MORPHOLOGICAL CHANGES IN PLACENTAL BLOOD VESSELS AT DIFFERENT SEVERITY LEVELS OF ECLAMPSIA

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Annotation: *Eclampsia is one of the leading pathologies during pregnancy. This condition is considered among the high-risk diseases that can lead to severe complications for both the mother and the fetus. In many cases, the development of eclampsia during pregnancy is associated with disturbances in placental formation. This study focuses on the microscopic changes in the structure of placental villi and their vascularization in eclampsia. Numerous studies have documented structural and vascular alterations in placental villi in both pre-eclampsia and eclampsia, highlighting their role in the progression of complications during pregnancy. In cases of pre-eclampsia and eclampsia, the length, volume, and surface area of villi, as well as the degree of vascularization, were observed to be underdeveloped.*

Keywords: *eclampsia, placenta, extragenital, vorsine-chorion.*

Introduction. In recent years, the issue of eclampsia has garnered special attention among obstetricians, gynecologists, embryologists, neonatologists, and morphologists. Eclampsia remains one of the leading pathologies during pregnancy. It often leads to multiple organ dysfunction, making the study of its etiology and pathogenesis a pressing topic for many researchers [10,13]. Eclampsia can cause complications of varying severity, including hemorrhage during obstetric procedures, placental abruption, placental insufficiency, fetal growth restriction, and severe disturbances in cerebral circulation [2,11,12]. Pregnant women affected by eclampsia and its complications experience a decrease in quality of life, which is associated with the risk of cardiovascular disease development. The progression of this pathological process in pregnant women is recognized as a major cause of perinatal morbidity and maternal mortality, as confirmed by contemporary statistical studies. According to statistics, pre-eclampsia accounts for 10.1% to 20% of pregnancy complications, and it represents 21.3% of maternal deaths. In developing countries, eclampsia remains the leading cause of maternal mortality (40–80%) [5,7,8]. Fischl F. and Grünberger W., in their studies, noted pathological changes in liver enzymes as an early warning sign of fetal distress due to pre-eclampsia.

In many countries, eclampsia is considered an “adaptation disorder” of pregnancy, manifesting at all stages of the general adaptation syndrome. Pathological changes developing in the pregnant woman’s body are closely linked with alterations in the immune, neurohumoral, and other regulatory systems that control vascular tone and microcirculation [1,3,6,14].

The development of eclampsia is influenced by genetic factors in combination with impaired immunological adaptation, such as inhibition of trophoblast migration and failure of spiral artery transformation. These factors, along with widespread vasospasm and imbalance in vasoconstrictor and vasodilator systems, lead to disrupted organ hemodynamics, including the uteroplacental-fetal circulation, and cause endothelial damage. Considering these factors, the severity of eclampsia requires detailed investigation of the pathomorphological changes in placental chorionic villi [4,8,9].

Aim of the Study: To morphologically investigate the changes in obliterative angiopathy of chorionic villi in the placenta in cases of varying severity of eclampsia.

Materials and Methods: The study examined 31 pregnant women in the main group, analyzing their medical records and obstetric history. Additionally, placental biopsy materials were collected from women who had died from eclampsia between 2019 and 2023. Each

patient and her biological material were registered in the RPAM "Adult Pathology" department. Two samples were taken from each placenta for pathomorphological examination, including both central and paracentral regions. Morphological and statistical methods were used in the study. The collected biological materials were fixed in a buffered 10% formalin solution for at least one day. The samples were then processed histologically, and paraffin blocks were prepared. Sections 3 μm thick were cut from the blocks and stained with hematoxylin-eosin. The sections were then covered with a cover slip and examined microscopically. Additionally, Van Gieson stain was used for histochemical examination to visualize the fibrous structure of the villi. The histological specimens were examined under a "LEICA LCD-400" microscope at magnifications of 10x, 20x, 40x, and 100x.

Depending on the severity of eclampsia in pregnant women, all placentas examined in the main group were divided into three subgroups. The first group included placentas from 10 pregnant women with early signs of eclampsia. The second subgroup consisted of placentas from 15 pregnant women with severe eclampsia. The third subgroup included 10 placentas from women exhibiting eclampsia symptoms in combination with various extragenital pathologies, such as arterial hypertension and chronic pyelonephritis.

Table 1.

Distribution of pregnant women by age in the study groups (absolute numbers, %)

Study group	16-25 years		21-30 years		25-30 years		31-40 years	
	Abc.	%	Abc.	%	Abc.	%	Abc.	%
Control group (n=25)	4	15,0	5	30,0	7	34,0	4	20,0
Main group (n=30)	5	34,5	10	28,2	24	40,0	6	17,3

Clinical Data Analysis: Analysis of clinical data showed that the most common extragenital comorbidities in the clinical groups were iron-deficiency anemia (25%), cardiovascular diseases (36%), and respiratory system disorders (21%). This is fully consistent with the literature regarding their role in the pathogenesis of eclampsia [3,8,12]. Eclampsia developed on the background of arterial hypertension and chronic pyelonephritis in 8 pregnant women. The placentas of 21 pregnant women aged 18 to 40 years (mean age 25 ± 1.1 years) were examined pathomorphologically. The pregnancies had proceeded normally. The birth weights of fetuses at 35–40 weeks ranged from 3040 g to 3760 g. Morphological measurements of the organs showed that the central region of the placenta typically had a thickness of 2.5–3.0 cm, while the edges were thinner, measuring 1.0–1.5 cm, forming the usual oval shape.

In the main group, the first subgroup consisted of 10 pregnant women aged 16–40 years (mean age 32 ± 1.4) with early eclampsia, whose placentas were examined pathomorphologically. In these cases, infants born at 37–38 weeks had weights ranging from 2560 g to 3130 g. The third subgroup of the main group included 8 women aged 18–35 years (mean age 30 ± 1) with eclampsia and extragenital pathologies, such as arterial hypertension and chronic pyelonephritis. Placental pathomorphological examinations were conducted in 7 cases.

Discussion of Results: Upon examination of the placentas, pathological foci were identified, though they were less pronounced in the control group. For example, thrombosis in the intervillous space was observed in only 1 out of 6 cases, and infarctions were also found in 1 out of 6 cases. These lesions were located within dense collagen layers, and the thickness of the muscular layer varied. In the arterioles, this layer consisted of 4–5 layers of myocytes, which extended into the dense collagen fibers beneath the syncytiotrophoblast covering the outer surface of the stroma of the stem villi. Additionally, a perivascular capillary network was observed on the outer surface of the stroma in the stem villi. Some of these capillaries were located directly beneath the epithelium and did not form syncytiocapillary membranes (Figure).

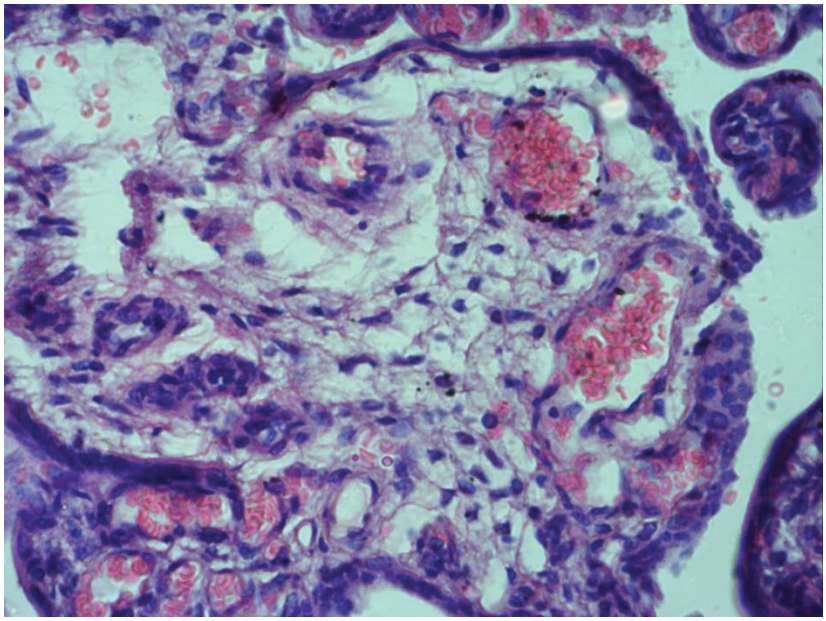


Fig. 1. **In a secondary stem villus, an arteriole (A) with a thin muscular wall is observed, alongside a venule (B). Beneath the epithelium, a perivascular capillary network (C) is visible. Staining was performed with hematoxylin and eosin, magnification ×200**

The presence of syncytiocapillary membranes is considered an objective indicator of full-term pregnancy. In this region of the placenta, these thin structures develop intensively during the final month of gestation. When stained with Schiff's reagent, a small accumulation of PAS-positive substances is clearly visible in the villous stroma (Figure 2).

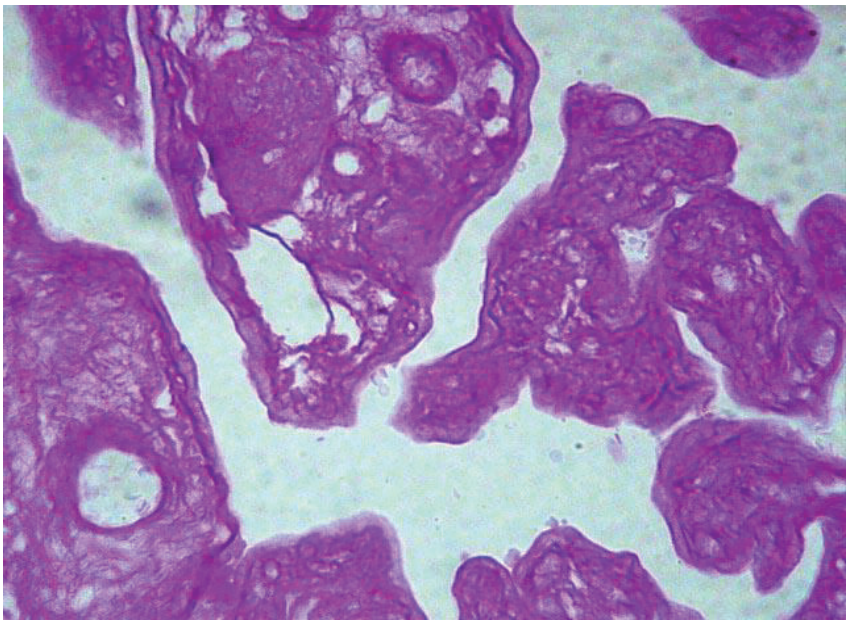


Fig. 2. **In physiological pregnancy, intermediate and terminal villi are observed, with only a small accumulation of PAS-positive material in the stroma of intermediate villi. PAS reaction, magnification ×10–40**

Histochemical studies showed that in all cases of acute eclampsia, the terminal villous syncytium stained a pale pink with the PAS reaction. Individual villous syncytia exhibited an intense PAS reaction, with positive substances accumulating in granules. These substances were identified in 22 cases. When sections of stem, intermediate, and terminal villi were stained according to Van Gieson and impregnated with silver, the distribution of collagen and elastic fibers was altered, reflecting the exacerbation of eclampsia compared to physiological pregnancy.

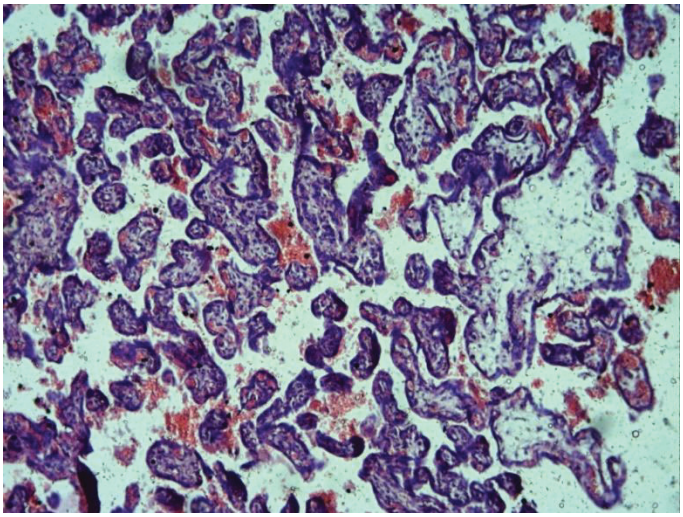


Fig. 3. **Pregnancy at 37 weeks. In early eclampsia, the number of blood vessels in terminal villi is increased, with a predominance of intermediately differentiated villi. Staining: hematoxylin and eosin. Magnification ×200**

In the presence of adequately developed arterioles, a noticeable increase in the area of venules can be observed in some chorionic villi (Figure 4). The lumens of these vessels contain the fetus's aggregated erythrocytes.

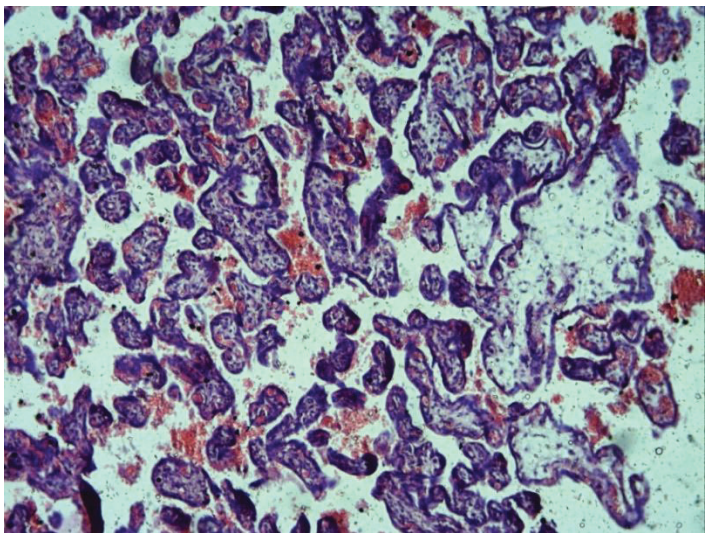


Fig. 4. **Pregnancy at 37 weeks. In early eclampsia, the number of blood vessels in terminal villi is increased, with a predominance of intermediately differentiated villi. Staining: hematoxylin and eosin. Magnification ×200**

A detailed study of terminal villi reveals weakening of cytotrophoblasts and hypovascularization in the stroma, with sparse central capillaries. These capillaries do not approach the epithelium, and the formation of syncytial layers is absent (Figure 5).

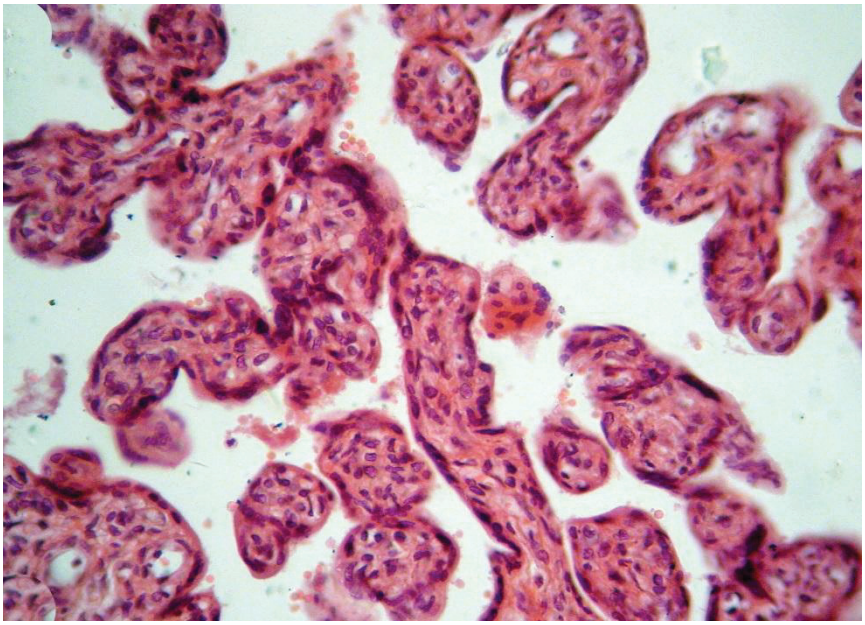


Fig. 5. **Pregnancy at 37 weeks. In severe eclampsia, the stroma of terminal villi shows sclerosis with increased density, hypovascularization, and absence of syncytial layers. Staining: hematoxylin and eosin. Magnification ×400 (10×40)**

In the capillary network surrounding obliterated arterioles and dilated venules, capillary dilation is observed, as well as capillary-sinusoidal structures located near the epithelial covering of the supporting villi. In the spaces around the capillary network, dense adhesive material composed of fetal erythrocytes is identified. In some supporting villi, the narrowed lumina of vessels indicate acute obliterative angiopathy.

The presence of pronounced hypoxia in the supporting villi is indicated by an increased number of fibroblasts and the predominance of involutive syncytial knots in the covering epithelium of these villi. Similarly, analogous changes in arteriolar vessels are observed in intermediate villi.

The placenta plays an active role in supporting the formation and growth of the fetus. The negative impact of eclampsia on fetal development manifests as delayed functional development, reduced adaptive responses, increased incidence of diseases, and the development of congenital deficiencies during the perinatal period. Morphological changes in placental vessels in eclampsia reflect a complex dynamic process aimed at maintaining the "mother–placenta–fetus" balance. During the progression of various pathologies, disruption of adaptive and compensatory mechanisms can lead to impaired blood supply to the placenta and, subsequently, to the fetus. Pathologies in placental vessels highlight the necessity of prenatal diagnostics, which allows for timely intervention, management of complex irreversible anomalies, and planning specialized care for the newborn or decisions regarding pregnancy continuation.

Conclusion. In various forms of eclampsia, angiopathy in the supporting, intermediate, and terminal villi and their vessels represents a clearly developed component of fetoplacental insufficiency. During the progressive course of eclampsia, concentric remodeling of blood

vessels is observed, resulting in lumen obliteration, thickening of the vessel walls, and a decrease in the geometric structural integrity of capillaries in terminal villi. This increases vascular resistance, leading to reduced diffusive-exchange function of the placenta and gradual development of placental insufficiency.

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