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ORIGINAL ARTICLE

Predictability of Fetal Doppler, Biophysical Profile, and Cardiotocography for Fetal Acidosis at Birth

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Abstract Fetal Doppler has allowed evaluation of the fetus in both physiological and pathological conditions which has helped in establishing the relationship between Doppler and fetal oxygenation. It is difficult to define pathological acidosis, but the threshold pH of <7 is the best independent predictor for unexplained seizures. Most infants tolerate acidemia well and recover without any remarkable long term sequelae. Worsening umbilical artery acidosis is directly and adversely related to worsening of neurological outcome, hypoxic ischemic encephalopathy, and multiorgan involvement with permanent neurologic injury. Hypoxic ischemic encephalopathy events are not limited to high-risk pregnancies but may occur in about 50 % of the low-risk population. Combination of low pH at birth with other abnormal clinical parameters, e.g., requirement for intubation, 5 min Apgar score ≤5 has 80 % positive predictability of seizures. Predictability of fetal Doppler examination for asphyxiated fetuses is in the tune of 86 %. High-risk pregnancies are screened antenatally by fetal Doppler, biophysical profile, and CTG to identify at-risk fetuses which are confirmed by the ABG analysis of cord blood immediately after birth. All these noninvasive modalities complement each other to identify, at the earliest, any clinical deterioration. Isolated abnormal, e.g., absent end diastolic flow in umbilical artery, abnormal biophysical profile, or nonreactive CTG are not adequately sensitive in identifying these fetuses which was observed in the present cohort. Thus, fetal Doppler in combination with biophysical profile supplemented with cardiotocograph helps in identifying at-risk fetuses for fetal acidosis and encephalopathy and helps in considering early intervention.

Keywords Fetal Doppler · Biophysical · Cardiotocograph · Hypoxemia · Acidosis · Apgar score · Predictability · Seizures

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Introduction

Fetal Doppler has allowed evaluation of the fetus in both physiological and pathological conditions which has helped in establishing the relationship between Doppler and fetal oxygenation. It is known that increased impedance to uteroplacental circulation is directly related to fetal hypoxemia. It stimulates preferential bloodflow to the brain, heart and adrenals; however, it does not necessarily correlate with hypercapnia and acidosis [1]. But, when this compensatory mechanism fails, it leads to increased pulsatility in the venous circulation which helps in optimizing the timing of delivery [2].

Acidosis is usually well tolerated by the fetus without any sequela. When fetal cord blood pH is >7.0 or base excess is less than minus 12 it does not necessarily have an adverse effect on the fetal cognition or long term complications [3]. It is difficult to define pathological acidosis, but Williams et al. have recommended the threshold pH of <7 as the best independent predictor for unexplained seizures [4].

Materials and Methods

Umbilical vein sampling was done immediately after the baby was born after doubly ligating the umbilical cord. Cord blood arterial blood gas analysis (ABG) was done to identify fetal acidosis after birth. ABG included pH, pCo_2, base excess and lactate. Fetal carbon di-oxide may be removed from the umbilical arterial blood and venous circulation has shown to have slightly higher pH and lower pCo_2 than the arterial blood [5]. Restriction of blood flow in the umbilical cord adversely affects the blood gases, as in cases of tight nuchal cord [6], cord prolapsed [7], or placental abruption with cord obstruction [8]. Even though these techniques have their limitations, yet if used within their specific domain, these may be of immense help to identify at-risk fetuses and to take appropriate measures to prevent neurological impairment.

Results

High-risk antenatal cases were followed-up prospectively in the department of Fetal Medicine at Artemis hospital with fetal Doppler, biophysical profile, and antenatal or intrapartum cardiotocograph (CTG). ABG analysis was done after birth in all the cases. All these antenatal screening tools were utilized for predicting worsening of the clinical situation and to identify at an earliest, the fetuses at risk of fetal hypoxemia and acidosis (Fig. 1).

This cohort of eight high-risk cases with their clinical presentation is depicted in Table 1; the fetal Doppler assessment and ABG analysis after birth are shown in Tables 2 and 3, respectively.

Cohort of high risk was defined by the fetal abdominal circumference or fetal weight <25th centile for the gestational age; abnormal Doppler when umbilical or uterine artery pulsatility index (PI) is >95th centile for the gestational age; abnormal biophysical profile by any score of 4/8 or less and nonreactive CTG (Figs. 2, 3).

Intrauterine growth restriction (IUGR) was identified in 4 cases (case no. 2, 6, 7, and 8). However, acidosis was identified in only 1 case (case no. 8), when the biophysical score dipped to 4/8 with nonreactive CTG. Two cases (case no 6 and 8) had biophysical score of 4/8 and nonreactive CTG (case no 4 and 8) but only 1 (case no. 4) had both of them abnormal and had severe respiratory acidosis. One case (case no. 6) with biophysical score of 4/8 had a reactive CTG and the other one (case no. 4) with normal biophysical score had nonreactive CTG but both of them had normal pH.

Absent end diastolic flow in the umbilical artery was identified in 2 preterm fetuses (case no. 2 and 3), but both of them had normal CTG and normal pH at birth. Fetal acidosis was identified more commonly in term pregnancies (case no. 4 and 8) as compared to preterm fetuses.

Identification of absent end diastolic flow led to the decision to expedite the delivery before ominous venous Doppler appeared to prevent irreversible fetal outcome. One case with nuchal cord with high resistance in the cord was identified to have fetal acidemia (case no. 4). Both the cases of fetal acidemia had high levels of lactate (cases no. 4 and 8, Table 2) depicting an intrinsic metabolic derangement.

Discussion

Predictability of fetal Doppler examination for asphyxiated fetuses is in the tune of 86 % [9]. Even then, most infants tolerate acidemia well and recover without any remarkable long term sequelae [10]. Worsening umbilical artery acidosis is directly and adversely related to worsening of neurological outcome, hypoxic ischemic encephalopathy, multiorgan involvement with permanent neurologic injury.

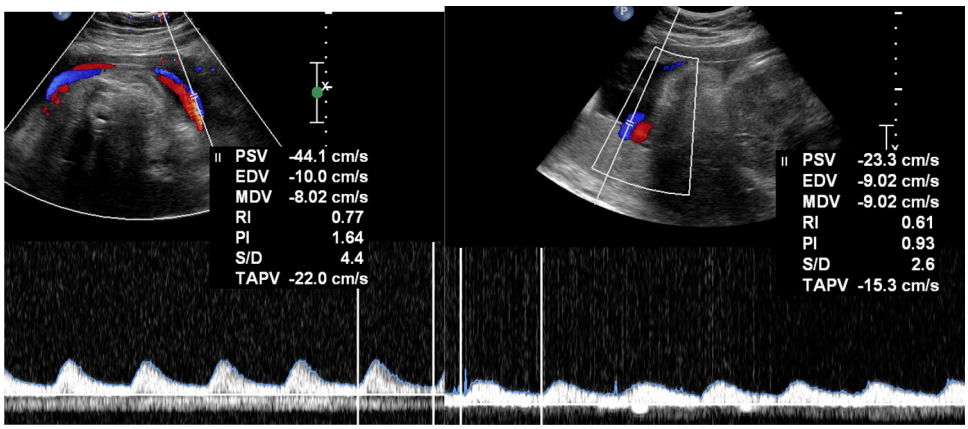
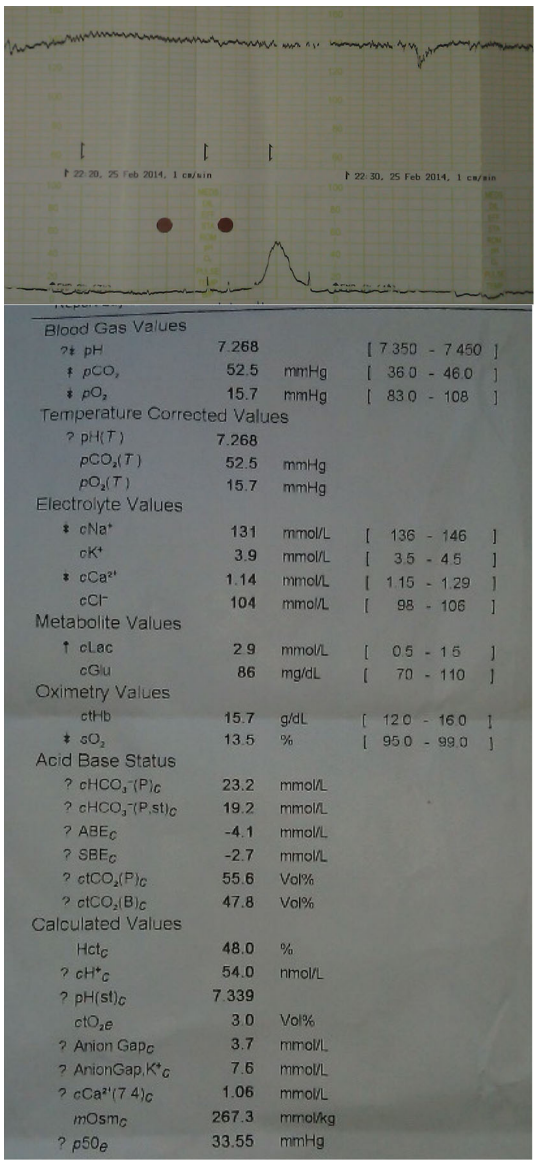


Fig. 1 Increased resistance to blood flow in the umbilical artery in nuchal cord and normal blood flow in the free loop of umbilical cord in case no. 4 (Table 1)

Table 1 Clinical profiles of the cases with their fetal Doppler assessment, biophysical score, CTG, and ABG analysis

Case no.	Period of gestation (weeks)	Confounding factors	AFI	Fetal Doppler	Biophysical profile	CTG	ABG	Birth weight (kg)	Mode of delivery
1	Term; 37 + 6	No	Oligohydramnios; AFI 6.2 cm	Brain sparing	Normal	Reactive	pH 7.355	3.36	LSCS; emergency
2	Pre-term; 32 + 6	IUGR	Normal; 12.9 cm	Absent end diastolic flow	Normal	Reactive	pH 7.32	1.39	LSCS; emergency
3	Pre-term; 33	GDM; high MSAFP at quadruple testing—2.7 MoM	Normal; 13 cm	Absent end diastolic flow	Normal	Reactive	pH 7.47	1.2	LSCS; emergency
4	Term; 37 + 6	IDDM; hypothyroidism; cholestasis; PIH	Normal; 11.9 cm	High umbilical artery resistance in loop around neck	Normal	Nonreactive; thick meconium	pH 7.26	2.76	Induction
5	Term; 38	Hypothyroidism; cholestasis	Oligohydramnios; AFI 9.0 cm	Normal	Normal	Reactive	pH 7.30	3.2	Vaginal
6	Pre-term; 36 + 3	Symmetrical IUGR; placenta previa type IV; absent fetal movements	Anhydramnios	Brain sparing	Score of 4/8	Reactive	pH 7.30	2.2	LSCS; emergency; placenta previa
7	Pre-trem; 35 + 1	Symmetrical IUGR; cholestasis	Oligohydramnios; AFI 8.5 cm	Normal	Normal	Reactive	pH 7.30	2.9	Elective LSCS
8	Term; 38	Hypothyroidism; Cholestasis; symmetrical IUGR	Oligohydramnios; AFI 9.2 cm	Normal	Score of 4/8	Nonreactive	pH 7.19	2.9	LSCS; emergency; impending eclampsia

ABG arterial blood gas analysis, *AFI* amniotic fluid index, *CTG* cardiotocograph, *GDM* gestational diabetes mellitus, *IDDM* insulin dependent diabetes mellitus, *IUGR* intrauterine growth restriction, *MSAFP* maternal serum alpha fetoprotein, *PIH* pregnancy induced hypertension



Blood Gas Values			
?‡ pH	7.268		[7.350 - 7.450]
‡ pCO_2	52.5	mmHg	[36.0 - 46.0]
‡ pO_2	15.7	mmHg	[83.0 - 108]
Temperature Corrected Values			
? pH(T)	7.268		
$pCO_2(T)$	52.5	mmHg	
$pO_2(T)$	15.7	mmHg	
Electrolyte Values			
‡ cNa^+	131	mmol/L	[136 - 146]
cK^+	3.9	mmol/L	[3.5 - 4.5]
‡ cCa^{2+}	1.14	mmol/L	[1.15 - 1.29]
cCl^-	104	mmol/L	[98 - 106]
Metabolite Values			
↑ cLac	2.9	mmol/L	[0.5 - 1.5]
cGlu	86	mg/dL	[70 - 110]
Oximetry Values			
ctHb	15.7	g/dL	[12.0 - 16.0]
‡ sO_2	13.5	%	[95.0 - 99.0]
Acid Base Status			
? $cHCO_3^-(P)_c$	23.2	mmol/L	
? $cHCO_3^-(P,st)_c$	19.2	mmol/L	
? ABE_c	-4.1	mmol/L	
? SBE_c	-2.7	mmol/L	
? $ctCO_2(P)_c$	55.6	Vol%	
? $ctCO_2(B)_c$	47.8	Vol%	
Calculated Values			
Hct_c	48.0	%	
? cH^+_c	54.0	nmol/L	
? $pH(st)_c$	7.339		
ctO_2e	3.0	Vol%	
? Anion Gap_c	3.7	mmol/L	
? $AnionGap,K^+_c$	7.6	mmol/L	
? $cCa^{2+}(7.4)_c$	1.06	mmol/L	
$mOsm_c$	267.3	mmol/kg	
? $p50_e$	33.55	mmHg	

Fig. 2 Nonreactive cardiotocograph (CTG) with loss of baseline variability with arterial blood gas (ABG) analysis of the fetus showing mixed acidosis in case no. 4 (Table 1)

Combination of low pH at birth with other abnormal clinical parameters, e.g., requirement for intubation, 5 min Apgar score ≤5 has 80 % positive predictability of seizures [11]. However, in the present series, both the acidotic fetuses responded very well to the treatment and recovered without any sequela.

Noninvasive antenatal techniques to predict mortality in preterm fetuses is almost similar to that of term fetuses. Inspite of absent end diastolic flow in umbilical artery in two preterm fetuses, none of them had acidosis stresses, the fact that surviving preterm fetuses tend to have higher umbilical artery pH as compared to those who die [12].

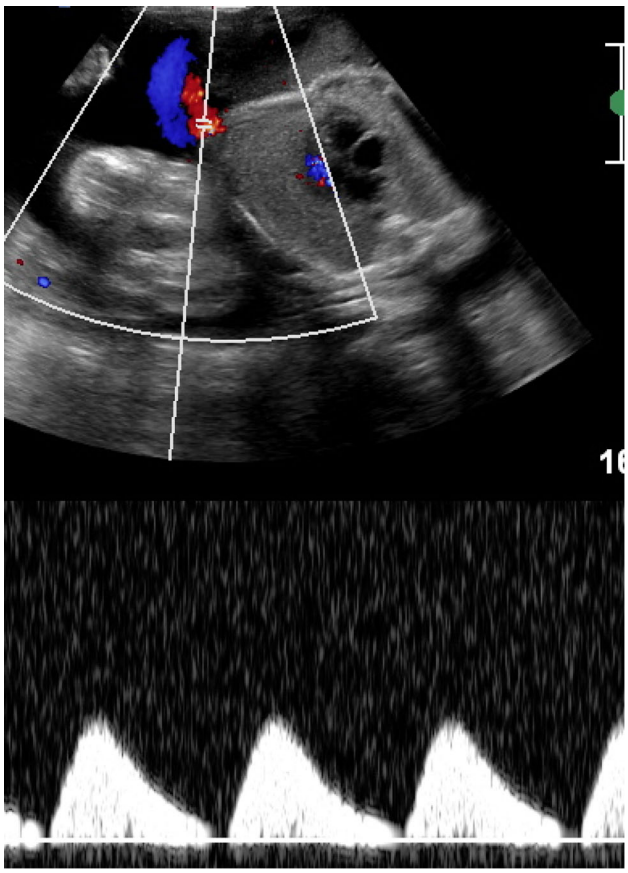


Fig. 3 Absent end diastolic flow in the umbilical artery in case no. 2 and 3 (Table 1)

Cord blood lactate has been identified to be almost fetal in origin and since it does not cross the placenta, it correlates very well with pH and base excess [13]. In the present series, both the cases of fetal acidemia had high lactate, which is a very specific sign of anaerobic metabolism and has high sensitivity (approaching 100 %) and specificity (95.4 %) for predicting neonatal encephalopathy [14].

In this series, none of the cases with absent end diastolic flow in umbilical artery had acidosis which is in concordance with the fact that markedly reduced or absent end diastolic flow of umbilical artery is an independent risk factor for fetal hypoxia and acidosis [15]. However, both the cases were delivered before the reversal of arterial flow or the venous changes appeared, as the fetuses with abnormal venous Doppler have worse prognosis [16] with almost all of them at risk of being asphyxiated [17].

Predictability of fetal biophysical score of ≤6 [18] and abnormal CTG trace for fetal acidosis (pH < 7.25) is only 50–65 % [19]. However, in the present series, when both low biophysical score was associated with loss of variability on CTG, the fetus was found to be acidotic. Growth

Table 2 Depicting fetal Doppler assessment of the high-risk cases

Case no.	Gestation (weeks)	Right MCA		Umbilical artery		Right uterine artery		Left uterine artery		Umbilical vein	Diastolic flow	Comment
		S/D ratio	PI	S/D ratio	PI (normal <3)	RI	PI	RI	PI			
1	37 + 6	2.7	1.13	2.9	1.2	0.48	0.77	0.56	0.99	Continuous forward flow	Present	Brain sparing
		2.9	1.13									
		2.8	1.10[a]									
2	32 + 6	3.4	1.30		2.12	0.54	0.82	0.59	1.07	Continuous forward flow	Absent	Absent end diastolic flow
					2.12							
					2.19							
					2.03[a]							
3	33	3.9	1.49		2.48	0.62	1.29	0.74	1.48	Continuous forward flow; preload index inferior vena cava—0.23 (Normal)	Absent	Absent end diastolic flow with high uterine artery resistance
4	37 + 6	2.2	0.9	3.3	1.24	0.54	0.99	0.58	0.95	Continuous forward flow	Present	High umbilical artery resistance in nuchal cord; normal flows in free loop S/D ratio = 2.7; PI = 1.11
		3.0	1.16	4.4	1.20							
				3.4	1.64							
				3.6[a]	1.32							
5	38	8.5	2.5	2.4	0.85	0.52	0.76	0.49	0.80	Continuous forward flow	Present	High uterine artery resistance
6	36 + 3	2.5	1.0	2.6	0.93	0.41	0.74	0.52	0.79	Continuous forward flow	Present	Brain sparing
		2.5	0.97									
		2.6[a]	0.98									
7	35 + 1	8.6	2.0	2.7	1.0	0.47	0.73	0.51	0.80	Continuous forward flow	Present	Normal Doppler
8	38	2.9	1.15	2.3	0.84		1.0	0.63	1.2	Continuous forward flow	Present	Brain sparing

[a] These different values were recorded 30 min apart in cases with abnormal Doppler values

MCA middle cerebral artery, *PI* pulsatility index, *RI* resistive index, *S/D ratio* systolic-diastolic ratio

Table 3 Depicting arterial blood gas analysis of the cases

Case no.	pH	pCO_2	Lactate	HCO_3	Base excess	Interpretation
1	7.3	44.0	1.5	23.9	−1.4	Mild mixed acidosis
2	7.3	45.0	2.0	22.6	−3.2	Mild mixed acidosis
3	7.4	40.0	3.5	23.5	0.4	Normal pH
4	7.2	52.5	2.9	23.2	−4.1	Mixed acidosis
5	7.36	45.0	1.3	25.3	0.2	Normal pH
6	7.3	42.2	2.0	18.0	−1.1	Metabolic acidosis
7	7.5	41.5	1.5	32.0	−2.4	Metabolic alkalosis
8	7.1	63.7	3.7	23.8	−6.1	Severe respiratory acidosis

restricted fetuses with brain sparing may have hypoxemia and, if complicated with abnormal fetal Doppler and ominous CTG, may have associated fetal acidosis.

Conclusion

As early as 1958, umbilical cord blood gas analysis was being utilized to identify fetal hypoxic stress [20]. Ever since, it has been widely accepted and now recommended by both American and British College of Obstetricians and Gynecologists to all the high-risk deliveries with its increasing clinical and medico-legal importance [21, 22]. Hypoxic ischemic encephalopathy events are not limited to high-risk pregnancies. They may occur in about 50 % of the low-risk population [23], and thus is recommended in all high-risk deliveries.

High-risk pregnancies are screened antenatally by fetal Doppler, biophysical profile, and CTG to identify at-risk fetuses which is confirmed by ABG analysis of cord blood immediately after birth. All these noninvasive modalities complement each other to identify at the earliest any clinical deterioration. Isolated abnormal, e.g., absent end diastolic flow in umbilical artery, abnormal biophysical profile, or nonreactive CTG are not adequately sensitive in identifying these fetuses which is observed in the present cohort.

Fetal Doppler in combination with biophysical profile supplemented with CTG helps in identifying at-risk fetuses for fetal acidosis. Umbilical cord blood gas analysis in combination with other clinical parameters, Apgar scores, need for ventilation, cardiopulmonary compromise, helps in identifying at-risk infants for encephalopathy and to consider early intervention.

Conflict of interest None.

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