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The Eyes in Pregnancy

By Mark Bona, MD, and Agnes Wong, MD, PhD, FRCSC

Pregnancy is associated with changes to multiple organ systems, including the visual system. The effects of pregnancy on vision can be divided into 3 broad categories:

- **physiologic changes**
- **pathologic changes that develop as a result of pregnancy (eg, central serous retinopathy, preeclampsia and eclampsia with associated hypertensive retinopathy, exudative retinal detachment, or cortical blindness)**
- **pre-existing conditions that alter over the course of pregnancy (eg, diabetic retinopathy, benign intracranial hypertension, meningioma, pituitary tumour).**

In addition, the use of ophthalmic drugs during pregnancy may adversely affect both the mother and the fetus. This issue of *Ophthalmology Rounds* discusses these conditions and current management strategies.

Physiologic changes

Several physiologic changes occur during pregnancy. Decreased intraocular pressure (IOP) has been demonstrated during the second half of pregnancy and this IOP reduction tends to persist for several months postpartum.[1,2] The reduced IOP is likely due to an increase in the facility of outflow[3,4] via one of several possible mechanisms, including increased uveoscleral outflow due to hormonal changes,[2] decreased episcleral venous pressure,[5] and decreased pressure in the upper extremities.[6] Consequently, pre-existing glaucoma has been reported to improve during pregnancy.[2]

The cornea also undergoes changes. Corneal sensitivity tends to decrease in the latter part of pregnancy and returns to normal sensitivity up to 2 months postpartum.[7] Corneal thickness also increases due to edema,[8] which may result in a change in the refractive index of the cornea.[9] Therefore, it is advisable that any changes in eyeglass prescriptions be postponed until several weeks postpartum. Pregnant patients have also described contact lens intolerance with previously comfortable lenses as a result of corneal edema.[10]

Pathologic changes that develop as a result of pregnancy

Central serous retinopathy

Central serous retinopathy (CSR) is characterized by neurosensory retinal detachment, with associated retinal pigment epithelial (RPE) detachment, RPE leakage, as well as RPE and choroidal hyperpermeability. In general, CSR is a sporadic, self-limiting disease with a predilection for young or middle-aged adult males with type A personalities, presumably due to elevated circulating cortisol and epinephrine that affect autoregulation of the choroidal circulation. Additional risk factors include untreated systemic hypertension, allergic respiratory conditions, as well as use of systemic steroids, antibiotics, or alcohol.[11]

Although CSR is much more common in males than in nonpregnant females with a ratio of 10:1, pregnancy has been shown to be an independent risk factor for CSR, with an odds ratio of 7.1.[11,12] It is, therefore, important to consider CSR in a pregnant patient who presents with the following symptoms: decreased visual acuity, central scotoma, micropsia, or metamorphopsia. Investigations include intravenous fluorescein angiogram (IVFA) or optical coherence tomography (OCT). OCT has a theoretical advantage over IVFA because the fetus is not exposed to fluorescein dye.[13]

CSR typically resolves spontaneously, with resorption of subretinal fluid and exudates within 1 to 6 months. Visual acuity returns to normal or near-normal (20/25) in most patients (80%-90%).[14] However, CSR in pregnancy is more likely to cause subretinal fibrinous exudates. These occur in 75%-100% of pregnant women, as compared to 17% of men and 0% of nonpregnant women.[14]

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FACULTY OF MEDICINE
University of Toronto

Department of Ophthalmology and Vision Sciences

Department of Ophthalmology and Vision Sciences, Faculty of Medicine, University of Toronto,
60 Murray St.
Suite 1-003
Toronto, ON M5G 1X5

Preeclampsia and eclampsia (toxemia)

Preeclampsia refers to new-onset hypertension (systolic blood pressure [BP] >140 mm Hg or diastolic BP >90 mm Hg) and proteinuria after 20 weeks gestation in a previously normotensive woman. It affects 5%-8% of pregnancies and is the second leading cause of maternal mortality (12%-18%) in North America. It is often accompanied by other signs of maternal end-organ damage, including oliguria, pulmonary edema, abdominal pain, liver dysfunction, thrombocytopenia, as well as visual and cerebral abnormalities.[15]

Eclampsia, characterized by the development of generalized tonic-clonic seizures in the setting of preeclampsia, occurs in up to 2% of women with preeclampsia. Eclampsia is an obstetrical emergency because both mother and fetus are at immediate risk of death or long-term neurologic complications. Prompt delivery of the fetus and placenta is the only cure. Symptoms usually resolve after delivery.

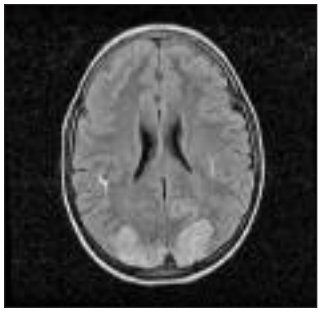
Visual symptoms in preeclampsia and eclampsia include decreased vision, photopsia, and visual field defects. The 3 most common visual complications of preeclampsia and eclampsia are hypertensive retinopathy, exudative retinal detachment, and cortical blindness. Possible explanations for these complications include coexisting or preexisting systemic vascular disease, changes in hormonal milieu, endothelial damage, abnormal autoregulation, hypoperfusion ischemia, or hyperperfusion edema. Each of these 3 visual complications is discussed in more detail below.

Hypertensive retinopathy is the most common ocular manifestation of preeclampsia and eclampsia, occurring in 60% of patients.[15] Focal arteriolar spasm and narrowing is commonly seen and may be associated with secondary changes (eg, diffuse retinal edema, hemorrhages, exudates, and nerve fiber layer infarcts [cotton wool spots]). The degree of retinopathy usually correlates with the severity of preeclampsia,[16] as well as APGAR scores[16] and fetal mortality.[17] Arterial narrowing is reversible following pregnancy in the majority of patients.[17]

Exudative retinal detachment occurs in 1% of preeclamptic patients and up to 10% of eclamptic patients.[13,14,18] It is thought to be caused by choroidal ischemia.[19] IVFA shows delayed filling of the choriocapillaries with normal retinal vasculature,[20] as well as choroidal non-filling with late fluorescein extravasation into subretinal and subpigment epithelial spaces.[21,22] Retinal pigment epithelium (RPE) lesions, called Elschnig spots, may also be found in preeclamptic patients with choroidal infarcts[23]. The prognosis is good, with visual symptoms and RPE changes resolve spontaneously within weeks of delivery.[17,23] OCT is an effective, non-invasive tool for following the evolution of fundus changes in these patients.[24]

Cortical blindness refers to reduced vision from bilateral damage to any portion of the visual pathways posterior to the lateral geniculate nucleus. Eye examination is typically normal, including a normal pupillary light reflex. It occurs in up to 15% of preeclampsia and eclampsia.[25,26] It can present both ante- and postpartum,[26] lasting from several hours to several days. Other presenting symptoms include headache, seizures, and loss of consciousness. Investigations include computed tomography (CT) or magnetic resonance imaging (MRI) of the brain. CT reveals bilateral low-density lesions in the occipital cortex,[25,27-29] while MRI shows hyperintense signals on T2-weighted images (Figure 1) and hypointense signals on T1-weighted images in the occipital cortex. These findings are consistent with transient ischemic events as a result of cerebral edema.[26,28-30]

Figure 1: Axial FLAIR magnetic resonance (MR) image showing hyperintense signal changes in the occipital lobe bilaterally in a preeclamptic patient with cortical blindness



Currently, there are 2 competing theories for the cause of cerebral edema. One theory is that cerebral vasospasm induced by severe hypertension results in cerebral ischemia and, consequently, cytotoxic edema. The second theory is that acute hypertension induces a loss of autoregulation, causing passive dilatation of cerebral arterioles, as well as an increase in vascular permeability, and consequently, vasogenic edema.[25] Diffusion-weighted imaging (DWI), utilizing the diffusion properties of water molecules, along with apparent diffusion coefficient (ADC) mapping, which measures the degree of translational freedom of water molecules, have been used to determine the etiology of cerebral edema.[31,32] Cytotoxic edema displays hyperintense signals on DWI and hypointense signals on ADC map. On the other hand, vasogenic edema displays variable signal intensities on DWI and hyperintense signals on ADC map.[33] To date, both vasogenic[31,32] and cytotoxic[33] edema have been observed in patients with cortical blindness.

Management of preeclamptic or eclamptic patients who develop cortical blindness is the same as for women without this visual complication, including magnesium sulfate for seizure prophylaxis, antihypertensives for severe hypertension, fluid restriction to avoid worsening of cerebral edema, ophthalmologic and neurologic consultation, as well as neuroimaging. Prompt delivery is curative, with resolution of neuroimaging findings.[25,30] Because acute visual changes may occur prior to eclamptic seizures, any visual loss in patients with preeclampsia should be considered a sign of impending eclampsia.[34]

Pre-existing conditions that are altered over the course of pregnancy

Diabetic retinopathy

Diabetic retinopathy tends to progress during pregnancy,[15,35] but the exact mechanism for this is unknown. Risk factors for progression include coexisting hypertension or preeclampsia,[36,37] severity of retinopathy before conception,[38,39] duration of diabetes before conception,[37,38,39] poor glycemic control before conception,[36,40] rapid institution of glycemic control,[41,42] and changes in retinal blood flow.[43] Interestingly, no association was found between the progression of retinopathy and level of glycosylated hemoglobin.[38,40] The progression rates of diabetic

Table 1: Progression rates of diabetic retinopathy stratified according to baseline characteristics

Severity of retinopathy before pregnancy	Two-step or greater progression of retinopathy	Progression to proliferative retinopathy
No retinopathy	10.3%	0%
Microaneurysm	21.1%	0%
Mild nonproliferative retinopathy	18.8%	6.3%
Moderate or severe nonproliferative retinopathy	54.8%	29%

Modified from Chew et al, 1995[39]

retinopathy depend on the severity of retinopathy at baseline before pregnancy, as shown in Table 1.

Management of pregnant women with diabetic retinopathy depends on the severity of the disease at conception (Table 2).[15] In patients who plan to have children, certain measures should be taken prior to pregnancy to reduce progression. They include tight control of blood sugars before pregnancy[44] and laser photocoagulation for preexisting proliferative diabetic retinopathy.[44,45] Reversal of pregnancy-induced retinal changes are common postpartum;[37, 41,46] however, progression could occur for up to 1 year after delivery. These patients are also at risk of developing complications such as vitreous hemorrhage and retinal detachment if not treated.[47] It is, therefore, important to continue following these patients closely after delivery.

Benign intracranial hypertension

Benign intracranial hypertension (BICH) is defined as raised intracranial pressure (ICP), in the absence of an intracranial mass or enlargement of the ventricles due to hydrocephalus. It is characterized by papilledema, headache, elevated ICP without any focal neurologic abnormalities (with the exception of abducens palsy) in an otherwise healthy individual. BICH is more prevalent in women, especially in young obese women of reproductive age.[48] In the past, BICH was thought to be more common in pregnancy; however, this is not supported by recent evidence.[49,50] BICH usually presents in the first trimester (82%), but it can present at any time during pregnancy.[49] Just as in nonpregnant patients, BICH can be asymptomatic or it may cause headache or visual symptoms. Visual field defects are the most common visual disturbances, occurring in up to 31% of patients.[49]

Visual outcome in pregnant women with BICH is the same as for those who are not pregnant.[49,50] BICH does not have a major negative impact on pregnancy; patients with BICH during pregnancy have the same spontaneous abortion rate as the general population.[49] The decision to treat is based on symptoms. Medical treatment of BICH in pregnancy is the same as in nonpregnant patients with a few exceptions:

- caloric restriction and weight reduction should be avoided because of the adverse effects of ketosis on the fetus
- corticosteroids should be used with caution because they may cause low birth weight
- repeated lumbar puncture may cause spontaneous abortion
- electrolytes should be monitored closely when using diuretics.[49-51]

If medical treatment fails, surgical options include lumboperitoneal shunt and optic nerve sheath decompression, both of which have been shown to be safe in pregnancy.[52,53] Intraoperative and postoperative uterine fetal monitoring is advised. Therapeutic abortion to limit progression of disease is not indicated. Subsequent pregnancy does not increase the risk of recurrence of BICH above the risk of recurrence in any other women with BICH.[49]

Meningioma

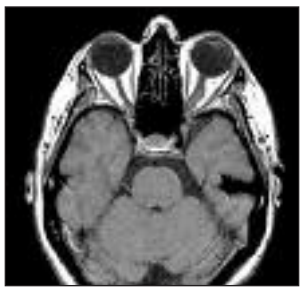
A meningioma is a benign, encapsulated, fibrous tumour that grows slowly, but it may ultimately compress and erode into adjacent structures. The clinical presentation of meningioma depends on its location. Orbital or sphenoid wing meningioma results in proptosis and compression of the optic nerve with monocular vision loss, ipsilateral relative afferent pupillary defect, optic disc swelling, and optociliary shunt vessels (Figure 2). Occipital lobe involvement produces a homonymous hemianopia. Olfactory groove involvement results in anosmia, ipsilateral optic atrophy, and contralateral papilledema (Foster-Kennedy syndrome). Meningioma in the cavernous sinus causes multiple cranial nerve palsies, including the oculomotor, trochlear, abducens as well as the first 2 divisions of the trigeminal nerves.

Table 2: Recommendations for monitoring of pregnant patients with diabetic retinopathy (DR)

First trimester	Second trimester	Third trimester
No DR		
• Dilated eye exam	• As needed for visual complaints	• As needed for visual complaints
Microaneuryms only		
• Dilated eye exam	• As needed for visual complaints	• As needed for visual complaints
Mild/Moderate NPDR		
• Dilated eye exam • Fundus photography	• Dilated eye exam • 1 x for mild • q4-6 wk for moderate	• Dilated eye exam q4-6 wk, more if needed
Preproliferative DR		
• Dilated eye exam • Fundus photography • Laser photocoagulation if severe	• Dilated eye exam q4-6 wk, more if needed • Laser photocoagulation if severe	• Dilated eye exam q4-6 wk, more if needed • Laser photocoagulation if severe
Proliferative DR		
• Dilated eye exam • Fundus photography • Laser photocoagulation	• Dilated eye exam • Fundus photography • Laser photocoagulation	• Dilated eye exam • Fundus photography • Laser photocoagulation

NPDR = non-proliferative diabetic retinopathy Modified from Schultz et al, 2006[15]

Figure 2: Axial T1-weighted MR image showing thickening of the left optic nerve consistent with an optic nerve sheath meningioma



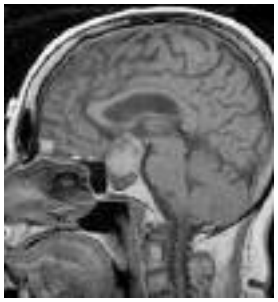
Meningiomas constitute 15%-20% of all intracranial tumours and they occur more often in women, with a female to male ratio of 3:1.[54] A relationship between meningioma and the reproductive cycle has been suggested because of the predominant presentation in women and the change in symptomatology during the menstrual cycle.[55] While its incidence does not increase in pregnancy,[56] meningioma demonstrates accelerated growth that may cause acute visual symptoms.[56,57] This may be attributed to the presence of progesterone and estrogen receptors in tumour cells. Over 70% of meningiomas express progesterone and androgen receptors, while fewer than 31% express estrogen.[56,58,59] However, it is unlikely that only progesterone is involved because the symptomatic meningioma growth is primarily confined to the second and third trimesters.[56] Meningioma has been found to remit postpartum.[56]

The management of a meningioma in pregnancy should be individualized and based on several factors, including location of tumour, tumour size, degree of visual loss, stage of pregnancy, viability of fetus, and the patient's desire to continue with the pregnancy. Because meningioma is generally resistant to radiation and chemotherapy, surgical excision remains the treatment of choice. If visual disturbances are mild and the pregnancy is close to term, no treatment is required since symptoms will resolve after delivery. Surgical management can then be considered after delivery. In the event of severe visual loss in a pregnancy close to term, the fetus should be immediately delivered by cesarean section, followed by surgical resection of the tumour.[60] For patients presenting with symptoms early in pregnancy, medical therapy (eg, steroids and hyperosmotic agents) can be used to reduce cerebral edema. This enables delaying the surgery until the fetus is sufficiently mature for delivery.[60] It is important to note that prolonged use of steroids has adverse effects on fetal growth and development. While hormonal therapy may have a role in the future management of unresectable meningioma,[54] it is not a viable option during pregnancy.

Pituitary tumour

Pituitary adenoma presents a potential risk for visual loss during pregnancy because the pituitary gland demonstrates physiologic growth during pregnancy. Autopsy studies demonstrate that the weight of the gland increases by 30% and the volume by 100% as a result of lactotrophic cellular hyperplasia.[61] This growth is corroborated by imaging studies that revealed a 45% increase in pituitary volume during the first trimester[62] and up to 136% at full-term.[63] Patients with microprolactinomas – defined as adenomas <1.0 cm – rarely exhibit visual disturbances (<2.3%)[64-66] and only a few exhibit asymptomatic enlargement (<4.5%).[64,65] However, patients with macroprolactinomas, defined as adenomas >1.0 cm (Figure 3), are at risk of clinically significant enlargement; up to 15.5%-23.3%[64-66] demonstrated symptomatic enlargement despite treatment with pregestational bromocriptine, 2.8% demonstrated symptomatic enlargement after prior radiation or surgical treatment,[67] and 8.9% demonstrated asymptomatic growth.[67]

Figure 3: Sagittal T1-weighted MR image showing a pituitary adenoma with internal apoplexy and compression of the optic chiasm



Headache is usually the first presenting symptom, followed by progressive visual field disturbances.[68] Typically, with a centrally-fixed optic chiasm, bitemporal hemianopia is most commonly seen, especially superiorly. Homonymous hemianopia can also be seen in advanced cases.[69,70] Other ophthalmic abnormalities include optic atrophy secondary to ischemia, as well as strabismus. Focal neurological signs such as cranial nerve palsies may also be present. Furthermore, harbouring an untreated pituitary adenoma appears to increase the risk of miscarriage, with an incidence of 27%.[71] For patients treated with bromocriptine early in pregnancy, the incidence of spontaneous abortion decreased to 7%, which is comparable to the rate found in normal women.[71] Untreated prolactinoma also increases the risk of prematurity.[72]

The management of pituitary adenoma in pregnancy depends on visual symptoms. Asymptomatic patients should have visual field testing every 3 months to monitor tumour growth and compression of the visual pathways.[69] Bromocriptine, a dopamine agonist, has been shown to inhibit prolactin production, decrease tumour volume and, consequently, reduce visual field defects.[73] Bromocriptine appears to be safe in pregnancy, with no increase in maternal or fetal morbidity or mortality.[74] Therefore, it may be given to asymptomatic patients as a preventative measure.[71] In symptomatic patients, in whom tumour expansion is suspected, confirmation can be made through MRI and visual field testing. Once confirmed, initiation of medical therapy (eg bromocriptine) is often indicated and these patients will likely require bromocriptine for the remainder of the pregnancy.[64,71] For patients who are not responsive to bromocriptine, other dopamine agonists, such as cabergoline, may be indicated. Recent studies on cabergoline reveal no significant complications in pregnancy. However, because there is much less safety data on cabergoline than on bromocriptine, it is not often used in pregnancy.[64] If visual field loss becomes severe or progressive despite medical therapy, trans-sphenoidal surgical decompression of the intracranial optic nerves and chiasm may be necessary during pregnancy.[64,71]However, surgery has been shown to

Table 3: Summary of commonly-used drugs during pregnancy

Drugs	Usage in pregnancy
Anti-infection drugs	
• Quinolone, gentamicin, erythromycin	• No teratogenic effect
• Acyclovir (HSV)	• No teratogenic effect
• Pyrimethamine and sulfadiazine (Toxoplasmosis)	• Pyrimethamine is potentially teratogenic but is still used by many; may use spiramycin instead
Anti-allergy drugs	
• Antihistamines	• No teratogenic effect
Corticosteroids	
• Systemic corticosteroids	• Systemic use — associated with infants with orofacial clefts, conotruncal heart defects, and neural tube defects
• Topical corticosteroids	• Topical use — no teratogenic effect
Anti-glaucoma drugs	
• Beta-blockers (eg, timolol)	• Associated with fetal cardiac arrhythmia and apnea • Teratogenic, especially in the first trimester
• Acetazolamide (glaucoma, BICH)	• Glaucoma literature reported neonatal teratoma, neonatal renal tubular acidosis and metabolic acidosis • Neuro-ophthalmic literature: no increased risk • Monitor blood level to prevent overdose and serious side-effects
• Travoprost (prostaglandin analogue)	• Probably contraindicated because prostaglandin can induce labour

increase the risk of abortion, with a 1.5-fold risk of fetal loss in the first trimester and a 5-fold risk of fetal loss in the second trimester.[75] After delivery, tumour reduction and decreased prolactin production have been described.[64] It is also important to note that breastfeeding does not increase the risk of tumour growth.[64]

Ophthalmic drugs

The use of ophthalmic medications during pregnancy presents potential risks to both the mother and the fetus. Unfortunately, limited data are available because of a lack of randomized controlled trials and meta-analyses. Most of the available evidence is based on individual case reports and animal studies. Table 3 summarizes the main published findings and may be used as a guide for commonly used ophthalmic drugs during pregnancy. As a general rule, the lowest possible dosage should be used. When using topical medications, nasolacrimal compression and temporary punctal occlusion could be performed to minimize systemic drug absorption. In situations that require the use of systemic or topical drugs that may have adverse side effects on pregnancy, consultation with the obstetrician before prescribing the medication is advisable.

Conclusions

Pregnancy may cause both physiologic and pathologic changes in the visual system. It may be associated with the onset of new diseases or it may alter the course of preexisting diseases. Recognizing the various visual symptoms and signs, as well as understanding the treatment strategies, are critical for the proper management of these patients. Caution should be exercised when prescribing ophthalmic drugs to pregnant women because of a lack of well-controlled studies in this area.

***Dr. Mark Bona** is an ophthalmology resident at Queen's University.*

***Agnes Wong, MD, PhD, FRCSC**, is an Associate Professor of Ophthalmology & Vision Sciences, Neurology, and Otolaryngology – Head & Neck Surgery, University of Toronto, and a Staff Ophthalmologist at the Hospital for Sick Children and Toronto Western Hospital*

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Department of Ophthalmology and Vision Sciences, University of Toronto

Upcoming events

October 13, 2007 International Ocular Blood Flow Symposium
Sutton Place Hotel, Toronto
Director: Dr. Neeru Gupta.
Contact: University of Toronto CME office – 416-978-2719

November 10-13, 2007 AAO, New Orleans, Louisiana

November 23-24, 2007 Department Walter Wright Program
The Revealing Retina
The Old Mill, Toronto
Course director: Dr. David Chow
Contact: U of T CME office – 416-978-2719

Note: This year's VPP rounds will be held at Mount Sinai Hospital, 18th Floor Auditorium, 600 University Avenue, Toronto. 5:30PM – 7:30PM.

Upcoming meeting

10-13 November 2007
American Academy of Ophthalmology
111th Annual Meeting
New Orleans, Louisianna
Contact: www.aao.org

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