НЕОНАТОЛОГІЯ, ХІРУРГІЯ ТА ПЕРИНАТАЛЬНА МЕДИЦИНА
NEONATOLOGY, SURGERY AND PERINATAL MEDICINE
Т. XV, №3(57), 2025
VOL. XV, №3(57), 2025
ISSN 2226-1230 (PRINT) ISSN 2413-4260 (ONLINE)
KEY TITLE: NEONATOLOGÌÂ, HÌRURGÌÂ TA PERINATAL'NA MEDICINA (ONLINE)
ABBREVIATED KEY TITLE: NEONATOL. HÌR. PERINAT. MED. (ONLINE)

UDK: 616-071+616-007.7+618.48+618.53
DOI: 10.24061/2413-4260. XV.3.57.2025.12

CLINICAL AND MORPHOLOGICAL FEATURES OF THE UMBILICAL CORD IN LATE MANIFESTATION OF FETAL GROWTH RESTRICTION

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Summary.

Placental and umbilical cord abnormalities can lead to negative perinatal outcomes: fetal growth restriction, intrauterine fetal death, low birth weight for gestational age, and emergency caesarean section. Some publications focus in detail on the assessment of pathological changes in the umbilical cord, but the relationship between macromorphological changes in the umbilical cord and fetal growth restriction remains controversial and unresolved.

***The aim of the study** was to investigate the clinical and macromorphological markers of umbilical cord blood flow disorders in late manifestations of fetal growth restriction through pathohistological examination of the umbilical cord.*

***Materials and methods of the study.** The study was conducted in two groups: the main group (89 patients with late manifestation of fetal growth restriction) and the comparison group (30 patients with normal fetometry parameters). Postnatally, umbilical cord tissue samples were collected for macroscopic and pathohistological examination: 32 samples in the main group and 10 samples in the comparison group.*

***Results of the study.** A comparative analysis of the course of pregnancy and its outcomes revealed a high proportion of complications and abdominal delivery (37-41.6%), in a larger proportion associated with preeclampsia (21-23.6%), and a combination of fetal growth restriction and preterm birth (14-15.7%). Macroscopic examination of the selected samples revealed excessive twisting/hypercoiling of the umbilical cord, excess of Wharton's jelly with a dense structure or its aplasia, in 10 samples (31.3%) there were cysts of Wharton's jelly, and in 5 (15.6%) increase in varicose umbilical vein with enlargement of the lumen and thinning of the wall was noted. In 26 (81.3%) samples of the umbilical cord of the main group, focal thinning of the umbilical vein wall, edema, vacuolar dystrophy of myocytes, myolysis and proliferation of connective tissue fibers in the muscular membrane of the umbilical vein were revealed. Venous thrombosis was common. Examination of the arteries revealed slit-like lumen and perivascular hemorrhages.*

***Conclusions.** A comparative analysis of the course of pregnancy revealed a high frequency of abdominal delivery (41.6%), mostly associated with preeclampsia (21-23.6%), a combination of fetal growth restriction and preterm birth (14-15.7%), and deterioration of the intrauterine condition of the fetus (33-37.1%) against the background of a high index of somatic pathology and placental dysfunction. The results of the study emphasize the dependence of umbilical cord abnormalities on the frequency of gestational complications and antenatal pathology, including those associated with fetal growth restriction. The characteristic pathohistological changes in the umbilical cord in such newborns were: short hypercoiled umbilical cord, aplasia and cysts of the Wharton's jelly, thinning and destructive changes in the wall of the umbilical vein, perivascular hemorrhages, and decreased arterial flow capacity.*

***Keywords:** Fetal Growth Restriction; Placental Dysfunction; Umbilical Cord; Macromorphological Markers of Umbilical Cord Pathology.*

Introduction

Placental and umbilical cord abnormalities can lead to various perinatal outcomes, including fetal growth restriction (FGR), low birth weight, intrauterine fetal death, too small weight for gestational age, and emergency caesarean section [1], with critical umbilical cord circulation being diagnosed in one in five stillborns during autopsy [2]. Many stillborn infants are phenotypically normal and show no signs of chronic placental damage (so-called «placental insufficiency»), and one of the presumed causes of death in these cases is hypoxic damage associated with mechanical disruption of the umbilical cord supply. At the same time, umbilical cord pathologies can manifest themselves as mechanical compression (umbilical cord entanglement and prolapse, true umbilical cord knots, hypercoiling/twisting, abnormally long or short umbilical cords, abnormal umbilical cord insertion or strictures), as well as structural abnormalities (single umbilical artery, velamentous, marginal or furcate insertion of the umbilical cord, excess or insufficiency of Wharton's jelly, etc.) [3-8]. In recent years, there has been a rapid growth in scientific research and publications devoted to the problem of fetal growth restriction, its early and late manifestations, perinatal consequences and long-term effects on the health of a baby [9-13].

A number of studies consider placental factors in the development of this problem, where ischemia and infection are significant factors [14, 15]. However, FGR remains a complex obstetric problem today, as the criteria of the new international classification, based on centile assessment of fetometry and Doppler parameters, remain variable in terms of prognosis, issues of prevention and prognosis of perinatal losses remain unresolved, and approaches to the timing and methods of delivery are still controversial [13, 16, 17].

Some publications focus in detail on the assessment of pathological changes in the umbilical cord (UC) [4, 5, 18, 19]. However, scientific positions are contradictory and controversial. Moreover, the question of the relationship

between macromorphological changes in the umbilical cord and types of FGR remains debatable and unresolved.

The aim of the study was to investigate clinical and macromorphological markers of umbilical blood flow disorders in late manifestation of fetal growth restriction by means of pathohistological examination of the umbilical cord.

Materials and methods

The study included patients with singleton pregnancies aged 18 to 35 years who consented to participate in the study, where the main group (89 patients with late manifestation of fetal growth restriction – after 32 weeks) and the comparison group (30 patients with normal fetometry parameters).

The diagnosis of fetal growth restriction was established in accordance with international criteria: birth weight <3 percentile or a combination of three criteria: birth weight <10 percentile; head circumference <10 percentile; prenatal diagnosis of FGR and prenatal risk factors associated with FGR [20]. Late-onset of FGR was diagnosed using the Delphi procedure [21] with Doppler and sonographic techniques at 32 weeks of gestation or later according to the following criteria: estimated fetal weight (EFW) and/or or abdominal circumference (AC) < 3rd percentile or two of three relative criteria (EFW and/or AC < 10th percentile, slowing of EFW and/or AC growth rate crossing more than two quartiles on growth percentile charts; cerebral-placental ratio < 5th percentile or pulsation index in the umbilical artery greater than 95th percentile) [10, 12, 21-23].

The criteria for inclusion in the main group were: age 18 to 35 years, late manifestation of fetal growth restriction, gestational age of 32 weeks or more, and the patient's consent to participate in the study. The criteria for inclusion in the comparison group were: physiological course of pregnancy, normal fetometry parameters, absence of uteroplacental blood flow disorders. Exclusion criteria: late reproductive age, multiple pregnancy, premature rupture of the amniotic membranes, planned selective deliveries associated with the intrauterine condition of the fetus, fetal malformations and chromosomal abnormalities, presence of severe somatic diseases, oncological diseases, refusal to participate in the study.

An abnormal condition at birth was determined according to the following criteria: Apgar score < 7 points at 5 minutes, umbilical artery pH < 7.0 or umbilical vein pH < 7.1, resuscitation measures and artificial lung ventilation, or ante-intranatal fetal death.

Significant neonatal morbidity was determined by the following criteria: neurological anomaly, periventricular leukomalacia, encephalopathy and seizure readiness, cardiovascular dysfunction or disseminated coagulopathy, respiratory disorders (prolonged respiratory support for more than 7-10 days, artificial lung ventilation, aspiration of meconium, persistent pulmonary hypertension, etc.); or clinical sepsis, meningitis, necrotic enterocolitis.

Dopplerometry of uteroplacental blood flow in patients of the study groups was performed using the Aloka SD SSD 3500 ultrasound diagnostic device (Japan) with colour Doppler mapping. The study was conducted dynamically at 32-34 weeks and 36-38 weeks. Dopplerography was used to measure the pulsation index (PI) in the uterine arteries (UA), umbilical cord arteries (UCA) and middle cerebral artery (MCA), where PI values in the UA and UCA above the 95th percentile and PI in the MCA below the 5th percentile were considered pathological. The cerebro-placental-uterine ratio (CPUR) was calculated using the formula PI MCA/PI UCA, where a value below the 5th percentile indicated centralisation of blood flow.

According to MacDonald T. M., low CPUR was associated with birth weight < 10th percentile with OR-9.1, < 5th percentile with OR-17.3 and < 3rd percentile with OR-57.0 ($P < 0.0001$ for all) compared to the cerebro-placental ratio or PI in UCA separately [24].

In the case of postnatal macro-histomorphological assessment of the placenta and umbilical cord, the placental weight, umbilical cord length, and placental-fetal ratio (PFR) were evaluated. The PFR is an indicator that reflects the ratio of placental weight to fetal weight and is determined by the formula: PPC = placental weight (g)/fetal weight (g), macroscopic features of the placental structure (shape, thickness, area, presence of infarctions, etc.).

Macroscopic and pathohistological examination of postnatally collected umbilical cord tissue samples was performed: 32 samples in the main group and 10 samples in the comparison group. Pathohistological examination was performed at the Educational and Scientific Laboratory of Morphological Analysis of Ivano-Frankivsk National Medical University. Samples were taken from the middle third of the umbilical cord, up to 2 cm in length. The obtained material was fixed for 24 hours in a 10% neutral buffered formaldehyde solution. After fixation and dehydration, the studied tissues were poured with paraffin. Serial sections were obtained on a sliding microtome and stained with haematoxylin and eosin; Masson's trichrome stain was used to differentiate connective tissue and muscles. Light microscopy was performed using a Leica DM 750 microscope with ×10, 20, ×40, ×63, and was photographed using a digital CCD camera with a resolution of 1200×1600 and saving photos in jpg format.

This work is a part of the research conducted by the Department of Obstetrics and Gynecology No. 1 of the Bogomolets National Medical University, entitled «Preserving and restoring women's reproductive health in conditions of rapid social and medical change» (state registration number 0123U100920) and interdepartmental research work of the Department of Obstetrics and Gynecology named after I. D. Lanovyi and the Department of Obstetrics and Gynecology of Postgraduate Education of the Ivano-Frankivsk National Medical University: «Development of diagnostic tactics and pathogenetic justification of effective methods for preserving and restoring reproductive potential and improving the quality of life of women with obstetric and gynecological pathologies» (state registration number 0121U109269, completion date 2021-2026), where the authors are co-executors of the study.

The research was conducted in accordance with the principles of the Helsinki Declaration on Biometric Research and the powers of the GCH ICH (1996), the Council of Europe Convention on Human Rights and the relevant laws of Ukraine on conducting experimental and clinical research, in accordance with biometric standards

ISSN 2226-1230 (PRINT) ISSN 2413-4260 (ONLINE)

and in compliance with the principles of confidentiality and ethics. This investigation is a randomised controlled prospective study, using a random sampling method.

Statistical data processing and graphical representation were performed using Statistica 6.0 software package with the application of arithmetic mean and standard deviations using Student's t-test. For statistical analysis of the relationship between independent variables and the condition, the odds ratio (OR) and 95% confidence interval (95% CI) were used.

Research results and discussion. The main group of pregnant women with late manifestation of fetal growth restriction generally demonstrated the following maternal and placental risk factors for the development of this pathology (Figure 1).

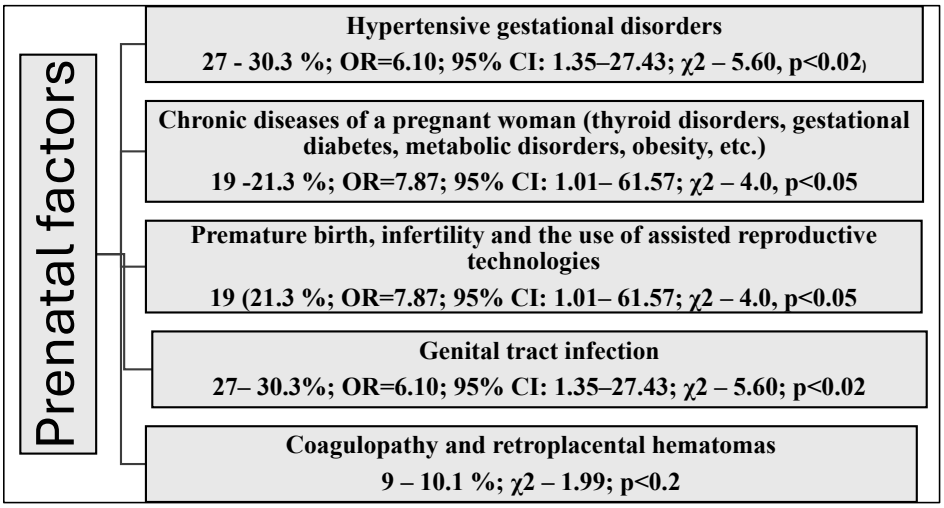


Figure 1. Main prenatal factors of placental dysfunction and late-onset fetal growth restriction, n=89, %.

The results obtained indicate the dominance of hypertensive gestational disorders in the group of prenatal factors of FGR (27-30.3%; OR=6.10; 95% CI: 1.35-27.43; χ2-5.60, p<0.02), chronic diseases of a pregnant woman (thyroidopathy, gestational diabetes, metabolic disorders, obesity, etc.) – 19 (21.3%; OR=7.87; 95% CI: 1.01-61.57; χ2-4.0, p<0.05), prematurity, infertility and the use of assisted reproductive technologies (ART) – 19 (21.3%); OR=7.87; 95% CI: 1.01-61.57; χ2-4.0, p<0.05), genital tract infections (27-30.3%; OR=6.10; 95% CI: 1.35-27.43; χ2-5.60; p<0.02), and to a lesser extent, coagulopathy and retroplacental hematomas (9-10.1%; χ2 = 1.99; p<0.2). The absence of prenatal risk factors was noted in 12 cases (7.9%).

A comparative analysis of pregnancy progression, obstetric and perinatal outcomes revealed abdominal delivery in 37 cases (41.6%) (Table 1), mostly associated with preeclampsia (21-23.6%), a combination of FGR and premature birth (14-15.7%), as well as deterioration of the intrauterine condition of the fetus (33-37.1%) against a background of a high index of somatic pathology and placental dysfunction.

The lowest proportion of operative delivery was observed in cases of fetal growth restriction against a background of treated infertility (4-4.5%), and in the absence of perinatal risk factors, the main indications were fetal distress during pregnancy and decompensated placental dysfunction (6-6.7%).

Table 1

Characteristics of the study groups, absolute numbers (%), n = 119

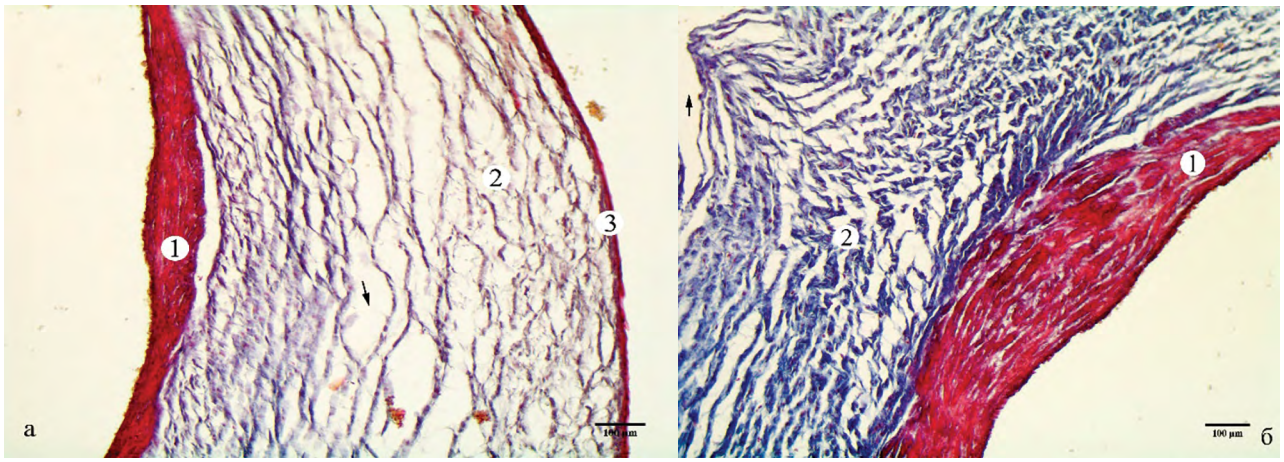
Indicators	Main group, n=89	Comparison group, n=30	χ2; p
Mother's age, years	26.7±1.3	28.1±1.2	p>0.05
First-time mothers	51 (57.3)	9 (30.0)	χ2=5.64; p<0.02
Pregnancy after ART	14 (15.7)	0	χ2=3.94; p<0.05
Gestational hypertensive disorders	27 (30.3)	2 (6.7)	χ2=5.60; p<0.02
Gestational diabetes	14 (15.7)	0	χ2=3.94; p<0.05
Obesity	19 (21.3)	1 (3.3)	χ2-4.0; p<0.05
Thyroid disorders	9 (10.1)	0	χ2 =1.99; p<0.2
Infectious conditions	14 (15.7)	0	χ2=3.94; p<0.05
Hemorrhagic factor	5 (5.6)	0	χ2 = 0.64; p<0.5
Premature birth + ART	19 (21.3)	1 (3.3)	χ2 = 4.0; p<0.05
Respiratory infections	17 (19.1)	2 (6.7)	χ2= 1.74; p<0,2
Bacteriuria and vaginitis	27 (30.3)	3 (10.0)	χ2= 3.90; p<0.05
Polyhydramnios	23 (25.8)	1 (3.3)	χ2= 5.73; p<0.02
Oligohydramnios	27 (30.3)	1 (3.3)	χ2=7.65; p<0.005
Caesarean section	37 (41.6)	4 (13.3)	χ2 = 6.72; p<0.01
Apgar score < 7 points	47 (52.8)	4 (13.3)	χ2=12.71; p<0.001
Transfer to NICU	33 (37.1)	0	χ2-13.60; p<0.001

The structure of postnatal abnormal conditions in newborns was as follows: the frequency of low Apgar scores < 7 points at 5 minutes was 52.8% (47), umbilical artery pH < 7.0 or umbilical vein pH < 7.1 – in 31.5% (28), resuscitation measures and artificial lung ventilation – in 11 cases (12.4%). Hospitalisation in the neonatal intensive care unit was required in one third of cases (33-37.1%). Abnormal conditions at birth were associated with premature delivery, hypertensive disorders and infection. It should be noted that six patients (6.7%) had a low Apgar score of < 7 points at 5 minutes where no prenatal risk factors for placenta-associated gestational complications were diagnosed. It indicates the need for further scientific research to optimise diagnostic approaches and delivery tactics in such pregnant women.

Cerebro-placental ratio parameters below unity were observed in 38 cases (42.7%; χ2-3.97; $p<0.05$) in the main group, while low cerebro-placental-uterine ratio parameters were observed in 49 cases (55.1%; χ2-6.83; $p<0.01$).

The assessment of the umbilical cord length allowed us to note slightly lower indicators compared to the data in the comparison group (21.4±2.2 cm versus 38.8±3.2 cm, respectively, $p<0.05$). When macroscopically examining the selected samples, excessive twisting/hypercoiling of the umbilical cord (UC), which led to the impossibility of dissection of vessels even on a 2 cm segment, as well as an excess of Wharton's jelly with a dense structure and/or its aplasia, which contributed to the development of vascular pathologies in it, should be noted. At the same time, UCs with excessive coiling, thinning of Wharton's jelly and their looseness prevailed. In 10 samples (31.3%) in the UC, cysts of Wharton's jelly were noted at autopsy.

In histological preparations, the change in the histoarchitecture of the umbilical vein is particularly striking. In 5 (15.6%) cases, varicose enlargement of the umbilical vein was observed, characterised by a sharp enlargement of the lumen and thinning of the walls (Fig. 2a). It should be noted that along the perimeter of such veins, there was an alternation of areas with sharp and moderate thinning of the media (Fig. 2 b). Also, around the UC vessels, there was a sharp thinning and swelling of the Wharton's jelly, and pseudocysts were diagnosed focally (Fig. 2 a).



a) b)

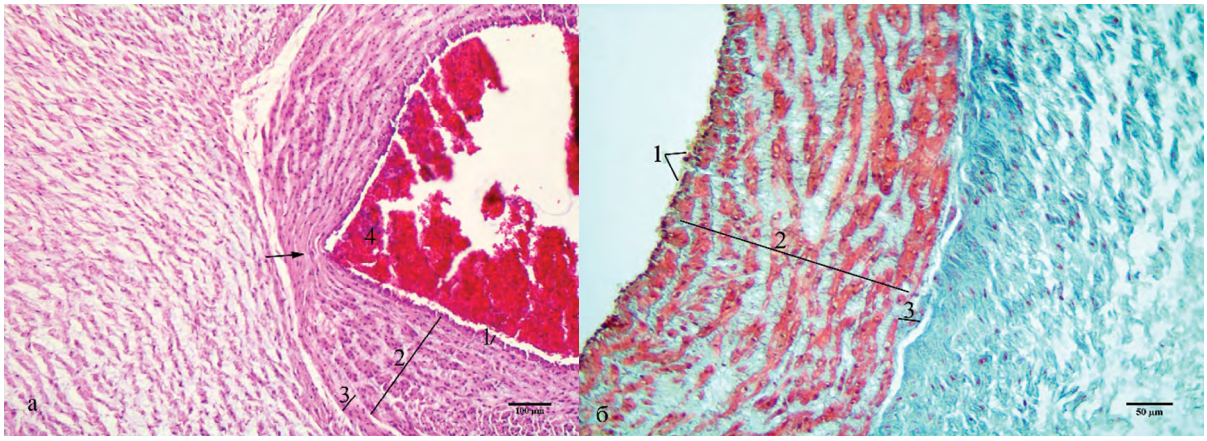
Figure 2. Features of the histostructure of the umbilical cord. Micrographs (a-b). Magnification: a-b) x100. Masson's trichrome stain (a-b). Varicose enlargement of the umbilical cord vein with thinning, swelling pseudocysts of Warton's jelly (patient Z., 25 years old. Diagnosis: Pregnancy I, 33 weeks. Fetal growth restriction (<3 percentile), late form). Designation: 1 – varicose thinning of the umbilical cord vein wall, 2 – swelling of the Wharton's jelly with pseudocysts (marked with an arrow), 3 – amniotic membrane.

In 26 (81.3%) samples of the umbilical cord collected postnatally in cases with FGR, we observed focal thinning of the umbilical vein wall (Fig. 3a), swelling, vacuolar dystrophy of myocytes, myolysis, and proliferation of connective tissue fibers in the muscular layer of the umbilical vein (Fig. 3b). The adventitial layer in the umbilical vein was sharply thinned and stratified in some places, which led to its single-layer structure in the form of separate connective tissue fibers and an unclear boundary between it and the Wharton's jelly (Fig. 3c). Venous thrombosis was common. The data obtained are confirmed by P. Tantbirojn's study, which shows an increase in the proportion of this finding in cases of umbilical cord anomalies to 28/102 (28.4%) compared to the control data – 6/84 (7.1%), which demonstrates the picture presented in some literature sources [19].

Macromorphological studies of the umbilical vein wall in the control group demonstrate the same thickness throughout the perimeter of the vessel, where three layers are clearly differentiated: the inner layer, which includes the endothelium lying on the basement membrane; the middle layer, which is the thickest and contains numerous concentric smooth muscle fibers with collagen fibers; and the third layer – the adventitia – with longitudinally arranged myocytes and collagen fibers (Fig. 3 d).

When examining UC arteries in samples collected postnatally during pregnancies complicated by FGR, it was found that most of them had slit-like lumens (Fig. 4a) and perivascular hemorrhages (4b).

In 4 samples (12.5%) of the UC in the case of FGR, unilateral varicose enlargement of the umbilical artery was detected. Such vessels were characterised by a sharply enlarged lumen, thinning and delamination of their wall membranes (Fig. 4).



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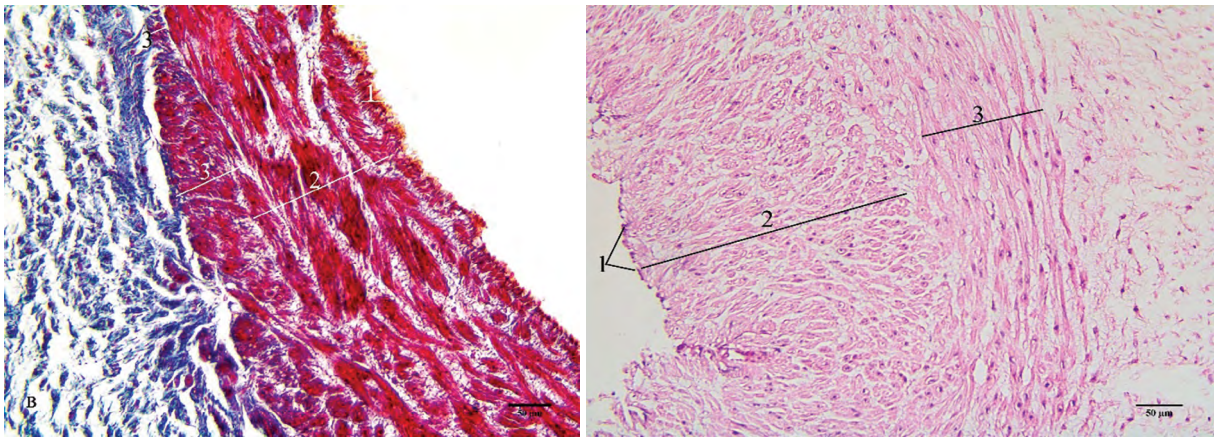
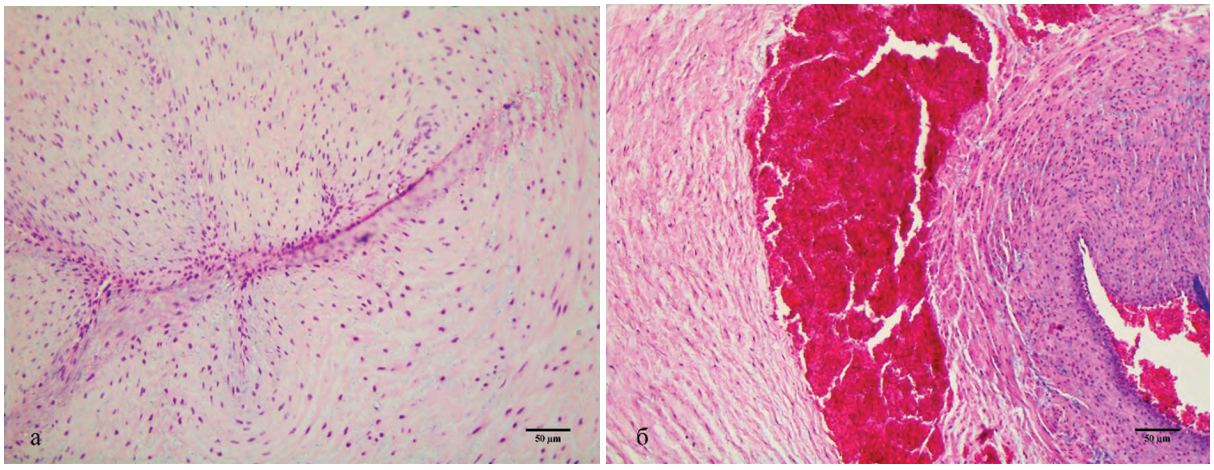


c) d)

Figure 3. Features of the histostructure of the umbilical cord. Micrographs (a-b). Magnification: a) x100, b-d) x200. Staining: a, d) haematoxylin and eosin, b-c) Masson's trichrome stain. Focal thinning of the wall (indicated by the arrow) of the umbilical cord vein and its thrombosis (a) (patient Z., 25 years old. Diagnosis: Pregnancy I, 33 weeks. Fetal growth retardation (<3 percentile), late form). Vacuolar dystrophy of myocytes, myolysis, and proliferation of connective tissue fibers in the muscular layer of the umbilical vein (b-c) (patient X, 27 years old. Diagnosis: Pregnancy II, 31 weeks. Fetal growth restriction (<3 percentile), late form. (b); patient B., 28 years old, diagnosis: Pregnancy I, 35 weeks. Fetal growth restriction (<5 percentile), late form (c). Structure of the umbilical vein wall of a patient in the control group (patient C., 24 years old. Diagnosis: Pregnancy I, 37 weeks. Delivery I, uncomplicated (d). Designations: 1 – inner membrane, 2 – muscular membrane, 3 – adventitial membrane, 4 – thrombus in the lumen of the vein.



a) b)

Figure 4. Features of the histostructure of the umbilical cord vessels. Micrographs (a-b). Magnification: a-b) x200. Staining: haematoxylin-eosin (a-b). Slit-like space of the artery (a) and perivascular hematoma (b) in samples in the case of FGR (patient K., 24 years old, diagnosis: Pregnancy I, 34 weeks. Fetal growth retardation (<3 percentile), late form).

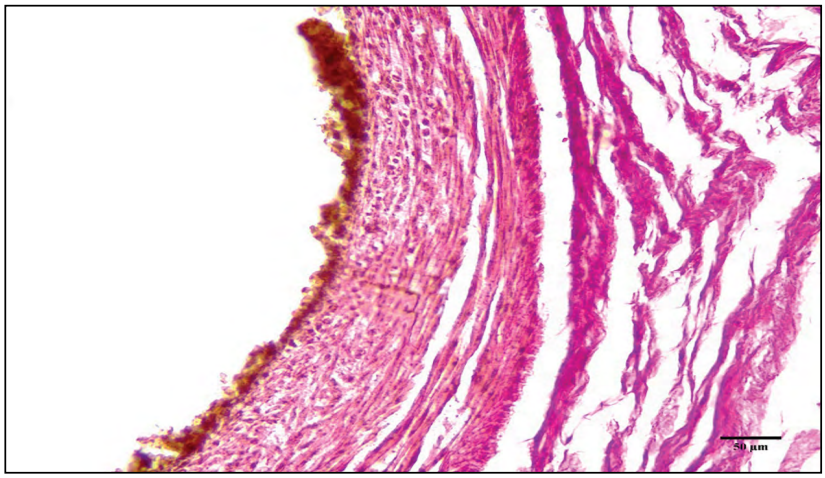


Figure 4. Varicose enlargement of the UC artery with FGR (patient Z., 25 years old. Diagnosis: Pregnancy I, 33 weeks. Fetal growth restriction (<3 percentile), late form). Microphotograph. Magnification: x200. Masson's trichrome stain. Designation: 1 – endothelium, 2 – muscular layer, 3 – stratified adventitia.

The muscular layer of the arteries becomes sharply thinned, and the myocytes in it lose their spiral arrangement. The adventitia is also thinned due to the loss of muscle layers and collagen fibres, often has layers that are stratified over a significant portion of the vessel perimeter and separated from the Warton's jelly by interstitial edema.

Discussion

The umbilical cord is the connecting link between the fetus and the mother and plays a key role in the proper functioning of fetal-placental blood circulation, ensuring adequate nutrition, oxygenation of the fetus and proper waste removal. In this study, we attempted to show the relationship between umbilical cord pathology and the development of FGR without taking into account changes in the placenta. First of all, we noted that all UC samples in newborns with FGR were hyperspiralised. According to scientific literature, the tortuous type of umbilical cord is important for feto-placental blood circulation, since umbilical cords with a segmented and knotted pattern can lead to chronic vascular obstruction of the fetus and stillbirth [2]. Moreover, the hypercoiled type of UC is associated with an increased risk of preterm birth, fetal health problems, the presence of meconium in the amniotic fluid, an Apgar score of < 7 at 5 minutes, intrauterine growth retardation, fetal and cardiac abnormalities, and fetal death [25].

According to our research, hypercoiled umbilical cord was associated with atrophy of the Wharton's jelly. As scientific publications demonstrate, the latter plays a major role in protecting the UC vessels from twisting and compression in response to fetal movements. The lack of adequate cushioning by Wharton's jelly in thin umbilical cords contributes to compression of the vessels and leads to disorders of fetal blood flow and growth [26]. A thin umbilical cord with a small amount of Wharton's jelly is associated with placental dysfunction, fetal growth retardation and low birth weight [27, 28]. Other researchers have linked obstructive umbilical cord lesions to a reduction in the area of the Wharton's jelly, which they interpret as risk factors for the development of fetal vascular malperfusion in the placenta [29]. Some scientists suggest that in early pregnancy, the area of Wharton's jelly increases linearly with gestational age, but after 32 weeks, the growth of Wharton's jelly volume stops [30]. The growth and structure of the Wharton's jelly (the composition of the gelatinous substance) change in cases of pregnancy complications such as preeclampsia [31] and gestational diabetes mellitus [32]. The fact of FGR and postnatal placenta examination were associated with a decrease in the area of the Wharton's jelly in the second half of pregnancy (second and third trimesters) [33].

Debebe SK et al. demonstrated that the width, length, and surface area of the placenta are directly proportional to the area of the Wharton's jelly. Moreover, the size of the umbilical vessels is closely correlated with both the area of the Wharton's jelly and other macroscopic parameters of the placenta (shape), confirming its important role in supporting placental growth and increasing blood flow in the UC with gestational age [27].

It is also worth paying attention to vascular abnormalities in the UC in newborns with FGR. Varicose enlargement of the umbilical vein was observed in 15.6% of cases of newborns with FGR. However, the data in the literature differ in their interpretation of this pathology. Navarro-González et al. argue that the presence of varicose enlargement of the umbilical vein as the only change usually has no consequences for the fetus [34]. However, in other studies, scientists associate the presence of intra-amniotic varicose enlargement of the umbilical vein with an increased risk of intra-amniotic hemorrhage, low birth weight and fetal death [35, 36].

Umbilical artery aneurysms were also diagnosed in a newborn with FGR, but such cases are very rare. They are identified in utero by turbulent pulsatile flow on ultrasound examination. They are usually associated with a condition known as single umbilical artery and are found in areas near the placental attachment site that are less protected by the Wharton's jelly, usually during the second or third trimester of pregnancy [37]. Umbilical artery aneurysms are associated with FGR, single umbilical artery, aneuploidy similar to trisomy 18, cardiac abnormalities, and fetal death [38].

Conclusions

A comparative analysis of pregnancy progression, obstetric and perinatal outcomes revealed a high frequency of abdominal delivery (41.6%), mostly associated with preeclampsia (21-23.6%), a combination of fetal growth restriction and preterm birth (14-15.7%), as well as deterioration of the intrauterine condition of the fetus (33-37.1%) against a background of a high index of somatic pathology and placental dysfunction. The results of our study emphasise the dependence of the UC abnormalities on the frequency of gestational complications and antenatal pathology, including those associated with fetal growth restriction. The characteristic pathohistological changes in the umbilical cord in such newborns were: short hypercoiled umbilical cord, aplasia and cysts of the Warton's jelly, thinning and destructive changes in the wall of the umbilical vein, perivascular hemorrhages, and reduced arterial flow capacity. Vascular anomalies, such as varicose veins and arteries, were quite rare in the morphology of the umbilical cord in newborns with FGR.

Prospects for further research

Changes in the umbilical cord are closely related to fetal programming and thus affect the health of a newborn at birth and in later childhood. Investigation of the spectrum of vascular changes in the placenta and their correlation analysis with UC and cardiovascular pathology diseases highlights the need for further research to establish ultrasound, anatomical, histological or plasma markers for the early diagnosis of fetal or prenatal pregnancy pathologies in order to prevent fetal morbidity and mortality.

Conflict of interest. The authors declare that there is no conflict of interest.

Ethical approval. This study was approved by the Ethics Committee of the HSEEU Ivano-Frankivsk National Medical University, Ivano-Frankivsk, Ukraine (approval ID: 146/24-26.09.2024).

Consent to participate. Written informed consent was obtained from the patients.

Data availability. Further data is available from the corresponding author on reasonable request.

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НЕОНАТОЛОГІЯ, ХІРУРГІЯ ТА ПЕРИНАТАЛЬНА МЕДИЦИНА
NEONATOLOGY, SURGERY AND PERINATAL MEDICINE
Т. XV, №3(57), 2025
VOL. XV, №3(57), 2025
KEY TITLE: NEONATOLOGÌÂ, HÌRURGÌÂ TA PERINATAL'NA MEDICINA (ONLINE)
ABBREVIATED KEY TITLE: NEONATOL. HÌR. PERINAT. MED. (ONLINE)
ISSN 2226-1230 (PRINT) ISSN 2413-4260 (ONLINE)

КЛІНІКО-МОРФОЛОГІЧНІ ОСОБЛИВОСТІ ПУПОВИНИ ПРИ ПІЗНІЙ МАНІФЕСТАЦІЇ ЗАТРИМКИ РОСТУ ПЛОДА

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Резюме.

Аномалії плаценти та пуповини можуть призвести до негативних перинатальних наслідків: затримка росту плода, внутрішньоутробна загибель плода, мала вага для гестаційного віку та екстрений кесарів розтин. Окремі публікації детально зупиняються на оцінці патологічних змін пуповинного канатику, але питання взаємозв'язку між макроморфологічними змінами пуповини і затримкою росту плода залишаються дискусійними і невирішеними.

Метою дослідження стало вивчення клініко-макроморфологічних маркерів розладів пуповинного кровотоку при пізній маніфестації затримки росту плода шляхом патогістологічного дослідження пуповинного канатику.

Матеріали та методи дослідження. Проведено дослідження у двох групах: основна група (89 пацієнток із пізньою маніфестацією затримки росту плода) та група порівняння (30 пацієнток з нормальними фетометричними показниками плода). Постнатально відібрано зразки тканини пуповинного канатику для макроскопічного та патогістологічного дослідження: 32 зразки – у основній групі та 10 зразків у групі порівняння.

Результати дослідження. Порівняльний аналіз перебігу вагітності та її наслідків дозволив відмітити високу частку ускладнень та абдомінального розродження (37-41,6%), у більшій частині асоційованого із прееклампсією (21,0-23,6%), поєднанням затримки росту плода та передчасних пологів (14,0-15,7%). При макроскопічному дослідженні відібраних препаратів відмітили надмірну скрученість/гіперспіралізацію пупкового канатику, надлишок Вартонієвих драглів із щільною структурою або його аплазію, у 10 зразках (31,3%) відмічались кісти Вартонієвих драглів, у 5 (15,6%) – варикозне розширення вени пуповин з розширенням просвіту та стоншенням стінки. У 26 (81,3%) зразках пуповинного канатику основної групи відмічали вогнищеві стоншення стінки пупкової вени, набряк, вакуольну дистрофію міоцитів, міоліз та розростання сполучнотканинних волокон у м'язовій оболонці пупкової вени. Нерідким було явище тромбозу вен. При дослідженні артерій виявлено щілиноподібний просвіт та периваскулярні крововиливи.

Висновки. Порівняльний аналіз перебігу вагітності дозволив відмітити високу частоту абдомінального розродження (41,6%), у більшій частці асоційованого із прееклампсією (21-23,6%), поєднанням затримки росту плода та передчасних пологів (14-15,7%), а також погіршенням внутрішньоутробного стану плода (33-37,1%) на тлі високого індексу соматичної патології та плацентарної дисфункції. Результати нашого дослідження підкреслюють залежність аномалій пуповинного канатику від частоти гестаційних ускладнень та антенатальної патології, у тому числі асоційованою із затримкою росту плода. Характерними патогістологічними змінами пуповинного канатику у таких новонароджених були: коротка гіперспіралізована пуповина, аплазія та кісти Вартонієвих драглів, стоншення та деструктивні зміни стінки пупкової вени, периваскулярні крововиливи, зменшення пропускної здатності артерій.

Ключові слова: затримка росту плода; плацентарна дисфункція; пуповина; макроморфологічні маркери патології пуповини.

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Received for editorial office on 09/07/2025
Signed for printing on 25/09/2025