Pregnancy is associated with various physiological and hemodynamic changes in the eye. An increase in corneal thickness and curvature and a decrease in corneal sensitivity are observed which may cause a temporary change in the refractive status. In the third trimester, there is a decrease in the intraocular pressure.

Though normal pregnancy is usually free of any significant retinal changes, a thorough retinal examination is warranted when a patient experiences ocular symptoms during the course of pregnancy. The important retinal conditions observed are worsening of pre-existing diabetic retinopathy, Preeclampsia and eclampsia associated with retinopathy (similar to hypertensive retinopathy), bullous retinal detachments, retinal pigment epithelium lesions and cortical blindness. Arterial and venous occlusive diseases are also not uncommon in these cases. Central serous chorioretinopathy and valsalva retinopathy may present during gestational period. Some cases of worsening of pre-existing non-infectious uveitis are also observed.

**Diabetic Retinopathy and Pregnancy**

Diabetic retinopathy is one of the major causes of preventable blindness in those aged between 24 and 64 years. Presumed mechanisms for worsening of retinopathy during pregnancy include changes in retinal hemodynamics and increased levels of various growth factors and hormones. Risk factors for the progression are poor metabolic control, duration of diabetes, baseline severity of diabetic retinopathy, pregnancy associated hypertension and preeclampsia, rapid normalization of glucose levels during pregnancy, and changes in retinal blood flow.

**Metabolic control**

The Diabetes in Early Pregnancy Study (DIEP), a prospective cohort study on 140 pregnant diabetic women showed that patients in whom retinopathy was most likely to progress had both the poorest control at baseline and the largest improvement during early pregnancy. However, it was impossible to separate these two risk factors as virtually all patients had improved metabolic control during early pregnancy.

**Duration of diabetes**

When the rate of development of retinopathy was compared in patients stratified by duration of diabetes, retinopathy progressed to proliferative levels in 39% of patients with more than 15 years of diabetes as opposed to 18% of patients with disease duration of less than 15 years.

**Baseline severity of retinopathy**

Risk of visual loss is low in those with no pre-existing retinopathy. According to the Diabetes in Early Pregnancy Study, 10.3% of women with no retinopathy and 21.1% of women with only microaneurysms had disease progression during or after pregnancy. Mild nonproliferative diabetic retinopathy (NPDR) (Fig 1B) progressed in 18.8% of diabetic women (6.3% to proliferative diabetic retinopathy (PDR), whereas moderate NPDR progressed in 54.8% of cases (29% to PDR) (Fig 1A). These findings indicate that severity of existing diabetic retinopathy profoundly influences the level of progression.

**Retinal blood flow**

Major changes in systemic vasculature are observed in pregnancy. The changes in the retinal blood flow are controlled by an autoregulatory mechanism in the retinal vasculature. A compensatory mechanism for the hyperdynamic circulation of the early pregnancy is present in both normal and diabetic women. Women with diabetes whose retinal blood flow remained unchanged developed no retinopathy. Those patients, in which the autoregulatory mechanism fails, will result in increased blood flow to the retinal vasculature and thereby inflict potential damage to endothelium at the capillary level.
Hypertension

Hypertension is a known risk factor for the progression of retinopathy and is additionally hazardous during pregnancy. In at least one major study, all patients with severe proliferative retinopathy also had proteinuria indicating a generalized vasculopathy.10

Other factors

Institution of rapid metabolic control will result in hypoglycemia which in turn causes retinal hypoxia and damage and the major changes observed are cotton wool spots and intra retinal microvascular abnormalities.6

Outcome

Some studies show that all of those with proliferative retinopathy at the start of pregnancy developed pregnancy induced hypertension or other obstetric complications resulting in fetuses with severe congenital malformations and/or fetal death.12 How ever, the long term consequences of diabetic retinopathy status during pregnancy does not show any deleterious effects attributable to pregnancy when compared to those who did not have progression during gestation.13

Management of diabetic retinopathy in pregnancy

Management of diabetic retinopathy depends on the severity of the disease at conception. The most important intervention is early education and good counselling of diabetic women in childbearing age. Ideally, patients should have good glucose control and diabetic retinopathy should be treated prior to conception.

Though regression of diabetic retinopathy is commonly seen during the postpartum period, some cases show progression for as long as 1 year postpartum with vitreous haemorrhage and retinal detachment complicating the condition. These complications along with neovascular glaucoma are associated with a worse visual outcome.15

Retinopathy in Pregnancy Induced Hypertension (PIH) (Preeclampsia/Eclampsia)

Since the early 19th century, retinal changes related to hypertension have been recognized. 25% of the patients with preeclampsia and 50% with eclampsia suffers visual

Tab1. Recommended management protocol for pregnant patients with diabetic retinopathy14

<table>
<thead>
<tr>
<th>Retinopathy prior to pregnancy</th>
<th>First trimester</th>
<th>Second trimester</th>
<th>Third trimester</th>
</tr>
</thead>
<tbody>
<tr>
<td>No DR</td>
<td>Dilated eye exam</td>
<td>As needed for visual complaints</td>
<td>As needed for visual complaints</td>
</tr>
<tr>
<td>Microaneurysms only</td>
<td>Dilated eye exam</td>
<td>As needed for visual complaints</td>
<td>As needed for visual complaints</td>
</tr>
<tr>
<td>Mild to moderate NPDR</td>
<td>Dilated eye exam Fundus photography</td>
<td>Dilated eye exam once for mild and every 4—6 weeks for moderate and severe NPDR (more frequently as needed)</td>
<td>Dilated eye exam every 4—6 weeks or more frequently as needed</td>
</tr>
<tr>
<td>Severe NPDR</td>
<td>Dilated eye exam Fundus photography Laser photocoagulation, if severe</td>
<td>Dilated eye exam every 4—6 weeks or more frequently as needed. Laser photocoagulation, if severe</td>
<td>Dilated eye exam every 4—6 weeks or more frequently as needed. Laser photocoagulation, if severe</td>
</tr>
<tr>
<td>Proliferative DR</td>
<td>Dilated eye exam Fundus photography Laser photocoagulation</td>
<td>Dilated eye exam Fundus photography Laser photocoagulation</td>
<td>Dilated eye exam Fundus photography Laser photocoagulation</td>
</tr>
</tbody>
</table>
disturbance including blurred vision, diplopia, photopsia, scotomata, amaurosis and chromatopsia and cortical blindness. The common ocular findings are focal constriction or spasm of the retinal arterioles leading to generalized narrowing, cotton-wool spots, intra retinal haemorrhages, retinal oedema, optic nerve oedema which resembles hypertensive retinopathy (Fig2). These findings in a patient with mild preeclampsia should raise suspicion about coexisting chronic diabetes and hypertension.

Putcher’s retinopathy developing after child birth has been reported in patients with preeclampsia and may be caused by compliment activated leukoembolus formation.

Choroidal involvement in PIH is marked by yellow-white focal lesions at the level of the retinal pigment epithelium (RPE), serous retinal detachment, and Elschnig’s spots (small, isolated areas of hyperpigmentation with surrounding yellow or red halos). The serous detachments are often bullous, and usually bilateral.

The prognosis for patients with visual disturbance associated with preeclampsia is good. Specific treatment for the ocular manifestations of PIH is not generally indicated. Systemic treatment of PIH consists of antihypertensive therapy, magnesium sulfate, and early delivery of the fetus when indicated.

Central Serous Chorioretinopathy (CSCR) in Pregnancy

This condition has a 10:1 male predominance outside the context of pregnancy. The etiopathophysiology is still not understood. Most cases occurred during the third trimester and spontaneously resolved during the early postpartum timeframe. There are reports of CSCR recurring in subsequent pregnancies. CSCR generally is unilateral with or without fibrin formation. Optical coherence tomography is the best non invasive diagnostic tool for CSCR in pregnancy which helps in the visualization of the retina, subretinal space, and retinal pigment epithelium without the risks of exposure of the fetus to fluorescein dye. Fortunately, CSCR itself is not associated with any adverse fetal outcomes. These patients are treated conservatively during pregnancy.

Other Retinal Manifestations in Pregnancy

Many women with non-infectious uveitis will experience a flare-up in disease activity within the first 4 months of pregnancy. Later pregnancy appears to be a time of relative disease inactivity. Many will experience a rebound in activity within 6 months of delivery. VKH and Behcet disease were the most frequent diagnoses. Of patients with active disease before pregnancy, most became inactive or at least less active by very early pregnancy. At 2 to 4 months of pregnancy, however, a flare-up was typical. Most flare-ups were effectively treated with corticosteroids.

Valsalva retinopathy is a unilateral or bilateral self-limiting condition that occurs when increased intra-thoracic or intra-abdominal pressure transmitted to the eye causes a sharp rise in the intra-ocular venous pressure, and rupture of superficial retinal capillaries (Fig4). Pregnancy is a known risk factor for Valsalva retinopathy, however, the diagnosis should be made only after excluding other causes of retinal haemorrhages.
Fundus photograph of a 29 year old primi showing valsalva retinopathy in her post partum period which resolved spontaneously in 3 months

Reference