JOURNAL OF ADVANCED SCIENTIFIC RESEARCH

ISSN: 0976-9595

Journal of Advanced Scientific Research (ISSN: 0976-9595)
Vol.5. Issue 8 page 27

Editorial Team

C/O Advanced Scientific Research,
8/21 Thamotharan Street,
Arisipalayam, Salem

Journal of Advanced Scientific Research (ISSN: 0976-9595)
Vol.5. Issue 8 page 28

OPHTHALMOLOGICAL DISORDERS IN HYPERTENSION COMPLICATIONS PREGNANCY

Utkurova F.Sh., Rakhmanova S.Sh., Saipova M.L.
Center for the development of professional qualification of medical workers

Keywords: preeclampsia; retinal detachment; chorioretinopathy; optical coherence tomography of the eye

Introduction. Ophthalmological and obstetric practice pays undeservedly little attention to changes in the visual organ during pregnancy. This is largely due to the reversibility of the processes occurring in all organs and tissues of pregnant women. However, on the one hand, knowledge of the subtleties and nuances of the eye condition during pregnancy can help an ophthalmologist choose the right tactics for managing a patient, on the other hand, ophthalmological data during examination of a patient with hypertensive complications of pregnancy can serve to prevent the development of formidable complications.

The aim is to study ophthalmological disorders in hypertensive complications of pregnancy. The paper considers physiological changes in the organ of vision during pregnancy, possible pathological changes against the background of pre-existing and emerging eye diseases during pregnancy, ophthalmological changes in hypertensive complications and preeclampsia, describes the possibilities of diagnosing these conditions in modern ophthalmology. Endothelial dysfunction plays a key role in the pathogenesis of vascular disorders in preeclampsia. Preeclampsia may be accompanied by hemorrhagic and ischemic retinal infarcts, retinal pigment epithelium detachment, corkscrew-like tortuosity of arterioles on the periphery of the retina, retinal detachment, edema of the optic nerve disc, vitreous hemorrhages, lesions of the cerebral cortex in the visual centers, which may resolve with improvement in general condition. In some women, retinal detachment and diabetic retinopathy are late complications of preeclampsia. Optical coherence tomography of the eye, Doppler sonography of the central retinal artery, orbital artery, posterior short ciliary arteries can be used to diagnose chorioretinal complications

With the advent of new devices, the expansion of the technical capabilities of ophthalmological studies, new facts of changes in the eyes during pregnancy appear. Prospects are opening up for a more detailed study of this problem. Changes in the organ of vision during pregnancy can be physiological, pathological, associated with pregnancy, or pathological, not associated with pregnancy, in addition, pregnancy can affect previously existing conditions. Hormonal, metabolic, hemodynamic, vascular and immunological changes that occur during pregnancy can affect the function of the eye. These changes are usually transient, but in some cases they can be permanent and have consequences after childbirth. The purpose of the work is to study ophthalmological disorders in hypertensive complications of pregnancy. Before talking about changes in the organ of vision as manifestations of hypertensive complications of pregnancy, it is necessary to determine the range of physiological restructuring of the organ of vision. The most common physiological changes are changes in corneal sensitivity and thickness, decreased tolerance to contact lenses, decreased intraocular pressure, hemeralopia, and refractive errors. As a result of

hormonal changes in the first trimester of pregnancy, there is a moderate thickening of the tissues of both the anterior and posterior segments of the eye. Such changes can lead to the development of myopia with a change in visual acuity of no more than one diopter. Myopic lens displacement occurs as a result of an increase in the curvature of the lens during pregnancy, which leads to a change in refraction. In addition, a temporary loss of accommodation may be observed in the immediate postpartum period. These changes in the lens also indicate that new glasses or refractive surgery should be avoided during pregnancy. The results of refractive eye surgery before, during, or immediately after pregnancy are unpredictable, and refractive surgery should be delayed until refraction has stabilized. The resulting blurred vision is often difficult to differentiate from complications of preeclampsia without the use of special fundus examination techniques [1]. Physiologic changes also include increased pigmentation of the eyelids, ptosis, as well as decreased conjunctival capillaries and increased granularity of the conjunctival venules. Melasma is a condition characterized by increased pigmentation around the eyes and cheeks. It is commonly seen during pregnancy and is a result of increased melanocytosis and melanogenesis due to hormonal changes during pregnancy.

Pathological changes in pre-existing and pregnancy-onset eye diseases may include worsening of diabetic retinopathy, central serous chorioretinopathy, increased risk of peripheral vitreochorioretinal dystrophies and retinal detachment, development of uveal melanoma, and a beneficial effect on non-infectious uveitis. A higher incidence of ocular melanoma and its rapid progression have been documented in pregnant women compared to non-pregnant women. The mechanism of tumor growth during pregnancy is unclear, as a hormonal correlation with the pathophysiology of melanoma has not yet been established [6]. Pregnant women are at increased risk of progression of pre-existing proliferative diabetic retinopathy, and women with diabetes should consult an ophthalmologist before pregnancy or early in the first trimester. Diabetic macular edema, a hallmark of severe diabetic retinopathy that leads to vision loss, may develop during pregnancy. The safety of intravitreal antivascular endothelial growth factor has not been established and is best avoided; intravitreal triamcinolone is a safe alternative [5]. Importantly, eyes with existing or worsening sequelae of diabetes, such as non-resolving vitreous hemorrhage and traction retinal detachment, may be considered for surgical intervention. Central serous chorioretinopathy is an uncommon but benign complication of pregnancy. White subretinal exudates and multiple recurrences are common in central serous chorioretinopathy of pregnancy [7]. Central serous chorioretinopathy is defined as a fluid-filled detachment of the sensorineural retina due to a focal leak at the level of the retinal pigment epithelium. Symptoms may range from halos and blurred vision to severe central vision metamorphopsia.

Material and methods. The first case was a 35-year-old patient at 16 weeks of pregnancy, and the second was a 26-year-old patient at 20 weeks of pregnancy. Due to pregnancy-related contraindications, subthreshold micro pulse laser photocoagulation was the therapeutic method. Functional and anatomical evolution was very good in both patients. In both cases, treatment of the disease was preferable to prevent important photoreceptor losses. After treatment, good anatomical and

functional results were obtained. Micro pulse laser is an effective solution for the treatment of central serous chorioretinopathy [9]. It is the only safe therapeutic solution during pregnancy. Central serous chorioretinopathy can be pregnancy-related, not necessarily suggesting preeclampsia.

Preeclampsia is one of the leading causes of maternal and fetal morbidity and mortality worldwide. The visual system is affected in approximately 25% of patients with preeclampsia and 50% of patients with eclampsia. Symptoms include visual impairment, visual field defects, and diplopia. Preeclampsia causes severe arteriolar spasm due to vasospasm with increased resistance to blood flow and general narrowing of the retinal arterioles. The severity of retinopathy in patients with preeclampsia is inversely proportional to the birth weight of the fetus.

Preeclampsia is a complication of pregnancy, characterized by the following manifestations: an increase in blood pressure to values above 140/90 mm Hg at least twice with an interval of six to eight hours. Arterial hypertension becomes a diagnostic criterion for preeclampsia if all other possible causes of hypertension, except pregnancy, have been excluded during the examination; significant proteinuria with excretion of more than 300 mg of protein per day in the urine [10]. Preeclampsia occurs after 20 weeks of gestation in 5-10% of all pregnancies. If symptoms of preeclampsia occur before 34 weeks of gestation, the pathology is considered early, after 34 weeks late [11]. Preeclampsia is a severe, life-threatening complication of pregnancy. The most significant risk factors for the development of preeclampsia are maternal age over 40 years, obesity, previous pregnancy arterial hypertension, kidney disease, diabetes mellitus, artificial insemination, systemic lupus erythematosus, multiple pregnancy and a history of preeclampsia [12]. The development of gestational hypertension and preeclampsia is dangerous due to its liver, kidney, hematological, neurological and vascular complications. The consequence of the development of preeclampsia is multiple organ failure with the development of symptoms such as headache, visual impairment, pulmonary edema, thrombocytopenia, and liver and kidney dysfunction. Due to the loss of protein in the urine and hypoproteinemia, preeclampsia is accompanied by the appearance of edema, from segmental in the lower extremities to anasarca. Another clinically significant condition accompanying the development of preeclampsia is HELLP syndrome a combination of hemolysis, increased activity of liver transaminases and thrombocytopenia. Preeclampsia complicated by tonic seizures is called eclampsia [12, 14]. Fundus signs of preeclampsia may include retinal arteriolar stenosis, tortuosity, central retinal vein occlusion, and retinal or vitreous hemorrhages, optic nerve edema. Clinical visual symptoms include blurred vision, diplopia, amaurosis, photopsies, and scotomas, including homonymous hemianopsia: photopsies associated with vitreous hemorrhages in HELLP syndrome; scotomas (loss of visual fields) as a result of optic nerve atrophy; diplopia associated with abducens (VI cranial) nerve palsy. The latter is sensitive to changes in blood pressure, as it has a large extent in the intracranial space. Other than lowering blood pressure and preventing further seizures with magnesium sulfate, no specific therapy is recommended for preeclamptic women who experience visual changes. Although in

most cases, visual acuity returns to normal within weeks to months after symptoms begin, permanent visual impairment may rarely occur.

The consequences of preeclampsia on the part of the mother are caused, first of all, by vascular reactions to instability of arterial pressure, which in the form of vascular catastrophes can be reflected in the functioning of all organ systems. The pathogenesis of vascular disorders in preeclampsia is closely associated with the formation of endothelial dysfunction. It is assumed that preeclampsia develops as a result of cytotrophoblast invasion disorders, which leads to widespread endothelial dysfunction in the mother's body. Endothelial dysfunction, according to modern concepts, is a consequence of the uncoupling of the processes of production of vasoconstrictors and vasodilators, antiproliferative and proliferative vascular factors, angioprotectors and prothrombotic components [15]. With the development of preeclampsia, endothelial dysfunction is characterized by an increase in the activity of such antiangiogenic factors as soluble tyrosine kinases, endothelial growth factor and endoglin. They are formed in the placenta and enter the maternal systemic bloodstream, affecting the reduction of the vasodilating activity of the nitric oxide signaling pathway. Due to spasm of small vessels, the development of thrombosis and, ultimately, ischemia of organs and tissues, the key role of endothelial dysfunction in the pathogenesis of vascular disorders in preeclampsia is realized [11].

Results. Endothelial dysfunction has a significant impact on the functioning of all organ systems of a pregnant woman. At the same time, after childbirth, and therefore, the relief of preeclampsia, molecular signs of endothelial dysfunction can persist for the next 15-25 years. The possibility of developing early and late vascular complications of preeclampsia is equally high for any organ system, including the organ of vision [16]. According to the review by Nagy ZZ [17], ophthalmologic symptoms after preeclampsia are found in one third of patients.

A 31-year-old woman with severe preeclampsia developed bilateral serous retinal detachment on the second day after delivery. A few days later, the subretinal fluid spontaneously resolved and visual acuity improved. Ophthalmologic examination did not reveal signs of hypertensive retinopathy. Some retinal pigment epithelial changes, which are considered to represent healed choriocapillaris infarcts, persisted. Spontaneous retinal detachment is a rare complication of preeclampsia, occurring both before and after delivery. Damage to the retinal vessels and choroid were involved in the pathogenesis. Treatment of retinal detachment as a complication in obstetrics is conservative, and the prognosis is good. Retinal detachment in preeclampsia may be an indication for termination of pregnancy.

A case report of a 17-year-old female patient who was admitted to the obstetrics department with symptoms of preeclampsia. The pregnancy was terminated by cesarean section at 38 gestational weeks. The patient complained of blurred vision in both eyes throughout the perinatal period. Ophthalmologic examination revealed serous retinal detachment in both eyes. Optical coherence tomography demonstrated a hyporeflexive area between the retinal pigment epithelium and the neurosensory retina. With blood pressure monitoring in the postpartum period, the serous retinal detachment resolved spontaneously and the patient's vision improved [18].

Ophthalmologic complications of preeclampsia may develop both de novo and against an already existing comorbid background, aggravated by the development of gestational arterial hypertension. In addition, visual impairment can be caused by the use of drugs for the pathogenetic therapy of preeclampsia (for example, magnesium sulfate, benzodiazepines and phentoin) [1]. At the molecular level, the development of preeclampsia is closely associated with the activity of AT1-AA. The study by Fang Liu et al. [14] demonstrates that in the in vivo modeling of preeclampsia, AT1-AA induces apoptosis of retinal cells due to the release of reactive oxygen species and caspases. To simplify the understanding of the basics of the pathogenesis of chorioretinopathy in preeclampsia, the mechanisms of pathological changes can be defined as the following main components. Vasoconstriction and deformation of arterioles. A characteristic consequence of the development of preeclampsia is the narrowing and deformation of the arterioles of the tissues of the posterior segment of the eye, twisting them in the form of a corkscrew. According to the report by Soma-Pillay P. et al. [17], in women with normal blood pressure in the early stages of pregnancy, the retinal vessels do not have pathological changes, whereas in the group of women with gestational arterial hypertension, the retinal vessels are subject to deformation already in the early stages of pregnancy. An increase in blood pressure by every 10 mm Hg in pregnant women, according to this report, is accompanied by a decrease in the caliber of retinal arterioles by 1.9 nm [19]. Often, when studying the mechanisms of development of vascular complications in preeclampsia, a comparison is made with the complications of malignant hypertension. In both of these cases, the choroid, retina, and optic nerve are primarily involved in the pathological process; The main difference is the reversibility of pathological changes in preeclampsia.

Reduced blood flow in spasmodic arterioles leads to ischemia and subsequent damage to the retina, choroid, and pigment epithelium. In combination with physiological gestational hypercoagulation, vasoconstriction and thrombosis of small vessels predispose pregnant women to hemorrhagic and ischemic retinal infarctions with a frequency three times higher than in non-pregnant women of the same age. He X. et al. [20] describe retinal hemorrhages as "flames" due to their characteristic shape and indicate that hemorrhagic and ischemic chorioretinal lesions of varying severity are observed in 100% of preeclampsia cases. Peripheral vasoconstriction, which occurs with systemic hypertension, leads to extravasation of the liquid portion of the blood into the extracellular space with the development of edema. With extravasation, diffuse macular edema is formed in the peripheral parts of the retina, and in the central zone, accumulations of exudate, resembling "cotton wool balls", can form, as well as lipoprotein deposits, which are usually called hard exudates [8]. Despite the possibility of spontaneous resolution of early complications of preeclampsia, the persistence of endothelial dysfunction after childbirth determines the possibility of developing late chorioretinal complications in women who have had preeclampsia. Kolenko O.V. et al. [21] in their works refer to data from a cohort study involving more than one million women; 21% of patients who have had preeclampsia, 4-6 years after delivery, seek medical help for various pathologies of the retina and choroid. At the same time, the most common late complications of preeclampsia are retinal detachment and diabetic retinopathy. Auger N. et al. [22]

note in their review that in a retrospective study of data on 1.1 million women, of whom 5.8% were diagnosed with preeclampsia, the risk of developing diabetic retinopathy was 8.4 times higher in the early form of preeclampsia and 3.6 times higher in its late form, compared with the control group of women who had normal blood pressure. The risk of developing late non-diabetic retinopathies in the same study in patients with preeclampsia was 4.6 and 1.9 times higher, respectively. Another study by Auger N. et al. [23] showed that women with a history of preeclampsia had a higher rate of cataract extraction during their lifetime compared to women who did not have preeclampsia (21/1000 versus 15.9/1000 cases, respectively). At the same time, the rate of cataract extraction was higher in women who had early preeclampsia (increased risk by 1.51 times versus 1.2 in women with late preeclampsia).

Discussion. In the case of moderate and severe preeclampsia, the risk was 20% higher. A similar study [24] conducted in the Russian Federation in 2020 showed a significantly higher incidence of late (developing within 3-11 years after delivery) retinopathies in groups of patients with gestational arterial hypertension and preeclampsia. Various types of fundus examination are used as instrumental diagnostics of retinal lesions in preeclampsia. One of the popular diagnostic methods is fluorescein angiography. This method allows one to assess the decrease in perfusion in the peripheral parts of the retina, while other methods allow one to examine mainly its central zone. Fluorescein angiography also allows us to detect damage to the pigment epithelium - in this pathological condition, multiple areas of fluorescein leakage into the subretinal layer may be observed, which resemble pinheads in appearance [12, 17]. Fluorescein angiographic observations confirm the hypothesis that retinal detachment is secondary to damage to the choroid. The most modern and accurate method for studying the vascularization of the posterior segment of the eye is OCT. Tomograms of patients with preeclampsia show thickening of the choroid with narrowing and deformation of its arterioles [25]. OCT allows us to assess the severity of decreased blood flow primarily in the capillaries of the central zone of the fundus. OCT in patients with preeclampsia may also show serous retinal detachment, subretinal deposits, and other choroidal vascular abnormalities [26]. A 33-year-old woman presented with conscious hypertension (170/90 mmHg) and proteinuria at 28 weeks of gestation. The patient complained of sudden and severe decrease in visual acuity. Fundus examination showed bilateral serous retinal detachments in the macula. Optical coherence tomography showed subretinal and intraretinal fluid. Intense arteriolar vasospasm has been implicated in the pathogenesis of serous retinal detachment. Optical coherence tomography showed the presence of both subretinal and intraretinal fluid during the acute phase of preeclampsia. The authors explain the detachment of the retinal pigment epithelium by age-related macular degeneration or other maculopathies; in young patients, it is described as idiopathic serous detachment of the retinal pigment epithelium [12].

Conclusions:

1. Physiological changes in the visual organ during pregnancy include pigmentation of the skin of the eyelids, ptosis, changes in clinical refraction in the

form of myopization of no more than 1 diopter, changes in the thickness, curvature and sensitivity of the cornea, a decrease in intraocular pressure.

2. Pathological changes in the comorbid background and eye diseases that arise during pregnancy include worsening of diabetic retinopathy, central serous chorioretinopathy, an increased risk of peripheral chorioretinal dystrophies and retinal detachment, and the development of uveal melanoma.

3. Signs of preeclampsia in the fundus may include narrowing of the retinal arterioles, tortuosity, occlusion of the central retinal vein and hemorrhages in the retina or vitreous body, edema of the optic nerve, serous retinal detachment.

4. Modern, safe and promising methods for detecting pathology of the eye vessels in hypertensive complications of pregnancy are optical coherence tomography of the retina and duplex scanning of the eye vessels.

References:

1. Stern EM, Blace N. Ophthalmic Pathology Of Preeclampsia. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2022 Jan

2. Garg P, Aggarwal P. Ocular changes in pregnancy. Nepal J Ophthalmol. 2012; 4(1): 150-161. DOI: 10.3126/nepjoph. v4i1.5867

3. Dinn RB, Harris A, Marcus PS. Ocular changes in pregnancy. Obstet Gynecol Surv. 2003; 58(2): 137-144. DOI: 10.1097/01. OGX.0000047741.79433.52

4. Roos NM, Wiegman MJ, Jansonius NM, Zeeman GG. Visual disturbances in (pre)eclampsia. Obstet Gynecol Surv. 2012; 67(4): 242-250. DOI: 10.1097/OGX.0b013e318250a457

5. Rzeszotarska A, Szczapa-Jagustyn J, Kociecki J. Ophthalmological problems in pregnancy – a review. Ginekol Pol. 2020; 91(8): 473-477. DOI: 10.5603/GP.2020.0080

6. Grant AD, Chung SM. The eye in pregnancy: ophthalmologic and neuro-ophthalmologic changes. Clin Obstet Gynecol. 2013; 56(2): 397-412. DOI: 10.1097/GRF.0b013e31828f273c

7. Androudi S, Ekonomidis P, Kump L, Praidou A, Brazitikos PD. OCT-3 study of serous retinal detachment in a preeclamptic patient. Semin Ophthalmol. 2007; 22(3): 189-191. DOI: 10.1080/08820530701501469

8. Ge G, Zhang Y, Zhang M. Pregnancy-induced hypertension and retinopathy of prematurity: a meta-analysis. Acta Ophthalmol. 2021; 99(8): e1263-e1273. DOI: 10.1111/aos.14827

9. Ochinciuc R, Munteanu M, Baltă G, Baltă F. Central serous chorioretinopathy in pregnancy. Rom J Ophthalmol. 2022; 66(4): 382-385. DOI:

10.22336/rjo.2022.68 10. Poon LC, Magee LA, Verlohren S, Shennan A, von Dadelszen P, Sheiner E, et al. A literature review and the best practice advice for second and third trimester risk stratification, monitoring, and management of pre-eclampsia. Int J Obstet Gynecol. 2021; 154(S1): 3-31. DOI: 10.1002/ijgo.13763

11. Belotserkovtseva LD, Kovalenko LV, Pankratov VV, Zinin VN. Pathogenetic Approach to Early Preeclampsia and the Feasibility of Pregnancy Prolongation. General Reanimatology. 2022; 18(2): 37-44. Russian (Белоцерковцева Л.Д., Коваленко Л.В., Панкратов В.В., Зинин В.Н. Ранняя преэклампсия и возможность пролонгирования беременности с точки зрения патогенетического

подхода //Общая реаниматология. 2022. Т. 18, № 2. С. 37-44). DOI: 10.15360/1813-9779-2022- 2-37-44

12. Chau FY, Pimentel MC, Bhagat N, Lim JI. Preeclampsia/eclampsia associated retinopathy. Am Acad Ophthalmol. 2022.

13. Yagel S, Cohen SM, Goldman-Wohl D. An integrated model of preeclampsia: a multifaceted syndrome of the maternal cardiovascular-placental-fetal array. Am J Obstet Gynecol. 2022; 226(2S): S963-S972. DOI: 10.1016/j.ajog.2020.10.023

14. Rana S, Lemoine E, Granger JP, Karumanchi S. Preeclampsia: pathophysiology, challenges, and perspectives. Circ Res. 2019; 124(7): 1094-1112. DOI: 10.1161/CIRCRESAHA.118.313276

15. Vlasov TD, Nesterovich II, Shimanski DA. Endothelial dysfunction: from the particular to the general. Return to the "Old Paradigm"? Regional blood circulation and microcirculation. 2019; 18(2): 19-27. Russian (Власов Т.Д., Нестерович И.И., Шиманьски Д.А. Эндотелиальная дисфункция: от частного к общему. Возврат к «старой парадигме»? //Регионарное кровообращение и микроциркуляция. 2019. Т. 18, № 2. С. 19-27). DOI: 10.24884/1682-6655-2019-18-2-19-27.

16. Moss HE. Neuro-ophthalmology and Pregnancy. Continuum (Minneap Minn). 2022; 28(1): 147-161. DOI: 10.1212/ CON.0000000000001059

17. Nagy ZZ. Review of the ophthalmic symptoms of preeclampsia. DHS. 2020; 3(1): 21-23. DOI: 10.1556/2066.2020.00005

18. Bartczak A, Kraśnicki P, Urban R, Laudaiński T, Mariak Z. Bilateral serous retinal detachment in preeclampsia-a case report. Klin Oczna. 2014; 116(1): 21-23.

19. Soma-Pillay P, Pillay R, Wong TY, Makin JD, Pattinson RC. The effect of pre-eclampsia on retinal microvascular caliber at delivery and post-partum. Obstet med. 2018; 11(3): 116-120. DOI: 10.1177/1753495X17745727

20. He Xinyi, Ji Yimei, Yu Meiting, Tong Yuhua. Chorioretinal alterations induced by preeclampsia. J Ophthalmol. 2021; 2021: 8847001. DOI: 10.1155/2021/8847001