International Journal of Clinical Obstetrics and Gynaecology

ISSN (P): 2522-6614
ISSN (E): 2522-6622

www.gynaecologyjournal.com
2021; 5(6): 155-158
Received: 07-09-2021
Accepted: 09-10-2021

Dr. Lalropuii
Obstetrician and Gynaecologist, Mamit District Hospital, Mamit, Mizoram, India

Dr. Sujatha TL
Professor, Department of OBG, SAT Hospital, Thiruvananthapuram, Kerala, India

Dr. Ayisha Begam
Assistant Professor, Department of OBG, Kanachur Institute of Medical Sciences, Mangalore, Karnataka, India

Corresponding Author:
Dr. Ayisha Begam
Assistant Professor, Department of OBG, Kanachur Institute of Medical Sciences, Mangalore, Karnataka, India

The fetal biometry and doppler parameters on ultrasound among pregnant women complicated with moderate and severe anemia in third trimester

Dr. Lalropuii, Dr. Sujatha TL and Dr. Ayisha Begam

DOI: https://doi.org/10.33545/gynae.2021.v5.i6c.1074

Abstract
Hypoxia-induced release of nitric oxide and opioids can account for much of the adenosine-independent cerebral vasodilatation in response to hypoxia in the fetus. Direct effects of hypoxia on cerebral arteries account for the remaining fraction, although the vascular endothelium contributes relatively little to hypoxic vasodilatation in the immature cerebral circulation. This was a hospital based cross sectional study and all cases of moderate to severely anaemic pregnant women in Obstetric and Gynaecology Department, with gestational age of 28 weeks and above were included in this study. In group 1, 67% had amniotic fluid index between 5-50thcentile, and there was no AFI <5th centile, while in group 2, AFI<5th centile was found in 21% which was statistically significant. The mean AFI for group 1 and 2 was 11.35+/-3.06cms and 9.19+/-2.34cms respectively (p=0.032).

Keywords: fetal biometry, doppler parameters, pregnant women

Introduction
Joseph Barcroff introduced an area of research on how the fetus withstands an environment of reduced oxygenation during intrauterine life, a century ago. Now a days, using ultrasonic transducers, the measurement of blood flow in the fetal carotid and femoral circulations are possible which permits investigation of the dynamics of the fetal brain sparing response [1]. Now we know that major components of fetal brain sparing during acute hypoxia are triggered exclusively by a carotid chemo reflex and that they are modified by endocrine agents and vascular oxidant tone. Despite intense interest into how the fetal brain sparing response may be affected by adverse intrauterine conditions, this area of research has been comparatively scant
When the late gestation fetus is exposed to acute hypoxia, fetal breathing movements cease. Foetal respiratory movements, and fetal heart rate decreases, both responses favouring a fall in fetal oxygen consumption. The reduction in fetal heart rate prolongs end diastolic filling time, increases end diastolic volume and thereby contributes to the maintenance of cardiac output and perfusion pressure despite bradycardia [2].
This causes increase in ventricular stretch that enhance sarcomere length, tension and contractility by means of the Frank–Starling mechanism, which has been shown to be operational in the late gestation fetus Through this mechanism, left and right ventricular stroke volumes are relatively well maintained in the face of increases in afterload, with the left ventricle having a greater reserve capacity for increases in afterload than the right ventricle. Although increases in preload and ventricular filling pressure may help maintain cardiac output during acute hypoxia, the Frank–Starling mechanism may be somewhat limited for increasing cardiac output above baseline during fetal life. This is partly due to the working point of the fetal circulation being close to or on the plateau of the ventricular function curves, thereby limiting the extent to which increases in ventricular stroke volume can actually lead to elevations in end diastolic ventricular filling pressures. Fetal heart decelerations also slow down the passage of blood through the circulation, increasing the efficiency of gaseous exchange in essential vascular beds [3].
In addition to fetal cardiac compensatory mechanisms, the fetal blood flow is redistributed in response to acute hypoxia away from peripheral vascular beds and prioritised towards essential circulations, such as those perfusing the brain – the so called 'brain sparing effect'. Since oxygen delivery is coupled to oxygen consumption, limiting blood flow to less essential vascular

beds such as the fetal intestines and fetal hind limbs also contributes to the overall decrease in oxygen consumption by the fetal tissues during acute hypoxia. Decreased oxygen delivery to the hind limbs increases lactate output, acidifying the fetal blood, which facilitates the unloading of oxygen from haemoglobin to the fetal tissues as the fetal blood becomes hypoxic [4].

Acute hypoxia promotes adenosine release, which depresses fetal cerebral oxygen consumption through action of adenosine on neuronal A1 receptors and vasodilatation through activation of A2 receptors on cerebral arteries. The vascular effect of adenosine can account for approximately half the vasodilatation observed in response to hypoxia. Hypoxia-induced release of nitric oxide and opioids can account for much of the adenosine-independent cerebral vasodilatation in response to hypoxia in the fetus. Direct effects of hypoxia on cerebral arteries account for the remaining fraction, although the vascular endothelium contributes relatively little to hypoxic vasodilatation in the immature cerebral circulation [5].

According to RCOG guidelines, growth restriction implies a pathological restriction of the genetic growth potential. As a result, growth restricted fetus may manifest evidence of fetal compromise(abnormal Doppler studies, reduced liquor volume).Diagnosis of SGA fetus usually relies on ultrasound measurement of fetal abdominal circumference or estimated fetal weight. Fetal growth restriction is used to describe fetus with an estimated fetal weight that is less than the 10th percentile for gestational age. Distribution of fetal blood flow is determined with C/U resistance ratio which is the ratio between the cerebral (CRI) and umbilical (URI) resistance index. This is always >1.1 during normal pregnancy, but decreases in hypoxic conditions due to increase in placental resistance and cerebral vasodilatation (brain sparing effect). This physiological change can be demonstrated by changes in doppler indices of middle cerebral artery and such cerebral vasodilation can be related to fetal pO2 decrease. Maternal anemia, being a hypoxic condition is also suspected to reduce fetal oxygen supply, despite there being no evidence of placental insufficiency [6].

Methodology

Study design

This was a hospital based cross sectional study.

Study population

All cases of moderate to severely anaemic pregnant women in Obstetric and Gynaecology Department, with gestational age of 28 weeks and above.

Study setting

Tertiary care Hospital setting.

Inclusion criteria

Antenatal patients with >28weeks of gestation Moderate and severely anaemic pregnant women ieHb<10gm/dl (as per ICMR) Age between 18-40 yrs.

Exclusion criteria

Those antenatal patients with any comorbidities affecting fetal growth and size (Toxoplasmosis, Rubella, Cytomegalovirus, Herpes infection, overt and gestational diabetes, gestational and chronic hypertension, malignancy, hereditary anemia, smoker or alcoholic, hemoglobinopathies, multiple gestation, Diagnosed renal or cardiac illness. etc) and those who are not willing to give consent.

Sample size

Sample size is fixed to 35.

Sampling technique

Consecutive cases satisfying the sample size.

Results

There was a disparity of >4 weeks between gestational age and ultrasound average gestational age among 64% of group 1(severe anemia), however, it was not observed in moderate anaemia.

Table 1: Showing disparity between Ultrasound and menstrual/corrected Gestational age

USSGA	Degree of anemia				Total		χ^2	df	p
	Group 1		Group 2						
	N	%	N	%	N	%			
<2 weeks	16	76	1	7.1	17	48.6	21.819	3	<0.001
2-4 weeks	5	24	4	29	9	25.7			
>4 weeks	0	0	9	64	9	25.7			
Total	21	100	14	100	35	100			

In group 2, AC <5th centile was found in 8 cases, while it was not found in group 2. In group 1, AC of 50-95th centile for gestatioal age was seen in 5 subjects, while it was not found in group 2, this incidence of significant decrease in AC in severe anemia is statistically significant.

Table 2: Abdominal circumference in moderate and severe anemia

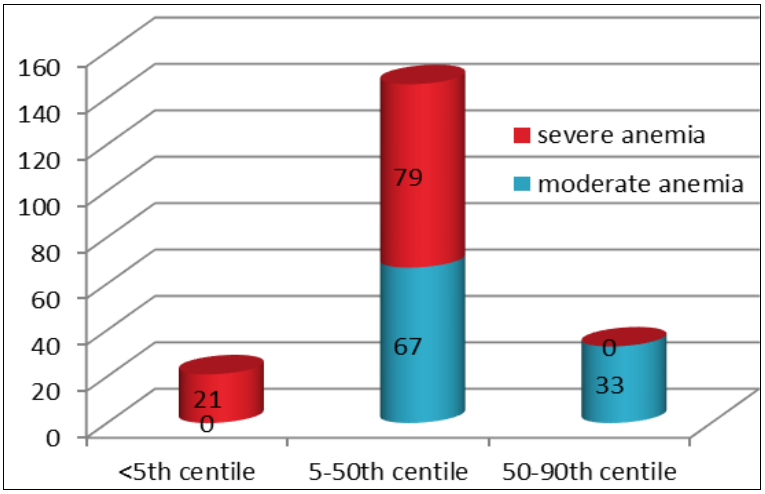
Abdominal circumference	Degree of anemia				Total		χ^2	df	P
	Group 1		Group 2						
	N	%	N	%	N	%			
<5th centile	0	0	8	57	8	22.9	16.818	2	<0.001
5-50th centile	16	76	6	43	22	62.9			
50-95 th centile	5	24	0	0	5	14.3			
Total	21	100	14	100	35	100			

Fetal growth restriction (estimated fetal weight was <10 th centile for the gestational age) was present in 42.9% of the total study group. FGR was observed in 9.5% in group 1 and 78.6% in group 2, which was statistically significant. The mean estimated fetal weight for group 1 and 2 was 2320.14+/-503grams and 1860.86+/-704.06grams (p =0.031) respectively

Table 3: Showing Estimated Fetal Weight in moderate and severe anemia

Estimated fetal weight	Degree of anemia				Total		χ^2	df	P
	Moderate		Severe						
	N	%	N	%	N	%			
<10th centile	2	9.5	11	78.6	13	37.1	17.153	1	<0.00
10-50th centile	19	90.5	3	21.4	22	62.9			
Total	21	100.0	14	100.0	35	100.0			

In group 1, 67% had amniotic fluid index between 5-50thcentile, and there was no AFI <5th centile, while in group 2, AFI<5th centile was found in 21% which was statistically significant. The mean AFI for group 1 and 2 was 11.35+/-3.06cms and 9.19+/-2.34cms respectively (p=0.032)



Graph 1: Amniotic fluid index distribution in moderate and severe anemia

Discussion

A study on "Maternal anemia and fetal cerebral hemodynamic response –Doppler assessment" by Milan Stefanovi *et al*. in 2005, where they studied 32 pregnancies complicated with anemia (moderate and severe) the biometry, amniotic fluid index, uterine, cerebral and Umbilical Doppler resistance index were measured and found that all parameters were lower in severe anemia complicating pregnancies in comparison to moderate anaemia [7].

A study conducted by carles G *et al*. on Doppler assessment fetal cerebral hemodynamic response to moderate or severe maternal anemia on 39 pregnancies complicated with moderate to severe anemia found that only severe maternal anemia (maternal hemoglobin level, <6 mg/L) triggered fetal cerebral vasodilation and reduced amniotic volume [8].

Recent evidence suggests that a high UA-PI and low MCA-PI, regardless of fetal size, is associated independently with intrapartum fetal compromise, low neonatal blood pH and neonatal unit admission MCA-PI, UA-PI and their ratio cerebroplacental ratio (CPR)) may have an important role to play in third trimester assessment of fetal wellbeing and screening for fetal hypoxemia [9].

A study on middle cerebral artery PSV Doppler by Mari G. *et al.* in 2007 have found that MCA PSV in IUGR fetuses provide better clinical information than does low MCA PI. They also mention that raised in PSV in anemic fetuses is due to increase cardiac output and decrease blood viscosity [10].

In order to investigate adaptive mechanisms by the fetus to overcome the growth disadvantage caused due to maternal nutritional limitations, Mahajan S Singh *et al*. have examined the quantitative variations in hormonal and growth factor profiles in paired cord blood and maternal samples obtained from neonates born to malnourished and/or anemic mothers. The results of their study showed a significantly higher levels of GH, PRL, HPL and IGF-1 in the cord blood of malnourished and anemic mothers indicative of an adaptive response on part of the fetus to overcome an in-utero growth disadvantage [11].

Mahajan S *et al*., in their study "Nutritional anaemia dysregulates endocrine control of fetal growth" have hypothesize that endocrine alterations may occur in the maternal-fetal milieu as a consequence of nutritional anaemia during pregnancy and examined the quantitative variations in hormonal profiles of maternal and cord blood samples obtained from mothers and their neonates. Their results show that: (1) 74.6% of the mothers in the study were anaemic, of which 85.2% had moderate anaemia and 14.7% had severe anaemia; (2) anthropometric parameters measured in the mothers showed that severely anaemic mothers had a significantly low pre- and post-pregnancy weight, a significantly decreased maternal fundal height and abdominal circumference; (3) anthropometric measures in the neonates born to severely anaemic mothers show a significant reduction in ponderal index, birth weight and placental weight; (4) significant increase in both maternal, fetal insulin-like growth factor 1, ferritin levels and increased maternal erythropoietin levels were observed with an increase in severity of anaemia [12].

H. Dhand *et al*. in 2011 conducted a study on "Middle cerebral artery Doppler indices better predictor for fetal outcome in IUGR" by comparing the role of middle cerebral artery and umbilical artery Doppler pulsatility indices in predicting the fetal outcome in intrauterine growth restriction. The predictive value of Doppler PI for detecting abnormal fetal outcome was 94% in middle cerebral artery as against 83% for umbilical artery. The sensitivity was 71% for middle cerebral artery versus 44% for umbilical artery. Thus middle cerebral artery doppler indices were a better predictor for fetal outcome in IUGR when compared with umbilical artery in terms of sensitivity and predictive value [13].

A prospective study on Middle cerebral artery to umbilical artery resistance index ratio in the prediction of neonatal outcome was done on 1999 to evaluate the usefulness of the middle cerebral artery to umbilical artery resistance index ratio (C/U ratio) as a predictor of adverse perinatal outcome where the subjects were categorized into two groups, Group A consisting of 35 small for gestational age (SGA) fetuses with a normal C/U ratio, and Group B comprising 35 SGA fetuses with an abnormal C/U ratio and found that the mean C/U ratio values for birth weight and gestational age were higher in group A than in group B. Fetuses born to mothers in group B stayed longer in the neonatal special care unit (NSCU), a higher percentage of mothers with an abnormal C/U ratio underwent ceasarean section [14].

Conclusion

- Fetal growth restriction (estimated fetal weight was <10 th centile for the gestational age) was present in 42.9% of the total study group. FGR was observed in 9.5% in group 1 and 78.6% in group 2, which was statistically significant.
- The mean estimated fetal weight for group 1 and 2 was 2320.14+/-503grams and 1860.86+/-704.06grams (p =0.031) respectively

References

1. Kalaivani K. Prevalence and consequences of anemia in pregnancy. Indian J med Res 2009;130(5):627-33.
2. Maternal mortality in India 1997-2003, Registrar General of India (available from http:/www.censusindia.net/)
3. Kennedy E, Dietary reforms intakes; development and uses for assessment of micronutrient status of women- a global prospective. Am J Clin Nutr 2005;81:1194-98.
4. Lopus AD, Dogers A, Vander HS, Murray C. Selected major risk factors and global and regional burden of disease. Lancet 2002;360(9343):1347-60.
5. Kawaljit Kaur. Anemia a silent killer among women in India: present scenario; Scholars research library. Euro J zool Res 2014;3(1):32-36.
6. Shrinivasa BM, Phillip RR, Krishnapalli VK, Suraj A, Sreelakshmi PR. Prevalence of anemia among tribal women of reproductive age- group in Wayanand district of Kerala. Int Health Allied Sci 2014;3:120-4.
7. Stefanovic M. Maternal anemia and fetal cerebral hemodynamic response-Doppler assessment. Facta universitatis. Medicine and Biology 2005;12(2):93-96.
8. Carles G, Tobal N, Arbeille P. Doppler assessment of the fetal cerebral hemodynamic response to moderate or severe maternal anemia. Ultrasound in Medicine & Biology 2003;29(5):S87
9. Akolekar R, Sarno L, Wright A, Wright D, Nicolaides K. Fetal middle cerebral artery and umbilical artery pulsatility index: effects of maternal characteristics and medical history. Ultrasound in Obstetrics & Gynecology 2015;45(4):402-408.
10. Mari G, Hanif F, Kruger M, Cosmi E, Santolaya-Forgas J, Treadwell MC. Middle cerebral artery peak systolic velocity: a new Doppler parameter in the assessment of growth-restricted foetuses. Ultrasound Obstet Gynaecol 2007;29:310-316.
11. Mahajan S, Singh S, Shah P, Gupta N, Kochupillai N. Effect of Maternal Malnutrition and Anemia on the Endocrine Regulation of Fetal Growth. Endocrine Research 2004;30(2):189-203.
12. Mahajan S, Aalinkeel R, Shah P, Singh S, Kochupillai N. Nutritional anaemia dysregulates endocrine control of fetal growth. British Journal of Nutrition 2008, 100-102.
13. Dhand H, Kansal H, Dave A. Middle cerebral artery Doppler indices better predictor for fetal outcome in IUGR. The Journal of Obstetrics and Gynecology of India 2011;61(2):166-71.
14. Accuracy of the Middle-Cerebral-To-Umbilical Artery Resistance Index Ratio in the Prediction of Neonatal Outcome in Patients at High Risk for Fetal and Neonatal Complications. (1995). Journal of Diagnostic Medical Sonography 1995;11(2):107.