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Umbilical and Main Portal Venous Blood-Flows of Fetal Liver in Normal and Growth Restricted Fetuses and the Impact of the Type of Umbilicoportal Anastomosis on the Main Portal Vein Blood-Flow

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Background: The fetal liver is perfused by the umbilical vein (UV) and the main portal vein (MPV), both of which are crucial for nutrient delivery. The configuration of the umbilicoportal anastomosis may influence MPV blood flow and potentially affect fetal liver perfusion in fetuses with fetal growth restriction (FGR).

Aims: To evaluate absolute and normalized UV and MPV blood flows in fetuses with normal growth and FGR, and to investigate the effect of umbilicoportal anastomosis type on MPV flow.

Study Design: Prospective case–control study.

Methods: Ultrasound was used to measure UV and MPV diameters, while Doppler ultrasound assessed time-averaged maximum velocities. Flow volumes were calculated as time-averaged maximum velocity volume and normalized to estimated fetal weight (TAMXVVN) and abdominal circumference. Anastomoses were categorized as T-, X-, or H-shaped. Z-scores were derived from AGA nomograms.

Results: Compared with AGA fetuses, FGR fetuses exhibited significantly smaller UV diameters, lower absolute UV flow, UV-TAMXVVN, and UV-TAMXVV/AC ($p < 0.05$), but higher MPV-TAMXVVN ($p < 0.05$), suggesting compensatory redistribution. Both UV and MPV flows showed strong correlations with gestational age ($r > 0.7$, $p < 0.001$). UV-TAMXVVN Z-scores decreased with gestation, whereas MPV-TAMXVVN Z-scores increased until 32 weeks before plateauing. Blood flow parameters did not differ significantly across anastomosis types in either group.

Conclusion: FGR fetuses demonstrate reduced UV perfusion with compensatory increases in MPV flow. The type of umbilicoportal anastomosis does not significantly affect MPV blood flow.

INTRODUCTION

The fetal liver receives blood from both arterial and venous systems. The arterial supply contributes only a minor fraction of total liver blood flow and enters directly into the hepatic sinusoids.[1] In contrast, the venous system constitutes the primary blood supply, comprising the umbilical and portal veins. The main portal vein (MPV) and right portal vein (RPV) branches originate embryologically from the vitelline veins, which are fundamental to the development of the portal venous system supplying the right liver lobe.[2] The umbilical vein (UV) enters the liver and continues as the left portal vein, predominantly perfusing the left liver lobe.[2] Approximately 75–80% of the oxygenated, nutrient-rich UV blood is distributed to the fetal liver, while 20–25% bypasses it, flowing directly into the systemic circulation via the ductus venosus (DV).[3] Overall, the fetal liver receives about 70–80% of its venous blood from the UV, with the remaining 14–20% supplied by the portal vein.[4,5]

The umbilical and portal venous systems converge at the portal sinus (PS). According to Kivilevitch et al.,[6] there are three principal types of umbilical-portal anastomosis. In the T-shaped variant, an end-to-side junction exists between the PS and MPV. The X-shaped variant features parallel PS and MPV vessels joined side-to-side, whereas the H-shaped variant is characterized by a thin connecting vessel between the two.[6]

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Several studies have explored the influence of venous liver perfusion on fetal growth in both normal and fetal growth restriction (FGR) fetuses. Adequate venous perfusion is essential for fetal growth, as it promotes hepatic cell proliferation through the expression of insulin-like growth factor I (IGF-I) and IGF-II mRNA.[7] In FGR fetuses, umbilical venous blood flow is significantly reduced.[8] As a compensatory mechanism, a greater proportion of blood is shunted through the DV into systemic circulation, reducing perfusion of the right liver lobe.[9] Additionally, MPV flow may increase in these fetuses, possibly reflecting enhanced arterial perfusion of the splanchnic region.[10] Prior research indicated that the X-shaped anastomosis occurs more frequently in late-onset FGR (LO-FGR), whereas the T-shaped variant predominates in normally growing fetuses.[11] However, hemodynamic patterns associated with each anastomosis type remain poorly understood.

This study aims to assess UV and MPV blood flows, as well as flow values normalized per kilogram of estimated fetal weight (EFW) and per millimeter of abdominal circumference (AC), in both normal and FGR fetuses. Furthermore, we investigated the potential impact of different umbilicoportal anastomosis types on MPV flow.

MATERIALS AND METHODS

This prospective observational case–control study was conducted at the maternal–fetal medicine department of a university hospital between March and December 2024. Ethical approval was obtained from the İstanbul University-Cerrahpaşa Ethics Committee (approval number: 2025/9, date: 08.01.2025), and all procedures were conducted in accordance with the principles outlined in the Declaration of Helsinki. Written informed consent was obtained from all participants, who were fully briefed on the study's objectives and procedures. The study included 151 pregnancies, divided into an appropriate-for-gestational-age (AGA) group (n = 108) and a FGR group (n = 43). The AGA cohort comprised pregnancies referred for routine fetal assessment between 18 and 37 weeks' gestation. FGR was diagnosed according to the Delphi consensus criteria, which encompass both early- and LO-FGR.[12] To ensure data independence, each participant underwent a comprehensive evaluation, and all measurements were obtained from distinct individuals. Inclusion criteria comprised singleton pregnancies with normal fetal ultrasound findings. Exclusion criteria included multiple pregnancies and maternal chronic diseases such as pregestational or gestational diabetes mellitus, chronic hypertension, rheumatologic disorders, or Rh alloimmunization. Fetuses were presumed to have a normal karyotype, as inferred from normal clinical outcomes at birth in the absence of prenatal genetic testing. Gestational age was determined based on the last menstrual period and confirmed by first-trimester crown–rump length measurements, with age rounded down to the nearest completed week. Ultrasound assessments were performed by two experienced physicians (R.M. and G.A.) using Voluson E10 and Voluson S8 systems (GE Healthcare, Zipf, Austria) equipped with 3.5- or 5.0-MHz transducers. The portal venous system and the type of umbilical-portal anastomosis were systematically evaluated according to the method described by Kivilevitch et al.[6] The standardized protocol included the following steps: first, acquisition of a standard AC section; second, visualization of the PS and RPV, including its anterior and posterior branches; and finally, oblique angling of the probe to identify the MPV and assess its anatomical relationship with the PS and RPV branches.

The distribution of anastomosis types was as follows: in the AGA group, T-shaped, X-shaped, and H-shaped types were observed in 74 (68.5%), 20 (18.5%), and 14 (13%) fetuses, respectively; in the FGR group, the distribution was 29 (67.4%), 8 (18.6%), and 6 (14%), respectively.

UV and MPV diameters (MPVDs) and total flow volumes were measured according to previously described methods.[13] Inner-to-inner vessel diameters were measured perpendicular to the longitudinal axis using grayscale imaging to calculate MPVD (Figure 1a) and UV diameter (UVD) (Figure 1b). For each vessel, two to three measurements were obtained along the vessel tract, and the mean value was used for statistical analysis.

Doppler measurements for both the UV and MPV were acquired with an insonation angle < 30° and in the absence of fetal movements. The MPV was sampled just below its bifurcation into right and left branches, at the segment closest to the PS, to minimize the influence of the hepatic artery (Figure 1c). The UV was sampled in a longitudinal view as close as possible to its placental insertion (Figure 1d). Blood-flow volume was calculated from vessel diameter and time-averaged maximum velocity (TAMXV) using the formula:

$$\text{TAMXVV (mL/min)} = h \times \left(\frac{D}{2}\right)^2 \times \pi \times \text{TAMXV (cm/s)} \times 60$$

where D is the vessel diameter (cm) and is a coefficient for the spatial blood velocity profile. A value of 0.5 was applied for h, as in previous studies.[5,13,14] For both UV and MPV, TAMXV volume (TAMXVV) was normalized to EFW and expressed as TAMXVVN. EFW was calculated using the Hadlock formula. Additionally, TAMXVV was divided by AC (mm) to obtain the TAMXVV/AC ratio.

Clinical management of FGR fetuses followed ISUOG guidelines.[12] Doppler assessments included the umbilical artery (UA) and middle cerebral artery, from which the cerebroplacental ratio was derived. Doppler evaluation of the DV was performed when clinically indicated. Delivery decisions were based on ISUOG recommendations, considering UA and DV Doppler findings, and, in the absence of computerized cardiotocography, the presence of repeated spontaneous fetal heart rate decelerations on conventional cardiotocography, stratified by gestational age.

Following delivery, key neonatal outcomes were recorded, including gestational age at birth, birthweight, 5-minute Apgar scores, and UA pH.

Statistical analysis

Data were analyzed using Statistical Package for the Social Sciences (SPSS) version 29.0 (IBM Corp., Chicago, IL, USA). Continuous variables are presented as mean ± standard deviation (SD) or

median (interquartile range), depending on distribution. Normality was assessed using the Shapiro–Wilk test. Variables with a normal distribution were compared using Student's t-test, whereas non-normally distributed variables were analyzed with the Mann–Whitney U test. Categorical variables were compared using the chi-square test. Correlations between continuous parameters were evaluated using Pearson's correlation coefficient.

To adjust for multiple comparisons between the AGA and FGR groups, *p* values were corrected using the Benjamini–Hochberg procedure to control the false discovery rate at 5%. For Mann–Whitney U tests, effect size (r) was calculated to quantify the magnitude of observed differences, using the formula:

$$r = \frac{Z}{\sqrt{N}}$$

where Z is the standardized test statistic from SPSS output and *N* is the total sample size (n = 151).

Percentiles (5th, 50th, and 95th) for relevant parameters across gestational ages were derived from regression analysis of the AGA control group. Corresponding FGR measurements were plotted on nomograms constructed from the control data. Quadratic regression models were used to describe the non-linear relationship between measured parameters and gestational age, expressed as:

$$Y = a + b_1(\mathrm{GA}) + b_2(\mathrm{GA})^2$$

Z-scores were calculated as:

$$Z = \frac{\text{measured value} - \text{mean predicted value}}{\text{SD of predicted AGA values}}$$

An a priori sample size calculation was performed for a two-sample, two-sided comparison at $\alpha = 0.05$. Based on previously published mean ± SD values for MPV-TAMXVVN, the corresponding effect size was Cohen's $d \approx 0.69$.[10] Approximately n = 43 participants per group (86 total) were required to achieve 90% power. Our final sample (AGA = 108, FGR = 43) exceeded this requirement, ensuring sufficient power to detect differences in MPV-TAMXVVN between groups.

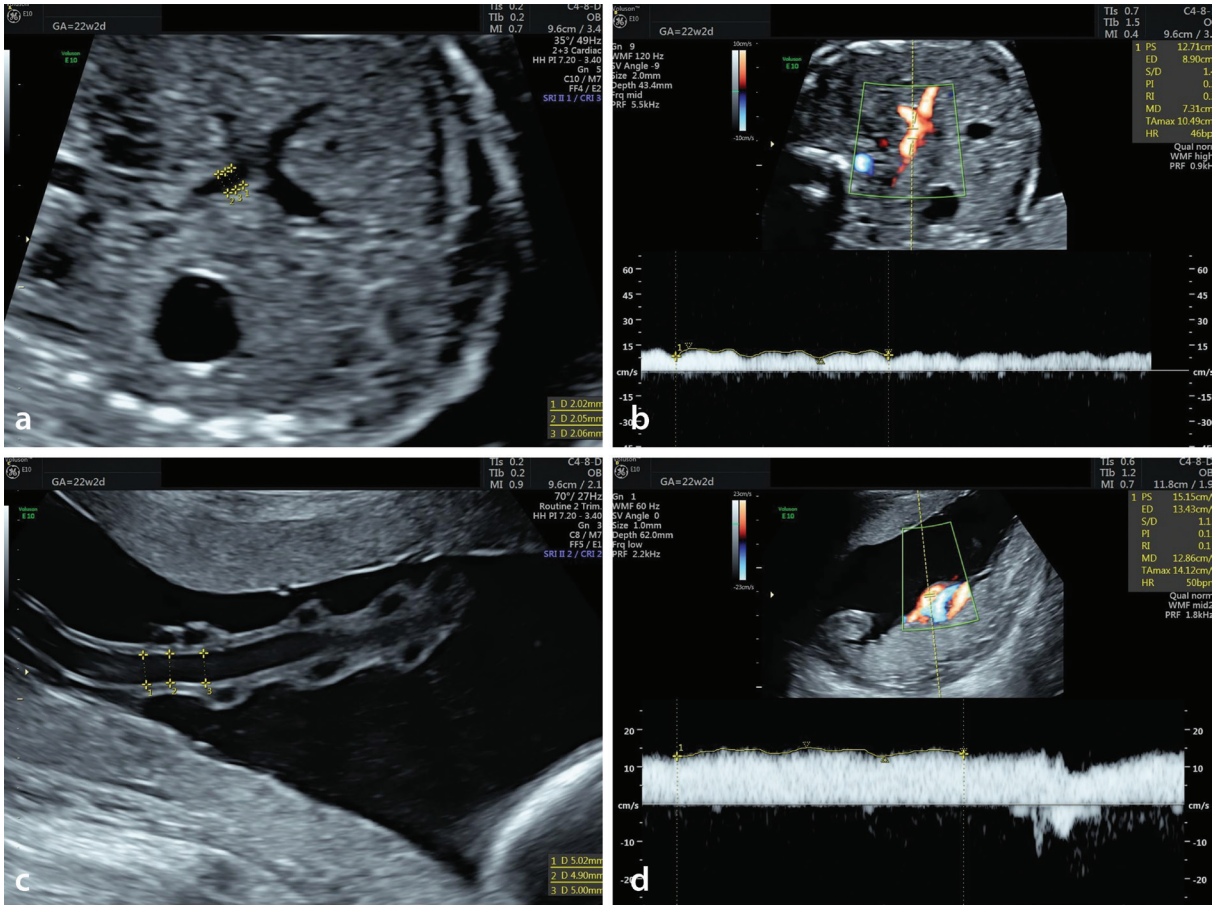

FIG. 1. Measurements of main portal vein and umbilical vein diameters (a, b) and corresponding pulsed-wave Doppler analyses obtained transabdominally (c, d).

All Doppler measurements were successfully obtained for all 151 participants, with no exclusions due to technical failure. Statistical analyses were conducted on the complete dataset.

RESULTS

The clinical characteristics and perinatal outcomes of AGA and FGR pregnancies are summarized in Table 1. There were no significant differences between groups in mean maternal age, nulliparity, gestational age at assessment, or UA pH ($p > 0.05$). Gestational age at delivery and birthweight were significantly higher in the AGA group ($p < 0.001$). Preterm delivery before 37 weeks occurred more frequently in FGR fetuses than in AGA fetuses ($p = 0.001$). Additionally, the incidence of 5-minute Apgar scores < 7 and admissions to the neonatal intensive care unit was significantly higher in the FGR group ($p = 0.026$ and $p = 0.001$, respectively). The FGR group also experienced two intrauterine deaths and one neonatal death due to respiratory distress syndrome associated with prematurity. Sonographic and Doppler measurements of the UV and MPV are presented in Table 2. Median UVD was significantly lower in FGR fetuses than in AGA fetuses ($p = 0.003$, r = 0.23). Although median MPVD was higher in the FGR group, this difference was not statistically significant ($p = 0.113$). FGR fetuses exhibited a significantly lower median UV-TAMXVV compared with AGA fetuses ($p = 0.002$, r = 0.26), while median MPV-TAMXVV values were similar between groups ($p = 0.376$).

After normalization to EFW, median UV-TAMXVVN was significantly lower and median MPV-TAMXVVN was significantly higher in FGR fetuses than in AGA fetuses ($p = 0.011$, r = 0.22 and $p = 0.002$, r = 0.32, respectively). Similarly, the median UV-TAMXVV/AC ratio was significantly lower in FGR fetuses compared with AGA fetuses ($p = 0.002$, r = 0.29), whereas the median MPV-TAMXVV/AC ratio did not differ significantly between groups ($p = 0.131$).

Z-scores for absolute blood flow per kilogram of EFW and per millimeter of AC in the UV and MPV are presented in Table 3. In FGR fetuses, UV-TAMXVVN Z-scores were significantly lower, whereas MPV-TAMXVVN Z-scores were significantly higher compared with AGA fetuses ($p = 0.006$ and $p = 0.002$, respectively). Similarly, median UV-TAMXVV/AC Z-scores were significantly lower in FGR fetuses than in AGA fetuses ($p = 0.002$), while MPV-TAMXVV/AC Z-scores did not differ significantly between groups ($p = 0.116$). Both UV and MPVDs increased with advancing gestational age, showing strong positive correlations with gestational week (r = 0.766 and 0.805, respectively; $p < 0.001$). Blood flow volumes (mL/min) of the UV and MPV across gestational ages in normal and growth-restricted fetuses, including 5th, 50th, and 95th percentile reference lines for the AGA population, are illustrated in Figures 2a, b. Blood flow values also correlated positively with gestational age for both veins (UV: r = 0.722; MPV: r = 0.703; $p < 0.001$). UV-TAMXVVN and MPV-TAMXVVN values across gestational weeks, plotted with 5th, 50t^{h}, and 95th percentile reference lines for the AGA population, are shown in Figures 3a, b. The mean Z-scores of UV- and MPV-TAMXVVN by gestational week in FGR and AGA fetuses are presented in Figures 4a, b. UV-TAMXVVN Z-scores demonstrated a progressive decrease with advancing gestation in both groups. In FGR fetuses, MPV-TAMXVVN Z-scores increased until approximately 32 weeks of gestation, after which they plateaued. In contrast, in AGA fetuses, MPV-TAMXVVN Z-scores decreased until around 32 weeks and then remained relatively stable until term.

MPV blood flow volumes (mL/min) were analyzed according to the

TABLE 1. The Clinical Characteristics and Perinatal Outcomes of the Pregnancies with Appropriate for Gestational Age and Fetal Growth Restriction.

	Appropriate for gestational age	Fetal growth restriction	*p*
n	108	43	
Maternal age (years)	30.1 ± 5.2	29.6 ± 5.6	0.649
Nulliparity	77, (71.3)	25, (58.1)	0.084
Gestational age at assessment (weeks)	29.4 (25.2-34.1)	31.1 (26.2-35.5)	0.225
Gestational age at birth (weeks)	38.2 (37-38.6)	36.5 (32.2-38)	0.001
Delivery < 37 gestational weeks	12, (11.1)	16, (37.2)	0.001
Birthweight (gram)	3040 (2628-3510)	2100 (1180-2600)	0.001
5-min Apgar score < 7	3, (2.7)	5, (12.2)	0.026
Umbilical artery pH	7.35 (7.32-7.38)	7.35 (7.32-7.37)	0.990
NICU admission	12, (11.1)	10, (24.4)	0.001
Intrauterine death	-	2, (4.6)	
Neonatal death	-	1, (2.4)	

Data are expressed as mean ± standard deviation, median (interquartile range), and n, (%) where appropriate.
NICU, neonatal intensive care unit.

three types of umbilicoportal anastomosis in both AGA and FGR fetuses. The distribution of MPV-TAMXVV by anastomosis type is shown in Figures 5a, b. Flow volumes were similar across all three anastomosis types, with nearly all measurements falling within the 5th to 95th percentile range for both groups. No significant effect of anastomosis type on MPV blood flow was observed in either AGA or FGR fetuses.

DISCUSSION

In the present study, absolute UV blood flow (mL/min) increased with advancing gestational age, rising from approximately 40 mL/min at 21 weeks to 200 mL/min at 36 weeks. This is consistent with the longitudinal observations of Kessler et al.,[15] who reported an increase from 44 mL/min at 21 weeks to 201 mL/min at 36 weeks. In contrast, normalized UV blood flow (UV-TAMXVVN, mL/min/kg) showed a declining trend, decreasing from approximately 120 mL/min/kg at 20 weeks to 95 mL/min/kg at 36 weeks. Similar trends were reported by Kessler et al.,[15] indicating that the rate of fetal weight gain outpaces the increase in umbilical venous flow, resulting in a relative decrease in normalized UV flow across gestation. This relative reduction may also reflect a decreasing proportion of fetal cardiac output directed to the placenta with advancing gestational age.[16,17] Absolute MPV blood flow increased from 10 mL/min at 24 weeks to 30 mL/min at 36 weeks, consistent with the findings of Kessler et al.,[14] who reported a similar rise from 10 mL/min to 30 mL/min over the same gestational period. Normalized MPV blood flow (MPV-TAMXVVN, mL/min/kg) exhibited a slight decline until approximately 32 weeks (from ~ 15 mL/min/kg at 20 weeks to ~ 12 mL/min/kg at 30 weeks), after which it remained stable until term, mirroring patterns observed in previous studies.[14] These findings indicate that MPV blood flow increases proportionally with fetal weight during the third trimester. The maintenance of normalized blood flow in the MPV underscores the high circulatory priority of the fetal liver during this critical period of growth.

In growth-restricted fetuses, both absolute and normalized UV flows were significantly lower than in AGA fetuses. UV-TAMXVVN in FGR

TABLE 2. Sonographic and Doppler Findings of the Umbilical and Main Portal Vein of the Appropriate for Gestational Age and Fetal Growth Restricted Fetuses.

	Appropriate for gestational age	Fetal growth restriction		
n	108	43	*p*	Adjusted *p*
UVD (cm)	0.64 (0.55-0.73)	0.56 (0.42-0.69)	0.003	0.006
UV-TAMXVV (mL/min)	127.62 (88.24-190.56)	80.8 (47.96-145.81)	0.001	0.002
UV-TAMXVVN (mL/min/Kg)	90.66 (72.19-115.74)	73.57 (58.72-95.39)	0.007	0.011
UV-TAMXVV/AC	0.514 (0.397-0.637)	0.392 (0.238-0.522)	0.001	0.002
MPVD (cm)	0.23 (0.19-0.28)	0.26 (0.2-0.31)	0.085	0.113
MPV-TAMXVV (mL/min)	14.65 (8.42-22.62)	15.39 (9.08-31.54)	0.376	0.376
MPV-TAMXVVN (mL/min/Kg)	10.77 (8.23-13.62)	15.03 (10.2-20.26)	0.001	0.002
MPV-TAMXVV/AC	0.062 (0.042-0.077)	0.062 (0.046-0.112)	0.115	0.131

Data represented as median (interquartile range).
UVD, umbilical vein diameter; MPVD, main portal vein diameter; UV-TAMXVVN, umbilical vein time-averaged maximum velocity volume per kilogram of EFW; MPV -TAMXVVN, main portal vein time-averaged maximum velocity volume per kilogram of EFW; EFW, estimated fetal weight; AC, abdominal circumference.

TABLE 3. Z-scores of Normalized Umbilical and Main Portal Vein Blood Flow per Kilogram of Estimated Fetal Weight and per Millimeter of Abdominal Circumference in AGA and FGR Fetuses.

	Appropriate for gestational age	Fetal growth restriction	*p*	Adjusted *p*
n	108	43		
UV-TAMXVVN	-0.16 (-0.72-0.60)	-0.71 (-1.19-0.01)	0.006	0.008
MPV-TAMXVVN	-0.12 (-0.78-0.62)	0.55 (-0.31-1.49)	0.001	0.116
UV-TAMXVV/AC	-0.1 (-0.72-0.51)	-0.69 [-1.59-(-0.05)]	0.001	0.002
MPV-TAMXVV/AC	-0.18 (-0.78-0.4)	-0.18 (-0.48-1.3)	0.116	0.002

Data represented as median (interquartile range).
UV-TAMXVV, umbilical vein time-averaged maximum velocity volume; UV-TAMXVVN, umbilical vein time-averaged maximum velocity volume per kilogram of EFW; MPV -TAMXVV, main portal vein time-averaged maximum velocity volume; MPV -TAMXVVN, main portal vein time-averaged maximum velocity volume per kilogram of EFW; EFW, estimated fetal weight; AC, abdominal circumference; AGA, appropriate-for-gestational-age; FGR, fetal growth restriction.

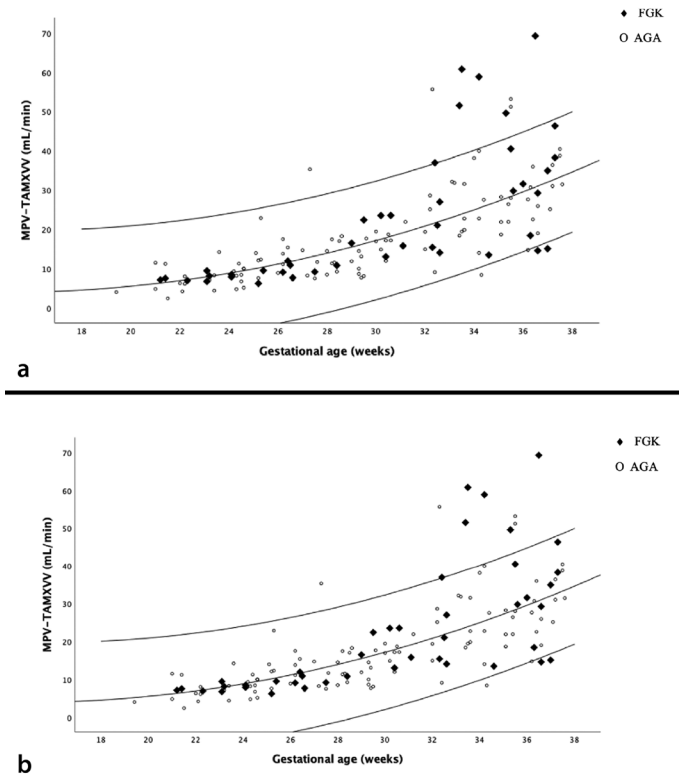

FIG. 2. Umbilical vein (a) and main portal vein (b) time-averaged maximum velocity volume (TAMXVV) (mL/min) across gestational ages in normal and growth-restricted fetuses, presented with 5th, 50th, and 95th percentile reference lines for the normal population.

MPV, main portal vein; FGR, fetal growth restriction; AGA, appropriate-for-gestational-age.

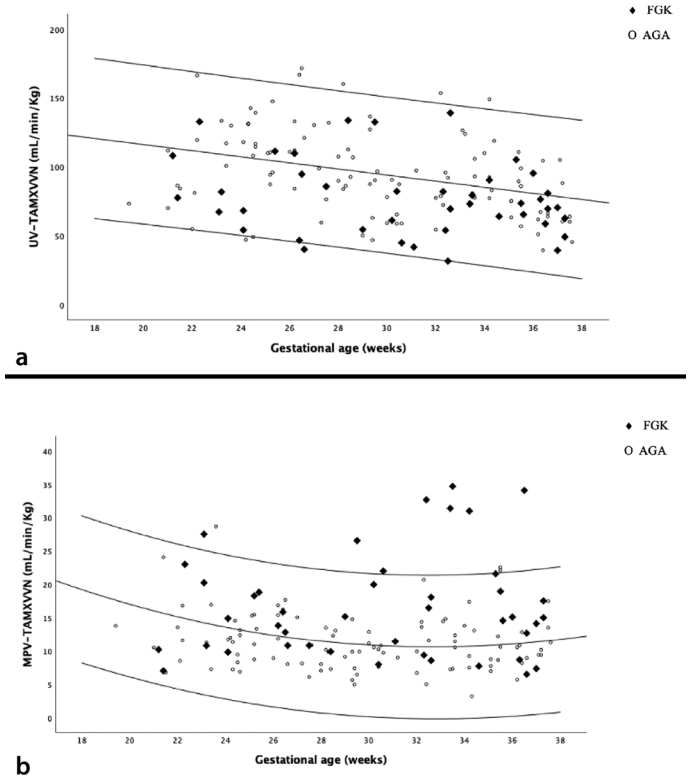

FIG. 3. Umbilical (a) and main portal vein (b) time-averaged maximum velocity volume per kilogram of estimated fetal weight (TAMXVVN) (mL/min/Kg) across gestational ages in normal and growth-restricted fetuses, presented with 5th, 50th, and 95th percentile reference lines for the normal population.

MPV, main portal vein; UV, umbilical vein; FGR, fetal growth restriction; AGA, appropriate-for-gestational-age.

fetuses exhibited a decreasing trend with advancing gestational age, similar to the pattern observed in AGA fetuses. These findings align with Ferrazzi et al.,[18] who reported comparable alterations in both UV-TAMXV and UV-TAMXVVN in growth-restricted fetuses. Reduced placental perfusion likely underlies the decreased UV flow observed in FGR. Conversely, absolute and normalized MPV flows were significantly higher in FGR fetuses compared with AGA fetuses. MPV-TAMXVVN in FGR fetuses increased until approximately 32 weeks of gestation and then stabilized toward term. This pattern contrasts with AGA fetuses, in whom MPV-TAMXVVN decreased until around 32 weeks and remained relatively stable thereafter. The increased contribution of MPV flow in FGR fetuses suggests an exaggerated compensatory mechanism in response to reduced umbilical venous inflow. Kiserud et al.[17] reported similar findings, highlighting a pronounced reduction in blood flow to the right hepatic lobe in FGR fetuses. Mechanistically, this phenomenon may be driven by reduced umbilical venous flow and enhanced shunting through the DV into the systemic circulation.[9,10] Previous studies indicate that the right and left hepatic lobes differ in circulatory and functional composition, with the right lobe being more vulnerable to hypoxia in FGR due to placental insufficiency.[19-21] These clinical observations are supported by experimental data. Tchirikov et al.[7] demonstrated that reduced afferent venous blood supply impairs tissue growth in animal models. Similarly, Popovici et al.[22] showed that hypoxia induces IGF-I binding protein synthesis, resulting in decreased IGF-I activity in human hepatocytes. Collectively, these findings underscore the critical role of venous perfusion in supporting normal hepatic development and overall fetal growth.

Kivilevitch et al.[23] investigated fetal growth in cases with intrahepatic umbilical–portosystemic venous shunt (IHUPSVS) and reported that compromised fetal growth was more frequent in the IHUPSVS group. In a subsequent prospective cross-sectional study, they identified IHUPSVS in 9.4% of 150 FGR fetuses, with Doppler findings suggesting an absence of placental insufficiency in these cases.[24] They proposed that a pre-existing shunt may reduce hepatic perfusion, thereby contributing to impaired tissue growth.

Data on the influence of umbilicoportal anastomosis type on fetal growth and the development of FGR remain limited. Kivilevitch et al.[6] were the first to suggest that variations in anastomosis configuration could affect intrahepatic flow dynamics, potentially influencing fetal growth patterns. To date, only one study has specifically addressed this issue, proposing that differences in anastomosis type may alter blood flow to the right hepatic lobe and contribute to LO-FGR.[11]

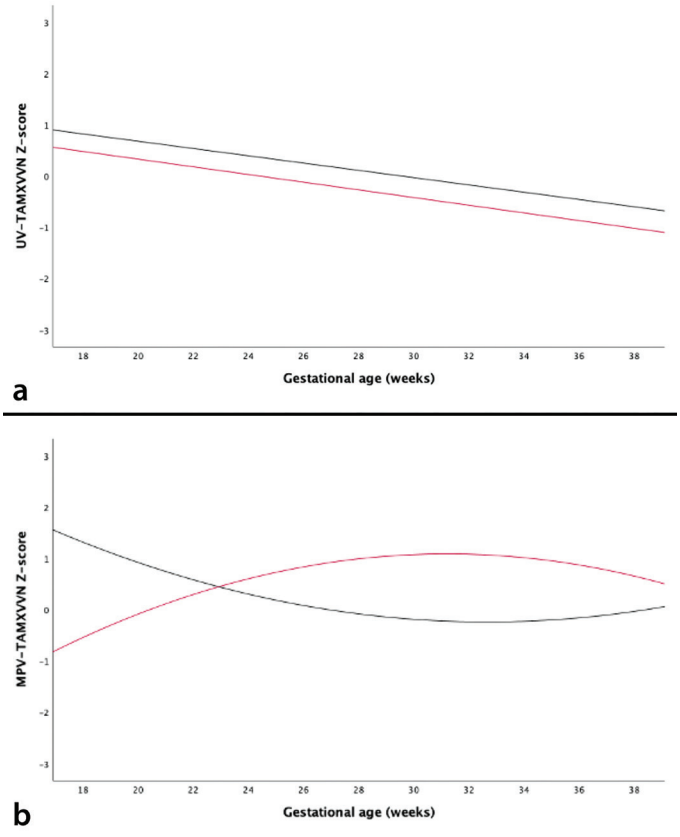

FIG. 4. The mean Z-scores of the umbilical (a) and main portal vein (b) time-averaged maximum velocity volume per kilogram of estimated fetal weight (TAMXVVN) (mL/min/Kg) according to gestational weeks in fetuses with growth restriction (red line) and normal growth (black line).

MPV, main portal vein; UV, umbilical vein.

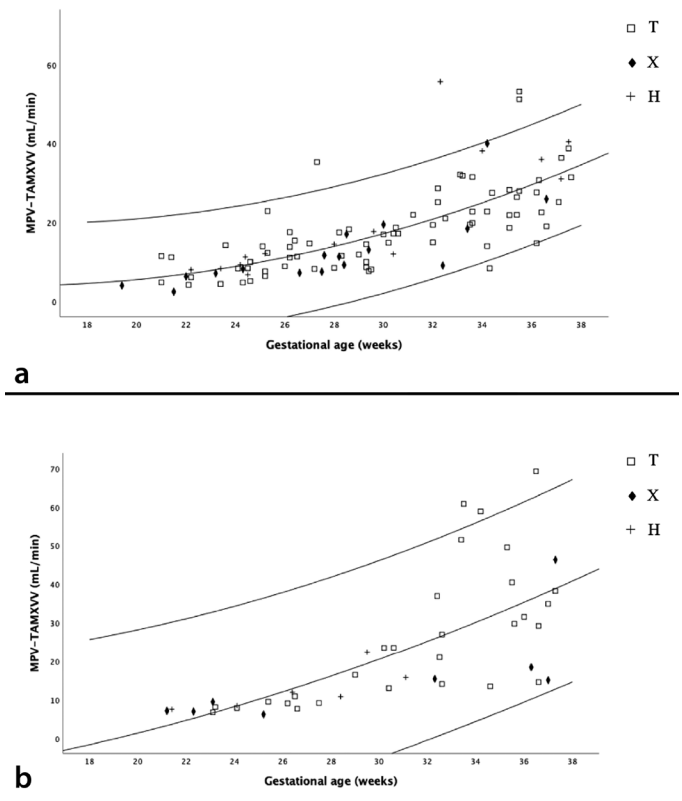

FIG. 5. Main portal vein time-averaged maximum velocity volume (mL/min) according to umbilicoportal anastomosis types (X, T, H) across gestational ages in normal (a) and growth-restricted (b) fetuses. Data are presented with the 5th, 50th, and 95th percentile reference lines for both normal (a) and growth-restricted (b) populations.

MPV, main portal vein.

In the present study, we examined absolute MPV blood flow across T-shaped, H-shaped, and X-shaped umbilicoportal anastomoses in both normal and growth-restricted fetuses. Our findings demonstrated no significant differences in MPV blood flow among the three anastomosis types in either group. However, the small number of fetuses in each anastomosis subgroup limits the strength of these conclusions, and further studies with larger sample sizes are warranted to confirm these observations.

To our knowledge, this is one of the few studies—and the most comprehensive to date—to systematically evaluate the morphological types of umbilicoportal anastomosis and their relationship with intrahepatic hemodynamic parameters in both normal and FGR fetuses. By assessing absolute blood flow as well as normalized flow values relative to EFW and AC, we provided a more precise evaluation of fetal venous perfusion adjusted for fetal size, thereby enhancing clinical relevance. Another strength of this study is the application of a standardized imaging protocol, with all Doppler assessments performed by two experienced maternal–fetal medicine specialists, ensuring consistency and reliability in data acquisition. Several limitations should be acknowledged. First, although the sample size was adequate to detect differences in overall hemodynamic parameters between normal and FGR fetuses, it may have been insufficient to detect subtle differences among umbilicoportal anastomosis subtypes, particularly in subgroup analyses. Second, the study was conducted at a single center, which may limit the generalizability of the findings to broader populations. Third, all Doppler measurements were obtained at a single time point, precluding the assessment of longitudinal changes in venous blood flow across gestation. Consequently, the reported trends cannot be interpreted as true longitudinal changes. Serial evaluations in future studies may provide deeper insight into dynamic vascular adaptations during fetal development.

In conclusion, FGR is associated with reduced umbilical venous flow and a compensatory increase in MPV perfusion. The type of umbilicoportal anastomosis, however, does not significantly influence MPV blood flow in either normal or growth-restricted fetuses. Further research with larger, multicenter cohorts and longitudinal designs is warranted to validate these findings and clarify the role of intrahepatic venous architecture in fetal growth.

Ethics Committee Approval: Ethical approval was obtained from the İstanbul University-Cerrahpaşa Ethics Committee (approval number: 2025/9, date: 08.01.2025), and all procedures were conducted in accordance with the principles outlined in the Declaration of Helsinki.

Informed Consent: Written informed consent was obtained from all participants prior to their inclusion in the study, and each participant was fully informed about the study's purpose and procedures.

Data Sharing Statement: The data that support the findings of this study are available from the corresponding author upon reasonable request.

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REFERENCES

1. Edelstone DI, Rudolph AM, Heymann MA. Liver and ductus venosus blood flows in fetal lambs in utero. *Circ Res.* 1978;42:426-433. [CrossRef]
2. Yagel S, Kivilevitch Z, Cohen SM, et al. The fetal venous system, part I: normal embryology, anatomy, hemodynamics, ultrasound evaluation and Doppler investigation. *Ultrasound Obstet Gynecol.* 2010;35:741-750. [CrossRef]
3. Rudolph AM. Hepatic and ductus venosus blood flows during fetal life. *Hepatology.* 1983;3:254-258. [CrossRef]
4. Kiserud T, Rasmussen S, Skulstad S. Blood flow and the degree of shunting through the ductus venosus in the human fetus. *Am J Obstet Gynecol.* 2000;182:147-153. [CrossRef]
5. Haugen G, Kiserud T, Godfrey K, Crozier S, Hanson M. Portal and umbilical venous blood supply to the liver in the human fetus near term. *Ultrasound Obstet Gynecol.* 2004;24:599-605. [CrossRef]
6. Kivilevitch Z, Gindes L, Deutsch H, Achiron R. In-utero evaluation of the fetal umbilical-portal venous system: two- and three-dimensional ultrasonic study. *Ultrasound Obstet Gynecol.* 2009;34:634-642. [CrossRef]
7. Tchirikov M, Kertschanska S, Stürenberg HJ, Schröder HJ. Liver blood perfusion as a possible instrument for fetal growth regulation. *Placenta.* 2002;23:S153-S158. [CrossRef]
8. Ferrazzi E, Rigano S, Bozzo M, et al. Umbilical vein blood flow in growth-restricted fetuses. *Ultrasound Obstet Gynecol.* 2000;16:432-438. [CrossRef]
9. Kiserud T, Kessler J, Ebbing C, Rasmussen S. Ductus venosus shunting in growth-restricted fetuses and the effect of umbilical circulatory compromise. *Ultrasound Obstet Gynecol.* 2006;28:143-149. [CrossRef]
10. Ebbing C, Rasmussen S, Godfrey KM, Hanson MA, Kiserud T. Redistribution pattern of fetal liver circulation in intrauterine growth restriction. *Acta Obstet Gynecol Scand.* 2009;88:1118-1123. [CrossRef]
11. Kaymak D, Madazli R. Evaluation of the type of fetal umblical-portal anastomosis in late-onset fetal growth restriction: a case-control study. *J Clin Ultrasound.* 2024;52:353-358. [CrossRef]
12. Lees CC, Stampalija T, Baschat A, et al. ISUOG Practice Guidelines: diagnosis and management of small-for-gestational-age fetus and fetal growth restriction. *Ultrasound Obstet Gynecol.* 2020;56:298-312. [CrossRef]
13. Kivilevitch Z, Gilboa Y, Gilad N, Kassif E, Achiron R. Afferent venous perfusion of fetal liver: umbilical and portal blood-flow volumes in fetuses born small-for-gestational age. *Ultrasound Obstet Gynecol.* 2023;62:813-820. [CrossRef]
14. Kessler J, Rasmussen S, Kiserud T. The fetal portal vein: normal blood flow development during the second half of human pregnancy. *Ultrasound Obstet Gynecol.* 2007;30:52-60. [CrossRef]
15. Kessler J, Rasmussen S, Godfrey K, Hanson M, Kiserud T. Longitudinal study of umbilical and portal venous blood flow to the fetal liver: low pregnancy weight gain is associated with preferential supply to the fetal left liver lobe. *Pediatr Res.* 2008;63:315-320. [CrossRef]
16. Kessler J, Rasmussen S, Godfrey K, Hanson M, Kiserud T. Fetal growth restriction is associated with prioritization of umbilical blood flow to the left hepatic lobe at the expense of the right lobe. *Pediatr Res.* 2009;66:113-117. [CrossRef]
17. Kiserud T, Ebbing C, Kessler J, Rasmussen S. Fetal cardiac output, distribution to the placenta and impact of placental compromise. *Ultrasound Obstet Gynecol.* 2006;28:126-136. [CrossRef]
18. Ferrazzi E, Rigano S, Bozzo M, et al. Umbilical vein blood flow in growth-restricted fetuses. *Ultrasound Obstet Gynecol.* 2000;16:432-438. [CrossRef]
19. Gruenwald P. Degenerative changes in the right half of the liver resulting from intra-uterine anoxia. *Am J Clin Pathol.* 1949;19:801-813. [CrossRef]
20. Cox LA, Schlabritz-Loutsevitch N, Hubbard GB, Nijland MJ, McDonald TJ, Nathanielsz PW. Gene expression profile differences in left and right liver lobes from mid-gestation fetal baboons: a cautionary tale. *J Physiol.* 2006;572:59-66. [CrossRef]
21. Jacobsson H, Jonas E, Hellström PM, Larsson SA. Different concentrations of various radiopharmaceuticals in the two main liver lobes: a preliminary study in clinical patients. *J Gastroenterol.* 2005;40:733-738. [CrossRef]
22. Popovici RM, Lu M, Bhatia S, Faessen GH, Giaccia AJ, Giudice LC. Hypoxia regulates insulin-like growth factor-binding protein 1 in human fetal hepatocytes in primary culture: suggestive molecular mechanisms for in utero fetal growth restriction caused by uteroplacental insufficiency. *J Clin Endocrinol Metab.* 2001;86:2653-2659. [CrossRef]
23. Kivilevitch Z, Kassif E, Gilboa Y, Weisbuch T, Achiron R. The intra-hepatic umbilical-Porto-systemic venous shunt and fetal growth. *Prenat Diagn.* 2021;41:457-464. [CrossRef]
24. Czeiger S, Weissbach T, Zloto K, et al. Umbilical-portal-systemic venous shunt and intrauterine growth restriction: an inquiry from a prospective study. *Am J Obstet Gynecol.* 2024;231:340.e1-340.e16. [CrossRef]