RESEARCH PAPER

ECHOCARDIOGRAPHIC ASSESSMENT OF PULMONARY HYPERTENSION IN PREGNANT WOMEN

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ABSTRACT

Background

pulmonary hypertension (PH) is hemodynamically defined as a resting mean pulmonary arterial pressure (mPAP) >20 mmHg measured via right heart catheterization**.**Pregnancy involves significant physiological cardiovascular adaptations, including increased circulating volume and cardiac output, which place additional stress on the pulmonary circulation and right ventricle. In women with pulmonary arterial hypertension (PAH), these changes may increase the risk of maternal cardiovascular complications and adverse pregnancy outcomes. Echocardiography provides a non-invasive method for assessing pulmonary hemodynamics and right ventricular function.

OBJECTIVES

The study aimed to assess pulmonary artery hypertension in pregnant women using transthoracic echocardiography and evaluate its association with maternal age and underlying clinical etiology. It also aimed to assess PAH severity using PASP/RVSP and compare its relationship with key right ventricular echocardiographic parameters.

Materials and Methods

A hospital-based cross-sectional observational study was conducted among 125 pregnant women aged 18–40 years, irrespective of trimester. Transthoracic echocardiography was performed using a LOGIQ P9 system. Tricuspid regurgitation velocity (TRV), tricuspid regurgitation pressure gradient (TRPG), pulmonary artery systolic pressure/right ventricular systolic pressure (PASP/RVSP), pulmonary acceleration time (PAT), tricuspid annular plane systolic excursion (TAPSE), inferior vena cava (IVC) diameter/collapse, and left ventricular ejection fraction (EF) were assessed. Participants were categorized according to PASP as normal, mild, moderate, or severe. Statistical analysis included chi-square test, analysis of variance, and Pearson correlation.

Results

Among the 125 participants, 46 (37.0%) were normal, 34 (27.2%) had mild, 25 (20.0%) had moderate, and 20 (16.0%) had severe PAH. Increasing maternal age was significantly associated with greater PAH severity (χ^2 = 152.988, $p < 0.001$). RVSP, TRV, TRPG, and IVC diameter progressively increased with increasing PAH severity, whereas PAT and TAPSE progressively decreased ($p < 0.001$). A significant association was also observed between PAH severity and etiology ($\chi^2 = 152.13$, df = 6, $p < 0.001$). Pearson correlation analysis demonstrated strong relationships between RVSP and the major echocardiographic parameters.

Conclusion

Increasing severity of pulmonary artery hypertension in pregnancy was significantly associated with advancing maternal age and changes in pulmonary hemodynamics and right ventricular systolic function. A multiparametric echocardiographic approach may provide useful non-invasive assessment for the identification, stratification, and monitoring of high-risk pregnant women.

Keywords

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Introduction

Pulmonary artery hypertension refers to the pathological increase in pressure within the pulmonary circulation. Based on haemodynamic criteria, pulmonary hypertension is identified when the mean pulmonary artery pressure exceeds 20 mmHg at rest, with confirmation obtained through right heart catheterisation. Pulmonary hypertension

is an important cardiovascular condition because persistent elevation of pulmonary pressure increases the workload on the right ventricle and may subsequently lead to right ventricular dysfunction and right heart failure. Pulmonary hypertension is clinically classified into five major groups according to the underlying disease mechanisms and associated clinical conditions. These include pulmonary arterial hypertension, pulmonary hypertension related to left heart disease, pulmonary hypertension associated with lung disease and hypoxia, pulmonary hypertension due to pulmonary artery obstruction, particularly chronic thromboembolic disease, and pulmonary hypertension with unclear or multifactorial mechanisms.Pulmonary arterial hypertension develops as a consequence of progressive pathological changes in the small pulmonary arteries. These changes cause narrowing and loss of the pulmonary vascular lumen, resulting in an increase in pulmonary vascular resistance. The increased resistance places a greater pressure load on the right ventricle. Persistent pressure overload may eventually result in right ventricular dysfunction and right heart failure. The disease process is influenced by multiple cellular and molecular mechanisms involving the pulmonary vascular system.As pulmonary vascular resistance increases, the right ventricle has to generate higher pressure to maintain pulmonary blood flow. Initially, the right ventricle may adapt to the increased afterload; however, persistent pressure overload can progressively affect right ventricular systolic function. Therefore, assessment of pulmonary pressure together with right ventricular function is important in the evaluation of pulmonary hypertension.Pulmonary arterial hypertension may develop without an identifiable underlying cause and is then classified as idiopathic PAH. A proportion of patients have a hereditary form related to genetic susceptibility. PAH may also occur secondary to other medical conditions, particularly connective tissue diseases, congenital heart disease, portal hypertension and HIV infection. Exposure to certain drugs and toxins has also been recognized as an associated cause of PAH.The underlying clinical condition may influence the development and severity of pulmonary vascular disease. Therefore, evaluation of the clinical etiology is important when assessing pulmonary hypertension, particularly in pregnant women in whom cardiovascular and haemodynamic changes may influence disease progression.Pregnancy is associated with substantial haemodynamic adaptations, including an increase in circulating blood volume and cardiac output. These physiological changes can impose additional stress on the right side of the heart in women with pulmonary arterial hypertension, particularly when pulmonary vascular resistance is already elevated. The limited ability of the pulmonary circulation and right ventricle to accommodate these changes contributes to the increased maternal and fetal risk associated with PAH during pregnancy.The cardiovascular system undergoes progressive changes throughout pregnancy to meet the increased circulatory requirements of the mother and developing fetus. Although these changes are well tolerated in healthy women, they may reveal previously unrecognized cardiac abnormalities or aggravate pre-existing cardiovascular conditions. In women with pulmonary hypertension, the increased haemodynamic demand may place additional stress on an already compromised pulmonary circulation and right ventricle.Pregnancy is accompanied by significant physiological adaptations in the cardiovascular system. Maternal blood volume and cardiac output progressively increase during pregnancy. Cardiac output begins to increase from the early stages of pregnancy and reaches its maximum during the early third trimester, when it is approximately one-third higher than the non-pregnant value. Maternal plasma volume also increases considerably, with the greatest expansion occurring during the later stages of gestation.Heart rate and stroke volume increase during pregnancy and contribute to the rise in cardiac output. At the same time, systemic vascular resistance decreases, allowing the maternal circulation to accommodate the increased blood flow associated with pregnancy. These changes are necessary to support the increased metabolic requirements of both the mother and fetus.The haemodynamic adaptations occurring during pregnancy can place additional stress on women with pulmonary arterial hypertension. The increase in circulating volume and cardiac output, together with changes in systemic vascular resistance, increases the demand placed on the right ventricle. In the presence of pre-existing pulmonary vascular disease, the right ventricle may have limited capacity to accommodate these changes. This may increase the risk of right ventricular dysfunction and heart failure during pregnancy.The interaction between pregnancy-related cardiovascular adaptations and pulmonary vascular disease makes assessment of pulmonary pressure and right ventricular function particularly important. Early identification of increased pulmonary pressures can help in recognizing pregnant women who may require closer cardiovascular monitoring.Pulmonary hypertension during pregnancy is associated with an increased risk of adverse outcomes for both the mother and fetus. Increased pulmonary vascular resistance and right ventricular pressure overload may compromise maternal cardiovascular adaptation during pregnancy. With increasing severity of pulmonary hypertension, the haemodynamic burden on the right ventricle may

become more pronounced and may contribute to cardiovascular complications.

Therefore, evaluation of pulmonary hypertension severity and right ventricular function is important in pregnant women. Echocardiographic assessment provides a non-invasive approach for evaluating pulmonary haemodynamics and detecting changes associated with increasing pulmonary pressure.The clinical presentation of pulmonary hypertension may vary according to the severity of disease and the underlying clinical condition. Symptoms may be nonspecific and may overlap with the normal physiological changes experienced during pregnancy. Consequently, clinical assessment alone may not be sufficient to identify the degree of pulmonary haemodynamic abnormality.The evaluation of pulmonary hypertension involves clinical assessment and cardiovascular investigations. Right heart catheterisation provides definitive haemodynamic assessment; however, echocardiography has an important role as a non-invasive method for estimating pulmonary pressures and evaluating cardiac structure and function.Transthoracic echocardiography is a useful non-invasive method for evaluating pulmonary hypertension. It provides information about pulmonary pressure estimates as well as the structural and functional response of the right ventricle to increased afterload. Important echocardiographic parameters include tricuspid regurgitation velocity, tricuspid regurgitation pressure gradient, pulmonary artery systolic pressure/right ventricular systolic pressure, pulmonary acceleration time, tricuspid annular plane systolic excursion and inferior vena cava .Assessment of these parameters together provides a more comprehensive evaluation of pulmonary haemodynamics and right ventricular function.Early recognition and assessment of pulmonary hypertension during pregnancy are clinically important because the physiological haemodynamic changes of pregnancy may increase the burden on the pulmonary circulation and right ventricle. Women with pulmonary hypertension are at increased risk of maternal cardiovascular complications and adverse pregnancy outcomes. Therefore, identification of elevated pulmonary pressures and assessment of right ventricular function are important for recognizing women who may require closer cardiovascular monitoring and specialized management.

Aim: To assess the pulmonary artery hypertension in pregnant women by using echocardiography.

Objectives:

- To assess the prevalence and severity of pulmonary artery hypertension among pregnant women using echocardiographic parameters.
- To evaluate the association between maternal age and the severity of pulmonary artery hypertension among pregnant women.
- To assess the relationship between pulmonary arterial pressure and right ventricular functional parameters using echocardiography.

Materials and Methods

A cross-sectional study was conducted among pregnant women attending Vels Medical College and Hospital during a 6-month study period from September 2025 to February 2026. The study included pregnant women who underwent transthoracic echocardiographic evaluation during the study period. A total of 125 pregnant women, aged 18–40 years and belonging to any trimester of pregnancy, were included in the study.

Inclusion and Exclusion Criteria

Pregnant women aged 18–40 years who were willing to participate, underwent transthoracic echocardiography during the study period, and provided informed consent were included. Women who were unwilling to participate, had inadequate echocardiographic images, incomplete clinical or echocardiographic data, severe systemic illness such as renal failure or sepsis, or poor echocardiographic windows were excluded.

Echocardiographic Assessment

Ultrasound evaluation was conducted using a GE logic P9 ultrasound system to assess the pulmonary artery hypertension in pregnant women. To obtain the best images, the patients should be in supine position and turn to left lateral position and the settings were manually changed. The American Society of Echocardiography (ASE) recommendations were followed for all two – dimensional (2D) and standard Doppler measure. Echocardiographic pulmonary hypertension was defined according to the prespecified echocardiographic criterion used in this study, based on estimated PASP/RVSP and/or TR velocity.

TAPSE was measured using M-mode at the lateral tricuspid annulus in the apical four-chamber view. IVC diameter were assessed using standard echocardiographic views. The inferior vena cava (IVC) diameter was assessed during quiet respiration, with IVC diameter less than 2.1 cm considered normal and diameter greater than 2.1 cm considered dilated. IVC respiratory collapsibility was also assessed to estimate right atrial pressure. PASP was estimated using the simplified Bernoulli equation from the TR velocity with estimated right atrial pressure derived from IVC characteristics. The current ASE echocardiographic severity ranges, PASP was categorized as normal (<30 mmHg), mildly elevated (30–50 mmHg), moderately elevated (50–70 mmHg), or severely elevated (>70 mmHg). PH was assessed echocardiographically using the study's predefined PASP/RVSP-based criterion. A

formal multiparametric echocardiographic probability algorithm was not used. Right-heart catheterization was not performed in any participant; therefore, the study assessed echocardiographic evidence suggestive of pulmonary hypertension rather than invasively confirmed pulmonary hypertension.

Statistical Analysis

Data were analyzed statistically to evaluate the distribution of pulmonary hypertension severity and its association with demographic and echocardiographic variables. The chi-square test was used to assess categorical associations, while analysis of variance (ANOVA) was used to compare echocardiographic parameters across severity groups. Pearson correlation analysis was performed to determine relationships between RVSP and selected echocardiographic parameters. A P value <0.05 was considered statistically significant.

Results

A total of 125 pregnant women aged 18–40 years were included in the study and categorized according to the study-defined pulmonary hypertension severity groups. Of the 125 participants, 46 (37%) were classified as normal, 34 (27.2%) had mild pulmonary hypertension, 25 (20.0%) had moderate pulmonary hypertension, and 20 (16.0%) had severe pulmonary hypertension.

Association Between Maternal Age and Pulmonary Hypertension Severity

The distribution of pulmonary hypertension severity differed significantly across maternal age groups. Among women aged 20–25 years, 41 were in the normal group and 1 had mild pulmonary hypertension. In the 25–30-year age group, 5 participants were normal, 31 had mild, 12 had moderate, and 3 had severe pulmonary hypertension. Among women aged >30 years, 2 had mild, 13 had moderate, and 17 had severe pulmonary hypertension. The association between maternal age group and pulmonary hypertension severity was statistically significant ($\chi^2 = 152.988$, df = 6, P < 0.001).

Echocardiographic Parameters According to Severity

RVSP increased progressively with increasing severity, from 23.63 ± 3.57 mmHg in the normal group to 32.79 ± 1.72 mmHg, 62.76 ± 4.28 mmHg, and 76.10 ± 2.85 mmHg in the mild, moderate, and severe groups, respectively (F = 167.47, P < 0.001).

PAT showed a progressive reduction across severity groups, with mean values of 132.52 ± 4.87 ms, 113.38 ± 5.18 ms, 90.84 ± 4.65 ms, and 73.35 ± 3.13 ms, respectively (F = 907.301, P < 0.001).

Similarly, TAPSE decreased progressively from 20.65 ± 2.59 mm in the normal group to 16.97 ± 0.39 mm, 14.92 ± 1.08 mm, and 11.45 ± 1.10 mm in the mild, moderate, and severe groups, respectively (F = 150.624, P < 0.001).

TR jet velocity increased from 2.322 ± 0.323 m/s in the normal group to 2.941 ± 0.093 m/s, 3.684 ± 0.191 m/s, and 4.840 ± 0.209 m/s in the mild, moderate, and severe groups, respectively (F = 587.053, P < 0.001).

TRPG also increased progressively, from 18.63 ± 3.57 mmHg in the normal group to 27.79 ± 1.72 mmHg, 52.96 ± 5.15 mmHg, and 66.55 ± 3.15 mmHg, respectively (F = 1132.482, P < 0.001).

Mean IVC diameter increased from 15.91 ± 3.15 mm in the normal group to 21.35 ± 0.54 mm, 24.24 ± 1.90 mm, and 28.85 ± 1.93 mm across increasing severity categories (F = 176.384, P < 0.001).

Mean EF was 62.22 ± 3.58%, 62.12 ± 3.35%, 58.00 ± 7.76%, and 58.45 ± 5.46% in the normal, mild, moderate, and severe groups, respectively. The difference was statistically significant (P = 0.001).

Association With Etiology

Pulmonary hypertension severity was significantly associated with the underlying broad etiological category ($\chi^2 = 152.13$, df = 6, P < 0.001). Mild, moderate, and severe pulmonary hypertension were predominantly observed among participants with secondary etiologies, whereas primary etiology was mainly associated with moderate and severe disease.

Correlation Analysis

RVSP demonstrated strong to very strong correlations with the principal echocardiographic parameters. RVSP showed a very strong negative correlation with PAT (r = −0.952, P < 0.001) and a strong negative correlation with TAPSE (r = −0.841, P < 0.001). Positive correlations were observed with TR jet velocity (r = 0.942, P < 0.001) and TRPG (r = 0.986, P < 0.001).

Table 1. Distribution of Participants According to Pulmonary Hypertension Severity

Severity category	n	%
Normal	46	37.0
Mild	34	27.2
Moderate	25	20.0
Severe	20	16.0
total	125	100

Table 2. Maternal Age Distribution According to Pulmonary Hypertension Severity

Age group(yr	Normal	Mild	Moderate	Severe

s)				
20-25	41	1	0	0
25-30	5	31	12	3
>30	0	2	13	17

$\chi^2 = 152.988$, df = 6, P < 0.001.

Table 3 Patient Distribution by Broad Etiology category

Variable	X^2 Value	df	P value	Interpretation
PAH vs Etiology	152.13	6	<0.001	Highly Significant

Table 4 Echocardiographic Parameters According to Severity Table

Parameter	Normal	Mild	Moderate	Severe	P value
RVSP(mmHg)	23.63 ±3.57	32.79 ±1.72	62.76 ±4.28	76.10 ±2.85	<0.001
PAT(ms)	132.52±4.87	113.38±5.18	90.84 ±4.65	73.35 ±3.13	<0.001
TAPSE(mm)	20.65 ±2.59	16.97 ±0.39	14.92 ±1.08	11.45 ±1.10	<0.001
TR jet velocity(m/s)	2.322 ±0.323	2.941 ±0.093	3.684 ±0.191	4.840 ±0.209	<0.001
TRPG(mmHg)	18.63 ±3.57	27.79 ±1.72	52.96 ±5.15	66.55 ±3.15	<0.001
IVC diameter(mm)	15.91 ±3.15	21.35 ±0.54	24.24 ±1.90	28.85 ±1.93	<0.001
EF(%)	62.22 ±3.58	62.12 ±3.35	58.00 ±7.76	58.45 ±5.46	0.001

Table 5 Pearson Correlation Analysis

Variables	Pearson's r	P value
RVSP vs PAT	-0.952	<0.001
RVSP vs TAPSE	-0.841	<0.001
RVSP vs TR jet velocity	0.942	<0.001
RVSP vs TRPG	0.986	<0.001

Figure 1 Distribution of Study Participants according to pulmonary artery hypertension severity

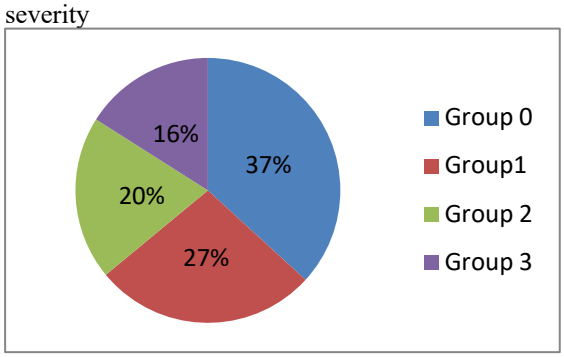


Figure 2 Age wise distribution of pulmonary artery hypertension severity

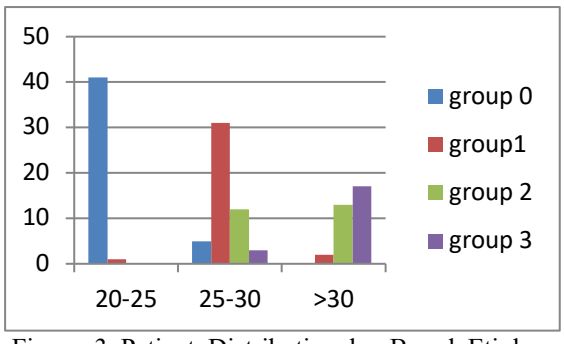
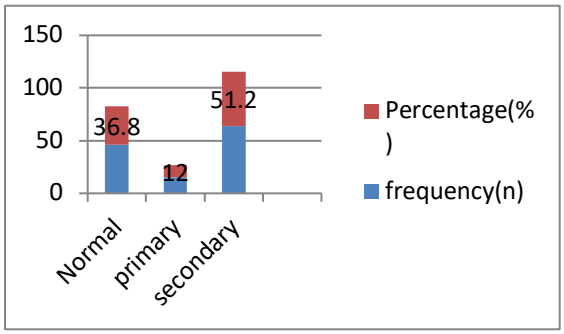


Figure 3 Patient Distribution by Broad Etiology category



Discussion

In the present study, maternal age demonstrated a statistically significant association with study-defined pulmonary hypertension severity (χ^2 = 152.988, df = 6, P < 0.001). The distribution showed a clear age-related pattern: among women aged 20–25 years, 41 were in the normal group and only 1 had mild pulmonary hypertension, with no moderate or severe cases. In the 25–30-year group, mild pulmonary hypertension predominated, while moderate and severe cases were also present. Among women aged >30 years, no participant was classified as normal, while 13 had moderate and 17 had severe pulmonary hypertension. This finding suggests that, within the present study population, increasing maternal age was associated with increasing echocardiographic pulmonary pressure severity.

A study from a tertiary institution evaluating pulmonary hypertension in pregnancy specifically examined the relationship between maternal age and severity. The authors reported that the severity of pulmonary hypertension and maternal morbidity appeared to increase with maternal age. Their data showed that women aged 36–40 years had severe pulmonary hypertension in 2 of 5 cases and a higher proportion of maternal morbidity compared with some of the younger age groups. The authors therefore discussed maternal age as a factor associated with increasing severity and morbidity in their cohort. This observation is closely related to the present study. Similarly, our study demonstrated that severe pulmonary hypertension was predominantly observed in women aged >30 years, whereas almost all women aged 20–25 years were in the normal group. Thus, the age-related distribution observed in our study is in agreement with the pattern described in this previous pregnancy cohort.Further evidence comes from a retrospective study of 154 pregnant women with pulmonary hypertension, in which patients were categorized into mild, moderate, and severe groups according to pulmonary artery systolic pressure. The authors found a statistically significant difference in maternal age among the three severity groups (P = 0.015). The mean ages were 31.2 ± 5.1 years in the mild group, 32.5 ± 4.8 years in the moderate group, and 29.1 ± 5.1 years in the severe group. Although this study demonstrated a significant difference in age across severity categories, it did not demonstrate the same progressive increase in age with increasing severity that was observed in our study. In fact, the severe group had a lower mean age than the mild and moderate groups. This difference may reflect variations in study population, underlying etiology, referral pattern, and clinical characteristics. Therefore, this study supports the relevance of maternal age as a variable in pregnancy-associated pulmonary hypertension but indicates that the relationship between age and severity may not be linear in every population.A separate retrospective study of 71 pregnant women with pulmonary hypertension demonstrated a pattern more closely resembling our findings. The authors reported mean maternal ages of 20.1 ± 5.2 years in the mild group, 25.2 ± 4.9 years in the moderate group, and 32.7 ± 5.3 years in the severe group, with a statistically significant difference between severity groups (P = 0.035). The progressive increase in mean age from the mild to the severe group is particularly relevant to the present study. In our population, the younger age group was predominantly normal or mildly affected, whereas moderate and severe categories were concentrated among older women. Therefore, both studies demonstrate a significant relationship between maternal age and pulmonary hypertension severity, although the age ranges and study populations differed.

In contrast, another multicenter retrospective study involving 181 critically ill pregnant women with pulmonary hypertension found a statistically significant difference in age across severity groups (P = 0.014), but the distribution did not demonstrate a simple progressive increase in age with increasing severity. The median age was lower in the severe group, and women aged ≤25 years represented a greater proportion of the severe group than in the mild group. This finding contrasts with the age pattern observed in our study. The difference may be explained by the fact that this study included critically ill pregnant women with established pulmonary hypertension, whereas our study included pregnant women undergoing echocardiographic assessment and classified them according to study-defined pulmonary pressure categories. Thus, differences in case selection and disease spectrum may influence the observed relationship between age and severity.

The findings of the present study can also be considered in relation to the larger systematic review and meta-analysis by El Iskandarani et al., which included 51 studies involving 4,583 pregnancies complicated by pulmonary hypertension. The reported mean maternal age was approximately 28 years, while the mean systolic pulmonary artery pressure was approximately 65 mmHg; the pooled population included mild,

moderate, and severe pulmonary hypertension. Although this meta-analysis did not establish maternal age as a direct predictor of pulmonary hypertension severity, it provides a broader reference for the demographic and hemodynamic characteristics of pregnant women with pulmonary hypertension. The mean age reported in that meta-analysis is comparable to the age range represented in the present study.

Taken together, previous studies demonstrate that maternal age has been associated with pulmonary hypertension characteristics during pregnancy, but the direction and strength of the relationship with disease severity are not uniform. Some studies have demonstrated increasing severity or morbidity with increasing maternal age, while others have demonstrated significant differences in age between severity groups without a linear age–severity relationship.The present study demonstrated a particularly clear age-related distribution, with moderate and severe study-defined pulmonary hypertension occurring predominantly in women aged >30 years. This finding is supported by the study in which severe pulmonary hypertension was associated with a higher mean maternal age, while it differs from studies in which younger women were also represented substantially among severe cases. These differences emphasize that maternal age alone should not be considered a determinant of pulmonary hypertension severity. Rather, age should be interpreted together with the underlying etiology, clinical condition, and echocardiographic findings.

Limitations

This study has several limitations. It was a single-center, cross-sectional study involving 125 pregnant women, which limits the generalizability of the findings and does not allow assessment of changes over time. Pulmonary pressure was evaluated using echocardiographic measurements rather than invasive hemodynamic confirmation. In addition, the severity categories were based on the pressure thresholds used in the present study and should therefore be interpreted within the context of this study. Longitudinal maternal and fetal outcomes were not assessed.

Conclusion

Our study concluded that an increasing PAH severity was observed with increasing maternal age. Older age groups showed a greater proportion of moderate and severe PAH compared with younger age groups. This finding suggest that maternal age is an important factor associated with PAH severity during pregnancy and also highlighting the importance of age wise assessment and early echocardiographic evaluation for appropriate monitoring of pregnant women.

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