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Use of Doppler indices in prediction of Acute Fetal Hypoxia – Proposed Staging System

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Abstract: *Background:* Till now multiple studies regarding Doppler indices in Obstetrics have concentrated on detection of chronic hypoxia, publications have been silent on early detection of fetal hypoxia before the damage sets in. *Objective:* To develop a novel, credible staging system for early detection of acute fetal hypoxia and its possible progression. A pilot study was undertaken to accomplish this larger objective. *Methods:* One hundred and thirty two singleton, uncomplicated, pregnancy between 34 to 42 weeks were included in over 1 year and followed up at a semi urban/ rural setup in Northern Karnataka region at 2 local nursing homes, 1 radiology centre and 1 tertiary care centre. A novel staging system was developed based on available patho-physiological knowledge of vascular Doppler changes secondary to change in LV function and applied with Doppler scan findings to detect and predict fetal distress. Standard statistical tests like p value, Chi square tests, Fischer's tests, and Spearman's correlation coefficient were utilized to come to conclusion. *Results:* Out of 132 subjects 32 subjects were detected with fetal distress (24.24%). Among those 32 subjects, 30 (22.7%) underwent LSCS and 2 had vaginal delivery. One subject continued pregnancy against medical advice and was later detected with IUFD. Proposed new staging of the AFH when applied showed consecutive increase in percentage of subjects with fetal distress with increase in stage. Stage 4 had a maximum percentage of fetal distress with 7 out of 8 subjects (88%). Similarly stage 0, 1, 2 and 3 had 20%, 16%, 19% and 33% respectively. Statistical significance was found between the different stages of AFH and subjects with fetal distress ($p<0.0004$). *Conclusion:* This novel staging method can be used to detect and predict acute fetal hypoxia and can help us in better management of the pregnancy.

Keywords: Acute Fetal Hypoxia (AFH), fetal distress, intrauterine growth restriction (IUGRs), Fetal Doppler indices

Abbreviations: AFH (Acute Fetal Hypoxia), FD (Fetal Distress), LSCS (Lower Segment Cesarean Section), LV (Left Ventricle), IUGRs (Intra Uterine Growth Restriction), MCA (Middle Cerebral Artery), MCAPI (Middle Cerebral Artery Pulsatility Index), TAo (Thoracic Aorta), TAoTAV (Thoracic Aorta Time Averaged Velocity), UTA (Uterine Artery), PI (Pulsatility Index), IUFD (Intra Uterine Fetal Death), UmA (Umbilical Artery), pH (Hydrogen ion concentration), E/A (Early Diastolic filling / Atrial Contraction).

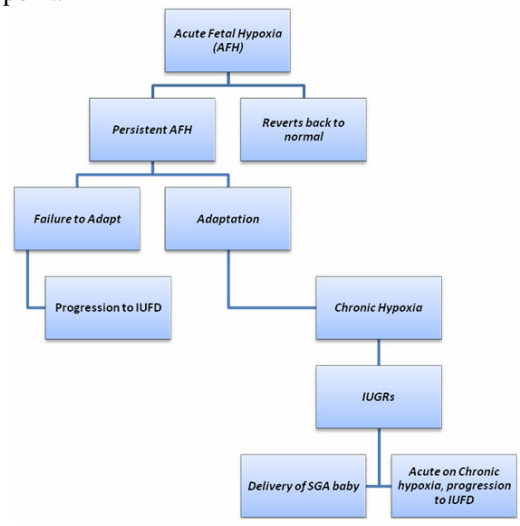
Introduction

Studies involving Doppler indices for diagnosing chronic fetal hypoxia and asphyxia have been conducted and documented extensively, where already fetal damage is done. Based on these extensive studies various Doppler guidelines have been in use for diagnosing Chronic Fetal Hypoxia and Asphyxia. Chronic Fetal Hypoxia invariably leads to Intra-uterine growth restriction (IUGRs). IUGRs is nothing but persistent hypoxia with premature exit of cell cycle together with enhanced apoptosis resulting in fewer number of cells in body. Progressive fetal deterioration manifests in sequential abnormalities starting with middle cerebral artery (MCA) dilatation, circulatory redistribution, oligohydramnios, disappearance of fetal heart variability, late

deceleration, and absence or reversal of umbilical artery (UmA) end diastolic velocity, acidemia, signs of cardiac failure and intra-uterine death (IUD). The UmA Doppler indices are more sensitive to asphyxia than hypoxia [1]. UmA Doppler indices are function of fetal cardio-vascular system.

Middle Cerebral artery indices are significantly sensitive to hypoxia. Middle Cerebral Artery (MCA) indices are function of maternal cardio-pulmonary system and Placenta (feto-maternal unit). Thoracic aortic (TAo) indices reflect fetal cardiac output. Thoracic Aorta (TAo) Time Averaged Velocity (TAV) is directly proportional to the cardiac output. In response to hypoxia, MCA Pulsatility Index (PI) decreases proportionally. Similarly initially in hypoxia, the TAoTAV increases proportionally to compensate hypoxia till a critical point is reached. Later once critical point is reached the TAoTAV decreases thereafter. In the initial phase of hypoxia the increase in TAoTAV is in response to the fall in MCA PI, as a part of compensatory mechanism to maintain cerebral flow. But in the later phase of hypoxia the compensatory mechanism fails and both TAoTAV and MCA PI decreases. MCA-PI and TAoTAV product value traces an increasing curvilinear path. The product of MCA Pulsatility Index (PI) with TAoTAV, initially shows compensation either maintaining curve or flattening and then gradually decreases indicating failure of fetal adaptation to hypoxia. Fetal adaptation to hypoxia is nothing but chronic hypoxia leading to IUGRs (Chart 1).

Chart-1: Patho-physiological progression of Fetal Hypoxia



According to Kingdom and Kauffmann [2], intra-uterine hypoxia can be classified into 3 subtypes;

1) Pre-placental hypoxia: where both mother and her fetus are hypoxic (Maternal Anemia, High altitude, Cynotic maternal heart diseases, etc.).
2) Utero-placental hypoxia, where the maternal oxygenation is normal but the utero-placental circulation is impaired (Pre-eclampsia, placental insufficiency, etc.).
3) Post placental hypoxia, where only fetus is hypoxic, mainly related to fetal diseases rather than to the direct impact on to the fetus [2].

A main consequence of chronic hypoxia is the failure of the fetus to achieve its genetically determined growth potential. About 10% of all babies grow poorly in-utero and are born small for gestational age. IUGRs is associated with fetal distress and asphyxia and a 6- to 10-fold increased perinatal mortality [3]. Frequent hypoxia-mediated complications include meconium aspiration, metabolic and hematologic disturbances, cognitive dysfunction, and cerebral palsy. Acute and chronic hypoxia is also associated with a variety of morphological and functional fetal cardiac changes that aim either to compensate for the reduced oxygenation of vital organs or are the result of hypoxia-mediated fetal tissue damage [4-6].

Apoptosis is a controlled active physiologic process that removes unwanted or defective cells by intrinsic programmed cell suicide [6]. In rat hearts exposed to oxidative stress, it could be shown that many genes that affect cell communication, survival and signaling were down regulated [6-7]. This down regulation is believed to be partly responsible for the long-term consequences of intrauterine hypoxia and leaves a persistent cardiovascular "imprint" that leads to cardiovascular disease in later life. With persistent hypoxia, premature exit of cell cycle is initiated, together with enhanced apoptosis resulting in fewer, but hypertrophied cardiomyocytes. This process aims for better energy efficiency during hypoxic conditions but also results in less compliant ventricles [8].

Bahado-Singh *et al* reported that an abnormally low cerebroplacental ratio is associated with increased perinatal morbidity and mortality and that the ratio improves the prediction of perinatal outcome compared with umbilical artery PI alone [9]. However, the cerebroplacental ratio did not appear to correlate significantly with outcome after 34 weeks. In third-trimester fetuses, the ratio of PI between the fetal descending thoracic aorta and the middle cerebral artery may be more useful [10].

Cardiac flow is greatly influenced by the modifications of arterial impedance to flow. Cerebral vasodilatation produces a decrease in left ventricle after-load, whereas increased placental and systemic resistance produce increased right ventricle after-load. Hypoxemia may also impair cardiac contractility directly, while changes in blood viscosity due to polycythemia may alter preload [11]. Consequently, growth-restricted fetuses' show, at the level of the atrioventricular valves, impaired ventricular filling (lower ratio of early passive to late active ventricular filling phase - E/A ratio) [12], lower peak velocities in the aorta and pulmonary arteries. These hemodynamic intracardiac changes are compatible with a preferential shift of cardiac output in favor of the left ventricle, leading to improved cerebral perfusion. Thus, in the first stages of the disease, the supply of substrates and oxygen can be maintained at near normal levels despite any absolute reduction of placental transfer [13].

Longitudinal studies of deteriorating growth-restricted fetuses have shown that peak velocity and cardiac output gradually decline, suggesting a progressive worsening in cardiac function [13]. Similarly, there is a symmetrical decrease in ventricular ejection force at the level of both ventricles, despite the dramatically different hemodynamic conditions present in the vascular district of ejection of the two ventricles (i.e. reduced cerebral resistances for the left ventricle and increased splanchnic and placental resistance for the right ventricle) [14]. This supports a pivotal role of the intrinsic myocardial function in the compensatory mechanism of the growth-restricted fetus following the establishment of the brain-sparing effect. Ventricular ejection force dramatically decreases in a short time interval (about 1 week), showing an impairment of ventricular force close to fetal distress. As a consequence, cardiac filling also is impaired. Fetal hypoxemia is associated with a reduction in umbilical venous blood flow, but, despite this decrease, a normal peak velocity in the ductus venosus is maintained [15].

In the growth-restricted fetus, the percentage of umbilical venous blood passing through the ductus venosus is increased from about 40% (in normal fetuses) to about 60% [16]. Therefore, there is redistribution in venous blood flow in favor of the ductus venosus at the expense of hepatic blood flow. Unlike peak velocity during ventricular systole, there were reduced or even reversed flow velocities during atrial contraction. One may speculate that increased end-diastolic right ventricular pressure would not influence ductus venosus blood flow velocities during atrial contraction, as flow is preferentially directed through the foramen ovale to the left atrium. However, the foramen ovale is closed during atrial contraction and blood flow velocity through the foramen ovale decreases to zero.

Alterations of venous flow velocity waveforms are in a closer temporal relationship to intrauterine fetal jeopardy, compared to changes in arterial flow, which may occur quite early during the course of impaired placental function. The degree of fetal acidemia can be estimated from Doppler measurements of pulsatility in both the arterial system and the ductus venosus. This was shown in a cross-sectional study of 23 severely growth-restricted fetuses, examining the relationship between Doppler measurements and umbilical venous blood gases obtained at cordocentesis [17].

With moderate acidemia (pH between -2 and -4 standard deviations from the normal mean for gestational age), almost all fetuses will have middle cerebral artery PI below two standard deviations, whereas there will be wide scatter of individual results for the ductus venosus, with the majority of measurements being still within the reference ranges. With increasing severity of hypoxemia and acidemia, ductus venosus PIs increases and, in the most severe cases, velocities with atrial contraction will be reduced to zero or

even become negative (deep or negative A wave). In a study investigating the association of arterial and venous Doppler findings with adverse perinatal outcome in severe fetal growth restriction, abnormal Doppler velocimetry of the ductus venosus was the only significant parameter associated with perinatal death and low 5-min APGAR scores [18].

Material and Methods

Our study was conducted in North Karnataka region of India, in a rural setup. This region is less urbanized and has poor access to health care facilities. This study is pilot study to test the validity of the proposed staging system of AFH. We conducted our study at 2 nursing homes, 1 tertiary care centre and 1 radiology centre for over 1 year period from May 2015 to May 2016. We studied and followed up 160 pregnant women and correlated Doppler findings with end obstetric result. After applying inclusion and exclusion criteria (Table 1), data of 132 patients were included in the study.

Table-1: Inclusion and Exclusion criteria for subjects selection for the study

Inclusion criteria	Exclusion criteria
• Pregnant patient between 34 weeks to 42 weeks • Precious pregnancy- married life >3y without successful conception or conceived by some ART help. • Uncomplicated / non high risk pregnancy • Relative Oligohydramnios • Past poor obstetrics history • Patients on continuous follow up post Doppler study	• IUGRestricted subjects • Multifetal pregnancy • Fetus with structural OR Chromosomal anomalies • CCF / Warning signs in first Doppler study • Pulsatile Umbilical vein • Loss of end diastolic flow in aorta • Umbilical PI >1.2 (cerebral odema) • TR, PR, Reverse `a' wave in DV Doppler • Fetal ascites, pleural effusion, pericardial effusion. • Placental odema • Patients with labour pains. • Patients with Chronic hypoxia

Informed consents were obtained from all the subjects. Subjects underwent Doppler study when they were referred by an obstetrician and followed the inclusion criteria. Subjects were allowed to rest for 10-15 mins in a left lateral recumbent position prior to commencement of the Doppler study. Doppler study was performed with Toshiba Xario model SSA-660A with a broadband matrix curvilinear 3.75 MHz tissue harmonics probe, by a single Sonologist to minimize the variability between the individuals and the reports. Patient's brief history was noted and then followed by measurement of fetal biometry; Doppler values of Umbilical Artery (UmA), Middle cerebral artery (MCA), Thoracic Aorta (TAo), Uterine Arteries (UTA) were recorded. Umbilical artery Doppler flow velocity waveforms were obtained from a free loop of chord. Measurements were acquired in the absence of fetal breathing or body movements with ultrasound beam/flow velocity angle less than 45^0. The machine has a software provision for auto tracing and manual angle correction post acquire. The Thoracic Aorta was visualized in the longitudinal view of the fetus; the transducer was tilted so that an angle of 45^0 or less was created between beam and Aorta. To obtain measurements from MCA, the transducer was positioned so that the sphenoid bones were seen in a transverse view of the fetal head. The MCA conveniently forms the angle of less than 45^0, and the Doppler indices were obtained.

The Doppler variables used in this study are MCA-PI, TAo-TAV and the product of MCA-PI and Tao-TAV. These values were plotted against the graphs for each subject according to their gestation age and determined whether the value lies within the normal range, above

or below the normal range. These graphs showing the normal range of the Doppler values are plotted against the gestation age. Based on the above position in the graph in all the three variables (MCA-PI, TAo-TAV and the product of MCA-PI and Tao-TAV) the staging of the hypoxia was determined as shown in the (Table 2). These graphs or nomograms are based on Kevin Harrington and Stuart Campbell [19].

Table-2: Proposed Doppler staging for Early detection of Acute Fetal Hypoxemia

Stages	Criteria			Hemodynamics	
	MCA-PI	TAo-TAV	MCV-PI*Tao-TAV	Circulation	Fetal Heart Rate
0	Normal	Normal	Normal	Normal fetal circulation	Normal
1	↓	Normal	Normal	Earliest sign of Fetal cerebral hypoxia	Tachycardia/ normal
2	↓	↑	Normal/↑	Early response to Fetal cerebral hypoxia	Tachycardia/ normal
3	↓	↓	Declining but visible within same normal range	Differential circulation by decreasing Aortic flow, beginning of increase in UmaPI	Tachycardia/Normal
4	↓/↑	↓	↓	Early Cardiac involvement	Bradycardia initiates here

In the acute fetal hypoxia (AFH) staging (Table 2), stage 0 is classified when values of all three variables within normal range. Stage I is defined when MCA-PI is low and TAo-TAV is normal and the product is normal. IInd stage consists of compensatory increase in TAo-TAV value in response to low MCA-PI. In IIIrd stage there is decline in MCA-PI and TAo-TAV, but the product is still within normal range. IVth stage involves all the three variables are below lower limit of normal range.

Stage 0 and stage I subjects were advised to come for regular follow-up at the clinic and stage II subjects were advised to repeat Doppler scan between 7 days to 14 days after 1st scan. Stage III and stage IV were advised admission and close supervision. Termination of pregnancy was decided based on obstetric clinical basis.

The fetal distress clinical criteria used are;

- Decreased fetal movement felt by the mother
- Meconium in the amniotic fluid ("meconium stained fluid")
- Non-reassuring patterns seen on cardiotocography.
- Decreased variability in the fetal heart rate.
- Persistent Bradycardia
- Variable deceleration
- Recurrent late decelerations
- Increased or decreased fetal heart rate (tachycardia and bradycardia), especially during and after a contraction.

Once fetal distress was clinically suspected, standard guidelines for termination of pregnancy either by vacuum, forceps or emergency lower segment cesarean section (LSCS) was performed. APGAR score (table 3) for the babies was recorded and specialist neonatology care was provided as per protocol.

APGAR score at 0 minute and 5 minute was recorded along with weight of the baby (Table 3). All the data was tabulated in Microsoft Excel spreadsheet and Statistical methods applied were Mean, Standard deviation, Chi square test, X^2/ Fisher's exact test, Relative risk, p-value (significance).

Table-3: APGAR score for new borns				
Score of 0	**Score of 1**	**Score of 2**	**Component of acronym**	
Complexion	Blue or pale all over	Blue at extremities body pink (acrocyanosis)	No cyanosis body and extremities pink	Appearance
Pulse rate	Absent	< 100 beats per minute	> 100 beats per minute	Pulse
Reflex irritability grimace	No response to stimulation	Grimace on suction or aggressive stimulation	Cry on stimulation	Grimace
Activity	None	Some flexion	Flexed arms and legs that resist extension	Activity
Respiratory effort	Absent	Weak, irregular, gasping	Strong, lusty cry	Respiration

Results

Our study is a pilot study which lasted for one year in which we included 160 subjects in study, out of which 28 subjects were either failed to follow up or ended up IUGRs; therefore they were excluded from the study as per exclusion criteria. In total there were 132 subjects which were followed up and included in the study. Out of 132 subjects, one subject had Intra Uterine Fetal Death (IUFD) at 39th week. This subject with IUFD was advised immediate admission after her last Doppler study but she delayed her admission by 8 days; two days after total loss of fetal movements. At admission it was found to be IUFD. All the subjects had undergone at least two routine obstetric scans before 3rd trimester and at least one Doppler study after 34 weeks. Only singleton pregnancies were included.

Mean age of the subjects was 25.8 years with standard deviation was 0.67. No significant variation between age groups of < 25years & > 25 years. Among the subjects 77 (58.3%) were primi para and 55 (41.6%) subjects were multipara. Out of 77 primies 27 subjects had fetal distress (35.06%). Among 55 multies 5 subjects had fetal distress (9.09%). The association between gravida with fetal distress showed high significance. Primies had 3.8 times higher risk as compared to multies [relative risk of 3.857(1.58-9.38)]. Statistically it was found that stages of acute fetal hypoxia (AFH), gravida, weight of baby after birth and APGAR score at zero minute were associated with fetal distress ($p \leq 0.005$). But APGAR score at 5 minutes, age of the subjects, gestation age of the pregnancy and gender of the fetus were not associated with fetal distress ($p \geq 0.005$) (Table 4, 5 & 6).

Table-4: Variables, their mean and standard deviation	
Variables	**Mean ± SD**
Age	25.8±.67
MCA PI	1.30±0.38
TAo TAV	41.19±12.56
MCA-PI * TAO-TAV	54.52±25.15
Weight	2.90±0.45
APGAR Score (0 MIN)	7.22±1,35
APGAR Score (5 MINS)	9.14±1.35

Table-5: Subjects classified into 5 stages of AFH and its statistical significance				
Stages	**Fetal Distress**		**Total Cases**	**Statistical Test**
	Yes	**No**		
0	5	20	25	$X^2 = 20.28$
1	4	21	25	P=0.0004
2	11	48	59	(Significant)
3	5	10	15	
4	7	1	8	

Table-6: Different variables and their statistical significance						
Stages		**Yes**	**No**	**Total**	**X^2/Fisher's Exact Test**	**RR (95%(CI)**
4 vs 0						
	4	7	1	8		
	0	5	20	25	***P: 0.0012***	4.375 (1.914 - 10.00)
	Total	12	21	33	(Significant)	
4 vs 1	**4**	7	1	8		
	1	4	21	25	***P: 0.0005***	5.469 (2.145 - 13.94)
	Total	11	22	33	(Significant)	
4 vs 2	**4**	7	1	8		
	2	11	48	59	***P: 0.0002***	4.693 (2.591 – 8.501)
	Total	18	49	67	(Significant)	
4 vs 3	**4**	7	1	8		
	3	5	10	15	***P: 0.0272***	2.625 (1.225 – 5.625)
	Total	12	11	23	(Significant)	
GRAVIDA						
PRIMI		27	50	77	***P=0.0006***	3.857(1.58-9.38)
MULTI		5	50	55	(Significant)	
AGE						
≤25		18	53	71	P=0.7482	1.105(0.6008-2.031)
>25		14	47	61	(Not significant)	
APGAR SCORE (At 0 min)						
≤7		20	30	50	***P=0.001***	2.733(1.465-5.099)
>7		12	70	82	(Significant)	
APGAR SCORE (At 5 min)						
≤7		5	1	6	P=0.1487	1.944(0.919-4.11)
>7		27	99	126	(Not significant)	
GENDER						
MALE		14	51	65	P=0.4752	0.8017(0.4358-1.475)
FEMALE		18	49	67	(Not significant)	
WEIGHT OF BABY						
<2.5		8	8	16		
2.5-3.0		16	59	75	***P=0.0364***	
>3		8	33	41	(Significant)	
GESTATION AGE						
≤37		17	35	52	P=0.0678	1.744 (0.9566-3.178)
>37		15	65	80	(Not significant)	

The study shows that stage IV neonate subjects had 88% incidence of fetal distress (Chart 2 & 3). It was observed that as the stage of AFH increases the incidence of the subjects with fetal distress increased. Stage Zero had relatively higher percentage of fetal distress than stage I and II (16% & 19% respectively) probably because of obstetric problems and missed cases of fetal distress due to transition from stage zero to Stage one correcting or compensating mechanisms (due to base reserve masking mechanism).

Chart-2: Percentage of cases with Festal Distress

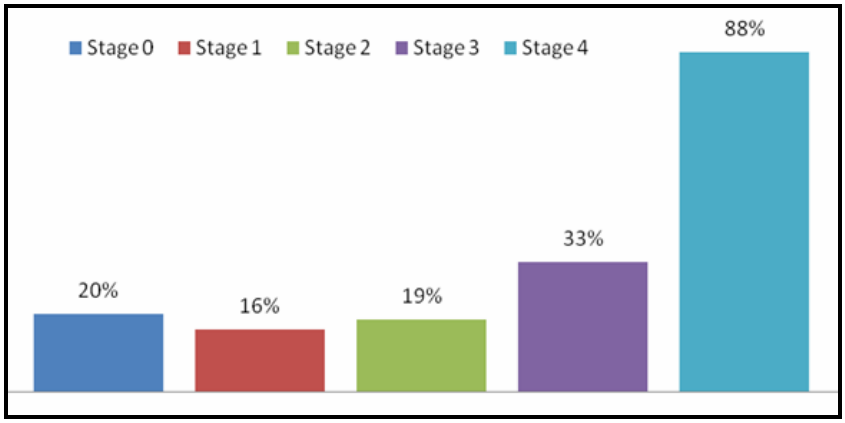
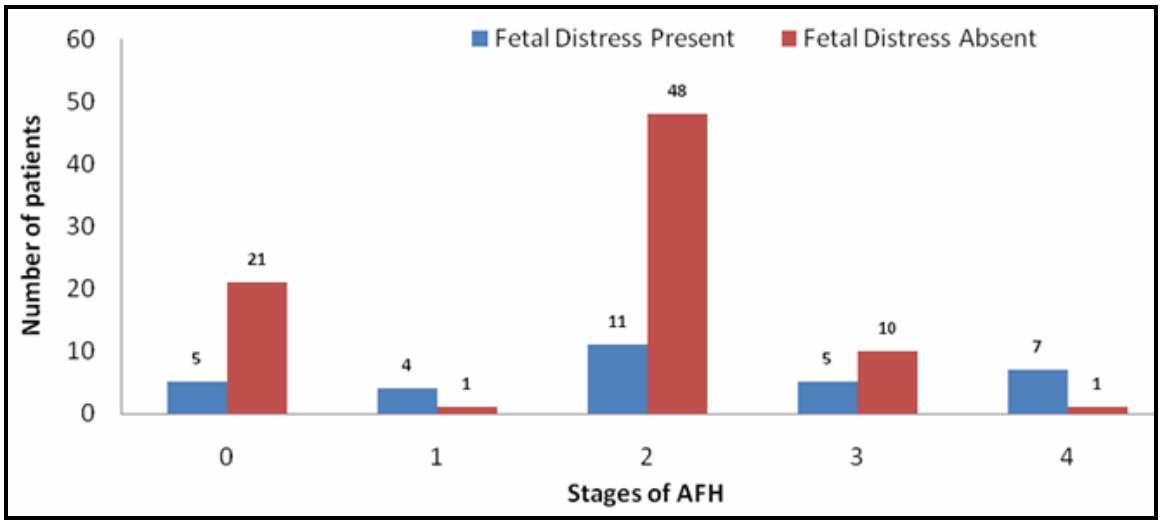


Chart-3: Distribution of Fetal distress in each stage



Out of 132 subjects, 50 neonates (37.87%) had APGAR score <7 at Zero minute, among those, 20 (40%) neonates with APGAR score <7 had typical clinical neonatal distress. No significant difference was found between male and female neonates who had (intrauterine) fetal distress. It was found that babies with <2.5 Kg weight had higher (50%) incidence of fetal distress as compared to babies with more than 2.5kg weight. Therefore lower the weight higher the rate of fetal distress, and statistically association was found to be significant (Table 6).

When stage IV is compared individually with other stages 0, I, II & III; the association is statistically significant ($p \leq 0.005$). The relative risk between stage IV and other stages ranges between 2.5 to 5.5 (stage IV & 0 is 4.375; stage IV & I is 5.469; stage IV & II is 4.693; stage IV & III is 2.625) (Table 6). In the scatter diagrams, Spearman's correlation co-efficient between 3 Doppler variables and the gestation age individually (Chart 4, 5 & 6) showed high significance ($p \leq 0.005$).

Chart-4: MCA PI vs Gestation Age

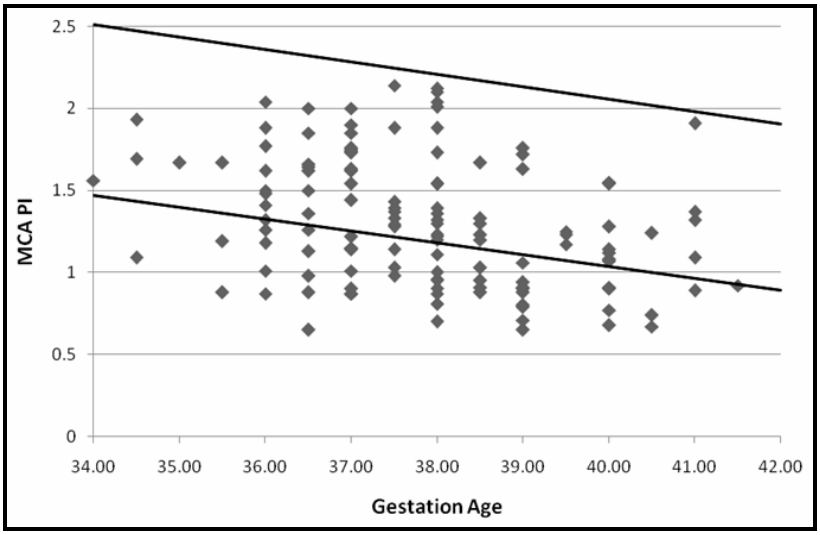


Chart-5: Tao TAV vs Gestation Age

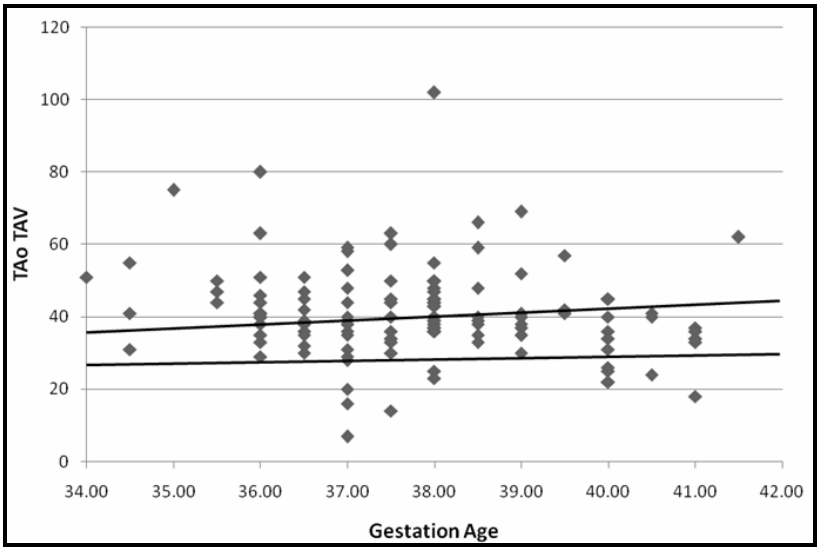
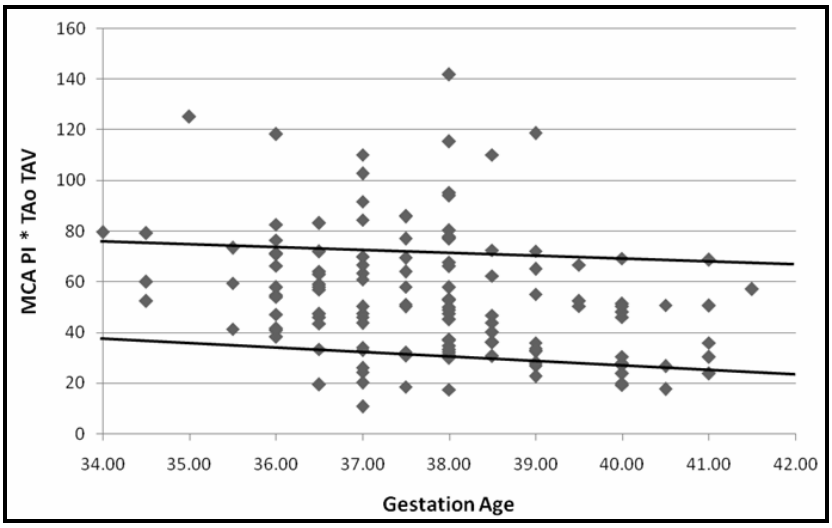


Chart-6: MCAPI*TAoV vs Gestation Age



Discussion

Taking all the previous research & clinical evidences it can be assumed that an event of acute fetal hypoxia (AFH) can have three distinct outcomes; first outcome could be AFH which reverts back to normal after the event, second outcome could be worsening of AFH & progression to IUFD or the third possible outcome is adaptation to hypoxia progressing to IUGRs/SGA. There is abundant research done with regards to chronic hypoxia or the third possible outcome. Very few attempts have been made to study acute fetal hypoxia and to create a staging system for predicting AFH. Some of the reasons for the fewer attempts in this regard is because of complete patho-physiology of AFH is not yet known; repeated Doppler scans are required over shorter duration to know the progression; the Doppler findings are variable from one sonologist to another sonologist due to skill of the sonologist, fetal position, maternal condition at the time of scan. In our study we have attempted to address this issue. This particular article is a pilot study which is actually part of bigger study lasting for 10 years.

The study data shows that out of 132 subjects 32 (24.24%) subjects had clinically detectable fetal distress for which standard protocols were followed, and 30 (22.72%) subjects delivered by LSCS and 2 delivered vaginally. Among those 2 vaginally delivered fetuses, one was IUFD. This high percentage of fetal distress as compared to general population average (6.8%) [20] was due to inclusion criteria like precious pregnancies, relative oligohydramnios, previous poor obstetric history there was high percentage of fetal distress leading to Cesarean section.

This study utilizes proposed new staging system for AFH jointly developed by authors Dr.P.T.Jadhav & Dr.R.P.Jadhav. This proposed staging system is based on the hemodynamic changes in the cerebral and systemic changes secondary to LV function. MCA PI is more sensitive to cerebral hypoxia & TAo TAV is more sensitive to LV function; product is sensitive to adaptation or failure to adaptation. Our study concentrates on stages of failure to adaptation to AFH. As the data shows that as the stage of the AFH increases the percentage of the subjects fetal distress increases, therefore there is a direct relation between AFH staging and fetal distress which is found to be highly significant statistically (p value : 0.0004). It is clear from the data that stage one has subjects with percentage of fetal distress of 16%; stage II has 19% subjects with fetal distress, stage III has 33% subjects and stage IV has maximum of 88% cases with fetal distress. It was also seen that one subject with stage IV classification had IUFD when pregnancy was continued against medical advice. All standard Doppler studies also say that approximately 6 days time to go for IUFD after detecting adverse Doppler indices.

This confirms our study and concludes that a staging system is useful in detecting a fetus in acute fetal hypoxia and appropriate measures can be taken to make sure the fetal morbidity and mortality can be reduced.

Conclusion

This study is first step towards creating a comprehensive staging system for Acute Fetal Hypoxia and formulating guidelines for the same. This staging can predict unexplained IUFD to some extent. This acute fetal hypoxia staging can be useful in predicting impending fetal distress, therefore the authors recommend stage 0, 1 and 2 subjects to have Doppler scan to be repeated after 1 week, and to be managed and followed up on OPD basis.

Stage 3 & 4 subjects to be admitted and observed in hospital for further management. This study is one of the first attempts to show that a staging system is possible to classify and predict fetal distress in advance, Doppler study is non invasive study and can be repeated without adverse effects on pregnancy, this study can be done in any rural setup, this is a cost effective diagnostic tool. As this is a pilot study, a smaller study population was included. A larger study population and longer duration study would be required.

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