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Correlation of Apgar Score and Umbilical Artery pH in Full-Term Newborns Delivered by Cesarean Section Due to Decreased Fetal Heart Rate

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ABSTRACT

Background & Objective: Despite the high efficacy of the Apgar score in finding respiratory distress, a low Apgar score doesn't necessarily indicate fetal hypoxia-asphyxia. Umbilical Artery pH (UApH) is one of the best indicators of fetal hypoxia. Therefore, it's so beneficial to consider these criteria and their relationship with the Apgar score for accurate diagnosis of prenatal respiratory distress retrospectively which reduces the unnecessary cesarean section (CS) rate.

Materials & Methods: 162 full-term (≥259 days) neonates delivered by CS with the diagnosis of decreased fetal heart rate (FHR) were evaluated. 1-min and 5-min Apgar scores and UApH were measured. The correlation between Apgar scores with UApH and the association between UapH and Apgar with the NICU admission were evaluated. The effect of other variables including mother's age, gravidity, gestational age, birth weight, newborn sex, and causes of decreased FHR on Apgar scores and UApH were studied as well.

Results: The most common cause of decreased FHR was fetal distress, boys had higher weight (P=0.033) and lower UApH (P=0.049) than girls. Other parameters were not different significantly between both sexes. There was a positive correlation between UApH and 1-min and 5-min Apgar scores (r=0.464 and r=0.370 respectively) when controlled for birth weight (P<0.0001). The RR for NICU admission in male acidemic neonates with abnormal 1-min Apgar was 14.05 (CI95%: 5.7-34.6) in comparison to females (RR=1.06, CI95%: 1-1.26).

Conclusion: Mild acidemia (UApH<7.2) at least in a male fetus would be a good predictor for postnatal complications and need for NICU admission. Future studies with more samples are suggested.

Keywords: Apgar, Prenatal Care, Fetal Hypoxia, Cesarean Section

Introduction

Normal fetal heart rate (FRH) is ranging from 120-160 beats per minute. Heart rate patterns in fetal distress (FD) including bradycardia (<120/min) and tachycardia (>160/min) indicate late onset and variable failure. Fetal problems are started before and during parturition, so screening women who are at high risk of FD is recommended. The most common cause of prenatal FD is uteroplacental failure **(1)** which is presented by intrauterine growth-retardation (IUGR), fetal hypoxia, and increased vascular resistance which leads to mixed respiratory and metabolic acidosis in severe forms.

Prenatal evaluation and monitoring aim to prevent intrauterine death, hypoxic brain injury, and also fetal acidemia **(1, 2)**. There are several methods to evaluate fetal health including non-stress test (NST) (heart rate response to fetal movements), oxytocin challenge test, biophysical profile scoring (which shows the best fetal evaluation), and Doppler assessment of aorta or umbilical cord artery **(1)**. Prenatal hypoxia due to insufficient oxygen supply is a life-threatening condition that increases the anaerobic metabolism and continues with imbalanced energy and accumulation of lactate and hydrogen ions **(3)**. Birth hypoxia is the most common cause of prenatal mortality and morbidity, as

well as ischemic-hypoxic brain injury, throughout the world **(4)**. Significant long-term morbidity and about 20% of neonatal death in India have also resulted from this condition **(5)**. There are 1.1 million stillbirths, more than one million neonatal deaths, and remaining, evolutionary disorders due to about 4-9 million estimated annual intrapartum hypoxia **(4)**.

The most popular evaluation method for neonates is an assessment of Apgar which is a rapid scoring system based on physiological response to the birth process, developed by Virginia Apgar in 1952. This method is highly applicable to assess the neonate's need for resuscitation, but it isn't a good criterion for evaluating the intrauterine fetal situation and doesn't provide complete information about the neonate asphyxia **(2, 5-7)**. Also, it is easily affected by many other factors, so it is not suitable for neonates undergoing endotracheal intubation. Since the Apgar score does not usually reflect the degree of acidosis at delivery, its value in the assessment of asphyxia must be questioned. Zaigham et al. have pointed out that a low Apgar score is not equivalent to neonatal asphyxia. If the Apgar score is simply used to diagnose neonatal asphyxia, it is a misunderstanding and abuse of the Apgar score. The Apgar score alone cannot predict the outcome of poor neonatal prognosis, nor can it predict neonatal mortality **(8, 9)**.

1-min and 5-min Apgar scores are assessed by an expert using each of the five physiological parameters including heart rate, respiratory effort, muscle tone, color, and reflex irritability. Full-term neonates with normal cardiopulmonary adaptation should catch an 8-9 Apgar score in the first and fifth minutes of birth. A 4-7 Apgar score indicates the need for monitoring to reveal whether this situation improved or any possible pathogenesis due to the parturition process and any neonate-related problems have led to a lower Apgar score.

Apgar score of 0-3 indicates cardiopulmonary arrest or diseases which cause bradycardia, hypoventilation, or failure of central nervous system (CNS) performance (10). Despite the high value of the Apgar score for diagnosis of FD, intrauterine asphyxia, airway obstruction, and CNS depression, the lower Apgar score doesn't necessarily indicate fetal hypoxia-asphyxia. Other causes including neonatal failure, the mother's use of narcotics, sedatives, and magnesium sulfate, congenital myopathy and neuropathy, spinal cord injuries, pulmonary disorders, diaphragmatic hernia, congenital athresia, and CNS disorders are also involved in lowering Apgar score. Also, the lower Apgar score doesn't predict the subsequent cerebral palsy **(11)**. So, it is important to consider other criteria for the diagnosis of prenatal asphyxia-acidosis and determine its correlation with the Apgar score to manage the low Apgar neonates **(12)**.

Umbilical artery pH (UApH) and metabolic acidosis are the most important criteria in the diagnosis of neonatal asphyxia and hypoxia during the parturition and also deciding on postnatal care **(3, 5, 13)**. Lactate and UApH are valuable parameters in differentiation between asphyxic or normal newborns and also there is a powerful correlation between UApH and lower Apgar score in the first and fifth minutes of birth as well as the need for resuscitation and NICU admission **(3, 5)**.

The normal UApH range is 7.4±0.2 and metabolic acidosis is defined by UApH<7 and Base deficit >12 mmol/l. There are also increased convulsion rates, need for intubation, NICU admission, and mortality with UApH less than 7 **(5, 14)**.

It is believed that using ABG analysis gives a good insight into the neonate situation in all deliveries and the hypoxic fetus also can be diagnosed to lower FD, mortality, and morbidity in this golden time to decrease unnecessary CS **(5)**.

Also, the clinical practice guideline of the Society of Obstetricians and Gynecologists of Canada (SOGC) recommended the umbilical cord ABG routine analysis as an appropriate criterion for determining suitable neonatal care at birth and after that **(3, 5)**.

Based on the above-mentioned outlines, this study aimed to assess the correlation of 1-min and 5-min Apgar scores and UApH and factors that affect this correlation in neonates delivered by CS due to decreased FHR to determine the degree of hypoxia during parturition to be applied in decreasing unnecessary CS and also to provide the best postnatal care **(15, 16)**.

Methods

In this cross-sectional study, 200 pregnant women referred to the women's ward of Vali-asr Hospital, Tehran, Iran, enrolled by convenient sampling after informed consent and assurance of keeping their records confidentially, from October 2016 to October 2017. Neonates were included if they had intrauterine tachycardia/ bradycardia, abnormal NST, and Meconium-stained amniotic fluid (MSAF). Exclusion criteria were reactive NST, <37 weeks gestational age, intrauterine death, and any anomalies. Accordingly, 42 participants were excluded, and subsequent evaluations were conducted on 162 full-term (gestation ≥259 days) women.

Prenatal heart rate was monitored and 1-min and 5-min Apgar scores (according to William's obstetrics textbook), sex, weight, and NICU admission of neonates delivered by CS due to decreased heart rate were measured by specialists in the operation room. Mother's factors including age, gestational age, and the number of pregnancies (gravidity) were also recorded.

To assess UApH immediately after delivery, two hemostat clamps were placed adjacent to the placenta, and at a distance of 10-20 cm, two other clamps in the vicinity of the baby, then 1-2 ml of isolated blood were drawn into a heparinized insulin syringe and sent to

laboratory quickly. PH was measured and acidemia was defined by the previously described cut-off of 7.2 **(17)**.

Data were analyzed by SPSS version 22 (IBM, USA), using Mann Whitney U and t-test for comparing the mean between groups. The Chi-square test was used for qualitative variables, and linear regression and Pearson correlation tests were used for quantitative variables.

Results

The mean (SD) age of included mothers was 27.25 (5.7) years, ranging from 17 to 42 years. 116 mothers (71.6%) were primigravid and 46 (28.4%) were multi-gravid. Mean gestational age was 275.04 (SD=7.76) days, 117 mothers (72.2%) had a gestational age between 259 and 280 days and 45 (27.8%) had more than 280 days.

The most common causes of decreased FHR in 162 (56.2% male) neonates delivered by CS, were FD followed by the nuchal cord, MSAF, and others as shown in Table 1. The means (SD) of all neonatal variables were not significantly different between the two groups of causes (Group1: FD and Group2: other causes) (P≥0.173).

Table 1. Diagnosed causes of decreased FHR

Causes	Frequency	Percent
Fetal distress	101	62.3
Nuchal cord	29	17.9
MSAF	20	12.3
Placental abruption	7	4.3
IUGR	3	1.9
Expired	2	1.2

The mean (SD) weight of neonates was 3.250 (47.24) kg, and as shown in Table 2, boys had higher weight (p=0.033) and lower UApH (p=0.049) than girls. Other parameters were not significantly different between both sexes. Abnormal (<8) 1-min and 5-min Apgar were seen in 8.6% and 1.2% of neonates, respectively. Abnormal UApH (<7.2) was seen in 6.2% of neonates, and 9.3% of all 162 neonates were also admitted to the NICU and two newborns (boys) were expired at birth.

Neonate's sex did not affect NICU admission (p=0.147) and also there were no significant mean differences of all neonatal parameters in different categories of gravidity (p>0.05) except the gestational age that was lower in multi-gravid mothers (p=0.048).

Table 2. Mean and SD of neonatal parameters in different sexes

Neonatal Parameter	Mean (SD)	Sex	Mean	Std. Deviation	p Value
Birth weight	3.250 (47.24) kg	M	3320.82*	478.23	0.033
		F	3161.05	452.67	
1-min Apgar	8.45 (1.22)	M	8.32	1.45	0.120
		F	8.6	0.81	
5- min Apgar	9.49(1.22)	M	9.35	1.53	0.108
		F	9.66	0.58	
UApH	7.32(0.095)	M	7.31	0.10	0.049
		F	7.34*	0.08	
Gestational age	275.04 (7.75) Days	M	275.03	7.48	0.985
		F	255.05	8.15	

***: significantly more**

There was a positive correlation between UApH and 1-min and 5-min Apgar scores with r=0.464 and r=0.370 respectively when controlling for birth weight (P<0.0001). The correlation between birth weight and 1-min and 5-min Apgar were also positive, r= 232 (P=0.005) and r=272 (P<0.001) respectively. The estimated correlations were stronger in male neonates, and UApH was not significantly correlated with the 5-min Apgar score in female neonates. Also, weight was not significantly correlated with 1-min and 5-min Apgar in female neonates (Table 3). There was no significant correlation between birth weight and UApH (p=0.735).

The ranks of UApH, 1-min, and 5-min Apgar scores were significantly different between NICU-admitted and non-admitted newborns (P<0.001). The rank of birth weight was not significantly different in NICU admitted and non-admitted newborns (P=0.055).

Table 3. Sex difference of estimated correlations between UApH, birth weight and Apgar score

Correlated variables	Sex	r	p value
UApH&1-min Apgar	M	0.509*	0.0001
	F	0.297*	0.012
UApH&5-min Apgar	M	0.406*	0.0001
	F	0.207	0.083
Weight&1-min Apgar	M	0.331*	0.002
	F	0.079	0.515
Weight&5-min Apgar	M	0.364*	0.0001
	F	0.176	0.141

***: Significantly correlated**

Of 10 neonates with acidemia (UApH<7.2), 6 (60%) were admitted to NICU (all were male and had 1-min Apgar<8) but those with normal UApH had only 6% NICU admission (Relative Risk [RR]=11.18, CI95%: 5.1-24.5). The RR for NICU admission in male acidemic neonates with abnormal 1-min Apgar was 14.05 (CI95%: 5.7-34.6) in compared to females (RR=1.06, CI95%: 1-1.26).

10 (83.3%) out of 12 neonates with 1-min Apgar<8 was admitted to NICU but only 3.3% of neonates with normal Apgar needed NICU admission (RR= 24.6, CI95%: 10.0-60.5). All admitted neonates had normal 5-min Apgar.

Of the total 15 NICU admitted newborns 11 (73%) were male and 4 (27%) were female (p= 0.147), 10 (67%) had abnormal 1-min Apgar, and 5 (33%) had normal 1-min Apgar (p<0.0001) and 6 (40%) had abnormal UApH and 9 (60%) had normal UApH (P<0.0001) (Table 4-5).

Table 4. Chi-2 analysis of NICU admission and 1-min Apgar

Sex			NICU admission		p value
			Admitted	Not admitted	
Male	1-min Apgar	<8	7	0	<0.001
		≥8	4	78	
Female	1-min Apgar	<8	3	2	<0.001
		≥8	1	65	
Total	1-min Apgar	<8	10	2	<0.001
		≥8	5	143	

Table 5. chi-2 analysis of NICU admission and UApH

Sex			NICU admission		p value
			Admitted	Not Admitted	
Male	UApH	<7.2	6	1	<0.001
		≥7.2	5	77	
Female	UApH	<7.2	0	2	0.89
		≥7.2	4	65	
Total	UApH	<7.2	6	3	<0.001
		≥7.2	9	142	

Mother's age had no significant correlation with any other variables, but gestational age had only a fair positive correlation with female birth weight, r=0.387 (P=0.001) (Figure 1).

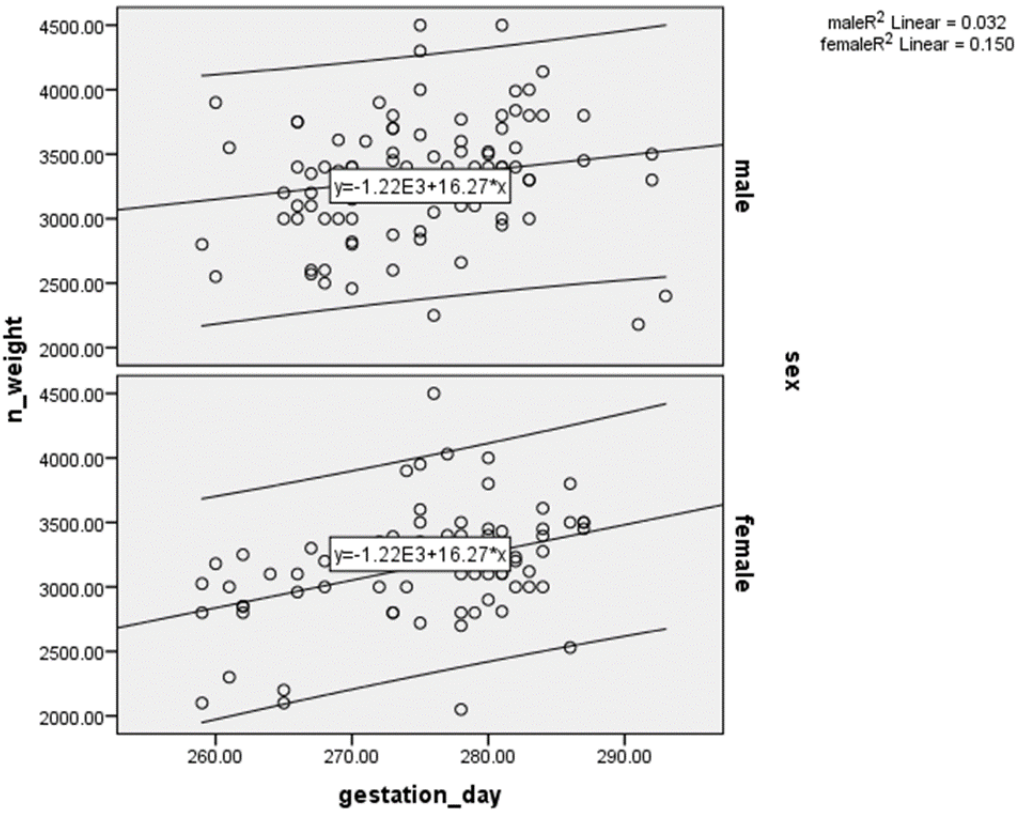

Figure 1. **linear regression analysis of birth weight and gestational age in different sexes**

Discussion

As shown in the results, this study demonstrated that UApH is positively correlated with 1-min abnormality in both sexes. That is consistence with previous studies as follows it is also recognized internationally that taking umbilical cord blood for blood gas analysis and detection after birth is a quantitative analysis method of fetal perinatal stress, which reflects the acid-base balance of newborns in the form of objective indicators, with strong specificity **(18, 19)**. A study by Taheripanah et al. showed that the assessment of UApH could help to predict intrapartum fetal asphyxia and there is a strong correlation between 1-min and 5-min Apgar and UApH and the occurrence of FD **(2)**. A study demonstrated the significant association between neonatal acidosis (UApH<7) and early outcomes including, seizures, increased risk of intubation, hypoxic-ischemic encephalopathy, and need for mechanical ventilation **(20)**. Another study showed that neonatal cardiac pattern (presence of low/absent variability for at least one hour only or along with late decelerations in the absence of accelerations) significantly predicts the development of acidemia **(21)**. A study demonstrated that low UApH<7.1 can predict a neonatal seizure and UApH<7 was more sensitive than a base excess of -16 in the prediction of the neonatal seizure (73.8% vs 52.5%) **(22)**. Another study concluded that newborns with severe umbilical acidemia (UApH<7) are at risk of abnormal neurologic outcomes including hypoxic-ischemic encephalopathy, and in this pH range the Apgar score cannot predict the asphyxia complications **(23)**. Despite all other studies which took the UApH cut-off as seven a study using ROC analysis calculated the most appropriate UApH cut-off as 7.2 to be more compatible with adverse outcomes in full-term newborns **(17)** which is more in line with our results. A Most recent study in this field with a large full-term participant stressed the importance of mild acidemia (UApH between 7.11-7.19) to be associated with an increased risk of some morbidity including FD and neonatal sepsis compared with normal neonates (UApH≥7.2) **(24)**. Also, a study by Modarressnejad et al. demonstrated the positive correlation between acidemia and 1-min Apgar and FD **(25)**. However, in the present study, the positive correlation of UApH and 5-min Apgar was seen just in male neonates. Also, the positive correlations of weight and Apgar (1-min and 5-min) were seen only in male neonates. This male factor also has been mentioned by an Iranian study by Kaveh et al. which demonstrated that male gender, CS, and gestational age over 37 weeks had a positive effect on the association between Apgar score and ABG **(12)**. Also, an old study implied that a low UApH is associated with male sex, grade three or four intraventricular hemorrhage, hyaline membrane disease, and neonatal death **(26)**. All boys in our study with abnormal 1-min Apgar who were admitted to NICU had abnormal UApH and two expired newborns were boys as well.

Conclusion

Although a low sample size led to low cases in the abnormal group and we had to use nonparametric analysis in some cases, It would be concluded that mild acidemia (UApH<7.2) at least in male fetuses would be a good predictor for postnatal complications and need for NICU admission. Future studies in this field with higher samples are suggested to determine the possible gender difference in association with fetal acidemia and postnatal complications.

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Conflict of Interest

The authors have no conflict of interest, and this paper has not been published and is not being considered for publication elsewhere.

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