

Review article

Ocular changes in pregnancy

Garg P , Aggarwal P

Maharishi Markandeshwar Institute of Medical Sciences and Research, Mullana, Ambala,
Haryana, India

Abstract

Pregnancy is often associated with ocular changes which may be more commonly transient but occasionally, permanent. The ocular effects of pregnancy may be physiological or pathological or may be modifications of pre-existing conditions. Physiological changes include increased pigmentation around the cheeks, ptosis, changes in cornea and refractive status, decreased intraocular pressure. These usually resolve post partum. Pre-existing diseases such as Graves' disease, Retinitis pigmentosa, Optic neuritis, should be monitored due to their remission or relapses in pregnancy. There may be worsening of Diabetic retinopathy, and Central serous chorio-retinopathy with increased risk of Retinal detachment. Conditions like Glaucoma and Non infectious uveal inflammatory disorders may even improve transiently. Pre-eclampsia and eclampsia could result in hypertensive retinopathy, exudative retinal detachment and cortical blindness. Neuro-ophthalmological disorders such as venous sinus thrombosis, benign intracranial hypertension, pituitary adenoma, meningioma and optic neuritis should be kept in mind as differential diagnosis in pregnant women presenting with visual acuity loss, visual field loss, persistent headaches or oculomotor palsies. Use of ophthalmic drugs can affect fetal health during pregnancy.

Key-words: diabetes, intracranial tumors, ocular changes, pregnancy, pre-eclampsia and eclampsia

Introduction

Women undergo a tremendous number of changes, both systemic and ocular, throughout pregnancy. During pregnancy, physiological changes occur in the cardiovascular, hormonal, metabolic, hematologic, and immunologic systems (Thornburg et al 2000; Sunness, 1988). Hormonal changes are among the most prominent systemic changes in pregnant women. The placenta, maternal endocrine glands and the fetal adrenal glands combine their productivity to make a high-powered hormone factory. The immune state is suppressed, leaving the

pregnant woman more susceptible to serious immunological disorders (Sheth et al, 2001). By some of these mechanisms, pregnancy causes ocular changes which may be more commonly transient but occasionally, permanent. The ocular effects of pregnancy may be physiological or pathological or may be modifications of pre-existing conditions. Visual changes in pregnancy are common, and many are specifically associated with the pregnancy itself. (Dinn et al, 2003)

The ocular effects of pregnancy may be divided into:

- Ocular changes and disorders that develop during pregnancy.
- The effects of pregnancy on pre-existing eye disorders.

Received on: 14.03.2011

Accepted on: 12.07.2011

Corresponding address: Dr Punita Garg, Assistant Professor

Maharishi Markandeshwar Institute of Medical Sciences and Research, Mullana, Ambala, Haryana, India

E-mail: doc_punita@yahoo.com

- c. Disorders of the eye associated with pregnancy-related diseases.
- d. Neuro-ophthalmological changes in pregnancy.
- e. Disorders related to labor and delivery

This review gives an overview on the changes that can affect the ocular health and vision of a pregnant woman and during childbirth.

Ocular changes and disorders that develop during pregnancy

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 (Pritchard et al, 1985). Spider angiomas, a type of telangectasia, commonly develop during pregnancy on the face and upper body. All these external changes resolve postpartum (Sheth et al, 2001; Pritchard et al, 1985)

Ptoxis (drooping of the eyelids) has been reported during and after normal pregnancy (Sanke, 1984) 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. Undertaking a search for preexisting underlying conditions such as Grave's disease may help to clarify the diagnosis (Sunness et al, 2001).

Effects on the cornea, lens and refractive status of the eye

Corneal sensitivity has been found to decrease in the later part of pregnancy and it usually returns to normal by eight weeks postpartum (Riss et al, 1981). Corneal thickness increases due to edema which may result in a change in the refractive index of the cornea (Weinreb et al, 1988; Fatt et al, 1973). The patient will complain of blurred vision. Contact lens users may find it increasingly difficult to continue wearing during pregnancy (Sheth et al, 2001; Park et al, 1992). This is relatively common in the last trimester of pregnancy. Transient loss of accommodation has been seen during and after pregnancy. Accommodative insufficiency and paralysis have been documented in association with lacta-

tion (Duke-Elder, 1971). The patient should be aware of possible fluctuations in vision, and that glasses prescribed during this time may become unsuitable. Therefore, it is advisable that any changes in eyeglass prescriptions be postponed until several weeks postpartum. Corrective procedures such as laser refractive surgery are contraindicated during this time (Dinn et al, 2003). There may be reduced tear production due to disrupted lacrimal acinar cells which increases the possibility of dry eye, infection and localized trauma (Schechter et al, 2002).

Krukenberg spindles on the cornea have been observed early in pregnancy and they tend to decrease in size during the third trimester and postpartum. They are not accompanied by other findings of pigment dispersion, such as increased angle pigmentation and iris trans-illumination defects (Riss et al, 1981). 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.

Effect on intraocular pressure

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 (Horven et al, 1974; Phillips et al, 1972). The reduced IOP is likely due to an increase in the facility of outflow (Becker et al, 1952; Paterson et al, 1963) via one of several possible mechanisms, including increased uveoscleral outflow due to hormonal changes, decreased episcleral venous pressure (Wilke, 1975) and decreased pressure in the upper extremities. Consequently, pre-existing glaucoma has been reported to improve during pregnancy (Phillips et al, 1972).

Ocular migraine

Migraine headaches are a common occurrence, affecting nearly 5 % of the general population. There is a greater prevalence of migraine headaches among women, suggesting that hormone levels es-



pecially estrogen influence the occurrence, frequency, and severity of migraine attacks. These changes cause a change in the blood flow of the body, which directly impacts on the “neurological” activities of the body. There may be a change in the frequency of migraine attacks during pregnancy. Both increased and decreased frequencies have been noted (Callaghan, 1968).

Changes in the visual field

There are conflicting reports on changes in the visual field. The classical field change is a bi-temporal hemianopsia. However, visual field changes varying from slight temporal or concentric contraction to complete homonymous hemianopsia have been reported (Brewington et al, 1974). 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. However, pregnant women with symptomatic visual field loss warrant further investigations.

Effects of pregnancy on pre-existing eye disorders

Disorders of uveal tissue – Non infectious 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 (Sunness et al, 2001). Postpartum endogenous candidal endophthalmitis, presumed to be related to intravascular dissemination around the time of delivery, has been reported (Cantrill et al, 1980).

Ocular toxoplasmosis

Primary infection during pregnancy may result in a congenital infection. Latent ocular toxoplasmosis may reactivate during pregnancy in the mother. Toxoplasmic retino-choroiditis is the most common cause of posterior uveitis in immunocompetent patients (Pleyer et al, 2007; Bonfioli et al, 2005). In adults decreased vision and floaters are most frequently reported. Active toxoplasmic retinochor-

oiditis typically presents as grey-white retinal necrosis with choroiditis, vasculitis and vitritis. However, atypical presentations including neuro-retinitis, papillitis, Fuch’s like anterior uveitis, scleritis and acute retinal necrosis have been described. Women with active infection during pregnancy should be monitored every three months by screening and their offspring followed up systematically (Bonfioli et al, 2005).

Uveal tumors

Recent evidence suggests that estrogen and progesterone do not have any role in the development or progression of uveal melanomas. Choroidal haemangiomas have been reported to undergo rapid growth during pregnancy but some can regress postpartum (Sunness et al, 2001; Foss et al, 1995).

Retinal disorders

In cases of retinitis pigmentosa, evidence of progression in some cases is seen. It does not always progress uniformly. There can be periods of more rapid worsening alternating with periods of relatively little change. It, therefore, becomes difficult to interpret whether changes reported are merely coincidental or truly related to pregnancy (Sunness et al, 2001).

Diabetic retinopathy: Diabetic retinopathy is the leading cause of blindness between the age of 24-64 years and half of this period corresponds to peak fertility and child-bearing years (Maayah et al, 2001). Pregnancy is a major risk factor for the progression of diabetic retinopathy and is definitely associated with increased prevalence and severity of retinopathy compared to non-pregnant diabetic women (Mallika et al, 2010). It is considered an independent risk factor for the development and progression of retinopathy. The presence of severe diabetic retinopathy may reflect the systemic status and has been considered a risk factor for adverse fetal outcome. Pregnancy is a prominent risk factor for the development and progression of retinopathy in women with insulin-dependent diabetes mellitus (IDDM) (Chen et al, 2004). They are encour-

aged to plan pregnancies early in life if possible. An important risk factor for the progression of Diabetic Retinopathy during pregnancy is the degree of retinopathy prior to conception. Progression was more significant in women with moderate and severe forms of retinopathy compared to women with mild or no retinopathy at conception (Chen et al, 2004). Multiple studies have demonstrated that a worsening of retinopathy in diabetic patients can occur during pregnancy (Impe, 2005).

In Diabetes in Early Pregnancy Study (DIEP), 54.8% of women with moderate to severe non-proliferative retinopathy demonstrated disease progression whereas in women with mild retinopathy 21.1% showed progression. Severe form of retinopathy changes is associated with poor peri-natal outcome and in women with severe proliferative changes before conception; pregnancy has to be deferred till the disease is treated (Mallika et al, 2010).

The American Academy of Ophthalmology recommends that the pregnant diabetic women should have an ophthalmological examination before conception to determine the baseline severity and then again during the first trimester. Subsequent examination should be every 3 months until delivery (Mallika et al, 2010; Impe, 2005)

Increasing severity of diabetic retinopathy has been shown to adversely affect outcome in pregnancy. Incidence of severe congenital malformations and/or foetal death is higher in patients with proliferative changes. Pregnancy does not cause any long term detrimental effects on the retina, kidney and peripheral nervous system. Retinopathy changes that have progressed during pregnancy have a tendency to regress after delivery (Mallika et al, 2010).

The progression of retinopathy in pregnancy depends on a variety of factors including severity of retinopathy at conception, adequacy of treatment, duration of diabetes, metabolic control before pregnancy, and the presence of additional vascular damage such as pre-existing or concomitant hyperten-

sive disorder (Best et al, 1997).

Management concerns that a diabetic women should have a pre-conception counseling and follow-up by multi-disciplinary team consisting of endocrinologist, ophthalmologist and perinatologist. Patients with severe non-proliferative and proliferative changes have a greater tendency for progression during pregnancy. So pregnancy should be deferred till the eye disease is treated and stabilized (Best et al, 1997).

If progression of the eye disease is noted during pregnancy, prompt laser photocoagulation is indicated in eyes with severe non-proliferative changes and should not be delayed till proliferative changes develop, because proliferative changes tends to progress despite photocoagulation in some eyes (Best et al, 1997).

Laser photocoagulation is the mainstay of treatment for DR with reasonable outcomes. Indications for surgery during pregnancy include: tractional retinal detachment, non-clearing vitreous haemorrhage and neovascular glaucoma. So as to conclude proper planning of pregnancy in young diabetic women and prompt laser photocoagulation of severe non-proliferative retinopathy can prevent serious sight threatening retinopathy (Mallika et al, 2010; Impe, 2005; Best et al, 1997).

Graves' disease

It is the most common cause of hyperthyroidism in pregnancy. It is an important cause of unilateral and bilateral proptosis. Pregnant women with Graves' orbitopathy are treated in a similar fashion to non-pregnant women. Graves' disease tends to remit late in pregnancy and relapse postpartum (Omoti et al, 2008). Rarely, the fetus can be affected because of transplacental passage of maternal IgG (Brown, 1996). Mild cases may be monitored, but moderate to severe cases must be treated. Thyroid inhibitors such as propylthiouracil, methimazole and carbimazole all cross the placenta and are excreted in breast milk, but the drug of choice in pregnant women is propylthiouracil (Brown, 1996).



Disorders of the eye associated with pregnancy-related diseases

1. Pre-eclampsia and eclampsia

Most common and most serious complications in pregnancy are seen in preeclampsia or fully developed eclampsia. Preeclampsia usually occurs after fifth month of pregnancy but may present anytime between 3rd and 9th month of pregnancy. Severity of retinal changes depends upon the degree of hypertension. 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 longterm neurologic complications. Prompt delivery of the fetus and placenta is the only cure (Cunningham et al, 1995). Retinal changes are likely to occur when diastolic BP is more than 100 mm of Hg and systolic BP is above 150 mm of mercury. Visual disturbances occur including scotoma, diplopia, diminished vision and photopsia. The three most common visual complications 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 (Cunningham et al, 1995).

Hypertensive retinopathy is the most common ocular manifestation of preeclampsia and eclampsia, occurring in 60% of patients. 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 (Tadin et al, 2001). Arterial narrowing is reversible following pregnancy in the majority of patients (Folk et al, 1981).

Exudative retinal detachment occurs in 1 % of pre-eclamptic patients and up to 10 % of eclamptic patients (Razai et al, 2004; Gass et al, 1991;

Sheth et al, 2001). It is thought to be caused by choroidal ischemia (Valluri et al, 1996). IVFA shows delayed filling of the choriocapillaries with normal retinal vasculature (Mabie et al, 1980) as well as choroidal non-filling with late fluorescein extravasation into sub-retinal and sub-pigment epithelial spaces (Sathish et al, 2000; Fastenberg et al, 1980). Retinal pigment epithelium (RPE) lesions, called Elschnig spots, may also be found in preeclamptic patients with choroidal infarcts (Saito et al, 1998). The prognosis is good, with visual symptoms and RPE changes resolve spontaneously within weeks of delivery (Sheth et al, 2001; Saito et al, 1998).

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 (Appollon et al, 2000; Cunningham et al, 1995). It can present both ante- and postpartum (Cunningham et al, 1995) lasting from several hours to several days. Other presenting symptoms include headache, seizures, and loss of consciousness MRI shows hyperintense signals on T2-weighted images 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 (Cunningham et al, 1995; Schwartz et al, 1992; Llovera et al, 2005; Do et al, 2002). Both vasogenic (Gregory et al, 2003; Schaefer et al, 1997) and cytotoxic (Na et al, 1994) edema have been observed in patients with cortical blindness.

Management includes magnesium sulfate for seizure prophylaxis, anti-hypertensives 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 (Appollon et al, 2000; Do et al, 2002). Acute visual changes may occur prior to eclamptic seizures, any visual loss in patients with pre-eclampsia should be considered a sign of impending eclampsia (Sharma et al, 2006).

HELLP syndrome

Approximately 10 % of women with severe pre-eclampsia develop the HELLP syndrome. It is characterized by haemolysis, elevated liver enzymes and low platelets. The syndrome is associated with poor maternal and fetal outcome. Ocular findings include bilateral serous retinal detachment with yellow/white sub-retinal opacities and sometimes vitreous hemorrhage (Sharma et al, 2006).

Disseminated intravascular coagulation (DIC)

Pregnancy is associated with a hyper-coagulable state and this can affect the retina and choroid. 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, leaving retinal pigment changes as a permanent feature (Sharma et al, 2006).

Thrombotic thrombocytopenic purpura (TTP) 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 edema (Sharma et al, 2006).

Other ocular findings such as retinal hemorrhages, exudates, sub-conjunctival hemorrhages, anisocoria (unequal pupils), motility disturbances, ischaemic optic neuropathy, homonymous hemianopia and scintillating scotoma may be noted (Sunness et al 2001).

Central serous retinopathy (CSR) is characterized by neuro-sensory retinal detachment, with associated retinal pigment epithelial (RPE) detachment, RPE leakage, as well as RPE and choroidal hyper-permeability. In pregnant woman it is often associated with sub-retinal exudation which is probably fibrinous in nature (Sunness et al, 1993; Khairallah et al, 1996). The affection resolves spontaneously at the end of pregnancy or after delivery, but may recur in the context or outside of subsequent pregnancy (Khairallah et al, 1996). The spe-

cial conditions of pregnancy, including haemodynamic, biological and psychological alterations may lead susceptible women to develop central serous chorioretinopathy. 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 (Razai et al, 2004).

4. Neuro-ophthalmic changes in pregnancy

Venous sinus thrombosis

Pregnancy and the puerperium have been recognised as periods of increased susceptibility to venous sinus thrombosis (Martin et al, 1941; Canu et al, 1993). Thrombosis of superior sagittal sinus or lateral sinus will result in increased intracranial tension leading to papilloedema. In case of lateral sinus and cavernous sinus thrombosis, it is unilateral to begin with but can become bilateral with the spread of thrombosis to opposite side which is quite common. Patient may present with clinical signs of Cavernous sinus thrombosis such as Proptosis, Chemosis, Oedema of eyelids; III, IV and VI cranial nerves palsy leading to ptosis and complete external ophthalmoplegia, a fixed and dilated pupil because of involvement of pupillary fibres. **Crowe's sign** - It is characterized by pressure on internal jugular vein of opposite side which will lead to engorgement of retinal veins. Homonymous hemianopia may be present. Cortical blindness may result by the extension of thrombosis by main sinus to the cortical veins of occipital lobe. The initial treatment should be intravenous heparin, with thrombolysis reserved for women who develop secondary deterioration.

Pituitary tumour

Pituitary adenoma presents a potential risk during pregnancy because the pituitary gland demonstrates physiologic growth during pregnancy. The weight



of the gland increases by 30 % and the volume by 100 % as a result of lactotrophic cellular hyperplasia (Foyouzi et al, 2004).

Patients with **micro-prolactinomas** – defined as adenomas <1.0 cm – rarely exhibit visual disturbances (<2.3 %). The patients with **macro-prolactinomas**, defined as adenomas > 1.0 cm, are at risk of clinically significant enlargement (Bronstein et al, 2002; Motlich et al, 1985; Albrecht et al, 1986). Headache is usually the first presenting symptom, followed by progressive visual field disturbances (Magyar et al, 1978). Bi-temporal hemianopia is most commonly seen. Homonymous hemi-anopia can also be seen in advanced cases (Bronstein et al, 1983; Halle et al, 1983). 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. Harbouring an untreated pituitary adenoma appears to increase the risk of miscarriage with an incidence of 27 % (Kupersmith et al, 1994). For patients treated with bromocriptine early in pregnancy, the incidence of spontaneous abortion decreased to 7 %.

Untreated prolactinoma also increases the risk of prematurity (Chiodini et al, 1981). Asymptomatic patients should have visual field testing every 3 months to monitor tumour growth and compression of the visual pathways (Bronstein et al, 2005). Bromocriptine, a dopamine agonist, has been shown to inhibit prolactin production, decrease tumour volume and, consequently, reduce visual field defects. Bromocriptine appears to be safe in pregnancy, with no increase in maternal or fetal morbidity or mortality (Krupp et al, 1987). Therefore, it may be given to asymptomatic patients as a preventative measure (Kupersmith et al, 1994). In symptomatic patients, in whom tumour expansion is suspected, confirmation can be made through MRI and visual field testing.

Despite medical therapy if visual field loss becomes progressive, trans-sphenoidal surgical decompression of the intracranial optic nerves and chiasm may

be necessary during pregnancy (Bronstein et al, 2002; Kupersmith et al, 1994). After delivery, tumor reduction and decreased prolactin production have been described. It is also important to note that breastfeeding does not increase the risk of tumor growth (Bronstein et al, 2002).

Meningiomas constitute 15 %-20 % of all intracranial tumours and they occur more often in women, with a female to male ratio of 3:1 (Whab et al, 2003). While its incidence does not increase in pregnancy, meningioma demonstrates accelerated growth that may cause acute visual symptoms (Roelvink et al, 1987; Cushing et al, 1938). This may be attributed to the presence of progesterone and estrogen receptors in tumour cells. Meningioma has been found to remit postpartum (Roelvink et al, 1987).

The management of a meningioma in pregnancy should be individualized and based on several factors including location of tumor, tumor size, degree of visual loss, stage of pregnancy, viability of fetus, and the patient's desire to continue with the pregnancy. Surgical excision remains the treatment of choice.

For mild visual disturbances and the pregnancy is close to term, no treatment is required. In the event of severe visual loss in a pregnancy close to term, the fetus should be immediately delivered by caesarean section, followed by surgical resection of the tumour. 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 (Wan et al, 1990).

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. BICH usually presents in the first trimester, but it can present at any time during pregnancy. It can be asymptomatic or it may cause headache or visual symptoms. Visual field defects are the most common visual disturbances (Digre et al, 1984). Visual outcome in



pregnant women with BICH is the same as for those who are not pregnant (Digre et al, 1984; Huna-Baron et al, 2002). It does not have a major negative impact on pregnancy. Medical treatment of BICH in pregnancy is the same as in non-pregnant 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 (Digre et al, 1984; Huna-Baron et al, 2002; Bagga et al, 2005).

If medical treatment fails, surgical options include lumbo-peritoneal shunt and optic nerve sheath decompression, both of which have been shown to be safe in pregnancy (Shapiro et al, 1995; Rush J, 1980).

Optic neuritis and neuropathy

Optic neuritis may be caused by multiple sclerosis (MS). The rate of relapse in MS decreases during pregnancy, and it rises significantly during the first three months post partum (Confavreux et al, 1999; Vukusic et al, 2006). Pregnancy does not appear to be a period at greater risk for exacerbations but, on the contrary it seems to act, on the whole, as a protective event (Bernardi et al, 1999). Multiple sclerosis must be considered as an etiology for acute puerperal lactation-associated blindness when there is no clear anatomic or infectious cause (Retzliff et al, 2001). Optic neuritis may also occur because of deficiency of vitamin B complex (Hyperemesis gravidarum). This is due vitamin loss or insufficient intake. Transient nerve palsies may occur because of polyneuritis (Sadovnick et al, 1994).

5. Disorders related to labour and delivery

A retinopathy similar to Purtscher's retinopathy has been reported within 24 hours of childbirth. It is

characterized by widespread cotton wool spots with or without intra-retinal hemorrhages, which represents arteriolar obstruction. The woman may experience severe unilateral or bilateral visual loss (Blodi et al, 1990). Bilateral retinal arteriolar occlusions from amniotic fluid particles have been reported (Chang et al, 1984). High myopia or previous retinal detachment surgery are not considered contraindications to spontaneous vaginal delivery (Inglesby et al, 1990; Neri et al, 1985). If there is retinal detachment, assistance during labour (for example, caesarean section, forceps delivery) would be preferable (Inglesby et al, 1990).

Orbital varices and haematomas have been reported during labour or in the early postpartum period. These can be associated with pain and diplopia (Jacobson et al, 1988).

Sheehan's Syndrome

Pituitary apoplexy occurs as a result of ischemic pituitary necrosis due to severe postpartum hemorrhage. It may be rarely seen without massive bleeding or after normal delivery. It is a complication of pituitary adenomas because of the sudden increase in pituitary size from infarction or hemorrhage. This condition may present as a sudden onset of headache, visual loss, and/or ophthalmoplegia. It is one of the most common causes of hypopituitarism. Enlargement of pituitary gland, small sella size, disseminated intravascular coagulation and autoimmunity have been suggested to play a role in the pathogenesis of Sheehan's syndrome in women who suffer from severe postpartum hemorrhage (Kelestimur, 2003).

Role of Ophthalmic drugs

The use of ophthalmic medications during pregnancy presents potential risks to both the mother and the fetus. 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 (Chung et al, 2004).

- Anti-infection drugs like Quinolone, gentami-



cin, erythromycin Acyclovir, anti-allergy drugs like Antihistamines are non teratogenic, Drugs like Pyrimethamine and sulfadiazine is potentially teratogenic but is still used by many; spiramycin may be used.

- Systemic use of corticosteroids in pregnancy is associated with infants with orofacial clefts, conotruncal heart defects, and neural tube defects.
- Use of Acetazolamide reported neonatal (glaucoma, BICH) teratoma, neonatal renal tubular acidosis and metabolic acidosis Monitoring of blood level is essential to prevent overdose and serious side-effects.
- Topical drugs like xylocaine drops and dilating drops have no teratogenic effect; however systemic use of them has been associated with minor fetal malformations. Beta-blockers are associated with fetal cardiac arrhythmia and apnea and is having teratogenic effect, especially in the first trimester.

Conclusion

In pregnancy, recognizing the various visual symptoms and signs, as well as understanding the treatment strategies, are critical for 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. Knowledge of ocular changes in pregnancy can help to differentiate the physiological changes from ocular manifestation of systemic disease and diseases pertaining to the eye in a pregnant woman. Their careful evaluation is a must for mother's eye and fetal health.

References

Albrecht BH, Betz G (1986). Prolactin-secreting pituitary tumors and pregnancy. *Contemp Issues Endocrinol Metabol: Prolactinomas*; 2: 195-219.

Appollon K.M., Robinson J.N., Schwartz R.B., Norwitz E. (2000). Cortical blindness in se-

vere preeclampsia: computed tomography, magnetic resonance imaging, and single photon-emission computed tomography findings. *Obstet Gynecol*; 95: 1017-1019.

Bagga R, Jain V, Das PC, Gupta KR, Gopalan S, Malhotra S (2005). Choice of therapy and mode of delivery in idiopathic intracranial hypertension during pregnancy. *Med Gen Med*; 7(4): 42.

Becker B, Friedenwald JS (1952). Clinical aqueous outflow. *Arch Ophthalmol*; 50: 557-571.

Bernardi S, Grasso MG, Bertollini R, Orzi F, Fieschi C (1991). The influence of pregnancy on Relapses in multiple sclerosis: a cohort study. *Acta Neurol Scand*; 84:403-406.

Best RM, Chakravarthy U (1997). Diabetic retinopathy in pregnancy. *British Journal of Ophthalmology*; 81:249-251.

Blodi BA, Johnson MW, Gass JD, Fine SL, Joffe LM (1990). Purtscher's-like retinopathy after childbirth. *Ophthalmology*; 97: 1654-1659.

Bonfioli AA, Orefice F (2005) Toxoplasmosis. *Semin Ophthalmol*; 20: 129-141.

Brewington TE, Clark CC, Amin N, Venable HP (1974). The Effect Of Pregnancy On The Peripheral Visual Field. *Journal Of The National Medical Association*; 66(4):330-331.

Brodsky JB, Cohen EN, Brown BW Jr, Wu M.L, Whitcher C (1980). Surgery during pregnancy and fetal outcome. *Am J Obstet Gynecol*; 138: 1165-1167.

Bronstein MD, Salgado LR, Musolino NRC (2002). Medical management of pituitary adenomas: the special case of management of the pregnant woman. *Pituitary*; 5: 99-107.

Bronstein MD (2005). Prolactinomas and pregnancy. *Pituitary*; 8: 31-38.

Brown RS (1996). Autoimmune thyroid disease in pregnant women and their offspring. *Endocr Pract*; 2(1): 53-61.



Callaghan N (1968). The migraine syndrome in pregnancy. *Neurology*; 18: 197–199.

Cantrill HL, Rodman WP, Ramsay RC, Knobloch WH (1980). Postpartum Candida endophthalmitis. *JAMA*; 243: 1163–1165.

Canu C, Barinagarmenteria F (1993). Cerebral venous thrombosis associated with pregnancy and puerperium: a review of 67 cases. *Stroke*; 24: 1880–1884.

Chang M, Herbert WN (1984). Retinal arteriolar occlusions following amniotic fluid embolism. *Ophthalmology*; 91:1634–1637.

Chiodini P, Liuzzi A, Cozzi R et al (1981). Size reduction of macroprolactinomas by bromocriptine or lisuride treatment. *J Clin Endocrinol Metab*; 53: 737-743.

Chung CY, Kwok AKH, Chung KL (2004). Use of ophthalmic medications during pregnancy. *Hong Kong Med J*; 10(3): 191-195.

Confavreux C, Hutchinson M, Hours M, Cortinovis-Tourniaire P, Grimaud J, Moreau T (1999). Multiple sclerosis and pregnancy: clinical issues. *Rev Neurol (Paris)*; 155:186-191.

Cunningham FG, Fernandez CO, Hernandez C (1995). Blindness associated with preeclampsia and eclampsia. *Am J Obstet Gynecol*; 172:1291-1298.

Cushing HW (1938). *Meningiomas: their classification, regional behaviour, life history and surgical end results*. Springfield, Illinois: Charles C. Thomas.

Digre KB, Varner MW, Corbett JJ (1984). Pseudotumor cerebri and pregnancy. *Neurology* 34:721-729.

Dinn RB, Harris A, Marcus PS (2003). Ocular changes in pregnancy. *Obstet Gynecol Surv* 58: 137-144.

Do DV, Rismondon V, Nguyen QD (2002). Reversible cortical blindness in preeclampsia. *Am J Ophthalmology* 134(6): 916-918.

Duke-Elder S (1971) *System of Ophthalmology*; Vol. 7 St. Louis: CV Mosby, 703.

Fastenberg DM, Fetkenhour CL, Choromokos E, Schoch DE (1980). Choroidal vascular changes in toxemia of pregnancy. *Am J Ophthalmol*; 89: 362-368.

Fatt I, Harris MG (1973). Refractive index of the cornea as a function of its thickness. *Am J Optom Physiol Opt*; 50: 383-386.

Folk JC, Weingeist TA (1981). Fundus changes in toxemia. *Ophthalmology*; 88: 1173–1174.

Foss AJ, Alexander RA, Guille MJ, Hungerford JL, McCartney AC, Lightman S (1995). Estrogen and progesterone receptor analysis in ocular melanomas. *Ophthalmology*; 102:431–435.

Foyouzi N, Frisbaek Y, Norwitz ER (2004). Pituitary gland and pregnancy. *Obstet Gynecol Clin North Am*; 31: 873-882.

Gass JD (1991). Central serous chorioretinopathy and white subretinal exudation during pregnancy. *Arch Ophthalmol*; 109(5): 677-678.

Gregory DG, Pelak VS, Bennet JL (2003). Diffusion-weighted magnetic resonance imaging and the evaluation of cortical blindness in preeclampsia. *Surv Ophthalmol*; 48(6): 647-650.

Halle AA, Drewry RD, Robertson JT (1983). Ocular manifestations of pituitary adenomas. *Southern Med J*; 76(6): 732-735.

Horven I, Gjonnaess H, Kroese A (1974). Corneal indentation pulse and intraocular pressure in pregnancy. *Arch Ophthalmol*; 91: 92-98.

Huna-Baron R, Kupersmith MJ (2002). Idiopathic intracranial hypertension in pregnancy. *J Neurology*; 249:1078-1081.

Impe JV (2005). Pregnancy and Its Effect on the Progression of Diabetic Retinopathy. *MJM*; 8(2):142-148.



- Inglesby DV, Little BC, Chignell AH (1990). Surgery for detachment of the retina should not affect a normal delivery. *BMJ*; 300:980.
- Jacobson DM, Itani K, Digre KB, Ossoinig K.C., Varner MW (1988). Maternal orbital hematoma associated with labor. *Am J Ophthalmol*; 105:547-553.
- Kelestimur F (2003). Sheehan's syndrome. *Pituitary*; 6:181-188.
- Khairallah M, Nouira F, Gharsallah R, Chachia N (1996). Central serous chorioretinopathy in a pregnant woman. *J Fr Ophtalmol*; 19:216-221.
- Krupp P, Monka C (1987). Bromocriptine in pregnancy: safety aspects. *Klin Wochenschr*; 65: 823-827.
- Kupersmith MJ, Rosenberg C, Kleinberg D (1994). Visual Loss in pregnant women with pituitary adenomas. *Ann Intern Med*; 12(7): 473-477.
- Llovera I, Roit S, Johnson A, Sherman L (2005). Cortical blindness, a rare complication of preeclampsia. *J Emerg Med*; 29(3): 295-297.
- Maayah J, Shammass A, Haddadin A (2001). Effect of Pregnancy on Diabetic Retinopathy. *Bahrain Med Bull*; 23(4):163-165.
- Mabie WC, Ober RR (1980). Fluorescein angiography in toxemia of pregnancy. *Br J Ophthalmol*; 64: 666-671.
- Magyar DM, Marshall JR (1978). Pituitary tumors and pregnancy. *Am J Obstet Gynecol*; 132:739-748.
- Mallika PS, Tan AK, Aziz S, Asok T, Syed Alwi SAR, Intan G. (2010). Diabetic retinopathy and the effect of pregnancy. *Malaysian Family Physician*; 5(1): 2-5.
- Martin J, Sheehan H (1941). Primary thrombosis of cerebral veins (following childbirth). *Br Med J*; 1:349-353.
- Motlich ME (1985). Pregnancy and hyperprolactinemic woman. *N Engl J Med* 312: 1364-1370.
- Na SJ, Hong JM, Park JH, Chung TS, Lee K.Y. (2004). A case of reversible postpartum cytotoxic edema in preeclampsia. *J Neurol Sci*; 22: 83-87.
- Neri A, Grausbord R, Kremer I., Ovadia J., Treister G. (1985). The management of labor in high myopic patients. *Eur J Obstet Gynecol Reprod Biol*; 19: 277-279.
- Omoti AE, Waziri-Erameh JM, Okeigbemen VW (2008). A Review of the Changes in the Ophthalmic and Visual System in Pregnancy. *Afr J Reprod Health*; 12(3): 185-196.
- Park SB, Lindahl KJ, Temnycky GO, Aquavella JV (1992). The effect of pregnancy on corneal curvature. *CLAOJ*; 18:256-259.
- Paterson GL, Miller SJH (1963). Hormonal influences in simple glaucoma. *Br J Ophthalmol*; 47:129-137.
- Phillips CI, Gore SM (1972). Ocular hypotensive effect of late pregnancy with and without high blood pressure. *Br J Ophthalmol*; 69:117-118.
- Pleyer U, Torun N, Liesenfeld O (2007). Ocular toxoplasmosis. *Ophthalmologie*; 104:603-616.
- Pritchard JA, MacDonald PC, Gran N.F. Williams Obstetrics. 17th ed. Norwalk, Conn. Appleton-Century-Crofts; 1985: 188-91.
- Razai KA, Elliott D (2004). Optical coherence tomographic findings in pregnancy associated central serous chorioretinopathy. *Graefes Arch Clin Exp Ophthalmol*; 242: 1014-1016.
- Retzliff M.G, Kobylarz E.J., Eaton C. (2001). Optic neuritis with transient total blindness during lactation. *Obstet Gynecol*; 98: 902-904.
- Riss B, Riss P (1981). Corneal sensitivity in pregnancy. *Ophthalmologica*; 183:57-62.
- Roelvink NC, Kamphorst W, Van Alphen HA, Rao BR (1987). Pregnancy-related primary brain and spinal tumors. *Arch Neurol*; 44: 209-215.

Rush J (1980). Pseudotumor cerebri. *Mayo Clin Proc* 55:541-546.

Sadovnick AD, Eisen K., Hashimoto SA, Farquhar R, Yee IM, Hooge J (1994). Pregnancy and multiple sclerosis - A prospective study. *Arch Neurol*; 51: 1120-1124.

Saito Y, Tano Y (1998). Retinal pigment epithelial lesions associated with choroidal ischaemia in pre-eclampsia. *Retina*; 18: 103-108.

Sanke RF (1984). Blepharoptosis as a complication of pregnancy. *Ann Ophthalmol*; 16: 720-722.

Sathish S, Arnold J (2000). Bilateral choroidal ischaemia and serous retinal detachment in pre-eclampsia. *Clin Exp Ophthalmol*; 28(5): 387-390.

Schaefer PW, Buonanno FS, Gonzalez GR, Schwamm LH (1997). Diffusion-weighted imaging discriminates between cytotoxic and vasogenic edema in patients with eclampsia. *Stroke*; 28: 1082-1085.

Schechter JE, Pidgeon M, Chang D, Fong YC, Trousdale MD, Chang N (2002). Potential role of disrupted lacrimal acinar cells in dry eye during pregnancy. *Adv Exp Med Biol*; 506:153-157.

Schwartz RB, Jones KM, Kalina P et al (1992). Hypertensive encephalopathy: findings on CT, MR imaging, and SPECT imaging in 14 cases. *AJR Am J Roentgenol*; 159:379-383.

Shapiro S, Yee R, Brown H (1995). Surgical management of pseudotumor cerebri in pregnancy: case report. *Neurosurgery*; 37(4): 829-831.

Sharma S, Wuntakal R, Anand A, Sharma TK, Downey G (2006). Pregnancy and the eye. *The Obstetrician & Gynaecologist*; 8: 141-146.

Sheth B, Mieler W (2001). Ocular complications of pregnancy. *Curr Opin Ophthalmol*; 12(6):455-463.

Somani S. Pregnancy special considerations.

www.emedicine.com/oph/topic747.htm.

Sunness JS, Haller JA, Fine SL (1993). Central serous chorioretinopathy and pregnancy. *Arch Ophthalmol*; 111:360-364.

Sunness JS, Santos A (2001). Pregnancy and the Mother's eye. In: *Duane's Clinical Ophthalmology on CD-ROM*. Philadelphia: Lippincott Williams & Wilkins.

Sunness JS (1988). The pregnant woman's eye. *Surv Ophthalmol*; 32: 219-238.

Tadin I, Bojic L, Mimca M, Karelovic D, Dogas Z (2001). Hypertensive retinopathy and pre-eclampsia. *Coll Antropol*; 25: 77-81.

Thornburg KL, Jacobson SL, Giraud GD, Morton MJ (2000). Hemodynamic changes in pregnancy *Semin Perinatol*; 24: 11-14.

Valluri S, Adelberg D, Curtis R et al (1996). Diagnostic indocyanine green angiography in preeclampsia. *Am J Ophthalmol*; 122: 672-677.

Vukusic S, Confavreux C (2006). Multiple sclerosis and pregnancy. *Rev Neurol (Paris)*; 62: 299-309.

Wan WL, Geller JL, Feldon SE, Sadun AA (1990). Visual loss caused by rapidly progressive intracranial meningiomas during pregnancy. *Ophthalmology*; 97: 18-21.

Weinreb RN, Lu A, Beeson C (1988). Maternal corneal thickness during pregnancy. *Am J Ophthalmol*; 105: 258-260.

Whab M, Al-Azzawi F (2003). Meningioma and hormonal influences. *Climacteric*; 6: 285-292.

Wilke L (1975). Episcleral venous pressure and pregnancy. *Acta Ophthalmol*; 125: 40-41.

Yung-Jen Chen, Hsi-Kung Kuo, Hsuan-We Huang. (2004). Retinal Outcomes in Proliferative Diabetic Retinopathy Presenting during and after Pregnancy. *Chang Gung Med J*; 27: 678-684.

Source of support: nil. Conflict of interest: none