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MORPHOGENESIS OF CHORIONIC VILLI IN PREGNANT WOMEN WITH CORONAVIRUS DISEASE (COVID-19)

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Abstract.

Villous chorion formation proceeds throughout gestation, encompassing stem, intermediate, and terminal villi. Exposure to adverse factors during pregnancy induces structural chorionic alterations that persist until delivery. SARS-CoV-2, the causative agent of coronavirus disease (COVID-19), directly activates endothelial cells, inducing microcirculatory disturbances alongside proliferative and inflammatory changes. Placental barrier integrity, nevertheless, permits delivery of neonates without detectable SARS-CoV-2 RNA by PCR and devoid of clinical signs of infection.

Objective. *To investigate chorionic villi morphogenesis in pregnancies complicated by COVID-19.*

Material and methods. *Placentas from pregnancies with confirmed maternal COVID-19, resulting in live-born, term neonates who were SARS-CoV-2 RNA-negative by PCR and presented with high Apgar scores, were examined (n = 144). Study groups were stratified according to the post-COVID interval (PCI), defined as the duration between maternal COVID-19 diagnosis and delivery. Group I (n = 77) comprised cases with PCI of 1-4 weeks, whereas Group II (n = 67) included those with PCI of 5-17 weeks. For comparison, placentas from physiological deliveries before the COVID-19 pandemic were analyzed (n = 53). Microscopic examination, immunohistochemical analysis with monoclonal antibodies against CD31 and smooth muscle actin (α-SMA), and statistical evaluation were performed. Quantification of the vascular component within chorionic villi and the percentage of stem and intermediate villi were assessed using the ONLINE JPG TOOLS service. The study was approved by the Bioethics Committee of Bogomolets National Medical University (Protocol No. 144, dated March 29, 2021). Statistical analysis was performed with SPSS IBM v.22 (Armonk, NY, USA). Descriptive statistics were presented as median with interquartile range (Me [Q1; Q3]). Differences between groups were assessed using the nonparametric Kruskal–Wallis test. A p-value < 0.05 was considered statistically significant. R&D project: «Study of morphogenesis and optimization of morphological diagnostics of the most common socially significant diseases» (State Registration No. 0124U000022, 2024-2026).*

Results. *Necrotic changes affecting the vascular endothelium of chorionic villi were observed in 98% of Group I cases, versus 6.1% in Group II. Stromal edema was present in all cases of Group I, accompanied by narrowing of the vascular lumen and a reduced vascular proportion in terminal villi to 29 (27-33) versus 68 (65-70) in the comparison group (p<0.001). In Group II, the vascular proportion increased to 46 (44-47). In stem (intermediate) villi, the arteriolar lumen diameter was reduced to 32 [18; 42] in Group I and 37 [14; 47] in Group II, compared with 48 [18; 80] in the comparison group (p<0.05). Arteriolar wall thickening corresponded to proliferative changes. Within the stroma of stem (intermediate) villi in Group I, telocyte-like cells exhibiting elongated morphology were observed, demonstrating negative or weak α-SMA expression. In Group II, cells with elongated α-SMA-positive processes corresponding to myofibroblasts were identified. Stromal fibrosis of stem and intermediate villi was identified in 89.4% of Group II cases compared with 4.7% in Group I; concomitantly, the proportion of these villi rose to 33 (23-47), versus 7 (7-9) observed in Group I and the comparison group, while the number of terminal villi was reduced in Group II.*

Conclusions. *COVID-19 during pregnancy induces remodeling of the villous chorion. Endothelial injury constitutes a central feature of this morphogenetic process, triggering phenotypic cellular transformation. Compensatory placental responses, including gradual restoration of endothelial integrity, vascular lumen, and focal hypoplasia of terminal villi, underlie the favorable neonatal outcomes reflected in high Apgar scores.*

Keywords: *COVID-19; SARS-CoV-2; Pregnancy; Placenta; Chorionic Villi; Telocytes; Remodeling.*

Introduction

Villous chorion development constitutes a continuous gestational process [1-3]. At term, placental architecture comprises stem, intermediate, and terminal villi [4], with terminal villi predominating and generating vasculosyncytial membranes that demarcate maternal–fetal circulatory compartments to facilitate efficient diffusion exchange [5].

Exposure to adverse intrauterine factors induces structural chorionic remodelling with persistence until parturition [6]. Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), targeting endothelial cells expressing angiotensin-converting enzyme 2 (ACE2) [7,8], triggers cellular activation [9] and and subsequent mediator release that disrupts microvascular luminal integrity [10-12].

Subendothelial migration of these mediators influences vascular smooth muscle cells and fibroblasts [13], thereby promoting arteriolosclerotic transformation [14,15]. Placental barrier function, nonetheless, permits delivery of neonates without virological or clinical evidence of SARS-CoV-2 infection [16,17].

Objective

To characterize morphogenetic features of chorionic villi in pregnancies complicated by COVID-19.

Materials and Methods.

Placentas from pregnancies with laboratory-confirmed maternal COVID-19, culminating in live-born, term neonates who were SARS-CoV-2 RNA-negative by PCR and exhibited high Apgar scores, underwent histopathological examination (n = 144). Maternal infection status was confirmed via positive polymerase chain reaction (PCR) testing; pregnancies complicated by preeclampsia were excluded.

Stratification into study groups was based on the post-COVID interval (PCI), defined as the elapsed time between

maternal diagnosis and delivery: Group I (n = 77) comprised PCI of 1-4 weeks; Group II (n = 67), PCI of 5-17 weeks.

Specimens were procured under a collaborative agreement with the Department of Pathological Anatomy, National Specialized Children's Hospital Ohmatdyt, Ministry of Health of Ukraine (2020-2022). Placentas from uncomplicated pre-pandemic deliveries served as comparators (n = 53).

Microscopic evaluation, immunohistochemical profiling with monoclonal antibodies targeting CD31 and smooth muscle actin (α-SMA) (Thermo Fisher Scientific, USA), and statistical modelling were employed..

Histological visualization was achieved via digital slide scanning with a Pannoramic DESK DW II system [18]; quantitative assessment of villous vascular components and and stem/intermediate villous ratio was conducted via the ONLINE JPG TOOLS service [19,20].

The study was conducted in accordance with bioethical standards regulated by the Declaration of Helsinki (1964), the Convention of the Council of Europe on Human Rights and Biomedicine (1997), and the current legislation of Ukraine, with formal approval granted by the Bioethics Committee of Bogomolets National Medical University (Protocol No. 144, dated March 29, 2021).

Statistical analysis was performed with SPSS IBM v.22 (Armonk, NY, USA). Descriptive statistics were presented as median with interquartile range (Me [Q1; Q3]); intergroup comparisons employed the nonparametric Kruskal–Wallis test, with statistical significance threshold set at $p < 0.05$.

R&D project: «Study of morphogenesis and optimization of morphological diagnostics of the most common socially significant diseases» (State Registration No. 0124U000022, 2024-2026).

Results and Discussion

Placental specimens obtained following delivery of live-born, term neonates from pregnancies complicated by COVID-19 at varying gestational stages were examined. Quantitative and qualitative distinctions emerged between study groups. Necrotic endothelial alterations and stromal edema affected chorionic villi in 98% of Group I cases versus 6.1% in Group II (Table 1; Figure 1).

Table 1

Pathomorphological changes in the placenta in pregnant women with COVID-19

Parameters	COVID-19 (N=144)			p
Groups	I (N=77)	II (N=67)	Comparison (N=53)	
Arteriolar endothelial necroptosis; n (%) [95% CI];	76 (98.7) [93-99.8]*	4 (6.1) [0.2-11.9]	0 (0) [0-6.7]	P_{1-2}<0.001
Narrowing of the lumen of the chorionic villi, n (%) [95% CI]; % of blood vessels in the terminal villus	77 (100) [95.3-100]; 29 (27-33)*	65 (97) [89.6-99.2]; 46 (44-47)*	0 (0) [0-3.8]; 68 (65-70)	p<0.001
Lumen diameter of the stem villus arterioles (μm)	32 [18; 42]*	37 [14; 47]*	48 [18; 80]	p < 0.05)
Wall thickness of the arterioles of the villi (μm)	35 [17; 48]*	22 [10; 35]*	13 [6; 28]	P_{1-2}<0.001 P_{1-3}<0.001
Proliferation of the muscular layer of the arterioles of the villi; n (%) [95% CI]	73 (94.8) [87.3-98]*	56 (83.6) [72.4-91]*	0 (0) [0-6.7]	p<0.001
Stromal fibrosis	4 (4.7) [0.1-9.3]	60 (89.4) [81-97.8]*	0 (0) [0-6.7]	p<0.001
Percentage of stem and intermediate villi in the chorion; %	7 (7; 9)	33 (23; 47)*	7 (7; 9)	p<0.001

*Note: *p<0,001 (Kruskal–Wallis test)*

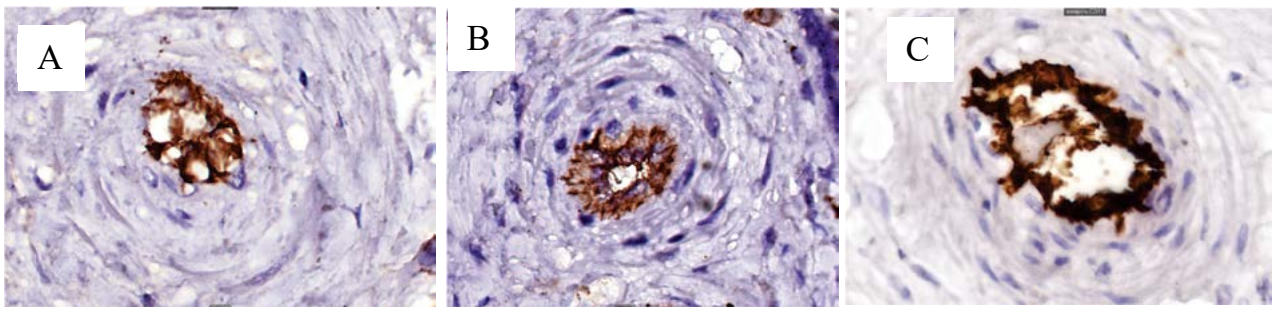

Figure 1. Structural changes in the arterioles of mature intermediate chorionic villi in COVID-19 patients and in the control group. A – Group I. B – Group II. C-Comparison group. Expression of monoclonal antibodies against CD31 in the endothelium of chorionic villi blood vessels. A, B, C ×1000.

Stromal edema, universal in Group I, coincided with vascular lumen narrowing and diminished vascular proportion within terminal villi [29 (27-33)] relative to the comparison group [68 (65-70); $p < 0.001$]. In Group II, the vascular proportion of terminal villi increased to 46 (44-47). In the main groups, quantitative changes in stem (intermediate) chorionic villi manifested as reduced arteriolar lumen diameter and an increased wall thickness (Table 1; Figure 1; Figure 2).

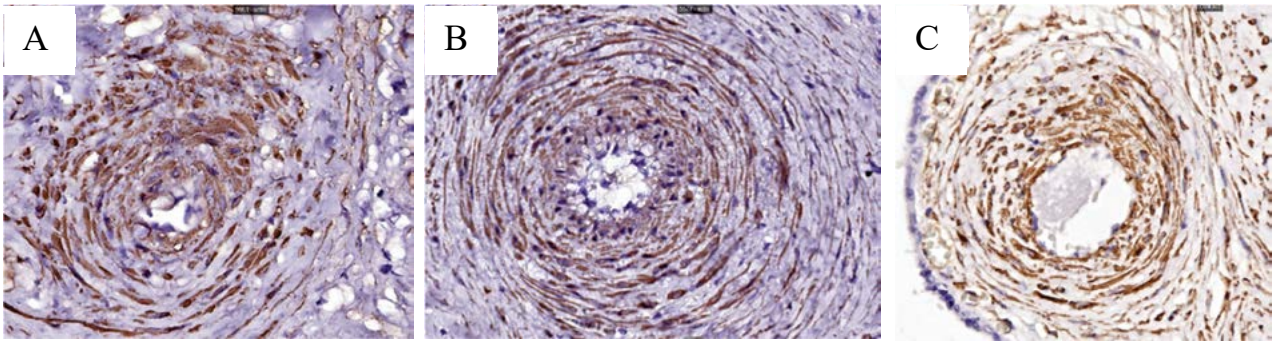

Figure 2. Structural changes in the arterioles of mature intermediate chorionic villi in COVID-19 patients and the control group. A – Group I. B – Group II. C-Comparison group. Expression of smooth muscle actin (α-SMA). A, B, C ×500.

Vessel wall thickness measured 35 [17; 48] in Group I and 22 [10; 35] in Group II, versus 13 [6; 28] in the comparison group (Table 1). Pairwise comparisons revealed significant differences between Group I and the comparison group ($p < 0.01$) and between Groups I and II ($p < 0.05$), with no statistically significant difference observed between Group II and the comparison group ($p > 0.05$) (Figure 3).

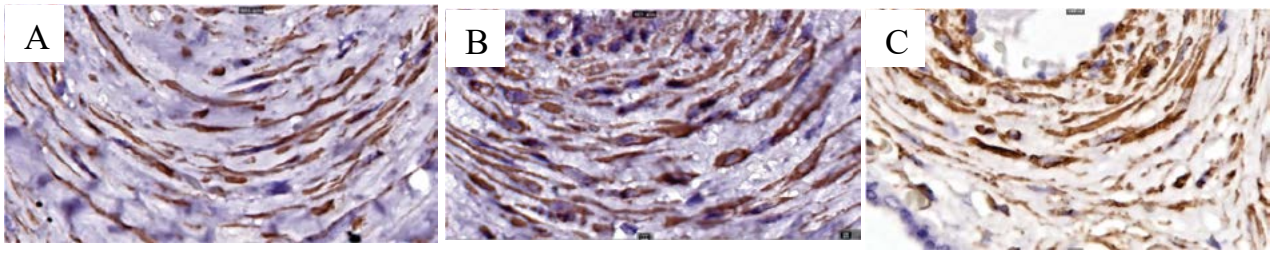

Figure 3. Structural changes in the wall of an arteriolar in a mature intermediate chorionic villus in a pregnant woman with COVID-19 and in the control group. A – Group I. B – Group II. C-Comparison group. Expression of smooth muscle actin. A, B, C ×1200.

Increased wall thickness corresponded to proliferative changes, detected in 94.8% of Group I cases and 83.6% of Group II cases. During the acute phase of maternal COVID-19, thin smooth muscle cells exhibiting elongated nuclei and moderate α-SMA expression were observed (Figure 3A). As the post-infectious interval lengthened, smooth muscle cell numbers increased, these cells exhibiting round nuclei and long processes with pronounced α-SMA expression (Figure 3B).

Within the stroma of stem (intermediate) villi, telocyte-like cells of elongated shape were observed, which demonstrated negative or weak α-SMA expression (Figure 4A), whereas Group II stroma contained cells with elongated α-SMA-positive processes consistent with myofibroblasts (Figure 4B).

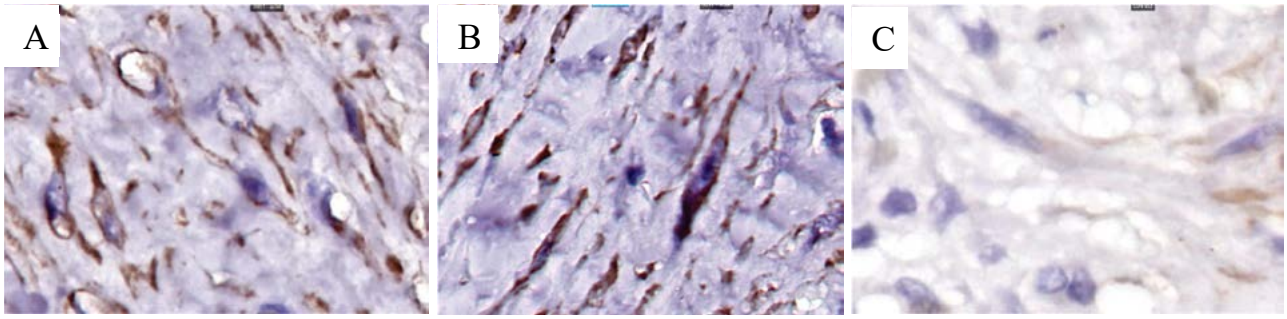

Figure 4. Structural changes in the stroma of mature intermediate chorionic villi in pregnancies complicated by COVID-19 and in the control group. A – Group I. B – Group II. C-Comparison group. A – Low expression of α-SMA in the processes of telocyte-like cells. B – marked expression of α-SMA in the processes of myofibroblasts. C – presence of α-SMA-negative cells, likely telocytes. Expression of smooth muscle actin (α-SMA). A, B, C ×1800.

Stromal fibrosis affecting stem and intermediate chorionic villi was identified in 89.4% of Group II cases, compared with 4.7% in Group I; concomitantly, the proportion of these villi in histological sections increased to 33 (23; 47) versus 7 (7; 9) observed in Group I and the comparison group (Figure 5). Terminal villi were reduced in number in Group II (Figure 5C).

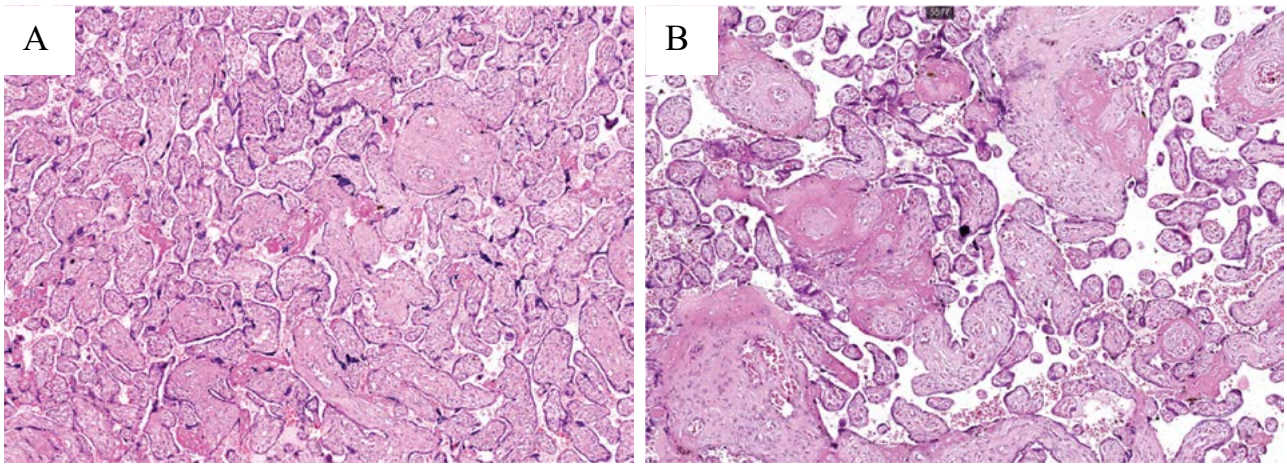

Figure 5. Remodeling of the villous chorion in a pregnant woman with COVID-19. A – Group I. Terminal chorionic villi predominate. An increased number of syncytial knots is observed among edematous, densely arranged terminal chorionic villi (A). B – Group II. Increased number of stem (intermediate) villi and stromal fibrosis; the number of terminal villi is reduced compared to Group I. H&E. A, B ×60.

Discussion of the results

SARS-CoV-2-induced endothelial injury was found to precipitate profound remodeling of the stroma and vasculature of chorionic villi. During the acute phase of maternal COVID-19, pathomorphological alterations in the chorionic villi were characterized by endothelial damage, vascular lumen narrowing, and predominance of contractile smooth muscle cells within arteriolar walls, as evidenced by elongated, spindle-shaped nuclear morphology. These alterations reflect vasoconstriction and impaired microcirculation, contributing to stromal hypoxia and edema. Prolongation of the post-infectious interval correlated with arterial wall remodeling in chorionic villi, wherein smooth muscle cells acquired a synthetic phenotype, manifested by increased cellularity, rounded nuclei, and enhanced α-SMA expression indicative of proliferative activation (Figure 3). Such phenotypic transformation of smooth muscle cells, plausibly mediated by hypoxia, inflammatory mediators, and growth factors, constitutes a central mechanism of vascular remodeling.

Extended PCI was associated with the emergence of α-SMA-expressing telocyte-like cells within the stroma, suggesting phenotypic transition of telocytes toward myofibroblast-like or fibroblast-like cells (Figure 4B). This interpretation is corroborated by the increased stromal fibrosis observed in Group II (Figure 5B).

Telocytes (TC), characterized by an elongated nucleus and extended cytoplasmic processes (telopodes), constitute the structural scaffold of chorionic villi and mediate intercellular communication, angiogenesis, and vascular tone regulation [21,22]. Irreversible TC alterations correlate with impaired angiogenic regulation [22] and terminal villous expansion, manifesting as delayed chorionic maturation [23], as observed in Group II. Several mechanisms underlying stromal fibrosis have been documented in preeclampsia. Irreversible TC damage and a reduced number of these cells precipitate stromal fibrosis, secondary to diminished regulatory control of fibroblast activity [24]. Alternatively, fibrotic progression stems from the phenotypic conversion of TC into fibroblast-like cells.

The remodeling process consequently entails coordinated contributions from both smooth muscle cells and TC. Transformation of telocyte-like cells promotes stromal fibrosis and subsequent stem villi remodeling; given that stem villi serve as the proliferative source for intermediate and terminal subtypes, this cascade precipitates delayed villous maturation and distal villous immaturity (Figure 5B).

The observed reduction in terminal villi following maternal COVID-19 diagnosis beyond 23 weeks' gestation did not compromise fetal status. This preservation likely reflects the prior establishment of mature intermediate villi, thereby resulting in focal rather than diffuse distal villous immaturity. Consistent with prior findings, maternal infection at earlier gestational stages (19-22 weeks) correlates with an elevated risk of perinatal loss, an outcome characterized by complete distal villous immaturity [25].

Prolongation of the PCI was accompanied by endothelial restoration, gradual normalization of vascular luminal caliber, and resolution of edema. The absence of adverse outcomes in the majority of maternal COVID-19 cases likely reflects inherent placental protective and compensatory mechanisms, a conclusion supported by the consistent delivery of neonates with high Apgar scores.

Conclusions. COVID-19 in pregnant women induces morphogenetic remodeling of the chorionic villi. Endothelial injury constitutes a primary driver of this process, initiating phenotypic cellular transformation. Compensatory placental adaptations – including gradual restoration of endothelial integrity and vascular luminal calibre, alongside focal distal villous immaturity – underlie the delivery of neonates with high Apgar scores.

Prospects for Future Research. Further studies should prioritize the characterization of TC structural alterations in pregnancies complicated by COVID-19 and concomitant antenatal fetal asphyxia, with particular emphasis on their contribution to placental dysfunction.

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Use of Artificial Intelligence. No artificial intelligence tools or methodologies were used in the preparation of this manuscript.

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НЕОНАТОЛОГІЯ, ХІРУРГІЯ ТА ПЕРИНАТАЛЬНА МЕДИЦИНА
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МОРФОГЕНЕЗ ВОРСИН ХОРІОНА ПРИ КОРОНАВІРУСНІЙ ХВОРОБІ (COVID 19) У ВАГІТНОЇ

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Резюме.

Формування ворсинчастого хоріона триває весь період свого існування: стовбурові, напівстовбурові та термінальні ворсини. Дія несприятливих чинників під час вагітності спричиняє зміни хоріона, що зберігаються до пологів. Пряма дія збудника коронавірусної хвороби (COVID-19), вірусу SARS-CoV-2, активує ендотеліоцити, спричиняючи порушення мікроциркуляції, проліферативні та запальні зміни. При цьому, бар'єрна функція плаценти забезпечує народження ПЛР негативних немовлят (визначення РНК вірусу SARS-CoV-2) без клінічних проявів інфікування.

Мета дослідження. Дослідити особливості морфогенезу ворсин хоріона при COVID-19 у вагітної.

Матеріали та методи дослідження. Досліджували плаценту при COVID-19 у вагітної при народженні ПЛР-негативного (визначення РНК вірусу SARS-CoV-2), живого доношеного плода з високими балами за шкалою Апгар (n =144). Основні групи формували в залежності від тривалості постковідного інтервалу (проміжок часу між діагностуванням COVID-19 у матері та пологами). Група I (n=77) був 1-4, група II (n=67) – 5-17 тижнів. Для порівняння даних досліджували плаценту (n=53) при фізіологічних пологах до епідемії COVID-19. Застосовували мікроскопічний, імуногістохімічні методи з використанням моноклональних антитіл проти CD31 та гладком'язового актину (α-SMA) і статистичні методи дослідження. Кількісні відмінності судин ворсин хоріона; відсоток стовбурових та напівстовбурових ворсин хоріона виявляли за допомогою сервісу ONLINE JPG TOOLS. Дослідження було схвалено Комітетом з біоетики Національного медичного університету імені І. І. Богомольця (протокол № 144 від 29 березня 2021 року). Статистичний аналіз результатів виконували з використанням програми SPSS IBM v.22 (Armonk, NY, USA), ліцензія НМУ імені О. О. Богомольця № 128 від 01.08.2016 р. Для статистичної обробки даних застосовували методи варіаційної статистики. Для множинного порівняння між групами середніх значень використовували непараметричний Kruskal–Wallis test. Рангові дані наведені у форматі медіана з нижнім та верхнім квартилями (Me [Q1; Q3]). Відмінності вважали статистично значущими при $p < 0,05$. Тема НДР: «Дослідження морфогенезу й оптимізація морфологічної діагностики найпоширеніших соціально значущих хвороб» (державний реєстраційний № 0124U000022, 2024-2026рр.рр.).

Результати. В групі I в 98% виявлялися некротичні зміни ендотелію судин ворсин хоріона проти 6,1% групи II. В групі I у всіх спостереженнях був наявний набряк строми ворсин зі зменшенням просвіту судин та відсотку судин у термінальній ворсині хоріона до 29 (27-33) проти 68 (65-70) у групі порівняння, $p<0,001$. У групі II відсоток судин термінальної ворсини хоріона збільшувався до 46 (44-47). У стовбурових (напівстовбурових) ворсинах зменшувався діаметр просвіту артеріол – 32 [18; 42] в групі I та 37 [14; 47] в II, проти 48 [18; 80] у групі порівняння, $p<0.05$. Збільшення товщини стінки артеріоли обумовлювалося проліферативними змінами в останній.

В стромі стовбурових (напівстовбурових) ворсин групи I були наявні телоцитоподібні клітини, видовженої форми, α-SMA-негативні або зі слабкою експресією α-SMA. У групі II виявлялися клітини з видовженими α-SMA-позитивними відростками – міофібробласти. При цьому фіброз строми стовбурових та напівстовбурових ворсин хоріона виявлявся у 89,4% випадків (проти 4,7% I у групі), а відсоток останніх в гістологічному зрізі був 33 (23; 47) проти 7 (7; 9) в групі I та групі порівняння. В групі II кількість термінальних ворсин виявилася зменшеною.

Висновки. COVID-19 у вагітної спричиняє ремоделювання ворсинчастого хоріона. Однією з ланок морфогенезу є ураження ендотелію, яке обумовлює фенотипову трансформацію клітин. Компенсаторні реакції плаценти, зокрема поступове відновлення цілісності ендотелію, просвіту судин, вогнищева гіпоплазія термінальних ворсин хоріона забезпечують сприятливі неонатальні результати, що підтверджується високими балами за шкалою Апгар.

Ключові слова: COVID-19; SARS-CoV-2; вагітність; плацента; ворсини хоріона; телоцити; ремоделювання.

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