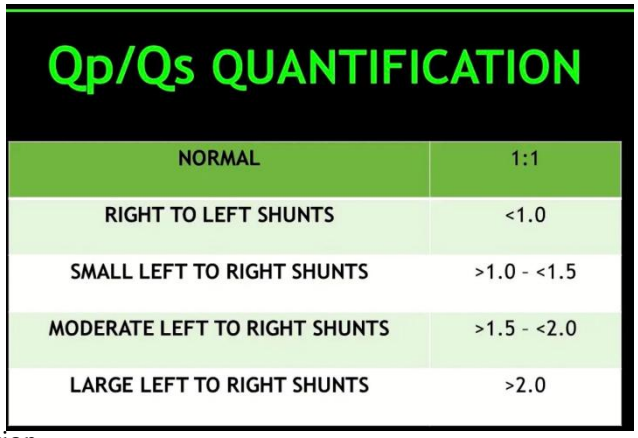


Figure (2-36) Qp/Qs quantification.

QP/QS can be classified in pediatrics and adults in the main five hemodynamic groups 1-with right side volume overload.

2-with pulmonary hypertension (mild/moderate/severe).

3-with Eseinmenger syndrome.

4-with severe PS.

RESULTS

Table (3-1) shows the Distribution of patients with ASD according to socio-demographic characteristics including (age, sex and Body surface area). Mean age of patients was (28.93 ± 21.38) years, older patient was 70 years and younger patient was 1 year. More than one third of patients (N=27, 37.0%) were children (≤ 14 years). Mean body surface area was (1.41 ± 0.54) m^2, maximum value was 2.14 m^2 and minimum value was 0.36 m^2. Majority of patients were females (N=57, 78.1%).

Table (3-1) Distribution of patients with ASD according to socio-demographic characteristics (N=73).

Socio-demographic characteristics	Number	%
Age (years)		
Children (≤ 14 years)	**27**	**37.0%**
Youth (15-24 years)	**5**	**6.8%**
Young adult (25-44 years)	**20**	**27.4%**
Middle age (45-59 years)	**13**	**17.8%**
Elderly (≥ 60 years)	**8**	**11.0%**
Total	**73**	**100.0%**
Sex		
Male	**16**	**21.9%**
Female	**57**	**78.1%**
Total	**73**	**100.0%**

***Mean body surface area was (1.41 ± 0.54) m^2, maximum value was 2.14 m^2 and minimum value was 0.36 m^2**

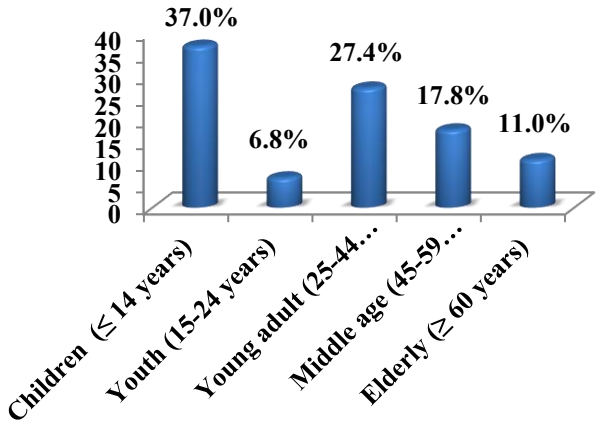


Figure 3-1: Distribution of patients with ASD according to age (N=74).

Table 3-2: Distribution of patients according to type and size of ASD. Majority of patients (N=68, 93.2%) presented with Seccundum ASD and majority of patients presented with large ASD (N=48, 65.8%).

Table 3-2: Distribution of patients with ASD according to type and size of ASD (N=73).

Study variables	Number	%
Type of ASD		
Seccundum	**68**	**93.2%**
Primum	**3**	**4.1%**
SVC	**2**	**2.7%**
Total	**73**	**100.0%**
Size of ASD		
Small	**15**	**20.5%**
Moderate	**7**	**9.6%**
Large	**48**	**65.8%**
Large and small (Two ASD)	**2**	**2.7%**
Moderate and small (Two ASD)	**1**	**1.4%**
Total	**73**	**100.0%**

Table 3: Distribution of patients according to Qp/Qs Quantification including (Normal 1:1, Right to left shunt < 1.0, Small left to right shunt >1.0 - <1.5, Moderate left to right shunt >1.5 - <2.0 and Large left to right shunt > 2.0). Majority of patients (N=29, 39.7%) presented with Large left to right shunt > 2.0.

Table 3-3: Distribution of patients with ASD according to Qp/Qs Quantification (N=73).

Qp/Qs Quantification	Number	%
Normal 1:1	**3**	**4.2%**
Right to left shunt < 1.0	**10**	**13.7%**
Small left to right shunt >1.0 - <1.5	**12**	**16.4%**
Moderate left to right shunt >1.5 - <2.0	**19**	**26.0%**
Large left to right shunt > 2.0	**29**	**39.7%**
Total	**73**	**100.0%**

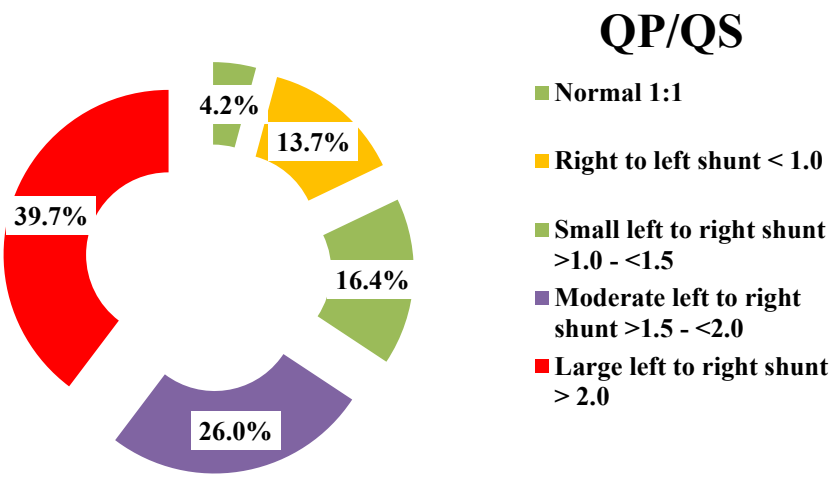


Figure 3-2: Distribution of patients with ASD according to Qp/Qs Quantification (N=73).

Table 3-4: Distribution of patients according to study variables including (pulmonary hypertension, pulmonary stenosis, Right side volume overload and Eseinmenger Syndrome). Severe pulmonary hypertension represent (N=10, 13.7%), Severe pulmonary stenosis represent only 3 patients (4.1%). Positive right side volume overload represent (N=63, 86.3%). Patients with Eseinmenger syndrome represent 8 patients (11.0%).

Table 3-4: Distribution of patients with ASD according to pulmonary hypertension, pulmonary stenosis, Right side volume overload and Eseinmenger Syndrome (N=73)

Study variables	Number	%
Pulmonary hypertension		
Negative	**42**	**57.5%**
Mild	**17**	**23.3%**
Moderate	**4**	**5.5%**
Severe	**10**	**13.7%**
Total	**73**	**100.0%**
Pulmonary stenosis		
Mild pulmonary stenosis	**13**	**17.8%**
Severe pulmonary stenosis	**3**	**4.1%**
No pulmonary stenosis	**57**	**78.1%**
Total	**73**	**100.0%**
Right side volume overload		
Positive	**63**	**86.3%**
Negative	**10**	**13.7%**
Total	**73**	**100.0%**
Eseinmenger syndrome		
Positive	**8**	**11.0%**
Negative	**65**	**89.0%**
Total	**73**	**100.0%**

Table 3-5: The mean differences of echo parameters including (RV base , RV mid D, TAPSE , FAC and RAA) according to pulmonary hypertension among pediatric age group (1-14 years). There was significant mean elevation of RAA among patients with severe PHT.

Table 3-5: The mean differences of echo parameters according to pulmonary hypertension among pediatric age group (N=27)

ECHO parameters	PHT	N	Mean	SD	P-value
RV base (cm/m^2)	Negative	19	4.53	1.83	0.872
	Mild	5	4.38	0.92	
	Severe	3	3.97	2.05	
RV mid D (cm/m^2)	Negative	19	4.02	1.51	0.813
	Mild	5	3.79	0.93	
	Severe	3	3.47	1.66	
TAPSE (cm/m^2)	Negative	19	2.21	0.73	0.817
	Mild	5	2.42	0.38	
	Severe	3	2.23	0.50	
FAC (%)	Negative	19	42.42	7.47	0.695
	Mild	5	44.20	12.43	
	Severe	3	39.00	3.60	
RAA (cm^2/m^2)	Negative	19	10.73	3.15	<0.001*
	Mild	5	15.40	4.78	
	Severe	3	26.23	7.61	

Table 3-6: The mean differences of echo parameters including (RV base , RV mid D, TAPSE , FAC and RAA) according to pulmonary hypertension among adult. There was significant mean reduction of TAPSE among patients with severe PHT. There was significant mean elevation of RAA among patients with severe PHT.

Table 3-7: The mean differences of echo parameters including (RV base , RV mid D, TAPSE , FAC and RAA) according to pulmonary stenosis among pediatric age group. There was no significant mean differences according to pulmonary stenosis.

Table 3-6: The mean differences of echo parameters according to pulmonary hypertension among adult (N=46)

ECHO parameters	PHT	N	Mean	SD	P-value
RV base (cm/m^2)	Negative	23	2.63	0.50	0.06
	Mild	12	2.81	0.54	
	Moderate	4	2.47	0.12	
	Severe	7	3.17	0.44	
RV mid D (cm/m^2)	Negative	23	2.23	0.49	0.23
	Mild	12	2.50	0.49	
	Moderate	4	2.16	0.16	
	Severe	7	2.55	0.44	
TAPSE (cm/m^2)	Negative	23	1.19	0.22	0.025*
	Mild	12	1.22	0.18	
	Moderate	4	1.26	0.11	
	Severe	7	0.92	0.30	
FAC (%)	Negative	23	40.47	6.72	0.227
	Mild	12	42.91	5.72	
	Moderate	4	42.75	5.43	
	Severe	7	36.42	8.75	
RAA (cm^2/m^2)	Negative	23	10.18	2.37	0.017*
	Mild	12	11.68	1.21	
	Moderate	4	11.15	2.82	
	Severe	7	14.28	5.45	

Table 3- 7: The mean differences of echo parameters according to pulmonary stenosis among pediatric age group (N=27)

ECHO parameters	Pulmonary stenosis	N	Mean	SD	P-value
RV base (cm/m^2)	Severe	3	3.71	1.92	0.435
	Mild or negative	24	4.53	1.67	
RV mid D (cm/m^2)	Severe	3	3.16	1.66	0.333
	Mild or negative	24	4.04	1.38	
TAPSE (cm/m^2)	Severe	3	1.60	0.10	0.064
	Mild or negative	24	2.33	0.64	
FAC (%)	Severe	3	40.00	10.00	0.60
	Mild or negative	24	42.67	8.03	
RAA (cm^2/m^2)	Severe	3	9.60	0.40	0.288
	Mild or negative	24	13.78	6.55	

Table 8: The mean differences of echo parameters including (RV base , RV mid D, TAPSE , FAC and RAA) according to Eseinmenger syndrome. There was significant mean reduction of FAC among patients with Eseinmenger syndrome. There was significant mean elevation of RAA among patients with Eseinmenger syndrome.

Table 3-8: The mean differences of echo parameters according to Eseinmenger syndrome (N=73).

ECHO parameters	Eseinmenger syndrome	N	Mean	SD	P-value
RV base (cm/m^2)	Positive	8	3.27	0.50	0.83
	Negative	65	3.38	1.43	
RV mid D (cm/m^2)	Positive	8	2.69	0.57	0.568
	Negative	65	2.96	1.25	
TAPSE (cm/m^2)	Positive	8	1.14	0.68	0.063
	Negative	65	1.61	0.66	
FAC (%)	Positive	8	36.25	8.11	0.037*
	Negative	65	41.93	7.03	
RAA (cm^2/m^2)	Positive	8	16.63	8.32	0.002*
	Negative	65	11.47	4.62	

Table 3- 9: The mean differences of echo parameters according to Right side volume overload among pediatric age group (N=27).

ECHO parameters	Right side volume overload	N	Mean	SD	P-value
RV base (cm/m^2)	Positive	25	4.40	1.72	0.739
	Negative	2	4.83	1.31	
RV mid D (cm/m^2)	Severe	3	3.88	1.44	0.659
	Positive	25	4.35	0.91	
TAPSE (cm/m^2)	Negative	2	2.28	0.65	0.318
	Positive	25	1.80	0.42	
FAC (%)	Negative	2	42.92	8.00	0.219
	Positive	25	35.50	7.77	
RAA (cm^2/m^2)	Negative	2	13.51	6.51	0.576
	Positive	25	10.85	2.33	

Table 3-9: The mean differences of echo parameters including (RV base , RV mid D, TAPSE , FAC and RAA) according to Right side volume overload among pediatric age group. There was no significant mean differences according to Right side volume overload.

Table 3-10: The mean differences of echo parameters including (RV base , RV mid D, TAPSE , FAC and RAA)

according to Right side volume overload among adult. There was significant mean elevation of RAA among adult patients with Right side volume overload.

Table 3-10: The mean differences of echo parameters according to Right side volume overload among adult (N=46).

ECHO parameters	Right side volume overload	N	Mean	SD	P-value
RV base (cm/m^2)	Positive	8	3.27	0.50	0.426
	Negative	65	3.38	1.43	
RV mid D (cm/m^2)	Positive	8	2.69	0.57	0.204
	Negative	65	2.96	1.25	
TAPSE (cm/m^2)	Positive	8	1.14	0.68	0.147
	Negative	65	1.61	0.66	
FAC (%)	Positive	8	36.25	8.11	0.761
	Negative	65	41.93	7.03	
RAA (cm^2/m^2)	Positive	8	16.63	8.32	0.005*
	Negative	65	11.47	4.62	

Table 3-11: The association between Qp/Qs Quantification and study variables including (pulmonary hypertension, pulmonary stenosis, Right side volume overload and Eseinmenger Syndrome). There was significant association between Qp/Qs Quantification and study variables including (pulmonary hypertension, Right side volume overload and Eseinmenger Syndrome). Majority (N=8, 80.0%) of patients with severe PHT presented with Right to left shunt. Less than half of patients with RS volume overload (N=29, 46.0%) presented with severe left to right shunt and all patients (N=8, 100.0%) with Eseinmenger syndrome presented with Right to left shunt.

Table 3-11: The association between Qp/Qs Quantification and pulmonary hypertension, pulmonary stenosis, Right side volume overload and Eseinmenger Syndrome (N=73).

Study variables	QP/QS					Total	P-value
	Normal	Right to left shunt	Small left to right shunt	Moderate left to right shunt	Severe left to right shunt		
PHT							
Negative	3 (7.1)	2 (4.8)	11 (26.2)	12 (28.6)	14 (33.3)	42 (100.0)	<0.001*
Mild	0 (0.0)	0 (0.0)	1 (5.9)	4 (23.5)	12 (70.6)	17 (100.0)	
Moderate	0 (0.0)	0 (0.0)	0 (0.0)	1 (25.0)	3 (75.0)	4 (100.0)	
Severe	0 (0.0)	8 (80.0)	0 (0.0)	2 (20.0)	0 (0.0)	10 (100.0)	
Total	3 (4.1)	10 (13.7)	12 (16.4)	19 (26.0)	29 (39.7)	73 (100.0)	
PS							
Severe	0 (0.0)	2 (66.7)	0 (0.0)	1 (33.3)	0 (0.0)	3 (100.0)	0.081
Mild or negative	3 (4.3)	8 (11.4)	12 (17.1)	18 (25.7)	29 (41.4)	70 (100.0)	
Total	3 (4.1)	10 (13.7)	12 (16.4)	19 (26.0)	29 (39.7)	73 (100.0)	
RS volume overload							
Positive	1 (1.6)	8 (12.7)	7 (11.1)	18 (28.6)	29 (46.0)	63 (100.0)	<0.001*
Negative	2 (20.0)	2 (20.0)	5 (50.0)	1 (10.0)	0 (0.0)	10 (100.0)	
Total	3 (4.1)	10 (13.7)	12 (16.4)	19 (26.0)	29 (39.7)	73 (100.0)	
Eseinmenger syndrome							
Positive	0 (0.0)	8 (100.0)	0 (0.0)	0 (0.0)	0 (0.0)	8 (100.0)	<0.001*
Negative	3 (4.6)	2 (3.1)	12 (18.5)	19 (29.2)	29 (44.6)	65 (100.0)	
Total	3 (4.1)	10 (13.7)	12 (16.4)	19 (26.0)	29 (39.7)	73 (100.0)	

Table 3-12: The association between pulmonary hypertension and ECG markers including (PWD, Axis, iRBBB-cRBBB, crochet sign in one inferior leads and crochet sign in all inferior leads). There was no significant association between pulmonary hypertension and ECG markers.

Table 3-12: The association between pulmonary hypertension and ECG markers (N=73).

ECG markers	Pulmonary hypertension				Total	P-value
	Negative	Mild	Moderate	Severe		
PWD						
40	**22 (52.3)**	**7 (41.2)**	**0 (0.0)**	**4 (40.0)**	**33 (45.2)**	**0.323**
60	**13 (31.0)**	**5 (29.4)**	**3 (75.0)**	**5 (50.0)**	**26 (35.6)**	
80	**7 (16.7)**	**5 (29.4)**	**1 (25.0)**	**1 (10.0)**	**14 (19.2)**	
Total	**42 (100.0)**	**10 (100.0)**	**4 (100.0)**	**10 (100.0)**	**73 (100.0)**	
AXIS						
Left	**6 (14.2)**	**3 (17.6)**	**1 (25.0)**	**0 (0.0)**	**10 (13.7)**	**0.88**
Right	**23 (54.8)**	**9 (52.9)**	**2 (50.0)**	**8 (80.0)**	**42 (57.5)**	
Superior	**2 (4.8)**	**0 (0.0)**	**0 (0.0)**	**0 (0.0)**	**2 (2.7)**	
Normal	**11 (26.2)**	**5 (29.5)**	**1 (25.0)**	**2 (20.0)**	**19 (26.0)**	
Total	**42 (100.0)**	**17 (100.0)**	**4 (100.0)**	**10 (100.0)**	**73 (100.0)**	
iRBBB-cRBBB						
cRBBB	**7 (16.7)**	**5 (29.4)**	**1 (25.0)**	**4 (40.0)**	**17 (23.3)**	**0.491**
iRBBB	**26 (61.9)**	**11 (64.7)**	**2 (50.0)**	**5 (50.0)**	**44 (60.3)**	
Negative	**9 (21.4)**	**1 (5.9)**	**1 (25.0)**	**1 (10.0)**	**12 (16.4)**	
Total	**42 (100.0)**	**17 (100.0)**	**4 (100.0)**	**10 (100.0)**	**73 (100.0)**	
Crochet sign in one inferior leads						
Positive	**12 (28.6)**	**6 (35.3)**	**2 (50.0)**	**6 (60.0)**	**26 (35.6)**	**0.239**
Negative	**30 (71.4)**	**11 (64.7)**	**2 (50.0)**	**4 (40.0)**	**47 (64.4)**	
Total	**42 (100.0)**	**10 (100.0)**	**4 (100.0)**	**10 (100.0)**	**73 (100.0)**	
Crochet sign in all inferior leads						
Positive	**29 (69.0)**	**11 (64.7)**	**2 (50.0)**	**4 (40.0)**	**46 (63.0)**	**0.338**
Negative	**13 (31.0)**	**6 (35.3)**	**2 (50.0)**	**6 (60.0)**	**27 (37.0)**	
Total	**42 (100.0)**	**10 (100.0)**	**4 (100.0)**	**10 (100.0)**	**73 (100.0)**	

Table 3-13: The association between pulmonary stenosis and ECG markers including (PWD, Axis, iRBBB-cRBBB, crochet sign in one inferior leads and crochet sign in all inferior leads). There was no significant association between pulmonary stenosis and ECG markers.

Table 3- 13: The association between pulmonary stenosis and ECG markers (N=73).

ECG markers	Pulmonary stenosis		Total	P-value
	Severe	Mild or negative		
PWD				
40	**3 (100.0)**	**30 (42.9)**	**33 (45.2)**	**0.295**
60	**0 (0.0)**	**26 (37.1)**	**26 (35.6)**	
80	**0 (0.0)**	**14 (20.0)**	**14 (19.2)**	
Total	**3 (100.0)**	**70 (100.0)**	**73 (100.0)**	
AXIS				
Left	**1 (33.3)**	**9 (12.9)**	**10 (13.7)**	**0.552**
Right	**2 (66.7)**	**40 (57.1)**	**42 (57.6)**	
Superior	**0 (0.0)**	**2 (2.9)**	**2 (2.7)**	
Normal	**0 (0.0)**	**19 (27.1)**	**19 (26.0)**	
Total	**3 (100.0)**	**70 (100.0)**	**73 (100.0)**	
iRBBB-cRBBB				
cRBBB	**0 (0.0)**	**17 (24.3)**	**17 (23.3)**	**0.528**
iRBBB	**2 (66.7)**	**42 (60.0)**	**44 (60.3)**	
Negative	**1 (33.3)**	**11 (15.7)**	**12 (16.4)**	
Total	**3 (100.0)**	**70 (100.0)**	**73 (100.0)**	
Crochet sign in one inferior leads				
Positive	**0 (0.0)**	**26 (37.1)**	**26 (35.6)**	**0.548**
Negative	**3 (100.0)**	**44 (62.9)**	**47 (64.4)**	
Total	**3 (100.0)**	**70 (100.0)**	**73 (100.0)**	

Crochet sign in all inferior leads				
Positive	**3 (100.0)**	**43 (61.4)**	**46 (63.0)**	**0.291**
Negative	**0 (0.0)**	**27 (38.6)**	**27 (37.0)**	
Total	**3 (100.0)**	**70 (100.0)**	**73 (100.0)**	

Table 3-14: The association between RS volume overload and ECG markers including (PWD, Axis, iRBBB-cRBBB, crochet sign in one inferior leads and crochet sign in all inferior leads). There was no significant association between RS volume overload and ECG markers.

Table 3-14: The association between RS volume overload and ECG markers (N=73).

ECG markers	RS volume overload		Total	P-value
	Positive	Negative		
PWD				
40	**27 (42.9)**	**6 (60.0)**	**33 (45.2)**	**0.74**
60	**23 (36.5)**	**3 (30.0)**	**26 (35.6)**	
80	**13 (20.6)**	**1 (10.0)**	**14 (19.2)**	
Total	**63 (100.0)**	**10 (100.0)**	**73 (100.0)**	
AXIS				
Left	**10 (15.9)**	**0 (0.0)**	**10 (13.7)**	**0.551**
Right	**36 (57.1)**	**6 (60.0)**	**42 (57.4)**	
Superior	**2 (3.2)**	**0 (0.0)**	**2 (2.7)**	
Normal	**15 (23.8)**	**4 (40.0)**	**19 (26.0)**	
Total	**63 (100.0)**	**10 (100.0)**	**73 (100.0)**	
iRBBB-cRBBB				
cRBBB	**15 (23.8)**	**2 (20.0)**	**17 (23.3)**	**0.312**
iRBBB	**36 (57.2)**	**8 (80.0)**	**44 (60.3)**	
Negative	**12 (19.0)**	**0 (0.0)**	**12 (16.4)**	
Total	**63 (100.0)**	**10 (100.0)**	**73 (100.0)**	
Crochet sign in one inferior leads				
Positive	**23 (36.5)**	**3 (30.0)**	**26 (35.6)**	**1.000**
Negative	**40 (63.5)**	**7 (70.0)**	**47 (64.4)**	
Total	**63 (100.0)**	**10 (100.0)**	**73 (100.0)**	
Crochet sign in all inferior leads				
Positive	**39 (61.9)**	**7 (70.0)**	**46 (63.0)**	**0.735**
Negative	**24 (38.1)**	**3 (30.0)**	**27 (37.0)**	
Total	**63 (100.0)**	**10 (100.0)**	**73 (100.0)**	

Table 3-15: The association between Eseinmenger syndrome and ECG markers including (PWD, Axis, iRBBB-cRBBB, crochet sign in one inferior leads and crochet sign in all inferior leads). There was no significant association between Eseinmenger syndrome and ECG markers.

Table 3-15: The association between Eseinmenger syndrome and ECG markers (N=73).

ECG markers	Eseinmenger syndrome		Total	P-value
	Positive	Negative		
PWD				
40	**2 (25.0)**	**31 (47.7)**	**33 (45.2)**	**0.266**
60	**5 (62.5)**	**21 (32.3)**	**26 (35.6)**	
80	**1 (12.5)**	**13 (20.0)**	**14 (19.2)**	
Total	**8 (100.0)**	**65 (100.0)**	**73 (100.0)**	
AXIS				
Left	**0 (0.0)**	**10 (15.4)**	**10 (13.7)**	**0.491**
Right	**7 (87.5)**	**35 (53.8)**	**42 (57.4)**	
Superior	**0 (0.0)**	**2 (3.1)**	**2 (2.7)**	
Normal	**1 (12.5)**	**18 (27.7)**	**19 (26.0)**	

Total	**8 (100.0)**	**65 (100.0)**	**73 (100.0)**	
iRBBB-cRBBB				
cRBBB	**4 (50.0)**	**13 (20.0)**	**17 (23.3)**	
iRBBB	**4 (50.0)**	**40 (61.5)**	**44 (60.3)**	**0.114**
Negative	**0 (0.0)**	**12 (18.5)**	**12 (16.4)**	
Total	**8 (100.0)**	**65 (100.0)**	**73 (100.0)**	
Crochet sign in one inferior leads				
Positive	**4 (50.0)**	**22 (33.8)**	**26 (35.6)**	**0.444**
Negative	**4 (50.0)**	**43 (66.2)**	**47 (64.4)**	
Total	**8 (100.0)**	**65 (100.0)**	**73 (100.0)**	
Crochet sign in all inferior leads				
Positive	**4 (50.0)**	**42 (64.6)**	**46 (63.0)**	**0.457**
Negative	**4 (50.0)**	**23 (35.4)**	**27 (37.0)**	
Total	**8 (100.0)**	**65 (100.0)**	**73 (100.0)**	

Table 3-16: The association between size of ASD and study variables including (age of patient, QP/QS, iRBBB-cRBBB, RV base and RV mid). There was significant association between size of ASD and age of patient, majority (N=9, 60.0%) of small ASD present in age <14 years, while three quarters of patients with large ASD (N=36, 75.0%) with age ≥ 14 years. There was significant association between size of ASD and iRBBB-cRBBB. Less than half of patients with small ASD (N=7, 46.7%) had negative results, while only two patients with large ASD (4.2%) had negative results. There was significant mean elevation of RV base and RV midline among patients with large ASD in both pediatric and adult.

Table 3-16: The association between size of ASD and study variables (N=73).

Study variables	Size of ASD				Total	P-value
	Small	Moderate	Large	Two ASD with different size		
Age (years)						
<14	**9 (60.0)**	**3 (42.9)**	**12 (25.0)**	**3 (100.0)**	**27 (37.0)**	**0.006***
≥ 14	**6 (40.0)**	**4 (57.1)**	**36 (75.0)**	**0 (0.0)**	**46 (63.0)**	
Total	**15 (100.0)**	**7 (100.0)**	**48 (100.0)**	**3 (100.0)**	**73 (100.0)**	
QP/QS						
Normal	**0 (0.0)**	**0 (0.0)**	**3 (6.3)**	**0 (0.0)**	**3 (4.1)**	
Right to left shunt	**1 (6.7)**	**1 (14.3)**	**8 (16.7)**	**0 (0.0)**	**10 (13.7)**	
Small left to right shunt	**4 (26.7)**	**2 (28.6)**	**6 (12.5)**	**0 (0.0)**	**12 (16.4)**	**0.905**
Moderate left to right shunt	**4 (26.7)**	**2 (28.6)**	**12 (25.0)**	**1 (33.3)**	**19 (26.0)**	
Large left to right shunt	**6 (40.0)**	**2 (28.6)**	**19 (39.6)**	**2 (66.7)**	**29 (39.7)**	
Total	**15 (100.0)**	**7 (100.0)**	**48 (100.0)**	**3 (100.0)**	**73 (100.0)**	
iRBBB-cRBBB						
cRBBB	**0 (0.0)**	**1 (14.2)**	**15 (31.3)**	**1 (33.3)**	**17 (23.3)**	
iRBBB	**8 (53.3)**	**3 (42.9)**	**31 (64.6)**	**2 (66.7)**	**44 (60.3)**	**0.001***
Negative	**7 (46.7)**	**3 (42.9)**	**2 (4.2)**	**0 (0.0)**	**12 (16.4)**	
Total	**15 (100.0)**	**7 (100.0)**	**48 (100.0)**	**3 (100.0)**	**73 (100.0)**	
RV base (cm/m^2) pediatric	**3.51 ± 1.37**	**2.78 ± 0.74**	**5.63 ± 1.43**	**4.06 ± 0.66**	**4.43 ± 1.68**	**0.002***
RV base (cm/m^2) adult	**2.15 ± 0.25**	**2.07 ± 0.47**	**2.92 ± 0.41**	**-**	**2.74 ± 0.51**	**<0.001***
RV mid D (cm/m^2) pediatric	**3.18 ± 1.20**	**2.42 ± 0.56**	**4.95 ± 1.08**	**3.46 ± 0.73**	**3.46 ± 0.73**	**0.001***
RV mid D (cm/m^2) adult	**1.71 ± 0.37**	**1.83 ± 0.48**	**2.51 ± 0.35**	**-**	**2.34 ± 0.47**	**<0.001***

Table 3-17: The mean differences of R/S mm in V1 according to study variables including (pulmonary hypertension, pulmonary stenosis, Right side volume overload and Eseinmenger Syndrome). There was significant mean elevation of R/S mm in V1 among patients with severe PHT, severe pulmonary stenosis and Eseinmenger Syndrome.

Table 3-18: The mean differences of RAA according to PWD including (40, 60 and 80). There was no significant mean differences of RAA according to PWD.

Table 3-17: The mean differences of R/S mm in V1 according to study variables (N=73).

Study variables	Number	Mean ± SD	P-value
PHT			
Negative	42	1.39 ± 1.83	<0.001*
Mild	17	1.02 ± 1.21	
Moderate	4	3.38 ± 1.49	
Severe	10	3.62 ± 2.01	
Pulmonary stenosis			
Severe	3	6.00 ± 1.73	<0.001*
Mild or negative	70	1.53 ± 1.71	
Right side volume overload			
Positive	63	1.78 ± 1.91	0.496
Negative	10	1.33 ± 2.02	
Eseinmenger Syndrome			
Positive	8	3.03 ± 1.43	0.04
Negative	65	1.55 ± 1.91	

Table 3-18: The mean differences of RAA according to PWD (N=73).

Study variable	PWD	N	Mean	SD	P-value
RAA (cm^2/m^2)	40	33	12.57	5.77	0.581
	60	26	11.29	2.92	
	80	14	12.15	4.27	

Table 3-19: The association between Qp/Qs Quantification and study variables including (PWD, iRBBB-cRBBB and R/S ratio). There was significant mean elevation of R/S ratio among patients with right to left shunt.

Table 3-20: The association between T inversion or POS and pulmonary hypertension, pulmonary stenosis and Eseinmenger Syndrome. There was significant association between T inversion or POS and pulmonary hypertension and Eseinmenger Syndrome. Majority (N=9, 90.0%) of those with severe PHT had positive results and all patients with Eseinmenger Syndrome (N=8, 100.0%) had positive results.

Table 3-19: The association between Qp/Qs Quantification and PWD, iRBBB-cRBBB and R/S ratio (N=73).

Study variables	QP/QS					Total	P-value
	Normal	Right to left shunt	Small left to right shunt	Moderate left to right	Severe left to right shunt		

				shunt			
PWD							
40	1 (33.3)	4 (40.0)	5 (41.7)	7 (36.8)	16 (55.2)	33 (45.2)	
60	1 (33.3)	5 (50.0)	4 (33.3)	9 (47.4)	7 (24.1)	26 (35.6)	0.795
80	1 (33.3)	1 (10.0)	3 (25.0)	3 (15.8)	6 (20.7)	14 (19.2)	
Total	3 (100.0)	10 (100.0)	12 (100.0)	19 (100.0)	29 (100.0)	73 (100.0)	
iRBBB-cRBBB							
cRBBB	2 (66.7)	4 (40.0)	1 (8.3)	4 (21.1)	6 (20.7)	17 (23.3)	
iRBBB	1 (33.3)	5 (50.0)	9 (75.0)	11 (57.9)	18 (62.1)	44 (60.3)	0.547
Negative	0 (0.0)	1 (10.0)	2 (16.7)	4 (21.1)	5 (17.2)	12 (16.4)	
Total	3 (100.0)	10 (100.0)	12 (100.0)	19 (100.0)	29 (100.0)	73 (100.0)	
R/S mm in V1	0.65 ± 0.21	3.82 ± 2.10	0.64 ± 0.21	1.93 ± 2.41	1.41 ± 1.39	1.72 ± 1.91	<0.001*

Table 3-20: The association between T inversion or POS and pulmonary hypertension, pulmonary stenosis and Eseinmenger Syndrome (N=73).

Study variables	T inversion or POS		Total	P-value
	Positive	Negative		
PHT				
Negative	10 (23.8)	32 (76.2)	42 (100.0)	
Mild	6 (35.3)	11 (64.7)	17 (100.0)	0.001*
Moderate	1 (25.0)	3 (75.0)	4 (100.0)	
Severe	9 (90.0)	1 (10.0)	10 (100.0)	
Total	26 (35.6)	47 (13.7)	73 (100.0)	
PS				
Severe	2 (66.7)	1 (33.3)	3 (100.0)	0.287
Mild or negative	24 (34.3)	46 (65.7)	70 (100.0)	
Total	26 (35.6)	47 (64.4)	73 (100.0)	
Eseinmenger syndrome				
Positive	8 (100.0)	0 (0.0)	8 (100.0)	<0.001*
Negative	18 (27.7)	47 (72.3)	65 (100.0)	
Total	26 (35.6)	47 (64.4)	73 (100.0)	

Table 3-21: The association between qR pattern and pulmonary hypertension, pulmonary stenosis and Eseinmenger Syndrome. There was significant association between qR pattern and pulmonary hypertension and Eseinmenger Syndrome. Majority (N=8, 80.0%) of those with severe PHT presented with POS and majority (N=7, 87.5%) of patients with Eseinmenger Syndrome had POS.

Table 3-21: The association between qR pattern and pulmonary hypertension, pulmonary stenosis and Eseinmenger Syndrome (N=73).

Study variables	qR pattern		Total	P-value
	POS	Negative		
PHT				
Negative	3 (7.1)	39 (92.9)	42 (100.0)	
Mild	1 (5.9)	16 (94.1)	17 (100.0)	<0.001*
Moderate	0 (0.0)	4 (100.0)	4 (100.0)	
Severe	8 (80.0)	2 (20.0)	10 (100.0)	
Total	12 (16.4)	61 (83.6)	73 (100.0)	
PS				
Severe	2 (66.7)	1 (33.3)	3 (100.0)	0.068
Mild or negative	10 (14.3)	60 (85.7)	70 (100.0)	
Total	12 (16.4)	61 (83.6)	73 (100.0)	
Eseinmenger syndrome				
Positive	7 (87.5)	1 (12.5)	8 (100.0)	<0.001*
Negative	5 (7.7)	60 (92.3)	65 (100.0)	
Total	12 (35.6)	61 (83.6)	73 (100.0)	

European Journal of Clinical Pharmacy
Print ISSN: 2385-409X **Online ISSN**: 3105-0409
Website: https://farmclin.com/

Data analysis

Statistical analysis was carried out using SPSS version 27. Categorical variables were presented as frequencies and percentages. Continuous variables were presented as (Means ± SD). Student t-test was used to compare means between two groups. ANOVA test was used to compare means among three groups or more. Pearson chi-square test and Fisher's Exact test were used to find the association between categorical variables. P value ≤ 0.05 was considered as significant.

DISCUSSION

The present study evaluated the correlation between echocardiographic parameters, particularly Qp/Qs ratio, right-sided chamber dimensions, right atrial area, TAPSE, and FAC, and electrocardiographic parameters including P-wave duration, frontal QRS axis, iRBBB/cRBBB, crochet sign, R/S ratio in V1, T-wave inversion/positive RV strain pattern, and qR pattern among patients with atrial septal defect (ASD) with hemodynamic effect. The study included both pediatric and adult patients, reflecting a broad clinical spectrum ranging from left-to-right shunting with right-sided volume overload to right-to-left shunting, severe pulmonary hypertension, and Eisenmenger physiology. Overall, echocardiographic variables QP/QS provided the most consistent assessment of hemodynamic severity, whereas selected ECG markers, particularly R/S ratio in V1, T-wave inversion/positive RV strain pattern, and qR pattern, were more closely related to advanced right ventricular pressure overload and Eisenmenger physiology than to simple volume overload alone.

This general pattern is supported by Cool et al., 2025, n=36, Indonesia [68], who evaluated echocardiographic flow ratio against catheter-derived flow ratio in uncorrected acyanotic adult congenital heart disease and emphasized the role of flow ratio in detecting Eisenmenger physiology,also Kuijpers et al., 2015, Netherlands [1], and Brida et al., 2022 [5], support this integrated approach, showing that late ASD expression depends on right-sided remodeling quantified by QP/QS , pulmonary vascular response, and rhythm consequences.

It is also supported by Umamy et al., 2025, n=98, Indonesia [59], who found that selected ECG markers predicted hemodynamic parameters in adults with uncorrected secundum ASD, particularly markers related to pulmonary vascular resistance and right-sided pressure burden. These findings place the present study within a consistent framework: echocardiography defines the anatomic and hemodynamic substrate, while ECG contributes supportive information mainly when right ventricular pressure overload or strain has developed

4.1 Socio-demographic characteristics

The present study showed a mean age of 28.93 ± 21.38 years, ranging from 1 to 70 years. Children aged ≤14 years represented 37.0%, whereas adolescents and adults formed the larger proportion, indicating delayed diagnosis or referral beyond early childhood. Kuijpers et al., 2015, Netherlands [1], and Brida et al., 2022 [5], support that secundum ASD may remain clinically silent until adolescence or adulthood, when right-heart dilatation, arrhythmia, exercise limitation, or pulmonary hypertension becomes evident. In contrast, Eryu et al., 2017, pediatric ASD sample, Japan [70], evaluated a younger cohort, reflecting screening and recruitment differences rather than different ASD physiology.

Females represented 78.1%, consistent with recognized female predominance in secundum ASD cohorts. Kuijpers et al., 2015, Netherlands [1], reported an approximate 2:1 female predominance, while Brida et al., 2022, international [5], described sex-related differences in adult ASD presentation. Iraqi reports by Rasheed et al., 2024, Kurdistan Region of Iraq [40], and Alaani et al., 2023, Iraq [41], provide regional congenital heart disease context, although not ASD-specific hemodynamic correlations. Mean body surface area was 1.41 ± 0.54 m^2, ranging from 0.36 to 2.14 m^2, as the cohort included children and adults. Thus, separating pediatric and adult echocardiographic analyses was appropriate because body size affects RV base, RV mid diameter, TAPSE, and RAA, making indexed values more reliable.

4.2 Type and size of ASD

Secundum ASD was dominant, accounting for 93.2% of cases; therefore, the results mainly represent secundum ASD physiology. This agrees with established descriptions that secundum ASD is the commonest anatomical subtype. Geva et al., 2014 [41], and Le Gloan et al., 2018, France [42], support that secundum ASD is the main form encountered clinically and the usual subtype assessed for closure suitability and right-heart remodeling.

Large ASD was present in 65.8%, indicating enrichment with anatomically large defects, while small ASD represented 20.5% and moderate ASD represented 9.6%. This indicates that the study population was enriched with clinically significant defects. Martin et al., 2014, review, USA [45], supported the interpretation that significant large ASD produces chronic right atrial and right

ventricular volume overload.. but the size should be separated from Qp/Qs or shunt volume, because anatomical size is not identical to hemodynamic magnitude. Silvestry et al., 2015, ASE/SCAI guideline, USA [43], emphasized defining ASD type, size, shape, location, rims, and number. Ribeiro et al., 2025, n=35 pediatric isolated ASD, Brazil [44], supported this anatomical approach using ASD diameter relative to interatrial septal and mitral annular dimensions. However, Refaei et al., 2017, n=141, Canada [1], reported that routine ECG parameters in pediatric isolated secundum ASD had limited ability to predict ASD size and hemodynamic outcomes. This difference indicates that ASD size may be strongly reflected by echocardiographic right-heart remodeling, while ECG changes may be less consistent, especially in children or less advanced disease.

4.3 Qp/Qs quantification

As showed in table (3-3) and figure (3-2)The largest subgroup had large left-to-right shunt with Qp/Qs >2.0, representing 39.7%, followed by moderate and small left-to-right shunts, confirming frequent pulmonary overcirculation. Qp/Qs should be interpreted as hemodynamic shunt magnitude, not anatomical ASD size. Torres, 2018, USA [45], explained that shunt direction and magnitude depend on defect size, interatrial pressure gradients, ventricular compliance, and pulmonary vascular resistance. Baumgartner et al., 2021, ESC guideline, Europe [46], also supports using Qp/Qs and right-heart volume loading when assessing ASD significance and closure suitability.

Right-to-left shunt was present in 13.7%, suggesting advanced pulmonary vascular disease or Eisenmenger physiology in a subgroup. Jain and Dalvi, 2018, India [47], and Gabriels et al., 2014, adult secundum ASD sample, Belgium [48], support that long-standing ASD may progress from pulmonary overcirculation to pulmonary vascular remodeling, increased resistance, bidirectional or reversed shunting, and altered operability. Figure 2 showed that large left-to-right shunt was most frequent, and the high frequency of right-sided volume overload in Table 4 ,but this should not be equated with anatomical large ASD because Table 16 showed no significant association between ASD size and Qp/Qs.According to ECG, Ocal and Saricam, 2020, adult secundum ASD cohort, Turkey [70], also supported the relevance of shunt size by evaluating the relationship between shunt size and right precordial ECG findings

4.4 Pulmonary hypertension, pulmonary stenosis, right-sided volume-overload subgroup, and Eisenmenger syndrome

Pulmonary hypertension was present in 42.5%, including 13.7% with severe PH, confirming the importance of pulmonary vascular assessment in ASD. Chinawa et al., 2022, pediatric ASD sample, Nigeria [49], reported PH among children with ASD and highlighted predictors of PH. In adults, Gabriels et al., 2014, adult secundum ASD sample, Belgium [48], and Jain and Dalvi, 2018, India [47], emphasized that ASD-associated PH requires integrated evaluation because operability depends on pulmonary vascular disease, not defect diameter alone.

Pulmonary stenosis was uncommon, with severe PS in only three patients. Severe PS causes RV pressure overload, but the small severe subgroup limited statistical associations; therefore, nonsignificant PS-related findings mainly reflect power limitation. Right-sided volume overload was present in 86.3% and should be understood as a right-sided volume-overload subgroup pathophysiologically consistent large secundum ASDs and increased left-to-right pulmonary flow. Baumgartner et al., 2021, ESC guideline, Europe [46], and Torres, 2018, USA [45], support evaluating significant ASD through shunt magnitude, right-heart volume loading, pulmonary vascular status, and clinical context, Dannenberg et al., 2020, n=113, Austria [69], supported the importance of right ventricular volume overload as an echocardiographic indicator of significant atrial shunt lesions. Eisenmenger syndrome was present in 11.0%, likely reflecting delayed diagnosis or referral, Brida et al., 2022 [5], and Jain and Dalvi, 2018, India [47], support progression of unrepaired ASD to pulmonary vascular disease and Eisenmenger physiology.

4.5 Echo parameters according to pulmonary hypertension among pediatric patients

Among pediatric patients, RV base, RV mid diameter, TAPSE, and FAC did not differ significantly by PH status, whereas RAA was significantly higher in severe PH, increasing from 10.73 ± 3.15 to 26.23 ± 7.61 cm^2/m^2. Thus, indexed RAA was the clearest pediatric echocardiographic marker of severe PH ,significant RAA finding may reflect that right atrial remodeling accumulates with chronic pressure and volume burden. Koestenberger et al., 2016, pediatric cohort, Austria/USA/Germany [50], provided RA size references in children with and without ASD or PH, supporting RA area assessment. Other parameters were probably nonsignificant because only three pediatric patients had severe PH. Murni et al., 2023, n=144, Indonesia [23], also supported the relevance of right-heart electrical and hemodynamic consequences in pediatric ASD with PH by showing that ECG criteria, including QRS axis ≥120°, P-wave abnormality, right precordial R-wave changes, RBBB, and qR/Q-wave features, could predict PH in children with ASD

4.6 Echo parameters according to pulmonary hypertension among adults

Among adults, severe PH was associated with significantly reduced TAPSE and increased RAA,

indicating impaired longitudinal RV systolic function and right atrial remodeling. This should be interpreted according to measured variables only, because RV-PA coupling was not assessed. Cossio-Aranda et al., 2016, adult ostium secundum ASD cohort, Mexico [51], supports more advanced right-heart involvement in severe PH. Pham et al., 2020, n=63 adult ostium secundum ASD patients, Mexico/Vietnam collaboration [52], also found lower RV fractional area change and higher right atrial volume with greater PH burden. Umamy et al., 2025, n=98, Indonesia [59], supported the association between ECG markers and hemodynamic burden in adults with uncorrected secundum ASD and showed that several ECG parameters differed according to pulmonary vascular resistance, including R/S in V1 and right ventricular strain and reduced Tapse with increasing RVSP. Thus, reduced TAPSE with increased RAA suggests both RV functional impairment and chronic atrial remodeling.

4.7 Echo parameters according to pulmonary stenosis among pediatric patients

No significant differences in RV base, RV mid diameter, TAPSE, FAC, or RAA were found by pulmonary stenosis among pediatric patients. TAPSE appeared lower in severe PS, but only three patients were in this subgroup; therefore, the nonsignificant result reflects subgroup size rather than absence of biological effect. Severe PS is a right ventricular pressure-overload lesion. Sirico et al., 2023, n=43 pediatric severe pulmonary valve stenosis patients, Italy [53], showed effects on RV dimension and function, with FAC improving after relief of obstruction while TAPSE did not improve immediately. Thus, the present negative result most likely reflects the very small severe PS subgroup.

4.8 Echo parameters according to Eisenmenger syndrome

Eisenmenger syndrome was significantly associated with lower FAC and higher RAA. FAC decreased to 36.25 ± 8.11%, while RAA increased to 16.63 ± 8.32 cm^2/m^2, indicating impaired RV systolic performance and right atrial enlargement in advanced pulmonary vascular disease. The discussion should focus on FAC and RAA because RV base, RV mid diameter, and TAPSE were not significant. Kalogeropoulos et al., 2010, adult Eisenmenger physiology cohort, USA [54], support quantitative RV function assessment including FAC. Moceri et al., 2012, adult Eisenmenger cohort, international [55], supports the prognostic relevance of RV function and RAA. Therefore, reduced FAC with increased RAA is consistent with transition from volume loading to pulmonary vascular disease with right-heart impairment.

4.9 Echo parameters according to right-sided volume-overload subgroup among pediatric patients

No significant mean differences were found according to right-sided volume-overload subgroup among pediatric patients. This is best explained by imbalance, as 25 of 27 children were positive and only two were negative. Thus, lack of significance does not exclude ASD-related volume-loading effects; it indicates underpowered comparison. Baumgartner et al., 2021, ESC guideline, Europe [46], and Torres, 2018, USA [45], support integrated hemodynamic assessment rather than reliance on a single parameter. Dannenberg et al., 2020, n=113, Austria [69], supported the importance of right ventricular volume overload as an echocardiographic sign of atrial shunt lesions. Martin et al., 2014, review, USA [8], also supported the pathophysiological relationship between significant ASD and right-sided chamber enlargement

4.10 Echo parameters according to right-sided volume-overload subgroup among adults

Among adults, RAA was significantly higher in the right-sided volume-overload subgroup, whereas RV base, RV mid diameter, TAPSE, and FAC were not significantly different. This suggests that right atrial remodeling may be a stable marker of chronic ASD-related volume burden in adults. Morton et al., 2003, adult ASD sample, Australia [56], supports that chronic right atrial stretch is associated with atrial structural and electrical remodeling. Guray et al., 2003, adult secundum ASD sample, Turkey [57], also supports altered atrial electrical properties. Therefore, increased RAA is physiologically consistent with chronic atrial stretch despite nonsignificant RV indices. TAPSE and FAC may remain preserved until later stages or may be more strongly affected by pressure overload than by volume overload alone.

4.11 Association between Qp/Qs and pulmonary hypertension, pulmonary stenosis, right-sided volume-overload subgroup, and Eisenmenger syndrome

Qp/Qs was significantly associated with pulmonary hypertension. Most severe PH patients had right-to-left shunt, whereas mild and moderate PH were more often left-to-right, supporting progression from pulmonary overcirculation to vascular disease and shunt reversal. Jain and Dalvi, 2018, India [47], Gabriels et al., 2014, adult secundum ASD sample, Belgium [48], and Baumgartner et al., 2021, ESC guideline, Europe [46], support that ASD shunt behavior changes with pulmonary vascular resistance, operability, and disease stage.

Qp/Qs was not significantly associated with pulmonary stenosis, but this does not mean PS is unrelated to RV pressure load; only three patients had severe PS. Qp/Qs was significantly associated with right-sided volume-overload subgroup, which is the expected hemodynamic connection: Qp/Qs reflects

pulmonary/systemic flow balance, while right-sided volume overload reflects the downstream impact of sustained left-to-right shunting. Nakayama et al., 2020, adult ASD cohort, Japan [71], supported this relationship by emphasizing RV dilatation as a consequence of ASD-related shunting, Torres, 2018, USA [45], and Baumgartner et al., 2021, ESC guideline, Europe [46], support interpreting Qp/Qs with right-heart volume loading and pulmonary vascular status. Qp/Qs was also significantly associated with Eisenmenger syndrome, as all Eisenmenger patients had right-to-left shunt, Cool et al., 2025, n=36, Indonesia [68], provides direct external support for this hemodynamic interpretation

4.12 Association between pulmonary hypertension and ECG markers

Pulmonary hypertension was not significantly associated with P-wave dispersion, frontal QRS axis, iRBBB/cRBBB, crochet sign in one inferior lead, or crochet sign in all inferior leads. However, right axis deviation and cRBBB were numerically more frequent in severe PH, making the pattern physiologically plausible though not statistically discriminative. Murni et al., 2023, n=144 children with ASD, Indonesia [58], showed that ECG criteria including QRS axis ≥120°, P-wave abnormality, right precordial R-wave changes, RBBB, and qR/Q-wave features could predict PH. Umamy et al., 2025, n=98 adults with uncorrected secundum ASD, Indonesia [59], also linked selected ECG markers with invasive hemodynamics.

Although P-wave dispersion did not differ significantly across PH categories, this does not exclude atrial electrical remodeling. Ho et al., 2001, pediatric secundum ASD sample, Singapore [60], and Salih and Brazinji, 2019, n=41 children with isolated secundum ASD, Iraq/Kurdistan [61], reported increased PWD in children with ASD, especially with right atrial dilation. Thus, PWD may reflect ASD-related atrial conduction heterogeneity more than PH grade in this cohort.

4.13 Association between pulmonary stenosis and ECG markers

Pulmonary stenosis was not significantly associated with P-wave dispersion, frontal QRS axis, iRBBB/cRBBB, or crochet sign. This should be interpreted cautiously because only three patients had severe PS. Severe PS is a pressure-overload lesion and may influence right-sided ECG patterns, but the study lacked enough severe cases to detect stable categorical differences. Fazelifar et al., 2023, severe pulmonary stenosis and pulmonary hypertension cohort, Iran [62], supports PS effects on QRS/R-wave and right-sided ECG patterns through RV pressure loading.

4.14 Association between right-sided volume-overload subgroup and ECG markers

Right-sided volume-overload subgroup was not significantly associated with P-wave dispersion, frontal QRS axis, iRBBB/cRBBB, or crochet sign. This nonsignificant result should be interpreted cautiously because most patients were volume-overload positive, leaving a small negative group. Therefore, the result does not exclude the established relationship between ASD-related right-heart volume loading and ECG changes.

Previous literature supports that right axis deviation and iRBBB/cRBBB are common ECG manifestations of secundum ASD related to right-sided volume loading. Raut, 2017, India [63], reported normal axis or right-axis deviation and incomplete RBBB among common ostium secundum ASD findings. Bayar et al., 2015, adult ASD sample, Turkey [64], reported the diagnostic value of right-sided conduction and axis abnormalities. Somura et al., 2015, n=100, Japan [9], linked ECG signs with shunt volume. Regarding atrial remodeling, Salih and Brazinji, 2019, n=41 children with isolated secundum ASD, Iraq/Kurdistan [61], and Ho et al., 2001, pediatric secundum ASD sample, Singapore [60], reported higher PWD in ASD, especially with right atrial dilation. Thus, the present nonsignificance likely reflects imbalance and categorical comparison.

4.15 Association between Eisenmenger syndrome and ECG markers

Eisenmenger syndrome was not significantly associated with P-wave dispersion, frontal axis, iRBBB/cRBBB, or crochet sign. However, right axis deviation and cRBBB were numerically more frequent among Eisenmenger patients, a pattern compatible with advanced right-heart pressure overload despite nonsignificance.

Murni et al., 2023, n=144 children with ASD, Indonesia [58], and Umamy et al., 2025, n=98 adults with uncorrected secundum ASD, Indonesia [59], support right-axis, RBBB, right precordial R-wave, and qR/Q-wave features as ECG markers of PH or abnormal hemodynamics. Therefore, routine ECG categories may not grade Eisenmenger physiology reliably, while later markers, especially R/S ratio in V1, T-wave inversion/age-adjusted RV strain, and qR pattern, were more informative.

4.16 Association between ASD size and age, Qp/Qs, iRBBB/cRBBB, RV base, and RV mid diameter

ASD size was significantly associated with age; most small ASDs occurred in children, while most large ASDs occurred in patients aged ≥14 years. This may reflect delayed diagnosis of larger defects or referral of more advanced cases. Kuijpers et al., 2015, Netherlands [1], and Brida et al., 2022 [5], support that secundum ASD may remain undiagnosed until adolescence or adulthood.

ASD size was not significantly associated with Qp/Qs. This is important because anatomical size and shunt magnitude are different. Silvestry et al., 2015, ASE/SCAI guideline, USA [43], supports separate anatomical assessment of size, shape, rims, and number, while hemodynamic assessment requires separate evaluation. Torres, 2018, USA [45], explained that Qp/Qs depends on interatrial pressure gradients, chamber compliance, and pulmonary vascular resistance in addition to defect anatomy. Therefore, this nonsignificant association is physiologically plausible.

ASD size was significantly associated with iRBBB/cRBBB, supporting that larger secundum defects may be linked to greater right-sided conduction effects, although not as a direct shunt-volume measure. Raut, 2017, India [63], Bayar et al., 2015, adult ASD sample, Turkey [64], and Somura et al., 2015, n=100, Japan [30], support frequent right-sided ECG changes in secundum ASD. RV base and RV mid diameter were also significantly higher in large ASD in pediatric and adult subgroups. Ribeiro et al., 2025, pediatric isolated ASD, Brazil [44], supports anatomical ASD size classification using structural echocardiographic measurements.

4.17 R/S ratio in V1 according to hemodynamic variables

R/S ratio in V1 was significantly elevated with severe PH, severe pulmonary stenosis, and Eisenmenger syndrome, but not with right-sided volume-overload subgroup. Mean R/S ratio was 3.62 ± 2.01 in severe PH, 6.00 ± 1.73 in severe PS, and 3.03 ± 1.43 in Eisenmenger syndrome. These numerical differences are clinically important because all three conditions increase right ventricular systolic load, but by different mechanisms. Severe PS produces fixed valvular obstruction and direct RV pressure overload, explaining the highest mean R/S value. Severe PH produces pulmonary vascular afterload and RV hypertrophy/strain, explaining the elevated but lower mean value. Eisenmenger syndrome represents advanced pulmonary vascular disease with shunt reversal and chronic RV remodeling; therefore, its R/S ratio remained high but was lower than severe PS in this cohort, possibly because Eisenmenger physiology includes mixed chronic remodeling, altered shunt direction, and small subgroup size.

This interpretation is supported by published ECG-hemodynamic data. Al-Naamani et al., 2008, n=282 patients with pulmonary hypertension, Canada [72], showed that ECG criteria based on R and S amplitudes in V1 were among the most predictive markers of pulmonary hypertension; R/S in V1 ≥1 had a positive predictive value of 72.7%, S in V1 ≤2 mm had a positive predictive value of 100%, and qR in V1 had a positive predictive value of 94.7%. Murni et al., 2023, n=144, Indonesia [58], also supports right precordial R-wave and qR/Q-wave features in predicting PH among children with ASD, while Umamy et al., 2025, n=98, Indonesia [59], supports the relationship between ECG markers and ASD hemodynamics. For pulmonary stenosis, Fazelifar et al., 2023, Iran [62], showed that R/S >1 in precordial leads and higher R-wave amplitude in V1 were more prominent in PS, supporting the particularly high R/S value in the present severe PS subgroup. Therefore, the gradient of R/S values across severe PS, severe PH, and Eisenmenger syndrome is physiologically coherent and supports R/S ratio in V1 as a pressure-overload marker rather than a simple volume-overload marker.

4.18 RAA according to P-wave dispersion

RAA did not differ significantly according to P-wave dispersion categories. Although PWD may be increased in ASD as a marker of atrial electrical heterogeneity, RAA measures anatomic chamber enlargement, whereas PWD reflects atrial conduction time but it did not show a graded relationship with indexed RAA in this cohort. This should not be interpreted as absence of atrial electrical remodeling. Atrial conduction can be affected by interatrial conduction pathways, autonomic tone, age, rhythm status, atrial fibrosis, measurement technique, and ECG sampling.

Ho et al., 2001, pediatric secundum ASD sample, Singapore [60], reported higher P-wave dispersion in children with ASD, especially with right atrial dilation. Salih and Brazinji, 2019, n=41 children with isolated secundum ASD, Iraq/Kurdistan [61], similarly found prolonged PWD and higher values among patients with right atrial dilation. Therefore, the nonsignificant association here may reflect categorical PWD groups, mixed pediatric-adult age distribution, and BSA-indexed RAA rather than absence of ASD-related atrial conduction abnormalities.

4.19 Association between Qp/Qs and PWD, iRBBB/cRBBB, and R/S ratio

Qp/Qs was not significantly associated with P-wave dispersion or iRBBB/cRBBB, but R/S ratio in V1 was significantly higher in right-to-left shunt. This indicates closer relation to advanced pulmonary vascular disease and RV pressure overload than to left-to-right shunt magnitude alone. Umamy et al., 2025, n=98 adults with uncorrected secundum ASD, Indonesia [59], supports the relationship between selected ECG markers and hemodynamic severity. Murni et al., 2023, n=144 children with ASD, Indonesia [58], also supports right precordial R-wave and qR/Q-wave features in predicting PH. In contrast, Somura et al., 2015, n=100, Japan [30], reported that RBBB was associated with higher Qp/Qs in ASD, and Ocal and Saricam, 2020, adult secundum ASD cohort, Turkey [65], supported a

relationship between shunt size and right precordial ECG findings.

The nonsignificant relationship between Qp/Qs and PWD should be interpreted carefully because PWD reflects atrial conduction heterogeneity and stretch rather than shunt magnitude alone. Ho et al., 2001 [60], Salih and Brazinji, 2019 [61], and Guray et al., 2003 [57], support increased PWD in secundum ASD, but it may not directly track Qp/Qs categories in a mixed-age cohort.

4.20 T-wave inversion or age-adjusted positive RV strain pattern according to pulmonary hypertension, pulmonary stenosis, and Eisenmenger syndrome

T-wave inversion or age-adjusted positive RV strain pattern was significantly associated with pulmonary hypertension and Eisenmenger syndrome. It was present in 90.0% of severe PH and 100.0% of Eisenmenger patients, indicating strong linkage with advanced RV strain and pulmonary vascular disease. This interpretation should be linked to RV pressure-overload literature rather than ASD diagnostic studies using T-wave abnormality alone.

Umamy et al., 2025, n=98 adults with uncorrected secundum ASD, Indonesia [59], is appropriate because it defined RV strain-related ECG markers and related them to hemodynamic parameters including PVR. Waligóra et al., 2021, pulmonary hypertension cohort, Poland [66], supports that extended precordial T-wave inversion is associated with RV enlargement and poor prognosis in PH. In children, interpretation must be age-adjusted because anterior T-wave patterns vary with age; Puchalski et al., 2006, pediatric cohort, USA [67], supports upright T wave in V1 and qR pattern as pediatric ECG evidence of RV hypertrophy. Pulmonary stenosis was nonsignificant, most likely because only three patients had severe PS.

4.21 qR pattern according to pulmonary hypertension, pulmonary stenosis, and Eisenmenger syndrome

qR pattern was significantly associated with pulmonary hypertension and Eisenmenger syndrome. It was present in 80.0% of severe PH and 87.5% of Eisenmenger patients, suggesting high specificity for advanced RV pressure overload. Murni et al., 2023, n=144 children with ASD, Indonesia [58], supports Q-wave/qR-related features in predicting PH. Umamy et al., 2025, n=98 adults with uncorrected secundum ASD, Indonesia [59], supports selected ECG markers as indicators of abnormal hemodynamics. Puchalski et al., 2006, pediatric cohort, USA [67], further supports qR pattern as an RV hypertrophy marker. The near-significant PS association was limited by the very small severe PS subgroup.

4.22 Overall interpretation

Echocardiography remains central for evaluating ASD anatomy and hemodynamic consequences. Anatomical ASD size, Qp/Qs, right-sided volume-overload subgroup, pulmonary hypertension, pulmonary stenosis, and Eisenmenger syndrome should not be merged. Large ASD refers to defect size; Qp/Qs to shunt magnitude; and right-sided volume-overload subgroup to downstream hemodynamic effect. The most consistent echocardiographic findings were increased RAA in severe pediatric PH, reduced TAPSE with increased RAA in adult severe PH, increased RAA in adult volume overload, and reduced FAC with increased RAA in Eisenmenger syndrome. Koestenberger et al., 2016 [50], supports RA size assessment in children with ASD/PH, while Moceri et al., 2012 [55], supports the prognostic importance of RV function and RA area in Eisenmenger syndrome.

ECG findings were more selective. P-wave dispersion, right axis deviation, iRBBB/cRBBB, and crochet sign were not significant discriminators of PH, PS, right-sided volume-overload subgroup, or Eisenmenger syndrome in several categorical analyses. However, previous ASD literature supports PWD, right-axis deviation, and iRBBB/cRBBB as common ASD-related markers of atrial conduction heterogeneity and right-sided volume loading. The nonsignificant results likely reflect mixed age distribution, categorical grouping, and subgroup imbalance, especially for right-sided volume overload. In contrast, R/S ratio in V1, T-wave inversion or age-adjusted positive RV strain pattern, and qR pattern were more strongly associated with severe PH, severe PS, Eisenmenger syndrome, or right-to-left shunt. These markers appear to reflect advanced RV pressure overload rather than simple volume loading. Therefore, ECG may help identify advanced pressure-overload physiology, but it cannot replace echocardiography for defining ASD anatomy, Qp/Qs, right-sided volume-overload subgroup, PH severity, pulmonary stenosis severity, or Eisenmenger physiology.

CONCLUSION

1. Atrial septal defect in the studied patients showed female predominance and a wide age distribution, with secundum ASD being the most common anatomical type and large ASD representing the predominant defect size.
2. Hemodynamically significant ASD was frequent, as many patients had large left-to-right shunt, right-sided volume overload, pulmonary hypertension, and a smaller but important proportion had Eisenmenger syndrome.
3. Larger ASD size was significantly associated with older age, iRBBB/cRBBB changes, and increased indexed right ventricular dimensions in both pediatric and adult patients.
4. Echocardiographic findings reflected disease

severity, particularly increased right atrial area with pulmonary hypertension and right-sided volume overload, reduced TAPSE in adults with severe pulmonary hypertension, and reduced FAC in patients with Eisenmenger syndrome.

5. ECG abnormalities showed variable relationships with ASD complications; however, R/S ratio in V1, T-wave inversion/POS, and qR pattern were significantly associated with severe pulmonary hypertension and Eisenmenger syndrome.

REFERENCES

1. Refaei M, Islam S, Mackie AS, Atallah J. Correlation of electrocardiogram parameters and hemodynamic outcomes in patients with isolated secundum atrial septal defects. Ann Pediatr Cardiol. 2017;10(2):152-157..
2. Moradian, Maryam, and Azin Alizadehasl, eds. Atlas of echocardiography in pediatrics and congenital heart diseases. Springer, 2021
3. Bartoszewska, Elżbieta, et al. "Atrial septal defects: From embryology to pediatric pulmonary hypertension." Journal of Clinical Medicine 14.21 (2025): 7698
4. Kashyap, Tejasvi, et al. "Transcatheter occluder devices for the closure of atrial septal defect in children: how safe and effective are they? A systematic review." Cureus 14.5 (2022).
5. Brida M, Chessa M, Celermajer D, Li W, Geva T, Khairy P, et al. Atrial septal defect in adulthood: a new paradigm for congenital heart disease. Eur Heart J. 2022;43(28):2660-2671.
6. Alaani, Samira Telfah Abdilghani, Firas Sadiq Alkubaisy, and Abdulqadir Abdulkareem Merie. "Epidemiological and clinical aspects of congenital heart disease in Fallujah Maternity and Children Hospital, Iraq." Iraq Medical Journal 7.2 (2023): 46-49.
7. Lelakowska, Maria, et al. "Transcatheter closure of atrial septal communication: impact on P-wave dispersion, duration, and arrhythmia in mid-term follow-up." Polish Heart Journal (Kardiologia Polska) 76.10 (2018): 1465-1473.
8. Martin, Seth S., Edward P. Shapiro, and Monica Mukherjee. "Atrial septal defects–clinical manifestations, echo assessment, and intervention." Clinical Medicine Insights: Cardiology 8 (2014): CMC-S15715.
9. Somura J, Nakagawa M, Ukiami M, Sagawa H, Furukawa O, Hoshino S, et al. Relationship between electrocardiographic signs and shunt volume in atrial septal defect. Pediatr Int. 2015;57(4):535-540.
10. Moss and Adam pediatric cardiology book/Atrial septal defect chapter28
11. Chen, Yu, et al. "A novel mutation in GATA4 gene associated with dominant inherited familial atrial septal defect." The Journal of thoracic and cardiovascular surgery 140.3 (2010): 684-687.
12. Sjöberg, P.; Clausen, H.; Arheden, H.; Liuba, P.; Hedström, E. Atrial septal defect closure in children at young age is beneficial for left ventricular function. Eur. Heart J. Imaging Methods Pract. 2024, 2, qyae058.
13. Borow, Kenneth M., and Robert Karp. "Atrial septal defect: lessons from the past, directions for the future." New England Journal of Medicine 323.24 (1990): 1698-1700.
14. Venkatram, Prabhakar. "Atrial Septal Defect: Types, Hemodynamic Changes, Diagnosis and Treatment Options." Heart Diseases and Echocardiogram: Principles in Practice. Cham: Springer Nature Switzerland, 2024. 193-205.
15. Bedford, D. Evan, Cornelio Papp, and John Parkinson. "Atrial septal defect." British Heart Journal 3.1 (1941): 37.
16. Uhley, M. H. "Lutembacher's syndrome and a new concept of the dynamics of interatrial septal defect." American Heart Journal 24.3 (1942): 315-328.
17. Barber JM, Magidson O, Wood P. Atrial septal defect: with special reference to the electrocardiogram, the pulmonary artery pressure and the second heart sound. British Heart Journal. 1950 Jul;12(3):277.
18. Campbell, Maurice. "Natural history of atrial septal defect." Heart 32.6 (1970): 820-826.
19. Laha, Somrita, et al. "Role of electrocardiogram in detecting congenital heart disease: prevalence and diagnostic accuracy of pathognomonic features." (2025).
20. MooRE, E. NEIL, et al. "Incomplete right bundle-branch block: An electrocardiographic enigma and possible misnomer." Circulation 44.4 (1971): 678-687.
21. Depasquale, N. P., and G. E. Burch. "Analysis of the RSR'Complex in Lead V1." Circulation 28.3 (1963): 362-367
22. Burch, George E., and Nicholas P. Depasquale. "Electrocardiogram and spatial vectorcardiogram of localized myocardial hypertrophy." Circulation 26.4 (1962): 544-552.
23. Fei Ho, Ting, et al. "Analysis of P wave and P dispersion in children with secundum atrial septal defect." Annals of noninvasive electrocardiology 6.4 (2001): 305-309.
24. Fei Ho T, Ling Chia E, Chin-Ling Yip W, Yee Chan K. Analysis of P wave and P dispersion in children with secundum atrial septal defect. Annals of noninvasive electrocardiology. 2001 Oct;6(4):305-9.
25. Khairy, Paul, and Ariane J. Marelli. "Clinical use of electrocardiography in adults with congenital heart disease." Circulation 116.23 (2007): 2734-2746.
26. Brink, A. J., and CATHERINE A. NEILL. "The electrocardiogram in congenital heart disease: with special reference to left axis deviation." Circulation 12.4 (1955): 604-611.

27. Ueda, Tadashi, et al. "Echocardiographic assessment of Qp/Qs in children with atrial septal defect or partial anomalous pulmonary venous connection." Japanese circulation journal 45.6 (1981): 639-645.
28. European Heart Journal (2023) 44 (Suppl 2) Imaging– Echocardiography, Congenital Heart Disease Qp/Qs in ostium secundum atrial septal defect mitigates right ventricular dysfunction in patients with secondary pulmonary hypertension E.C. Guerra1, N. Espinola-Zavaleta2,
29. Chinawa, Josephat, et al. "Predictors of pulmonary hypertension among children with atrial septal defects (ASD)." Libyan Journal of Medicine 17.1 (2022): 2007603.
30. Behjati-Ardakani, Mostafa, et al. "The clinical course of patients with atrial septal defects." Iranian journal of pediatrics 26.4 (2016): e4649.
31. Haque, Md Serajul, et al. "Pulmonary Artery Pressure Profile in Atrial Septal Defect (ASD) Patients." Bangladesh Heart Journal 30.1 (2015): 27-28.
32. Lammers AE, Apitz C, Amedro P, Bonnet D, Budts W, D'Alto M, et al. Echocardiographic assessment of the right heart in children and adults: Recommendations from the Association for European Paediatric and Congenital Cardiology. Cardiol doi:10.1017/S1047951122000209
33. Rudski LG, Lai WW, Afilalo J, Hua L, Handschumacher MD, Chandrasekaran K, et al. Guidelines for the echocardiographic assessment of the right heart in adults: A report from the American Society of Echocardiography. J Am Soc Echocardiogr. 2023;36(2):137181. doi:10.1016/j.echo.2022.10.006.
34. Grygier, Marek, et al. "Percutaneous closure of atrial septal defect: a consensus document of the joint group of experts from the Association of Cardiovascular Interventions and the Grown-Up Congenital Heart Disease Section of the Polish Cardiac Society." Polish Heart Journal (Kardiologia Polska) 78.10 (2020): 1066-1083.
35. Cardoso, Cristiano Oliveira, et al. "Effectiveness of the Amplatzer™ device for transcatheter closure of an ostium secundum atrial septal defect." Arquivos brasileiros de cardiologia 88 (2007): 384-389.
36. Li, L. I., et al. "Defective T wave combined with incomplete right bundle branch block: a new electrocardiographic index for diagnosing atrial septal defect." Chinese Medical Journal 125.6 (2012): 1057-1062.
37. Heller, Joseph, et al. ""'Crochetage"(notch) on R wave in inferior limb leads: a new independent electrocardiographic sign of atrial septal defect." Journal of the American College of Cardiology 27.4 (1996): 877-882.
38. Kuijpers JM, Mulder BJM, Bouma BJ. Secundum atrial septal defect in adults: a practical review and recent developments. Neth Heart J. 2015;23(4):205-211.
39. Eryu Y, Hata T, Nagatani A, Funamoto Y, Uchida H, Fujino M, et al. Electrocardiographic RR and QT interval variability in patients with atrial septal defect and healthy children. Pediatr Cardiol. 2017;38(5):993-1001.
40. Rasheed ZA, Sharif AM, Mohammad AM. Outcomes of congenital heart diseases in Duhok, Kurdistan Region of Iraq. SEEJPH. 2024;25(S1):1522-1529.
41. Geva T, Martins JD, Wald RM. Atrial septal defects. Lancet. 2014;383(9932):1921-1932.
42. Le Gloan L, Legendre A, Iserin L, Ladouceur M. Pathophysiology and natural history of atrial septal defect. J Thorac Dis. 2018;10(Suppl 24):S2854-S2863.
43. Silvestry FE, Cohen MS, Armsby LB, Burkule NJ, Fleishman CE, Hijazi ZM, et al. Guidelines for the echocardiographic assessment of atrial septal defect and patent foramen ovale. J Am Soc Echocardiogr. 2015;28(8):910-958.
44. Ribeiro LC, Silva RMFL, Tonelli HAF, Guimarães AFM, Araújo FDR, Meira ZMA, et al. Classifying the size of an atrial septal defect according to echocardiographic parameters and its association with the clinical presentation in pediatrics. Arq Bras Cardiol: Imagem Cardiovasc. 2025;38(2):e20240089.
45. Torres AJ. Hemodynamic assessment of atrial septal defects. J Thorac Dis. 2018;10(Suppl 24):S2882-S2889.
46. Baumgartner H, De Backer J, Babu-Narayan SV, Budts W, Chessa M, Diller GP, et al. 2020 ESC guidelines for the management of adult congenital heart disease. Eur Heart J. 2021;42(6):563-645.
47. Jain S, Dalvi B. Atrial septal defect with pulmonary hypertension: when/how can we consider closure? J Thorac Dis. 2018;10(Suppl 24):S2890-S2898.
48. Gabriels C, De Meester P, Pasquet A, De Backer J, Paelinck BP, Morissens M, et al. A different view on predictors of pulmonary hypertension in secundum atrial septal defect. Int J Cardiol. 2014;176(3):833-840.
49. Chinawa J, Chinawa AT, Ossai E, Duru C. Predictors of pulmonary hypertension among children with atrial septal defects. Libyan J Med. 2022;17(1):2007603.
50. Koestenberger M, Burmas A, Ravekes W, Avian A, Gamillscheg A, Grangl G, et al. Echocardiographic reference values for right atrial size in children with and without atrial septal defects or pulmonary hypertension. Pediatr Cardiol. 2016;37(4):686-695.
51. Cossio-Aranda J, Attie F, Espinola-Zavaleta N, Buendía-Hernández A, Zabal C, García-Montes JA, et al. Echocardiographic correlates of severe pulmonary hypertension in adult patients with

ostium secundum atrial septal defect. Echocardiography. 2016;33(12):1891-1898.
52. Pham T, Bracamontes-Castelo G, Antonio-Villa NE, Avendaño-Pérez L, Zamora KDV, Keirns C, et al. Evaluation of regional left and right ventricular function in patients with ostium-secundum atrial septal defect using speckle tracking echocardiography. Arch Clin Biomed Res. 2020;4(4):274-291. doi:10.26502/acbr.50170104.
53. Sirico D, Spigariol G, Mahmoud HT, Basso A, Cuppini E, Avesani M, et al. Acute changes in right ventricular function in pediatric patients with pulmonary valve stenosis undergoing percutaneous valvuloplasty: a speckle-tracking study. J Clin Med. 2023;12(13):4344. doi:10.3390/jcm12134344.
54. Kalogeropoulos AP, Border WL, Georgiopoulou VV, Pernetz MA, Howell S, McConnell M, et al. Right ventricular function in adult patients with Eisenmenger physiology: insights from quantitative echocardiography. Echocardiography. 2010;27(8):937-945. doi:10.1111/j.1540-8175.2010.01194.x.
55. Moceri P, Dimopoulos K, Liodakis E, Germanakis I, Kempny A, Diller GP, et al. Echocardiographic predictors of outcome in Eisenmenger syndrome. Circulation. 2012;126(12):1461-1468.
56. Morton JB, Sanders P, Vohra JK, Sparks PB, Morgan JG, Spence SJ, et al. Effect of chronic right atrial stretch on atrial electrical remodeling in patients with an atrial septal defect. Circulation. 2003;107(13):1775-1782.
57. Guray U, Guray Y, Yilmaz MB, Mecit B, Sasmaz H, Korkmaz S. Evaluation of P wave duration and P wave dispersion in adult patients with secundum atrial septal defect during normal sinus rhythm. Int J Cardiol. 2003;91(1):75-79.
58. Murni IK, Kato T, Wirawan MT, Arafuri N, Hermawan K, Hartopo AB, et al. An electrocardiographic score to predict pulmonary hypertension in children with atrial septal defect. BMC Pediatr. 2023;23:287.
59. Umamy KN, Martiana AK, Myrtha R, Irnizarifka I, Nursidiq AA. Electrocardiographic markers predict hemodynamic parameters in adults with uncorrected secundum atrial septal defect. Egypt Heart J. 2025;77:8.
60. Ho TF, Chia EL, Yip WCL, Chan KY. Analysis of P wave and P dispersion in children with secundum atrial septal defect. Ann Noninvasive Electrocardiol. 2001;6(4):305-309.
61. Salih AF, Brazinji SH. Value of P-wave dispersion in pediatric patients with secundum atrial septal defect. Electron J Gen Med. 2019;16(2):em116.
62. Fazelifar AF, Talebian F, Ghaffarinejad Z, Habibi MA, Pasebani Y, Mazloomi AA, et al. Electrocardiographic manifestations of pulmonary stenosis versus pulmonary hypertension. J Electrocardiol. 2023;81:117-122.
63. Raut MS. Electrocardiographic crochetage sign in atrial septal defect. Ann Card Anaesth. 2017;20(3):347-348.
64. Bayar N, Arslan S, Köklü E, Cagirci G, Cay S, Erkal Z, et al. The importance of electrocardiographic findings in the diagnosis of atrial septal defect. Kardiol Pol. 2015;73(5):331-336.
65. Goncalvesova E, Vargova V, Stenova E, Lazurova I, Payer J. Electrocardiographic signs of right ventricular hypertrophy in pulmonary arterial hypertension. Bratisl Lek Listy. 2011;112(7):381-385.
66. Waligóra M, Gliniak M, Bylica J, Pasieka P, Łączak P, Podolec P, et al. Extended precordial T wave inversions are associated with right ventricular enlargement and poor prognosis in pulmonary hypertension. J Clin Med. 2021;10(10):2147. doi:10.3390/jcm10102147.
67. Puchalski MD, Lozier JS, Bradley DJ, Minich LL, Tani LY. Electrocardiography in the diagnosis of right ventricular hypertrophy in children. Pediatrics. 2006;118(3):1052-1055.
68. Cool CJ, Khalid AF, Sukmadi N. Utilization of the flow ratio measured by echocardiography compared to the flow ratio by right heart catheterization for detecting Eisenmenger syndrome in uncorrected acyanotic adult congenital heart disease. BMC Med Imaging. 2025;25:136.
69. Dannenberg V, Goliasch G, Hengstenberg C, Binder T, Gabriel H, Schneider M. Detection of atrial shunt lesions with a single echocardiographic parameter. Wien Klin Wochenschr. 2020;132(11-12):295-300
70. Ocal A, Saricam E. Relationship between shunt size and right lead electrocardiographic findings in patients with secundum atrial septal defect. EJMI. 2020;4(2):265-268.
71. Nakayama R, Takaya Y, Akagi T, Nakagawa K, Watanabe N, Nobusada S, et al. Impact of right ventricular dilatation in patients with atrial septal defect. J Interv Cardiol. 2020;2020:9509105
72. Al-Naamani K, Hijal T, Nguyen V, Andrew S, Nguyen T, Huynh T. Predictive values of the electrocardiogram in diagnosing pulmonary hypertension. Int J Cardiol. 2008;127(2):214-218.