

ROLE OF PULMONARY ARTERY ACCELERATION TIME IN INFANTS OF DIABETIC MOTHERS IN ASSESSEMENT OF PULMONARY ARTERY PRESSURE

Fatma Mostafa Yahia Abdl Aal¹, Waleed Mohamed Elguindy², Mohamed Omar Dawoud² and Eman Mohamed EL sayed²

ABSTRACT:

¹Eltahrir Hospital

²Department of Pediatrics; Faculty of Medicine, Ain Shams University. Cairo, Egypt

Corresponding author:

Fatma Mostafa Yahia Abdl-Aal.
Mobile: +002-01005710601

E-mail:

fatmamostafa081@gmail.com

Received: 24/11/2022

Accepted: 4/3/2023

Online ISSN: 2735-3540

Background: *Infants of diabetic mothers (IDMs) have impaired myocardial performance and are at risk of pulmonary hypertension.*

Aim of the work: *to evaluate the precision of doppler echocardiography-derived pulmonary artery acceleration time (PAAT) in estimating pulmonary artery pressure in infants of diabetic mothers and to compare it to the conventional method by using tricuspid regurgitation velocity.*

Studied cases and techniques: *A comparative cross-sectional study, which included (30) infants of diabetic mothers, with a mean age of 3.4 ± 1.5 days, who were admitted to the neonatal intensive care unit (NICU) of Ain Shams University's hospitals, because of elevated pulmonary pressure, as a case group, and (30) healthy age- and gender-matched neonates as a control group. Transthoracic echocardiography & doppler echocardiography-derived PAAT was done at 1st week of life.*

Results: *The study revealed a substantially significant decrease in the level of PAAT ($p < 0.001$) and an increase in the level of systolic pulmonary artery pressure (SPAP) in the group of IDM diagnosed with pulmonary arterial hypertension compared with the control group. Also, according to research, there was a substantial negative relationship found among the level of PAAT (ms) & level of SPAP (mmHg) in the cases group ($r = -0.984$ and $p < 0.001$). PAAT ≤ 72 ms had 96.67% sensitivity and 93.33% in identifying studied cases with pulmonary arterial hypertension in IDMs.*

Conclusions: *PAAT measures can be used as an alternative to the conventional method. PAAT ≤ 72 ms can serve as a strong, non-invasive predictor of pulmonary arterial hypertension in infants of diabetic mothers.*

Keywords: *pulmonary arterial hypertension, IDM, pulmonary artery acceleration time, systolic pulmonary artery pressure*

INTRODUCTION:

Diabetes mellitus (DM) is a common pregnancy medical problem, affecting 0.5 percent to five percent of all pregnancies. It can happen before pregnancy or throughout pregnancy for the first time^[1].

Maternal diabetes mellitus is linked to an improved risk of neonatal respiratory distress syndrome, accounting for approximately 2.2 percent to twenty percent of persistent pulmonary hypertension (PPHN) cases in late preterm & term infants (> 34 weeks gestational age)^[2].

PPHN is a syndrome characterised by a sustained elevation of pulmonary vascular resistance with severe hypoxemia caused by extrapulmonary shunting of deoxygenated blood right-to-left across patent ductus arteriosus & patent foramen ovale [3].

occurrence of PPHN is ~2/1,000 live births, with term & late preterm infants being most affected. With mortality rate of four-thirty three percent, PPHN remains 1 of leading reasons for neonatal morbidity & mortality[4].

IDMs are more likely to have the foetal circulation condition persist, which can cause cardiorespiratory distress in the first 24 hours of life. Why high pulmonary resistance continues is a mystery. Atypical respiratory distress syndrome, hypoglycemia, hyperviscosity, and idiopathies are possible causes[5].

Pulmonary arterial hypertension (PAH) monitoring by pulsed-wave doppler echocardiography using pulmonary artery acceleration duration (PAAT) is non-invasive, quantitative, & easily evaluated than tricuspid regurgitation measurement[6]. This is due to the fact that PAAT is available in more than ninety percent of studied cases, but the tricuspid regurgitation (TR) envelope is frequently insufficient, frequently unavailable, and only detectable in < 0.5 of studied cases with bronchopulmonary dysplasia (BPD) & PAH[6-7]. Many people mistake the pressure gradient for the TR quantity. The right ventricular systolic pressure (RVSP) won't represent the SPAP if there is any right ventricular outflow obstruction or significant left-to-right shunts [8].

In cases of severe TR, right atrial pressure will rise quickly throughout systole, underestimating the RVSP. Values shouldn't be reported unless there is clear trace since if not there is a possibility of major underestimate. Many patients will have no or just a minimal TR with an unclear spectral doppler TR trace or ventricular

septal defect (VSD) ejecting into site of tricuspid valve. Additionally, doppler interrogation beam must be 20° or less from the jet direction. Some unusual jets make this impossible sometimes[8].

In case of right ventricular failure & right ventricular outflow tract obstruction, the calculation of SPAP by measuring TR is unreliable. It is possible to miss tricuspid valve regurgitation, which affects 60 to 85% of PPHN patients[9].

According to the pulmonary artery (PA) pressure used to characterize pulmonary hypertension (PH), a meta analysis of twenty nine researches comparing echo-derived TR gradient and cardiac catheterization-derived PA pressure measures indicated sensitivity of eighty three percent & specificity of seventy two percent for diagnosis of PH [10]. However, a pediatric investigation demonstrated only a slight association ($R^2 = 0.36$, $P < 0.01$) between direct measurements by cardiac catheterization and estimated right ventricular pressures by echo[11]. A prospective study done by *Roushdy et al.*, [12] tricuspid regurgitation velocity (TRV) cutoff value of >3.96 m/s had 66.7 percent sensitivity & one hundred percent specificity to define pulmonary vascular resistance (PVR) by cardiac catheterization ($PVR_{CATH} > \text{six WU/m}^2$ in study of 175 studied cases with PH secondary to congenital heart disease (CHD).

AIM OF THE STUDY:

Aim of the present study was to assess the precision of Doppler echocardiography-derived PAAT in estimating pulmonary arterial hypertension in infants of diabetic mothers & compare it to the conventional method.

PATIENTS & METHODS:

The current research was a comparative cross-sectional study that was conducted on

30 infants of diabetic mothers with a mean age of 3.4 ± 1.5 days as a case group and 30 age- and gender-matched healthy neonates of non-diabetic mothers as a control group. All cases were recruited from the neonatal intensive care unit, Ain Shams University's hospitals, Cairo, Egypt, over a 1-year period (April 2021 to April 2022).

Infants were considered eligible for inclusion if born to a diabetic mother during pregnancy and diagnosed with pulmonary arterial hypertension. We excluded neonates with multiple congenital anomalies and infants diagnosed with right ventricular outflow obstruction. The control group will be recruited from the observatory room. Relevant maternal and neonatal demographic data would be extracted from the medical records. A thorough clinical test was completed on both cases and control groups, laying stress on anthropometric measures (weight in kg, length in cm), vital data (heart rate), blood pressure, and cardiac examination, and searching for signs of pulmonary hypertension.

Echocardiography Assessment:

The most commonly used echocardiographic method for pulmonary artery pressure (PAP) estimation is a measurement of the pressure gradient caused by tricuspid regurgitation among right ventricle and right atrium. All transthoracic echocardiography was done using a MH70 machine (Sumsung Medical Systems). Pulmonary artery acceleration time was measured by positioning the cursor just beneath the pulmonary valve annulus in the center of the distal right ventricular outflow tract. This site decreases patent ductus arteriosus (PDA) flow contamination of the doppler signal at the mean pulmonary artery. Pulsed wave sweep speed was set to maximum, and gain was adjusted to generate a clean, uniform doppler profile in order to enhance flow features and measurement accuracy. The resulting envelope was then used to calculate PAAT.

Statistical Analysis:

Data were collected, revised, coded, & entered into IBM Statistical Package for Social Science (SPSS) version twenty three Statistical Package for Social Science. Means, standard deviations, and ranges of quantitative data with a parametric distribution were presented. Qualitative variables were also presented numerically and as percentages. When the predicted count in any cell was < five, **chi-square test & fisher exact test** were used to compare groups with qualitative data.

An independent t-test was used to compare 2 independent groups with quantitative data & parametric distribution.

Pearson relationship coefficients were used to evaluate the relationship among 2 quantitative parameters in the same group. A p-value of <0.05 was considered significant.

The receiver operating characteristic (ROC) curve was used to evaluate the best cut-off point for PAAT (ms) & SPAP (mm Hg) with its sensitivity, specificity, positive predictive value, negative predictive value, and area under the curve.

RESULTS:

The study involved 60 neonates: 30 babies with pulmonary hypertension and diabetes from diabetic moms and 30 babies as controls. The control group had a mean age of 4.5 ± 1.8 days and 46.7% of its members were female, compared to the IDM group's mean age of 3.4 ± 1.5 days with 50% of its members being female. There was no substantial variation in their gender among them ($p=0.796$).

The case group's mean weight was 3.63 ± 0.76 kg, while the control group's mean weight was 3.27 ± 0.28 kg. When compared to the healthy control group, there was a substantial rise in the diabetic group's infants' birth weight and length (p-values = 0.018 and 0.007, respectively). The case group's mean systolic blood pressure (SBP)

was 66.3 ± 8.4 mmHg and its mean diastolic blood pressure (DBP) was 42.2 ± 6.0 mmHg, whereas the control group's mean systolic blood pressure was 69.1 ± 6.8 mmHg and its mean diastolic blood pressure was 43.3 ± 4.2 mmHg. Regarding blood pressure (BP), there were no substantial variations among the case group and control group (SBP, DBP, and mean BP).

The IDM group's gestational age was lower than the control group's (p-value = 0.002), with a mean gestational age of 36.4 ± 2.0 weeks (wks) in the case group and 37.7 ± 0.9 wks in the control group. There was a substantially higher rate of caesarean sections (CS) among the case group with $p=0.040$, as there were 28/30 cases (93.3%) with a CS as the mode of delivery compared to 22/30 of the controls (73.3%). All IDM cases with a pulmonary hypertension diagnosis required oxygen support. In 6/30 of the cases (20%), nasal oxygen was required; 9/30 of the cases (30%) required continuous positive airway pressure; and 15/30 of the cases (50%) required invasive mechanical ventilation as oxygen support, as shown in Table 1.

The mean systolic pulmonary arterial pressure derived from tricuspid regurgitation in the case group was 67.27 ± 18.86 mm Hg. while the mean level of SPAP in the control group was 28.83 ± 9.68 mm Hg. The mean

value of pulmonary artery acceleration time in the IDM group was 44.6 ± 17.69 ms, while the mean value of PAAT in the control group was 113 ± 13.13 ms, with a substantially higher level of PAAT and a higher level of SPAP in the group of IDM diagnosed with pulmonary arterial hypertension than the control group (p-value <0.001 and < 0.001). Our study revealed a substantial increase in the percentage of moderate and severe pulmonary hypertension in the cases group (23.3 percent and 70.0 percent, respectively) compared to mild pulmonary hypertension. As found in Table 2.

There was a substantial negative relationship found among the level of PAAT (ms) and level of SPAP (mmHg) in case group with ($r=-0.984$) and ($p <0.001$): as shown in Table 3.

A ROC curve analysis was used to define specific cut-off values for PAAT and SPAP in predicting pulmonary hypertension. PAAT 72 msec had a sensitivity of 96.67% and a specificity of 93.33%, with an area under the ROC curve of 0.978 (0.94-0.99, a 95% confidence interval (CI)). SPAP > 36 mmHg resulted in a sensitivity of 93.33% and a specificity of 90.0%, with an area under the ROC curve of 0.928 (0.90- 0.93, ninety-five percent CI), as shown in Figure 1.

Table (1): Comparison between control group and cases group regarding demographic data neonatal characteristics and neonatal outcomes.

		Control (n=30)	Cases (n=30)	Test value	P value	Sig.
Gender	Male	16(53.3%)	15(50.0%)	0.070*	0.796	NS
	Female	14(46.7%)	15(50.0%)			
Age (days)	Mean±SD	4.5 ± 1.8	3.4 ± 1.5	2.470•	0.017	S
	Range	2 – 7	2 – 6			
Weight (Kg)	Mean±SD	3.27 ± 0.28	3.63 ± 0.76	2.440•	0.018	S
	Range	2.8 – 4.0	2.2 – 5.5			
Length (cm.)	Mean±SD	45.9 ± 1.6	47.7 ± 3.1	2.790•	0.007	HS
	Range	43.0 – 49.0	43 – 56			
Gestational age (weeks)	Mean±SD	37.7 ± 0.9	36.4 ± 2.0	3.270•	0.002	HS
	Range	35 – 39	32 – 39			
SBP (mmHg)	Mean±SD	69.1 ± 6.8	66.3 ± 8.4	1.420•	0.162	NS
	Range	58 – 82	54 – 86			

Role Of Pulmonary Artery Acceleration Time In Infants Of Diabetic Mothers In Assesment Of..

DBP (mmHg)	Mean±SD	43.3 ± 4.2	42.2 ± 6.0	0.800•	0.429	NS
	Range	36 – 53	29 – 60			
Mean BP (mmHg)	Mean±SD	51.9 ± 4.1	51.0 ± 7.0	0.630•	0.531	NS
	Range	46 – 61	35 – 69			
Method of delivery	CS	22 (73.3%)	28 (93.3%)	4.320*	0.040	S
	NVD	8 (26.7%)	2 (6.7%)			
Oxygen support	Nasal	0 (0.0%)	6 (20.0%)	71.260*	<0.001	HS
	CPAP	0 (0.0%)	9 (30.0%)			
	IMV	0 (0.0%)	15 (50.0%)			
	No	30 (100.0%)	0 (0.0%)			

*: Chi-square test; •: Independent t-test

Table (2): Comparison between control group and cases group regarding transthoracic echocardiography parameters.

		Control (n=30)	Cases (n=30)	Test value	P value	Sig.
PAAT	Mean±SD	113.27 ± 13.13	44.60 ± 17.69	17.073	<0.001	HS
	Range	68 – 130	25 – 110			
SPAP severity	Normal	30 (100.0%)	0 (0.0%)	52.941	<0.001	HS
	Mild	0(0.0%)	2 (6.7%)			
	Moderate	0 (0.0%)	7(23.3%)			
	Severe	0 (0.0%)	21 (70.0%)			
SPAP	Mean±SD	28.83 ± 9.68	67.27 ± 18.86	-9.933	<0.001	HS
	Range	19 – 60	25 – 108			

PAAT: Pulmonary artery acceleration time; **SPAP:** Systolic Pulmonary Artery Pressure. Independent-Samples T Test

Table (3): Correlation between level of PAAT (ms) and level of SPAP (mmHg) in cases group and control group.

		PAAT (ms)	
		r	p-value
SPAP (mmHg)	Cases	-0.968**	0.000
	Controls	-0.941**	0.000

**Pearson correlation coefficients

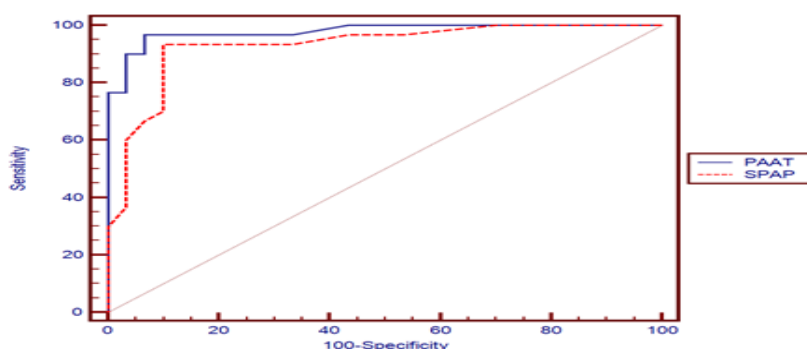


Figure 1: Receiver Operating Characteristic curve (ROC) for PAAT and SPAP to differentiate between control group and cases group

DISCUSSION:

Pulmonary artery acceleration time is a promising alternative tool for non-invasive

estimation of SPAP. It is simple to get & is not dependent on occurrence of TR or a structural heart abnormality [13].

The current study's objective was to compare the Doppler echocardiography-derived PAAT to the common method in estimating pulmonary arterial hypertension in newborns of diabetic mothers.

Cases and controls were divided into four categories: normal SPAP (SPAP 35 mmHg), mild (SPAP thirty six-forty five mmHg), moderate (SPAP forty six-fifty five mmHg), & severe PH (SPAP > 55 mmHg) [14].

Among the two groups, our study found that 30 neonates had normal pulmonary pressure (SPAP 35 mmHg), 2 neonates had mild pulmonary hypertension (SPAP 36–45 mmHg), 7 neonates had moderate pulmonary hypertension (SPAP forty six–fifty five mmHg), & 21 neonates had severe pulmonary hypertension (SPAP > 55 mmHg).

Additionally, compared to mild pulmonary hypertension, our study found a substantially increase in percentage of moderate and severe pulmonary hypertension in the case group (23.3 and 70.0%, respectively). Similar outcomes were shown by *Arshad et al.* [15], who discovered that severe PPHN was the most common form, occurring in 63/122 (51.6%) cases, followed by moderate PPHN in 40/122 (32.8%) cases, and mild PPHN in 19/122 (15.6%).

13.5 % of newborns with PPHN had mild PPHN, 54.1% had intermediate PPHN, & 32.4% had severe PPHN, according to a retrospective observational study by *Aprianto et al.* [16] on newborns with PPHN.

Our study demonstrated a substantially significant decline in PAAT and an increase in SPAP in the group with IDM and pulmonary arterial hypertension when compared to control group (p-values <0.001 & 0.001).

Outcomes are consistent with those of *Smith et al.* [17], who discovered that, in comparison to the control group with the

greatest PAAT, gestational diabetes mellitus (GDM) infant group had lower PAAT and lower PAAT to right ventricular ejection time (RVET) ratios (PAATi) ($P < 0.001$). Similar outcomes were found by *Tai et al.* [18] in recent trial with forty two studied cases < thirty six months of years old who were undergoing cardiac catheterization, both with & without a prior suspicion of PH. They found that studied cases with PH (mPAP > 20 mmHg) had shorter PAAT (74 msec) than individuals without PH (mPAP 20 mmHg) (111 msec).

In a prospective multi-center study, PAAT was evaluated on days one, two, 5-7, 32 & thirty six weeks post-menstrual age, as well as at one year corrected age in 239 preterm infants (29 weeks at birth). In line with our findings, it was discovered that, when compared to newborns without pulmonary hypertension at both time points (thirty two weeks postmenstrual age (PMA) & thirty six weeks PMA), all of pulmonary hypertension-affected infants had lower PAAT & PAAT/RVET ($p < 0.001$) [19].

Numerous factors, including greater early pulmonary ejection, improved pulmonary vascular resistance, & loss of lung compliance, which induce quick rise & decrease in flow velocity, may cause PAAT to be shortened in people with PH. In fact, PAAT stands for pulmonary flow acceleration, which rises as vascular resistance rises, based on Newton law of motion [20].

Also, according to outcomes, there was a substantial negative relationship found among level of PAAT (ms) & level of SPAP (mmHg) in case group with ($r = 0.984$) and ($p < 0.001$), & also in control group with ($r = 0.955$) and ($p < 0.001$). Our findings are in line with cohort research of studied cases one year of years old that looked at the correlation among cardiac catheterization-measured pulmonary artery pressures and PAAT, which is determined by echocardiography. An negative relationship

among PAAT & mean PAP ($r = 0.34$, $P = 0.01$) and SPAP ($r = 0.31$, $P = 0.02$) was demonstrated by linear regression analysis [21].

In a recent retrospective study, 82 newborns in the NICU and nursery's medical records and echocardiographic data were reviewed (age 0–4 months). Maximum velocity of tricuspid valve regurgitation doppler was used to determine a reported good connection between PAAT and SPAP ($r = 0.83$). To calculate SPAP in newborns and young babies, an equation was applied: $SPAP = 82.6 - 0.58 \times PAAT + \text{right atrium (RA) mean pressure}$ [22]. findings of *Dasgupta et al.* [24] found that healthy controls without PH have PAAT > 120 ms; PAAT ninety ms reliably identifies great pulmonary artery pressure (PAP); and value of 40 ms indicates severe PAP. A PAAT of 70 ms in preterm infant indicates raised PVR & potential BPD-PH. Additionally, *Tai et al.* [18] reported a strong inverse relationship among PAAT & sPAP ($r = 0.63$, $p < 0.001$).

Another resaerch by *Levy et al.* [22] showed that PAAT ninety ms in newborns is greatly predictive of pulmonary hypertension, which involved 75 children (median age, 5.3 years). The PAAT value fluctuates according on the child's age. A PAAT of 110 ms indicates pulmonary hypertension in older kids. It is simple to calculate the ratio of PAAT to RVET, & result of 0.31 indicates pulmonary hypertension with sensitivity & specificity of more than ninety%.

Also, *Koestenberger et al.* [25] stated that PAAT > 120 ms was defined in children to differentiate among PAH studied cases & healthy controls in a prospective echocardiographic examination on 756 healthy children (years old one day to eighteen years) & 54 children with PH.

We determined the optimal cutoff point for PAAT to diagnose pulmonary hypertension to be 72 ms with sensitivity of

96.67% & a specificity of 93.33% using ROC curve analysis. When detecting (6/7) moderate and (21/21) severe pulmonary hypertension (PHT), or pulmonary systolic pressure greater than 45 mmHg, a PAAT of 72 ms was more effective ($P < 0.001$). Therefore, PAAT shorter than 72 ms is more suitable for the detection of moderate and severe PHT than mild PHT, according to this finding. This is consistent with the findings of *Tai and his coworkers* [18], who discovered that the optimal cutoff point of PAAT to diagnosis PVR index 3 Wu*m2 was 77 msec with poor sensitivity (59%), but great specificity (94%).

Additionally, *Patel et al.* [19] discovered that a PAAT forty seven msec & a PAAT/RVET 0.28 at 32 weeks PMA resulted in a sensitivity of ninety one percent & a specificity of ninety five percent, with area below receiver operating characteristic curve of 0.93 (ninety five percent CI, 0.88-0.97) for the recognition of late pulmonary hypertension at 36 weeks PMA.

Similar findings were reported by *Levy et al.* [22], who said that PAAT of ninety msec & PAAT:RVET 0.31 produced sensitivity & specificity of ninety seven percent & 95%, respectively, for diagnosis of pulmonary hypertension (mean PAP > twenty five mmHg).

Additionally, a Leuven, Belgium observational study that involved 98 adult patients undergoing cardiac surgery between August and December 2013 found that PAAT is effective at differentiating among studied cases with & without pulmonary hypertension, with cut-off of < 107 ms identifying pulmonary hypertension with sensitivity of seventy five percent & specificity of 94.8% [13].

Great probability of pulmonary hypertension (SPAP above 38 mmHg) is also indicated by values of pulmonary acceleration time of 100 ms, according to

Granstam et al. [26], who reported this with a sensitivity of 89% & specificity of 84%.

Additionally, in research of 56 adult patients, *Tossavainen et al.* [27] discovered that pulmonary acceleration time 90 had a positive & negative predictive value of eighty eight percent & eighty one percent, in detecting studied cases with PVR three WU (mean age 61 ±13 years).

According to *Lanzarini et al.*[28], a PAAT value of ninety three ms recognized 67.4percent of studied cases with pulmonary hypertension.

Our study has certain limitations, such as a relatively small sample size; only thirty studied cases were contained in research. More research on a larger number of diabetic mothers' infants may yield more significant results. In addition, research lacked longitudinal follow-up to evaluate if accuracy of detection of PAH by pulmonary artery acceleration time changed over time.

Conclusion:

Our study of pulmonary arterial hypertension in infants of mothers with DM over 1st few days of life illustrated a decrease in the levels of PAAT and an increase in the level of SPAP. There was significant negative relationship found among the levels of PAAT (ms) & level of SPAP (mmHg) in IDM diagnosed with PAH. Also, PAAT levels can be helpful for the prediction of pulmonary hypertension, with the best cutoff point being 72 ms.

Ethics approval and consent to participate:

Both the institutional review board and the local committee of ethics approved the protocol of this research at the Faculty of Medicine of Ain Shams, which was performed based on the Helsinki Declaration. An informed written consent for participation in the study was obtained from the parents or legal guardians of the studied subjects prior to their involvement in

this study. The data supporting our study findings are provided by the corresponding author upon a reasonable request. All authors approved the final manuscript version. (FMASU-MS-216/2021)

Conflicts of interest:

The authors affirm that they have no conflicts of interest.

REFERENCES:

1. Anjum, S. K., & Yashodha, H. T. A study of neonatal outcome in infants born to diabetic mothers at a tertiary care hospital. *Int J Contemp Pediatr*, 2018; 5(2), 489-92.
2. Steurer, M. A., Jelliffe-Pawłowski, L. L., Baer, R. J., Partridge, J. C., Rogers, E. E., & Keller, R. L. Persistent pulmonary hypertension of the newborn in late preterm and term infants in California. *Pediatrics*, 2017; 139(1).
3. Mandell, E., Kinsella, J. P., & Abman, S. H. Persistent pulmonary hypertension of the newborn. *Pediatric Pulmonology*, 2021; 56(3), 661-669.
4. Martinho, S., Adão, R., Leite-Moreira, A. F., & Brás-Silva, C. Persistent pulmonary hypertension of the newborn: pathophysiological mechanisms and novel therapeutic approaches. *Frontiers in Pediatrics*, 2020; 8, 342.
5. Al-Biltagi, M. Cardiac changes in infants of diabetic mothers. *World Journal of Diabetes*, 2021; 12(8), 1233-1247.
6. Trittmann, J. K., Almazroue, H., Nelin, L. D., Shaffer, T. A., Celestine, C. R., Green, H. W., et al. PATET ratio by Doppler echocardiography: noninvasive detection of pediatric pulmonary arterial hypertension. *Pediatric Research*, 2021; 1-6.
7. Gaulton, J. S., Mercer-Rosa, L. M., Glatz, A. C., Jensen, E. A., Capone, V., Scott, C., et al. Relationship between pulmonary artery acceleration time and pulmonary artery pressures in infants. *Echocardiography*, 2019; 36(8), 1524-1531.

8. Skinner,G.J. Echocardiographic Assessment of Pulmonary Arterial Hypertension for Pediatricians and Neonatologists. *Front. Pediatr., Sec. Pediatric Cardiology*, 2017.
9. Aggarwal, S. Natarajan, G. Echocardiographic correlates of persistent pulmonary hypertension of the newborn. *Early Hum. Dev.* 2015; 91, 285–289.
10. Janda S, Shahidi N, Gin K, Swiston J. Diagnostic accuracy of echocardiography for pulmonary hypertension: A systematic review and meta-analysis. *Heart*, 2011; 97:612–22.
11. Hill KD, Lim DS, Everett AD, Ivy DD, Moore JD. Assessment of pulmonary hypertension in the pediatric catheterization laboratory: Current insights from the magic registry. *Catheterization & Cardiovascular Interventions*, 2010; 76:865–73.
12. Roushdy A. M., Ragab I., Abd el Raouf W. Noninvasive assessment of elevated pulmonary vascular resistance in children with pulmonary hypertension secondary to congenital heart disease: A comparative study between five different Doppler indices. *J Saudi Heart Assoc*, 2012; 24:233–24.
13. Cowie, B., Kluger, R., Rex, S., & Missant, C. The relationship between pulmonary artery acceleration time and mean pulmonary artery pressure in patients undergoing cardiac surgery: an observational study. *European Journal of Anaesthesiology| EJA*, 2016; 33(1), 28-33.
14. Deshpande, S., Suryawanshi, P., Holkar, S., Singh, Y., Yengkhom, R., Klimek, J., et al. Pulmonary hypertension in late onset neonatal sepsis using functional echocardiography: a prospective study. *Journal of Ultrasound*, 2021; 25(2), 233-239.
15. Arshad, M. S., Adnan, M., Anwar-ul-Haq, H. M., & Zulqarnain, A. Postnatal causes and severity of persistent pulmonary Hypertension of Newborn. *Pakistan Journal of Medical Sciences*, 2021; 37(5), 1387.
16. Aprianto, M., Rahman, M., & Utomo, M. T. Characteristics, Diagnosis, Management and Output of Persistent Pulmonary Hypertension of the Newborn at Dr. Soetomo Hospital. *Indian Journal of Forensic Medicine & Toxicology*, 2021; 15(1).
17. Smith, A., Franklin, O., McCallion, N., Breatnach, F., & Afif, E. K. Effect of Gestational Diabetes Mellitus on Neonatal Myocardial Function. *Neonatology*, 2021; 118(1), 64-72.
18. Tai, C., Hsieh, A., Moon-Grady, A. J., Keller, R. L., Teitel, D., & Nawaytou, H. M. Pulmonary artery acceleration time young children is determined by heart rate and transpulmonary gradient, but not by pulmonary blood flow: A simultaneous echocardiography—cardiac catheterization study. *Echocardiography*, 2022; 39(7), 895-905.
19. Patel, M. D., Breatnach, C. R., James, A. T., Choudhry, S., McNamara, P. J., Jain, A., et al. Echocardiographic assessment of right ventricular afterload in preterm infants: maturational patterns of pulmonary artery acceleration time over the first year of age and implications for pulmonary hypertension. *Journal of the American Society of Echocardiography*, 2019; 32(7), 884-894
20. Dammassa, V., Corradi, F., Colombo, C. N. J., Mojoli, F., Price, S., & Tavazzi, G. Pulmonary artery acceleration time accuracy for systolic pulmonary artery pressure estimation in critically ill patients. *The Ultrasound Journal*, 2022; 14(1), 1-9.
21. Mohammad Nijres, B., Bokowski, J., Mubayed, L., Jafri, S. H., Davis, A. T., & Abdulla, R. I. Utility of pulmonary artery acceleration time to estimate systolic pulmonary artery pressure in neonates and young infants. *Pediatric cardiology*, 2020; 41(2), 265-271.
22. Levy, P. T., Patel, M. D., Groh, G., Choudhry, S., Murphy, J., Holland, M. R., et al. Pulmonary artery acceleration time provides a reliable estimate of invasive pulmonary hemodynamics in children. *Journal of the American Society of Echocardiography*, 2016; 29(11), 1056-1065.

23. Yared, K., Noseworthy, P., Weyman, A. E., McCabe, E., Picard, M. H., & Baggish, A. L. Pulmonary artery acceleration time provides an accurate estimate of systolic pulmonary arterial pressure during transthoracic echocardiography. *Journal of the American Society of Echocardiography*, 2011; 24(6), 687-692.
24. Dasgupta, S., Richardson, J.C., Aly, A. M., Jain, S. Role of functional echocardiographic parameters in the diagnosis of bronchopulmonary dysplasia-associated pulmonary hypertension. *Journal of Perinatology*, 2021; 42,19–30.
25. Koestenberger, M., Grangl, G., Avian, A., Gamillscheg, A., Grillitsch, M., Cvirn, G., et al. Normal reference values and z scores of the pulmonary artery acceleration time in children and its importance for the assessment of pulmonary hypertension. *Circulation: Cardiovascular Imaging*, 2017; 10 (1), e005336.
26. Granstam, S. O., Björklund, E., Wikström, G., & Roos, M. W. Use of echocardiographic pulmonary acceleration time and estimated vascular resistance for the evaluation of possible pulmonary hypertension. *Cardiovascular ultrasound*, 2013; 11(1), 1-7.
27. Tossavainen, E., Söderberg, S., Grönlund, C., Gonzalez, M., Henein, M. Y., Lindqvist, P. Pulmonary artery acceleration time in identifying pulmonary hypertension patients with raised pulmonary vascular resistance. *European Heart Journal–Cardiovascular Imaging*, 2013; 14(9), 890-897.
28. Lanzarini, L., Fontana, A., Campana, C., & Klersy, C. Two simple echo-Doppler measurements can accurately identify pulmonary hypertension in the large majority of patients with chronic heart failure. *The Journal of heart and lung transplantation*, 2005; 24(6), 745-754.

وقت تسريع الشريان الرئوي عند الرضع من أم مصابة بمرض السكر تم تشخيصهم بارتفاع ضغط الدم الرئوي وأهميته لتقدير ضغط الشريان الرئوي الانقباضي

*فاطمة مصطفى يحيى عبد العال و** ايمان محمد السيد و** وليد محمد الجندى و** محمد عمر داوود

*قسم الأطفال بمستشفى التحرير العام ** قسم الأطفال بطب عين شمس

المقدمة: الأطفال المولودين لأمهات مصابات بداء السكري (DM) يعانون من ضعف في أداء عضلة القلب وهم معرضون لخطر ارتفاع ضغط الدم الرئوي.

الهدف من البحث: تقييم دقة تخطيط صدى القلب الدوبلري المشتق من زمن تسريع الشريان الرئوي (PAAT) في تقدير ضغط الشريان الرئوي عند الرضع من الأمهات المصابات بداء السكري.

المرضي وطرق البحث: دراسة مقطعية مقارنة شملت (٣٠) رضيعاً لأم مصابة بالسكري ، تتراوح أعمارهم بين ٠ - ٧ أيام تم إدخالهم إلى المحضن في مستشفيات جامعة عين شمس ، وتم تشخيص إصابتهم بارتفاع ضغط الدم الرئوي كمجموعة حالة ، و (٣٠) العمر الصحي والجنس المطابقين لحديثي الولادة من أم غير مصابة بالسكري كمجموعة ضابطة. تم إجراء تخطيط صدى القلب عبر الصدر وتخطيط صدى القلب الدوبلري المشتق من PAAT في الأسبوع الأول من العمر.

النتائج: كشفت الدراسة عن انخفاض معتد به إحصائياً في مستوى وقت تسريع الشريان الرئوي ($p < 0.001$) وزيادة في مستوى ضغط الشريان الرئوي الانقباضي ($p < 0.001$) في مجموعة الرضع من أم مصابة بمرض السكر المشخصة بارتفاع ضغط الدم الشرياني الرئوي عن المجموعة الضابطة. أيضاً ، وفقاً لدراستنا ، كان هناك ارتباط سلبي ذي دلالة إحصائية بين مستوى PAAT (ملي ثانية) ومستوى SPAP (مم زئبق) في مجموعة الحالات ($r = -0.984$ and $p < 0.001$). كان لدى $PAAT \leq 72$ حساسية ٩٦,٦٧٪ وخصوصية ٩٣,٣٣٪ في تحديد المرضى الذين يعانون من ارتفاع ضغط الدم الشرياني الرئوي عند الرضع من أم مصابة بداء السكري.

الاستنتاجات: تدابير PAAT تنخفض بشكل ملحوظ في حالات ارتفاع ضغط الدم الرئوي. يمكن أن يكون PAAT - 72 ملي ثانية بمثابة مؤشر قوي غير جراحي لارتفاع ضغط الدم الشرياني الرئوي عند الرضع من أم مصابة بداء السكري