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Original Article

Superior mesenteric artery blood flow in infants of very preterm and/or very low birthweight and related factors

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Abstract

Background Significant hemodynamic changes in preterm infants during early life could have consequences, especially on intestinal blood flow. Decreased of superior mesenteric artery (SMA) blood flow may lead to impairment in gut function and feeding intolerance.

Objective To assess SMA blood flow velocity in very preterm and/or very low birth weight (VLBW) infants in early life and to elucidate potential influencing factors.

Methods This cross-sectional study was conducted in the NICU at Cipto Mangunkusumo Hospital, Jakarta, including very preterm infants (28-32 wk GA) and/or VLBW (1,000-1,500 g) infants. Superior mesenteric artery (SMA) blood flow was evaluated by peak systolic velocity (PSV), end diastolic velocity (EDV), and resistive index (RI) measurement using Color Doppler ultrasonography (US) at <48 hours after birth. Maternal and neonatal characteristics that could be potentially associated with SMA blood flow were analyzed.

Results We examined 156 infants eligible for inclusion. The PSV, EDV, and RI of SMA blood flow were not associated with gestational age or birth weight. Small for gestational age (SGA) infants had significantly lower median EDV [15.5 (range 0.0-32.8) *vs.* 19.4 (range 0.0-113.0), respectively; (P=0.003)] and higher RI [0.80 (range 0.58-1.00) *vs.* 0.78 (range 0.50-1.00), respectively; (P=0.009)] compared to appropriate for gestational age (AGA) subjects. Infants born to mothers with preeclampsia (PE) had lower median PSV [78.2 (range 32.0-163.0) *vs.* 89.7 (range 29.2-357.0), respectively; (P=0.038)]) and EDV [16.2 (range 0.0-48.5) *vs.* 19.4 (range 0.0-113.0), respectively; (P=0.022)] compared to those without maternal PE. Infants with absent/reverse end-diastolic velocity (AREDV) had a lower median EDV [16.9 (range 0.0- 32.4) *vs.* 19.4 (range 0.0-113.0), respectively; (P=0.041)] compared to those without AREDV. Furthermore, infants with hemodynamically significant patent ductus arteriosus (hs-PDA) had lower median EDV [16.2 (range 0.0-113.0) *vs.* 19.4 (range 0.0-71.1), respectively; (P=0.027)] but higher RI median [0.80 (range 0.50-1.00) *vs.* 0.78 (range 0.55-1.00), respectively; (P=0.032)] compared to those without hs-PDA. No difference in SMA blood flow across other factors was observed.

Conclusion Superior mesenteric artery blood flow in very preterm and/or VLBW infants can be assessed using Color Doppler US by measuring PSV, EDV and RI. Changes in these parameters are significantly associated with SGA, preeclampsia, AREDV, and hs-PDA. **[Paediatr Indones. 2023;63:80-7; DOI: https://doi.org/10.14238/pi63.2.2023.80-7**].

Keywords: *intestinal blood flow; superior mesenteric artery; very preterm infants; color Doppler ultrasound*

Color Doppler US has been used to measure blood flow velocity in peripheral vessels to evaluate circulation in the organs, including intestinal circulation, since SMA is the main artery which supplies blood to the small intestine and most of the large intestine. Intestines have circulatory autoregulation that

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keeps SMA blood flow velocity steady in the face of fluctuations in arterial perfusion pressure,[1] but infants with hypotension may overcome the autoregulation, leading to decreased velocity.[2] Any alteration of SMA blood flow could have an impact on gut function, which can lead to feeding intolerance.[3]

Significant hemodynamic changes that occur at birth and during the first week of life in preterm infants are influenced by many factors, both in the womb and after birth. Such changes are thought to reflect redistribution of systemic blood flow to maintain perfusion of vital organs at the expense of abdominal organs, including gastrointestinal tract. Peak systolic velocity (PSV), end diastolic velocity (EDV), and resistive index (RI) are the most commonly used SMA blood flow parameters in neonates. The aim of this study was to assess SMA blood flow velocity in very preterm and/or very low birth weight (VLBW) infants in early life and to elucidate the factors influencing it.

Methods

This cross-sectional study was conducted in the NICU at Cipto Mangunkusumo Hospital (CMH), Jakarta, a tertiary referral and academic hospital in Indonesia. This study was conducted from March 2021 to August 2022 after approval by the Ethics Committee of the Universitas Indonesia Faculty of Medicine.

We included very preterm infants (28 to < 32 weeks gestational age) and VLBW (1,000 to < 1,500 g) infants who were born in Cipto Mangunkusumo Hospital. Infants with lethal and complex congenital disorders (e.g., gastroschisis, gastrointestinal tract atresia, critical congenital heart disease, and syndromes) as well as those who were unable to undergo ultrasound examination during the study period, were excluded.

Subjects' SMA blood flows were assessed by peak systolic velocity (PSV), end diastolic velocity (EDV), and resistive index (RI) examinations using Color Doppler US at the age of < 48 hours after birth. We used an *Affiniti 50* (*Philips*, USA) US machine with 8-5 MHz transducer. Infants were placed in a supine position during the examination and given a non-nutritive sucking pacifier or provided with sucrose if they were agitated. Using a real-time two-dimensional US machine with a longitudinal abdominal approach at the epigastrium region, the sampling volume of the color Doppler was obtained a few millimeters distal to the origin of the SMA. Angle correction was used when necessary. When five stable consecutive wave forms were obtained, the curve was traced and the PSV, EDV, and RI ([PSV-EDV]/PSV) in the SMA were automatically calculated.

Potential factors associated with SMA blood flow were sex, gestational age, birth weight, small for gestational age, any current intrauterine infection, placental blood flow, preeclampsia (PE) with or without $MgSO_4$ administration, antenatal steroid usage, history of neonatal resuscitation, APGAR score, respiratory distress syndrome (RDS), surfactant administration, patent ductus arteriosus (PDA), and highest ventilatory support. All data were obtained from either direct observation or medical records. Placental blood flow data were obtained from US performed by obstetricians. Echocardiography was performed by a certified pediatrician. Patent ductus arteriosus status was defined as hemodynamically (hs-PDA) or non-hemodynamically significant. Hyaline membrane disease was diagnosed based on chest x-ray.

To avoid bias, the examiner, an expert in pediatric ultrasound, was unaware of both the maternal and infant history prior to US. The minimum required sample size calculated to estimate a mean value for PSV, EDV, and RI with 5% of type I error was 76 subjects.

Numerical data were presented as mean and standard deviation if normally distributed, or as median and range if not normally distributed. Relationships between SMA blood flow parameters and gestational age and birth weight were displayed as scatter plots and correlation tests. Bivariate analyses using independent T-test (parametric) or Mann-Whitney U test (non-parametric) were conducted. Results were considered to be statistically significant for P values < 0.05. Data were analyzed using *SPSS version 24* for *Windows* software.

Results

One hundred fifty-six infants were included in this study. Their mean gestational age was 30.3 (SD 1.9) weeks and mean birth weight was 1,278 (SD 273) grams. About 98% of infants required active

resuscitation after birth and among them 54.5% needed mechanical ventilation. Complete subject characteristics are shown in **Table 1**.

All three SMA blood flow parameters showed no specific pattern with regards to gestational age and birth weight in scatter plots, and all the above analyses revealed very weak correlations (**Figures 1** and **2**). These findings indicated that SMA blood flow parameters were independent to gestational age and birth weight, and so, diminished flow was not uniformly present in very preterm and VLBW populations.

Table 2 shows PSV, EDV, and RI values across different patient characteristics. We found that infants with SGA had significantly lower EDV (P=0.03) and higher RI (P=0.09). Infants born from mothers with preeclampsia had lower PSV (P=0.038) and EDV (P=0.022), but no significant difference was found for RI. AREDV was significantly associated with lower EDV (P=0.041) compared to those with normal placental blood flow. Another significant finding was that infants with hs-PDA had lower EDV but higher RI than those without hs-PDA, indicating a worse SMA blood flow possibly due to a stealing effect. Otherwise, there were no significant differences in SMA blood flow across the other factors studied.

Table 1. Baseline characteristics of study participants

Characteristics	(N = 156)
Sex, n (%)	
Male	72 (46.2)
Female	84 (53.8)
Gestational age, n (%)	
< 28 weeks	5 (3.2)
28-32 weeks	135 (86.5)
≥ 32 weeks	16 (10.3)
Mean (SD), weeks	30.35 (1.93)
Birth weight, n (%)	
< 1000 g	22 (14.1)
1000 - <1500 g	104 (66.7)
>1500 g	30 (19.2)
Mean (SD), g	1,278.30 (2753)
Active neonatal resuscitation, n (%)	
Yes	153 (98.1)
No	3 (1.9)
APGAR score at 1 min, n (%)	
0-3	37 (23.7)
4-6	82 (52.6)
7-10	37 (23.7)
APGAR score at 5 min, n (%)	
0-3	1 (0.6)
4-6	42 (26.9)
7-10	113 (72.5)
Hyaline membrane disease, n (%	
Yes	134 (85.9)
No	22 (14.1)
Surfactant administration, n (%)	
Yes	81 (51.9)
No	75 (48.1)
Hemodynamically significant PDA, n (%)	
Yes	53 (34.0)
No	103 (66.0)
Maternal use of antenatal steroid, n (%)	
Yes	75 (48.1)
No	81 (51.9)
Placental blood flow, n (%)	
Reverse/absent	31 (19.9)
Normal	79 (50.6)
Not examined	46 (29.5)

Discussion

To the best of our knowledge, this study is the first in Indonesia to examine SMA blood flow using US in very preterm and/or VLBW infants. We found that SMA blood flow parameters were independent of gestational age and birth weight. The SGA, maternal preeclampsia, AREDV, and hs-PDA were significantly associated with differences in SMA blood flow parameters. These findings are valuable in the assessment of hemodynamic changes in early neonatal life and, may strengthen the utility of US in evaluating intestinal blood flow.

In our study, there was no significant difference in PSV between SGA and AGA infants, but EDV was lower in SGA than AGA subjects, and RI was higher in SGA than AGA subjects. These results were in accordance with previous reports which mentioned that SMA blood flow velocity was lower in SGA infants compared to AGA infants.[4-6] Maruyama *et al.*[7] reported that PSV and EDV were lower in SGA than AGA infants, and RI tended to be higher in the SGA group compared to the AGA group. However, Van Bel *et al.*[8] showed the opposite results. These varying results may have been due to differences in subject characteristics and factors that influence SMA blood flow velocity.

The SMA blood flow velocity was reported to be lower in infants with PDA.[2,7,9,10] A study reported that postprandial SMA blood flow velocity was lower

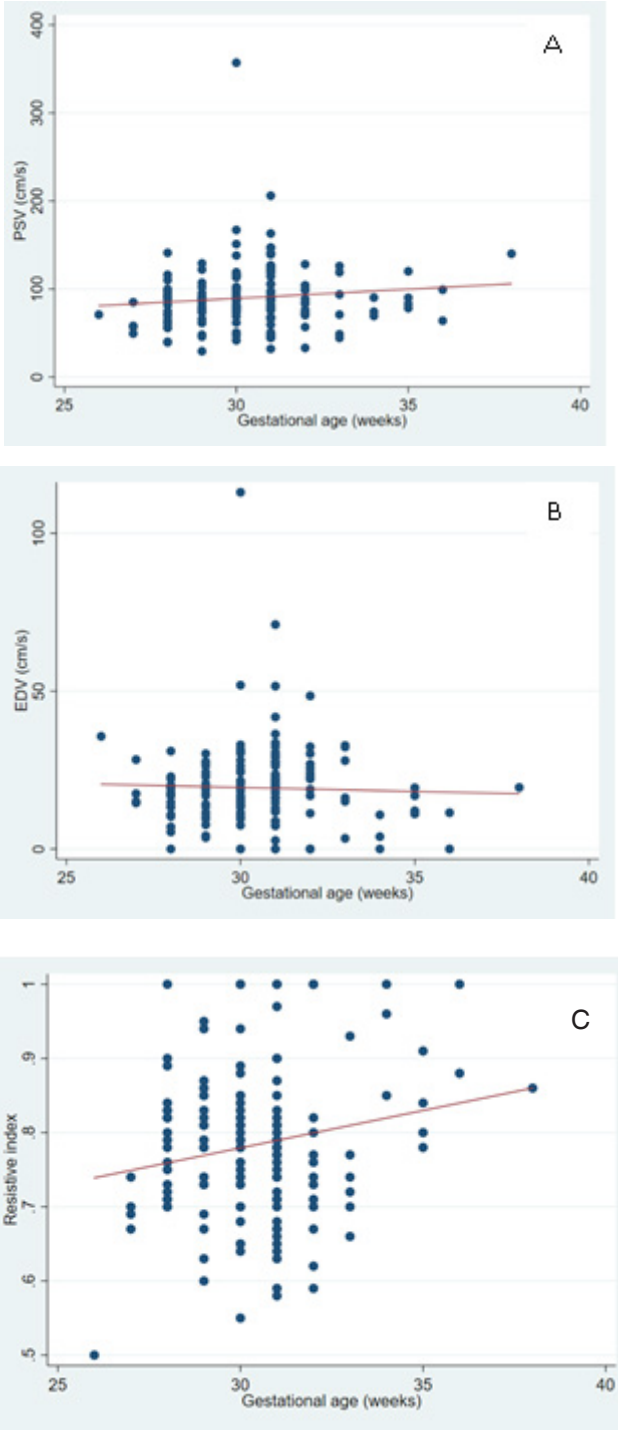

Figure 1. Relationship between SMA blood flow and gestational age
A. PSV (R = 0.11), B. EDV (R = -0.03), C. RI (R = 0.19)

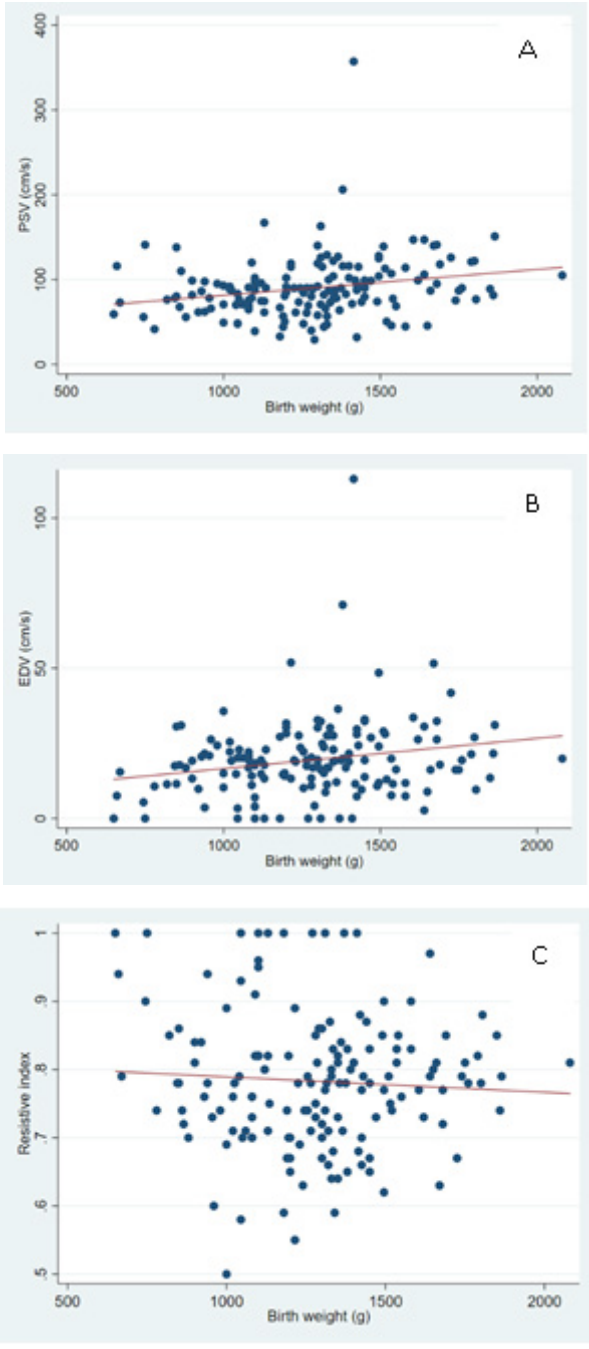

Figure 2. Relationship between SMA blood flow and birth weight
A. PSV (R = 0.23), B. EDV (R = 0.21), C. RI (R = -0.06)

in subjects with large PDAs.[11] Although this finding was not statistically significant, it took longer times for infants with large PDA to reach full enteral intake compared to those with small PDAs. The incidence of death was higher in infants with large PDA due to necrotizing enterocolitis (NEC). In our study, infants with hs-PDA before enteral feeding had significantly lower EDV and higher RI, but no significant difference in PSV compared to those without hs-PDA. This result was in agreement with previous studies.[7,12] Another study reported that EDV was significantly lower in infants with PDA at 1st day of age compared

Table 2. Analysis of potential maternal and infant factors and SMA blood flow parameters

Factors	Median PSV (range)	P value	Median EDV (range)	P value	Median RI (range)	P value
Gender						
Male (n=72)	82.7 (33.0-357.0)	0.268	17.2 (0.0-113.0)	0.101	0.80 (0.50-1.00)	0.104
Female (n=84)	88.3 (29.2-163.0)		19.5 (0.0-51.9)		0.78 (0.55-1.00)	
Small for gestational age						
Yes (n=39)	78.2 (33-141.0)	0.106	15.5 (0.0-32.8)	0.003	0.80 (0.58-1.00)	0.009
No (n=117)	89.0 (29.2-357.0)		19.4 (0-113.0)		0.78 (0.50-1.00)	
Intrauterine infection						
Yes (n=12)	94.0 (60.7-140.0)	0.175	19.5 (13.3-41.8)	0.155	0.78 (0.66-0.86)	0.562
No (n=144)	86.5 (29.2-357.0)		18.7 (0.0-113.0)		0.78 (0.50-1.00)	
Placental blood flow (n=110)						
AREDV (n=31)	80.2 (33.0-167.0)	0.143	16.9 (0.0-32.4)	0.041	0.78 (0.58-1.00)	0.200
Normal (n=79)	89.0 (29.2-357.0)		19.4 (0.0-113.0)		0.78 (0.50-1.00)	
Preeclampsia						
Yes (n=39)	78.2 (32.0-163.0)	0.038	16.2 (0.0-48.5)	0.022	0.78 (0.58-1.00)	0.94
No (n=117)	89.7 (29.2-357.0)		19.4 (0.0-113.0)		0.78 (0.50-1.00)	
Preeclampsia treated with MgSO4 (n=39)						
Yes (n=18)	85.0 (32.0-141.0)	0.310	17.2 (0.0-32.4)	0.507	0.78 (0.60-1.00)	0.631
No (n=21)	74.8 (40.0-163.0)		14.8 (0.0-48.5)		0.79 (0.58-1.00)	
Antenatal steroid						
Yes (n=75)	88.9 (39.2-167.0)	0.173	16.9 (0.0-51.9)	0.408	0.79 (0.55-1.00)	0.116
No (n=81)	84.3 (29.2-357.0)		19.4 (0.0-113.0)		0.76 (0.50-1.00)	
APGAR score at 1 min						
0-3 (n=37)	89.1 (41.6-357.0)	0.728	17.8 (0.0-113.0)	0.868	0.79 (0.59-1.00)	0.869
4-6 (n=82)	83.8 (32.0-151.0)		18.4 (0.0-51.9)		0.78 (0.50-1.00)	
7-10 (n=37)	89.0 (29.2-206.0)		19.4 (0.0-71.1)		0.78 (0.62-1.00)	
APGAR score at 5 min						
0-3 (n=1)	74.1	0.575	10.8	0.471	0.85	0.589
4-6 (n=42)	89.4 (41.6-357.0)		19.5 (0.0-113.0)		0.79 (0.50-1.00)	
7-10 (n=113)	86.6 (29.2-206.0)		17.9 (0.0-71.10)		0.78 (0.58-1.00)	
Highest ventilatory support <48 hours						
None (n=2)	120.5 (113.0-128.0)	0.081	38.3 (28.1-48.5)	0.067	0.69 (0.62-0.75)	0.267
CPAP (n = 34)	90.0 (32.0-151.0)		19.4 (0.0-33.6)		0.79 (0.58-1.00)	
Positive pressure ventilation (n=35)	86.9 (29.2-206.0)		19.9 (0.0-71.1)		0.77 (0.59-1.00)	
Endotracheal intubation (n=85)	79.8 (33.0-357.0)		16.9 (0.0-113.0)		0.78 (0.50-1.00)	
PDA						
Hemodynamically significant	86.3 (33.0-357.0)	0.546	16.2 (0.0-113.0)	0.027	0.80 (0.50-1.00)	0.032
Non-significant	87.6 (29.2-206.0)		19.4 (0.0-71.1)		0.78 (0.55-1.00)	
HMD						
Yes (n=136)	86.8 (29.2 - 206.0)	0.197	18.1 (0.0 - 71.1)	0.513	0.78 (0.50 - 1.00)	0.556
No (n=20)	91.8 (44.3 - 357.0)		19.5 (0.0 - 113.0)		0.79 (0.62 - 1.00)	
HMD treated with surfactant (n=136)						
Yes (n=73)	88.9 (39.2-167.0)	0.251	17.5 (0.0-51.6)	0.765	0.78 (0.59-1.00)	0.447
No (n=63)	84.8 (29.2-206.0)		19.2 (0.0-71.1)		0.78 (0.50-1.00)	

AREDV=absent/reverse end-diastolic velocity umbilical artery; CPAP=continuous positive airway pressure; HMD=hyaline membrane disease; PDA=patent ductus arteriosus; data are presented as median (range)

to those without PDA.[2] Further study is needed to study the impact of low EDV and high RI in infants with PDA and whether these two conditions can be used to predict feeding intolerance and NEC before feeding in early life.

Preeclampsia (PE) was associated with important maternal vascular changes and has a direct effect on SMA blood flow in preterm infants. The PE effects are not modified by PDA.[12] Changes in the blood flow of the gastrointestinal tract of infants due to

PE may impair feeding tolerance and increase the risk for complications such as NEC.[12,13] Of our 156 subjects, 39 infants of mothers with preeclampsia showed significantly lower PSV and EDV compared to infants of mothers without preeclampsia, however, no difference was noticed in RI between the two groups. Further investigation is needed to assess the impact of these findings on both feeding intolerance and NEC.

Administration of $MgSO_4$ as a tocolytic agent to treat preeclampsia may have various effects on the fetus and newborn. Little is known about antenatal $MgSO_4$ effects on fetal and newborn circulatory systems. Most studies on the hemodynamic effects of antenatal $MgSO_4$ have been focused on cerebral vasculature, with few reports on its effects on the gastrointestinal system. Of the 39 infants whose mothers had preeclampsia in our study, 18 were exposed to $MgSO_4$. Although PSV and EDV in these 18 infants were higher than in the 21 infants without exposure to $MgSO_4$, the difference was not statistically significant. Havranek *et al.*[14] also reported no significant differences in PSV, EDV, and mean velocity (MV) of SMA between groups who were exposed and not exposed to antenatal $MgSO_4$.

The AREDV during pregnancy is a strong indication of placental insufficiency, and has been associated with intraventricular hemorrhage, bronchopulmonary dysplasia, respiratory distress syndrome, necrotizing enterocolitis, long-term neurodevelopmental impairment, and perinatal mortality.[15] In our study, infants with AREDV umbilical blood flow had a significantly lower EDV than those with normal placental blood flow. However, no differences were observed in PSV and RI between groups. The relationship between AREDV in the umbilical artery and RDS is still controversial. A previous study found that mother with abnormal umbilical artery velocity had significantly higher incidence of RDS,[16] and another study found that umbilical artery with AREDV in the presence of IUGR was associated with significantly increased incidence of RDS.[17] In contrast to other studies, umbilical artery AREDV did not increase the risk of RDS and they concluded that lung maturation was not related to placental insufficiency.[18-20] In our study, 136 of 156 infants had RDS but only 31 of 110 infants had AREDV. This implies that there might be other factors that contribute to the incidence of RDS.

A previous study found that VLBW infants with RDS who were treated with surfactant had increased left ventricular output, decreased splanchnic and renal blood flows, and abnormal wave forms of blood flow velocities of the organ arteries when hs-PDA developed.[9] We found no difference in PSV, EDV, or RI SMA blood flow between RDS and non-RDS infants, nor between surfactant and non-surfactant treated RDS. This result might have been due to aggressive surfactant administration among infants with respiratory distress while measurements of SMA blood flow were done after surfactant therapy.

In conclusion, Color Doppler US can be used to assess SMA blood flow by measuring PSV, EDV, and RI in very preterm and/or VLWB infants. Significant differences in these parameters are related to SGA, preeclampsia, AREDV, and hs-PDA. Further study is needed to assess the effects of changes in SMA blood flow as predictors of feeding intolerance.

Conflict of interest

None declared.

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