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Pregnancy and Eye
Gebelik ve Göz

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ABSTRACT
Visual obscurations are common during pregnancy. The ocular effects of pregnancy may be physiological, pathological or may be modifications of pre-existing conditions. While most of the described changes are transient in nature, others extend beyond delivery and may lead to permanent visual impairment. Also, pregnancy can affect vision through systemic disease that are either specific to the pregnancy itself or systemic diseases that occur more frequently in relation to pregnancy. Neuro-ophthalmological disorders should be kept in mind in pregnant women presenting with visual acuity or field loss. Therefore, it is important to be aware of the ocular changes in pregnancy in order to counsel and advice women who currently are, or are planning to become pregnant.
Key words: Ocular, pregnancy, blindness.

ÖZET
Göz kararmaları gebelikte yaygın olarak görülür. Gebeliğin göze etkileri fizyolojik, patolojik ya da önceden var olmuş modifikasyonlar olabilir. Tanımlanan değişikliklerin çoğu doğal olarak ortaya çıkarken genelde geçicidir fakat diğerleri doğumdan sonrasına aktarılarak kalıcı görme bozukluğuna neden olabilir. Ayrıca gebelik ya da gebeliğin kendine özgü sistemik hastalıklar veya gebelik ile ilişkili daha sık meydana gelen sistemik hastalıklar aracılığıyla görüşü etkileyebilir. Nöro-oftalmolojik bozukluklar gebe kadınlarda görsel netlik ya da görsel alan kaybı ile başvurduklarında akılda bulundurulmalıdır. Bu bağlamda gebelik planlaması yapan kadınlara danışma ve tavsiye verme için gebelikteki oküler değişikliklerinin farkında olmak çok büyük önem arz etmektedir.
Anahtar kelimeler: Oküler, gebelik, körlük.

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Introduction

Pregnancy implies progressive anatomical and physiological changes that are not only confined to the reproductive organs, but also to all the systems of the body[1]. Pregnancy results in a lot of hormonal changes in the body and the eyes are of no exception[2]. In general, the ocular effect of pregnancy can be divided in to physiologic and pathologic changes[3] . Most of the physiologic changes that occur as a result of pregnancy are usually marked in the third trimester. This is because at this period, hormonal activity is at its peak. However, these changes are transient because several weeks at postpartum, all hormonal activities return to their pre-pregnant state[4].

The aim of this study is to give an overview on the changes that can affect the ocular health and vision of a pregnant woman and keep the ophthalmologist/physician aware of the physiologic changes of pregnancy, the effect of pregnancy on pre-existing ocular disease and the ocular manifestations of systemic diseases in pregnant women. The physiological changes in eyes include the following systems.

Oculo-Cutaneous

Chloasma which is also known as mask of pregnancy is a hormonal mediated process, characterized by increased pigmentation around the eyes and cheeks. The pigmentation changes tend to fade slowly postpartum[5]. It is caused by increased pigmentation related to increased estrogen and progesterone[6]. It is postulated that hormonal variations of pregnancy increase melanin as a result of an increase in both melanogenesis and melanocytosis[3].

Ptosis (drooping of the eyelids) has been reported during and after normal pregnancy and is thought to be related to fluid retention and hormonal changes. It requires no treatment. Ocular motility defects can present for the first time during pregnancy[6].

Cornea

Corneal sensitivity has been found to decrease in the later part of pregnancy. Krukenberg spindles on the cornea have been observed early in pregnancy and they tend to decrease in size during the third trimester and postpartum. The mechanism presumably is related to hormonal changes such as low progesterone levels, however, by the third trimester, an increase in progesterone and aqueous outflow often result in decreased or absence of Krukenberg spindles [5]. The loss of corneal sensitivity during pregnancy cannot account for the difficulty encountered by some women in wearing their contact lenses. It is more likely to be

due to a variation in corneal topography as a result of oedema[7]. Park et al. showed that there was a statistically significant increase in corneal curvature during the second and third trimesters which resolved completely after delivery or after the cessation of breast feeding[3].

Pre-existing conditions, such as keratoconus may progress during pregnancy[8]. Only a few cases of iatrogenic keratectasia and keratoconus occurring during pregnancy have been reported. There is growing evidence that the massive estrogen increase in late pregnancy increases the risk of keratectasia in predisposed individuals[9]. In the literature, four cases of keratoconus progression were reported as developing during pregnancy without any accompanying factors. Various studies have demonstrated elevated levels of collagenolytic and gelatinolytic activities in keratoconic corneas. Matrix metalloproteinase (MMP) levels are increased, whereas tissue inhibitors of MMPs (TIMPs) are decreased in the keratoconic corneas as well as during pregnancy too[10]. Several clinical observations suggest that thyroid gland dysfunction is associated with keratoconus pathophysiology[11]. Corrective procedures such as laser refractive surgery are contraindicated during this time[5].

Contact lens intolerance: The sensitivity of the pregnant mother's cornea decreases significantly which may cause problems for contact lens wearers who may traumatize their corneas more than usual[12].

Change in refraction: The tendency of fluid retention affects your refraction. It is usually a temporary change and you need not get your eyes re-tested during the later stages of pregnancy and for at least the first 6 weeks after child birth[12]. Pregnant women usually have about twofold increase in secretion of aldosterone. This, along with the actions of estrogens, causes a tendency to reabsorb excess sodium from the renal tubules and to retain fluid. Also, the bone marrow becomes increasingly active and produces extra red blood cells to go with the excess fluid volume. Fluid retention can cause the curvature of the cornea to become steeper, causing light rays from objects to become focused in front of the retina, as it happens in myopia[4].

Cornea-Conjuctiva

Dry eyes: Some women experience dry eyes during pregnancy. This is usually temporary and goes away after delivery[12]. This may occur as a result of the direct disruption of lacrimal acinar cells through pregnancy enhanced immune-reactivity of prolactin, transforming growth factor beta 1, and epidermal growth factor in ductal cells[3]. In one study, a decrease in tear

production occurred during the third trimester of pregnancy in approximately 80% of pregnant women[6].

Conjunctival blood vessels: Changes in conjunctival blood vessels have been described toward the end of pregnancy. These changes include a granularity of conjunctival venules, mild spasm of conjunctival arterioles, and decreased visualization of conjunctival capillaries. Excessive vomiting during pregnancy can cause conjunctival petechiae[12].

Glaucoma

Intra-ocular pressure: The normal intra-ocular (IOP) pressure may decrease slightly and may persist for several months postpartum. This could be advantageous to patients suffering from glaucoma[12]. The reduced IOP is likely due to an increase in the facility of outflow via one of several possible mechanisms, including increased uveo-scleral outflow due to hormonal changes, decreased episcleral venous pressure and decreased pressure in the upper extremities[5]. Estrogen causes dilatation of the vessels of the circulatory system leading to decreased arterial pressure and thus a reduction in aqueous humor production[2]. Progesterone has glucocorticoid antagonistic properties and this antagonistic action helps in the lowering of the IOP[1].

Visual field (VF) changes: The classical field change is a bitemporal hemianopsia. However, visual field changes varying from slight temporal or concentric contraction to complete homonymous hemianopsia have been reported. Proposed mechanisms are equally diverse and include changes to the pituitary gland that may affect the optic chiasm. These asymptomatic visual field changes were shown to be completely reversible postpartum[5]. Akar et al. found that the visual field mean threshold sensitivity increased significantly in the third trimester[3].

Crystalline Lens and Uvea

Lens: The curvature of the crystalline lens can increase, causing a myopic shift in refraction[6]. Pizzarell reported a worsening of myopia with pregnancy[4] .

Accomodation: Transient loss of accommodation has been seen during and after pregnancy. Accommodative insufficiency and paralysis have been documented in association with lactation[5].

Uveitis: The immunosuppressive effects and high steroid levels present in pregnant women may cause improvement of uveitis during pregnancy but there is a risk of exacerbation

postpartum[5]. The development of anterior uveitis associated with ankylosing spondylitis can be more common in the early postpartum period. Postpartum endogenous candidal endophthalmitis, presumed to be related to intravascular dissemination around the time of delivery, has been reported[6]. In some cases, patients with Vogt-Koyanagi-Harada (VKH) disease became pregnant and their ophthalmic findings improved during pregnancy in spite of tapering of systemic corticosteroids[13].

Pathological effects of pregnancy on eyes and diseases modified by pregnancy are as follows :

Oculo-Vascular Changes

Diabetes mellitus: This is the most common ocular condition modified by pregnancy[6]. Gestational diabetes poses a very low risk for the development of retinopathy. In patients who had nonproliferative diabetic retinopathy (DR), studies demonstrated that as many as 50% of them may show an increase in their nonproliferative retinopathy. Approximately 5-20 % of these patients develop proliferative changes. An ophthalmologic examination at least once every trimester is recommended. Studies on patients with proliferative diabetic retinopathy have shown that a progression of disease may occur in as many as 45% of them. In patients with proliferatve diabetic retinopathy, monthly ophthalmic examinations are warranted. Proliferative diabetic retinopathy may regress at the end of the third trimester or postpartum. Patients with proliferative diabetic retinopathy, cesarean section should be considered to prevent vitreous hemorrhage due to Valsalva maneuver used during labor. Diabetic macular edema may develop or worsen during pregnancy[12].

Factors that have been shown to influence the progression of DR in pregnancy include, the pregnant state itself, duration of diabetes, degree of retinopathy at time of conception, metabolic control of diabetes, and the presence of co-existing hypertension. The exact pathogenesis for the progression of DR during pregnancy remains controversial[2]. Some studies demonstrated a decrease in retinal venous diameter and volumetric blood flow in diabetic patients during pregnancy and hypothesized that this may exacerbate retinal ischemia and hypoxia3. Chen et al. measured volumetric blood flow in the major retinal veins during pregnancy in diabetic women, found a connection between progression of retinopathy and increased volumetric total blood flow in a retinal quadrant[14].

Occlusive vascular disorders: It is well appreciated that pregnancy represents a hypercoagulable state in which both clotting factors and clotting activity are increased. Both branch and central retinal artery occlusions have been reported to occur in pregnancy[12].

Gonzalvo et al. reported a case of central retinal vein occlusion associated with pre-eclampsia and HELLP (haemolysis, elevated liver enzymes and low platelets) syndrome, a thrombotic microangiopathic vasculopathy following caesarean section[15].

Pregnancy induced hypertension (pre-eclampsia)/eclampsia: Von Graefe first described retinal changes in pre-eclamptic women in 1855[15]. Ocular involvement reported in these patients includes conjunctival vascular anomalies, hypertensive retinopathy, exudative retinal detachment, vitreous and preretinal haemorrhage, ischaemic optic neuropathy and hypertensive choriodopathy. Out of the visual symptoms blurred vision is most common[16]. Patients may present with symptoms of headache and visual disturbances in the form of scotoma. Visual disturbances develop in perhaps 25% of women with severe pre eclampsia[17]. The development of complete blindness is rare and seen in only 1%-3% of cases[18]. Rasdi et al from Malaysia studied a group of patients with hypertensive disorders of pregnancy. The retinal changes were seen in 21.5% of preeclampsia/eclampsia. The most common abnormality seen in the fundus is narrowing of retinal arterioles. In a study, Reddy from India has reported retinal changes in 53.4% preeclampsia[19]. The mechanism of such changes remain unknown, but is believed to be a result of a combination of factors, including pre-existing vascular disease, hormonal changes, endothelial damage, alterations in cerebral autoregulation and hypoperfusion-induced ischaemia 15. Pre-eclampsia is associated with choroidal ischaemia and increased microvascular permeability, a combination that is likely to increase the fragility of new choroidal vessels. The Valsalva manoeuvre during labour might also provoke choroidal haemorrhage[20].

In eclampsia, retinal changes are likely to occur when diastolic blood pressure (BP) is more than 100 mm of Hg and systolic BP is above 150 mm of mercury[5]. Reversible cortical blindness and extraocular muscle palsy, though rare, have been well documented in the eclamptic patients[16]. The three most common visual complications of preeclampsia and eclampsia are hypertensive retinopathy, exudative retinal detachment and cortical blindness[21].

Disseminated intravascular coagulation (DIC) can occur with severe pre-eclampsia. The choroidal involvement causes a serous retinal detachment, which resolves with the resolution of DIC[5].Placental thromboplastin may release into maternal circulation and activate the extrinsic coagulation system with resultant DIC[22].

Exudative retinal detachment occurs in 1% of pre-eclamptic patients and up to 10 % of eclamptic patients. It is thought to be caused by choroidal ischemia[5]. The majority of patients who manifest serous detachment during pregnancy have, with clinical management,

complete recovery within weeks after delivery, and there is no need for any surgical intervention[23].

HELLP syndrome: Approximately 10 % of women with severe pre-eclampsia develop the HELLP syndrome. Ocular findings include bilateral serous retinal detachment with yellow/white sub-retinal opacities and sometimes vitreous hemorrhage[5].

Cortical blindness: Cortical blindness refers to reduced vision from bilateral damage to any portion of the visual pathways posterior to the lateral geniculate nucleus [19]. The most common etiology is bilateral posterior cerebral artery infarction[24]. Cortical blindness is a clinical syndrome characterized by intact pupillary reflexes and normal fundoscopic findings. Neuroimaging findings in cortical blindness range from normal to typical findings such as bilateral cortical occipital lesions with hypodensity on computerized tomography (CT) or hyperdensity on T2-weighted magnetic resonance imaging (MRI)[21]. Hinchey J et al between 1988 and 1994, found only three cases that had reversible cortical blindness following eclampsia. Some other studies suggest vascular endothelial damage as the underlying mechanism in this case of preeclampsia related transient cortical blindness[25]. Isolated cortical blindness has been thought to occur in only 1% to 3% of pregnancies complicated by preeclampsia-eclampsia[26].

Thrombotic thrombocytopenic purpura (TTP): It is rare but can develop in association with pregnancy. Visual symptoms occur in approximately 10% of these women and are generally related to serous retinal detachment, arteriolar constriction and optic disc oedema[6].

Antiphospholipid syndrome (APS): It is an autoimmune disorder characterized by either a history of vascular thrombosis or pregnancy morbidity in association with the presence of antiphospholipid antibodies. Conjuctival telangiectasia or conjuctival microaneurysms, episcleritis, limbal or filamentary keratitis and iritis have been described as the APS ocular features from the anterior segment, vitritis, retinal detachment, posterior scleritis, branch or central retinal vein occlusion, bilateral choroidal infarction, cilioretinal artery occlusion, venous tortuosity, retinal haemorrhages, cotton-wool spots and central serous type chorioretinopathy from the posterior segment and monocular or bilateral transient visual loss, transient visual field loss, ischemic optic neuropathy and progressive optic nerve atrophy as the neuro-ophthalmologic features of APS[27].

Venous sinus thrombosis: Pregnancy has increased susceptibility to venous sinus thrombosis. Significant increased risk is associated with caesarean delivery, increasing

maternal age, hyperemesis gravidarum, intercurrent infection and maternal hypertension. Common signs and symptoms are headache, focal or generalised seizures, paresis and papilloedema[6].

Macular Changes

Central serous retinopathy (CSR): Chumbley and Frank reported central serous retinopathy in a 34-vear-old woman during 4 consecutive pregnancies, with remission after delivery or spontaneous abortion. Probably circulatory changes in the choriocapillaris can provoke central serous choroidopathv in pre-disposed eyes[28]. Kitzmann et al. reported the results of a population based study on the incidence of CSR in Olmsted, Minnesota from 1980 to 2002 and found 11 females with CSR confirmed by fluorescein angiography, one of who was pregnant (9%), Kitzmann et al. also reported nine cases of CSR without confirmation by fluorescein angiography, pregnancy was one of the risk factors affecting this group (one patient)[2]. The macula is especially affected by pregnancy even when healthy − for example, two studies reported infrequent central serous chorioretinopathy in the third trimester in healthy pregnant women[29].

Toxoplasmic retinochoroiditis: Silveira et al. have reported a case of maternofetal transmission in a preconceptionally immunised woman. This finding could be accounted for by a down-regulation of the T-cell-mediated immune response that is observed during pregnancy. This contention is supported by the findings of Ramchani et al. who reported a case of acquired ocular toxoplasmosis occurring during pregnancy without transmission of the disease to the child[30].

Choroid neovascularisation (CNV): Healthy pregnancy is associated with increased activity of many angiogenic factors, including vascular endothelial growth factor (VEGF), placental growth factor (PlGF), erythropoietin (EPO) and nitric oxide, which can all stimulate retinal neovascularisation and CNV[20]. CNV occurrence during pregnancy has been reported as a complication of presumed ocular histoplasmosis syndrome or punctuate inner chorioretinopathy[31].

Optic Nerve Changes

Optic neuritis and neuropathy: The association of optic neuritis and pregnancv has previously been suggested[28]. There appears to be a decreased incidence of optic neuritis during pregnancy, perhaps because of the immunosuppressive effects[6].

Other Oculo-CNS Changes

Retinitis pigmentosa progression in some cases is seen. Choroidal haemangiomas have been reported to undergo rapid growth during pregnancy but some can regress postpartum[5]. With pregnancy, previously asymptomatic pituitary adenomas or microadenomas may enlarge and result in various ophthalmic symptoms such as headache, visual field change, and/or visual acuity loss[12]. The tumor and intrasellar contents may expand superiorly, compressing the optic chiasm, optic tracts, and optic nerves, producing decreased visual acuity in 52% and visual field defects in 64% of patients[8]. Meningiomas may have a very aggressive growth pattern during pregnancy that is difficult to manage. They may regress postpartum but may regrow during subsequent pregnancy [12]. The ophthalmic findings reported are decreased visual acuity, visual field defects (for example, bitemporal hemianopia), oculomotor palsy and disc oedema[6].

Sheehan syndrome/Pituitary apoplexy: Sheehan's syndrome (SS) is a pituitary failure occurring in women after labour. The prevalence of SS in India is estimated to be 2.7–3.9% among parous women older than 20 years[32]. It is considered a potentially visually-threatening disorder as a result of sudden increase in pituitary size from infarction or hemorrhage. The classic VF defect is a bitemporal superior quadrantic defect. Ophthalmoplegia occurs in 78% of cases. It results from compression of the cavernous sinus, which makes cranial nerves III, IV, and VI vulnerable to injury. Oculomotor nerve is involved most commonly. Horner syndrome may develop from damage to the sympathetic fibers[3] . Common predisposing factors include closed head trauma, hypotension, hypertension, history of pituitary irradiation, cardiac surgery, anticoagulant therapy and pregnancy. Clinical features of pituitary apoplexy include sudden onset of headache, nausea, vomiting, visual symptoms, ptosis, altered mental status and endocrinologic dysfunction[8].

Idiopathic intracranial hypertension (IIH): IIH is a syndrome of raised intracranial pressure in the absence of clinical, laboratory, or radiological evidence of intracranial space-occupying lesions[33]. Common symptoms are headache, pulsatile tinnitus, nausea, transitory visual obscurations, diplopia and blurred vision. In theory, IIH may be attributed to the following factors; parenchymal edema, increased cerebral blood volume, excessive cerebrospinal fluid (CSF) production, compromised CSF resorption, and venous outflow obstruction. Both pregnancy and exogenous estrogens are thought to promote IIH or worsen it. Since Quincke first related menstrual irregularity and pregnancy to IIH over a century ago, a number of isolated case reports and a few larger series have described patients who developed IIH during

pregnancy[34]. The condition was considered 'benign' in comparison with cases of tumour but it has been argued that loss of visual function in up to 25% of cases and progression to blindness if untreated means that it should not be considered 'benign' as far as visual function is concerned[35]. Decreased flow in the optic nerve results in papilledema and vision changes. When the abducens nerve is involved, diplopia occurs. Most patients have some evidence of optic nerve disease, such as slightly reduced visual acuity, color deficiency,a visual field defect, or an afferent pupillary defect[36].

A few other diseases also need attention during pregnancy:

Graves' disease: Hyperthyroidism occurs in 2/1000 pregnancies, the commonest cause (85%) being Graves' hyperthyroidism[37]. It is an important cause of unilateral and bilateral proptosis. Graves' disease tends to remit late in pregnancy and relapse postpartum[5].

Myaesthenia gravis (MG): The effect of pregnancy on MG varies considerably among women and even between pregnancies in the same woman. During pregnancy, symptoms worsened for 41% of women with MG, while 30% showed no change, and 29% had remission of symptoms. Improvement of symptoms during the second and third trimesters has been attributed to normal immunosuppressive changes. Thus, women with MG should delay pregnancy for at least 2 years after disease onset [38].

Conclusion

This article provides a practical overview for pregnant women and the ophthalmologist. Recognizing various ocular symptoms and signs in pregnancy, as well as understanding the treatment strategies, are critical for proper management of these patients. Doctors should treat each pregnant woman on an individual basis and should have a firm understanding of the various ocular changes associated with pregnancy and the implication they may have for management.Therefore in pregnant and postpartum women, a high index of suspicion and a thorough ophthalmic evaluation can often be vision and life saving.

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