Review Article

Pregnancy and retinal and retinal vascular complications

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Abstract: Pregnancy requires several changes in the body of the mother to successfully carry and deliver a child. Multiple alterations occur, including changes in cardiovascular system to meet the increased demands of the mother and placenta, the tilting of the hypercoagulable status to a more pro-coagulable state to prevent excessive blood loss post-delivery, and immunologic manipulations to protect the mother and fetus and decreasing the risk of a fatal immunologic response to the allogeneic fetus. These alterations are physiologically normal and expected, but can become pathologic when thresholds are crossed. Pregnancy may cause or exacerbate underlying retinal vascular diseases, a class of disorders compromised predominantly of retinal vein occlusion (RVO), retinal artery occlusion (RAO), central serous retinopathy (CSR), diabetic retinopathy, and hypertensive-related retinopathy, which includes pre-eclampsia, eclampsia, and hemolysis, elevated liver enzymes, and low platelets (HELLP) syndrome. The majority of the literature on retinal changes associated with pregnancy has focused on diabetic retinopathy, while the knowledge regarding the pathogenesis and treatment options of other pregnancy-related vascular diseases remains scarce. Understanding the implications pregnancy has on these rare, but severe, retinal vascular complications can help guide clinical management and potential treatment modalities. This paper aims to serve as a review of the retinal manifestations of diseases outside of diabetic retinopathy.

Keywords: Pregnancy; retinal vascular complications; central serous chorioretinopathy (CSCR)

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Introduction

Background

Pregnancy, a state in which the human body goes through physiologic changes in order to support the adapting mother and growing fetus, can come with systemic and ocular complications. These ocular manifestations can range from mild to visually destructive.

Rationale and knowledge gap

Out of protection for the fetus, more invasive studies are prohibited, which limits our knowledge of deeper pathophysiology, cost-benefits ratio of treatments, and certain treatment outcomes in pregnant patients.

Objective

This review article aims to highlight certain retinal vascular conditions more common in pregnancy, such as central or branch retinal artery occlusion (CRAO and BRAO, respectively), central or branch retinal vein occlusion (CRVO and BRVO, respectively), central serous retinopathy (CSR), pre-eclampsia related retinopathy and retinopathy secondary to hemolysis, elevated liver enzymes, and low platelets (HELLP) syndrome. Overall, the current regimens and outcomes are anecdotal and require the cooperation of hematologists, cardiologists, obstetricians,

and ophthalmologists.

Normal physiologic changes during pregnancy

Pregnancy induces several physiological changes in the mother to both nurture the developing fetus and help prepare the mother for labor and delivery. These include, but are not limited to: cardiovascular manipulations to meet the placental metabolic demands, the induction of a hypercoagulable state to prevent excessive blood loss post-delivery, and immunologic adaptations to protect the mother and fetus while avoiding a fatal immunologic response to the allogeneic fetus (1). These alterations are physiologically normal, but can become pathologic when protective thresholds are crossed.

Early on, the mother's cardiovascular system changes drastically: cardiac output increases around 40% due to an increase in stroke volume and heart rate, while peripheral vasodilation leads to a 25–30% decrease in systemic vascular resistance due to estradiol and nitric oxide upregulation (2). Despite the increase in plasma volume and cardiac output, pregnant women are physiologically hypotensive due to progesterone release, which relaxes the blood vessel walls and increases blood flow to mother and fetus. The coagulation system is altered during pregnancy to produce a hypercoagulable state to ensure hemostasis after delivery. Clotting factors such as factors VIII, IX, and X increase while endogenous anticoagulants such as antithrombin and protein S decrease (3). While the maternal immune system has not been fully understood, it's known that a balance between pro-inflammatory and anti-inflammatory changes leads pregnant women to being susceptible to infections (4).

Retinal vascular occlusion

Retinal vascular occlusions, which can occur in either arteries (RAOs), or veins (RVOs), typically occur in the fifth decade or later and are attributed to various systemic risk factors, such as hypertension, hyperlipidemia, and diabetes. Other risk factors include inherited disease states that predispose to clot formation, including, but not limited to: patent foramen ovale, antiphospholipid syndrome, factor V Leiden mutation, low protein S, and increased factor VIII (5). Although pregnancy occurs in ages younger than the fifth decade, the physiological changes associated with gestation can lead to spontaneous occurrences of RAOs and RVOs. The prevalence of retinal vascular occlusions is not well documented. Maurya *et al.*, report a 2-year prospective study at an outpatient setting for ophthalmology and for obstetrics and gynecology and found 16.36% of pregnant-related vascular disorders to be that of RVO (6). In another study by Park *et al.* over a 4-year period found out of the 1.8 million births, 33 pregnancies were complicated by RVO (7).

CRVO

The risk of RVO instances during pregnancy is reported to be between 0.05% and 1.8% and occurs most often in the third trimester (8). Patients typically present with acute, painless visual decline as the blocked, damaged vasculature leads to macular edema. Additionally, the lack of oxygenation induces a release of vascular endothelial growth factor (VEGF), with subsequent abnormal neovascularization, and in certain cases, vitreous hemorrhage or tractional retinal detachments (9).

A few reports exist in the literature detailing RVOs in pregnancy. Li *et al.* report a case of a 30-year-old woman who developed a bilateral CRVO at month 7 in her pregnancy (10). The patient was monitored but not treated. Post-partum, her sight returned to normal in both eyes at the 1-year mark. Several other reports have opted for observation as well (10-14). When treatment was initiated, therapies ranged from intravitreal corticosteroid injections, panretinal photocoagulation, and systemic administration of anti-coagulation (5,15-19).

RAO

Pregnancies with RAO are usually complicated with pre-eclampsia or hypercoagulability due to acquired or congenital deficiency in hemostatic factors. The incidence of RAO is also not well documented; in one retrospective case series of 147 patients who developed acute visual loss during the pregnancy, 4.1% (n=2) were due to RAO (20). Treatment of pregnancy-induced RAOs in the literature has mainly been anticoagulants and observation (5,11,12,18).

Interestingly, the development of a RAO in pregnancy has led to the diagnosis of a systemic condition. Kurtz *et al.* identified a unilateral BRAO during pregnancy which led to a previously undetected diagnosis of familial thrombophilia (11). The authors describe a 32-year-old, who presented with a BRAO at 13 weeks of gestation. She had no prior thrombotic events, though family history was significant for deep venous thrombosis and lethal pulmonary embolism in maternal grandmother. At the time of presentation, she was evaluated for thrombophilia-hypofibrinolysis and

revealed a protein S deficiency and a high factor VIII count. Likewise, Askim *et al.* describe the first case of a BRAO due to hereditary hemorrhagic telangiectasia (HHT) during pregnancy (21). In this case, a pregnant woman presented with sudden scotoma in her left eye. A work-up revealed HHT that ran through her family. A thoracic computed tomography (CT) and pulmonary angiography revealed a pulmonary arteriovenous malformation (PAVM). The authors propose that HHT should be screened for PAVMs prior to pregnancy; if already pregnant, the patient should undergo an echocardiography to confirm PAVM.

Central serous chorioretinopathy (CSCR)

CSCR is reported to be ten times more common in men, but pregnancy is a risk factor for CSCR development (8). CSCR is characterized by elevated endogenous cortisol and catecholamines leading to a permeable blood-retinal barrier (22). An accumulation of subretinal fluid leads to neurosensory retinal detachment in the macula at the level of the retinal pigment epithelium (RPE) (23).

In pregnant women specifically, CSCR can present with white subretinal exudation. One study reported white fibrous subretinal exudates are found in 90% of pregnancy-associated CSCR (pCSCR) compared to 20% of general cases (24). Risk factors during pregnancy include glucocorticoid use, Cushing syndrome, obstructive sleep apnea, hypertension, and emotional stress. In pregnancy in particular, elevated cortisol levels, preeclampsia, and hypertension are risk factors (25).

There are cases which document the spontaneous resolution of CSR after delivery (23,26).

However, there are also reports detailing permanent visual loss in patients. CSCR can also recurrently present in subsequent pregnancies and, over time, lead to subretinal fibrin-fibrosis and scarring of the macula (27,28).

Pre-eclampsia-complicated retinopathy

Pre-eclampsia is a leading cause of both maternal and fetal morbidity and mortality worldwide and is characterized by hypertension, proteinuria, and maternal organ dysfunction between mid-gestation to 6 weeks post-partum. Mild pre-eclampsia is reported to impact 0.5% of women and severe pre-eclampsia impacts 2–3% of women (29). Ophthalmologic complications associated with pre-eclampsia include cortical blindness, serous retinal detachment, Purtscher-like retinopathy, CRVOs, and retinal or vitreous hemorrhages (30). Most reports document the improvement of vision after delivery but in some cases, Purtscher-like retinopathy resulted in permanent vision loss (31,32).

While vision changes can be transient in the settings of pre-eclampsia, some courses of pregnancy can lead to permanent vision loss as was the case in Kotlyar *et al.* (33). A 27-year-old primigravida patient presented at 31 weeks with pre-eclampsia. She was treated with hydralazine and labetalol to maintain blood pressure control. Unfortunately, after delivery, she developed decreased visual acuity that was limited to counting fingers. Fundus exam showed diffuse retinal whitening, optic nerve pallor, and retinal hemorrhages. At 3-month follow-up, the patient had visual acuity limited to 20/150 and 20/250.

HELLP-complicated retinopathy

HELLP syndrome is a pregnancy complication that usually presents in women with underlying pre-eclampsia or eclampsia. Risk factors are shared with pre-eclampsia and include pre-existing hypertension or diabetes, advanced maternal age. The pathophysiology of Purtscher-like retinopathy is not well understood in the pregnant state. In the setting of HELLP syndrome, it is thought chronic inflammation of the placenta may cause complement activation leading to neutrophil and leukocyte embolization. Interestingly, in 15% of HELLP patients, no signs of pre-eclampsia are present (34).

Much like the rare case of Purtscher-like retinopathy in the setting of pre-eclampsia, Stewart *et al.*, report on a 25-year-old primigravida patient who developed Purtscher-like retinopathy in the setting of HELLP syndrome (35). Unfortunately, her vision remained impaired on follow up, with fundus exam showing severe optic atrophy and retinal thinning. In another instance, a 23-year-old primigravida patient developed HELLP syndrome and 2 days after delivery, noticed diminution of her vision. A fundus exam revealed bilateral serous detachment involving the macula. Her vision recovered completely 3 weeks post-partum (36).

HELLP syndrome has also been documented in the setting of posterior reversible encephalopathy syndrome (PRES). In this case, a 26-year-old pregnant female developed acute, bilateral vision loss due to serous retinal detachments and cortical vision loss during her post-partum period (37). She also developed systolic blood pressure in the 200s and laboratory tests showed thrombocytopenia, diffuse intravascular coagulopathy, and elevated liver

function tests. She continued to deteriorate with worsening headaches and acute encephalopathy. HELLP syndrome was diagnosed and subsequent magnetic resonance imaging (MRI) confined vasogenic cerebral edema and PRES. Unfortunately, her visual acuity never improved.

Disseminated intravascular coagulation (DIC)-complicated retinopathy

DIC itself is associated with pregnancy complications such as pre-eclampsia, HELLP syndrome, placental abruption, retained stillbirth, and amniotic fluid embolism. Pregnancy-associated DIC secondary is estimated to be around 1–5% of all pregnancies in countries in Western hospitals (38). Two reports in the literature detail vision loss in the context of DIC in pregnancy. In one case, vision improved, while in the other, it did not. Both cases presented with serous retinal detachments, which were thought to be secondary to intravascular coagulation occluding the choriocapillaris in the poster pole, subsequently damaging the RPE-cells and their ability to supply the retina and subretinal space (39).

Amniotic fluid embolism-complicated retinopathy

Amniotic fluid embolism is a dangerous and life-threatening complication of pregnancy where the amniotic fluid enters the maternal pulmonary circulation shortly after giving birth and causes cardiorespiratory collapse. Risk factors include advanced maternal age, multiparity, trauma, and induction of labor (40). Amniotic fluid embolism occurs in 2 to 8 per 100,000 deliveries and is the second leading cause of peripartum maternal death in the United States (41). These amniotic fluid emboli can also occlude retinal arteries in pregnant patients, causing occlusion and serious vision consequences. Kim and Choi present a dramatic case of amniotic fluid embolism in a 28-year-old female who was admitted for an elective abortion at 16 weeks (42). Three hours later, the patient reported reduced vision in her left eye with initial best-corrected visual acuity (BCVA) was count fingers. Fluorescein angiography showed an occlusion in the infratemporal branch retinal artery. Three days later, two more arteries were occluded. The final BCVA never improved.

Current treatments

Out of ethical constraints for the well-being of the fetus, exploration of effective treatment during pregnancy are mostly anecdotal, leading to limitations on consensus for treatment. Anti-VEGF has growing literature supporting its use and is further discussed in the diabetic retinopathy section review paper (15,16,43).

Conclusions

One proposed intervention is laser photocoagulation, especially in the setting of neovascular complications which can arise from ischemia (8). Intravitreal corticosteroids are also well tolerated and can be used to target macular edema (16,17). Systemic anti-coagulants such as low-molecular-weight heparin (LMWH) have been used as well in an effort to lessen the burden of ischemic vascular injury, but the evidence for this is sparse. The literature also documents several cases of observation, as spontaneous resolution has been documented with time and/or after delivery. These treatments and outcomes are listed in *Table 1*.

Table 1 Pregnancy and retinal and retinal vascular complications

Authors	Number of patients	Demographics	Retinal findings (CRVO, BRVO, CRAO, BRAO, CSR, etc.)	Vision in impacted eye	Ocular complications (CME, NVE, etc.) or remarkable systemic findings	Treatment	Resolution with delivery	Final vision
Jürgens *et al*., 2022	3	Patient 1: 32 YO, 19 weeks	CLRAO w/non-ischemic CRVO	5/200	No	Acetylsalicylic acid (100 mg PO)	Vision improved, while the inner retina layers and superior half of optic disc showed atrophy	20/20
		Patient 2: 30 YO, 10 weeks; PFO identified during work-up	CLRAO	20/20	No	Acetylsalicylic acid (100 mg PO)	Vision unimpacted, while the inner retina layers on OCT revealed progressive atrophy	20/20
		Patient 3: 32 YO, 16 weeks	PAMM	Not available	Not available	Not available	Not available	Not available
Wester and Murray, 2010	1	26 YO, 26 weeks	Retinal AVM with multiple BRVOs	20/50-2	CME	Bevacizumab	No, poor visual and anatomical outcomes	6/200E
Li *et al*., 2019	1	30 YO, 7 months	CRVO bilaterally	20/63, 20/40	CME bilaterally	Not available	No, poor visual outcome	20/125, 20/200
Kurtz *et al*., 2016	3	Patient 1: 35 YO, 8 weeks	BRAO—left eye	Not reported	Not available	Enoxaparin 40 mg BID, switched to once a day	Not available	Not available
		Patient 2: 32 YO, 13 weeks	BRAO—right eye	Not reported	Not available	Enoxaparin 1.5 mg/kg per day	Not available	Not available
		Patient 3: 55 YO, 15 years post delivery	CRVO left eye	Not reported	Not available	–	Not available	Not available
Pokroy *et al*., 2022	1	39 YO, 7 weeks	CRVO right eye	20/200	CME	Deferred treatment until 2nd trimester: intravitreal dexamethasone implant at 16 weeks	Yes, visual recovery	VA of 20/30
Ozdamar *et al*., 2010	1	25 YO, 1 week post delivery	Combined CLRAO and incomplete CRVO of right eye	20/20, 20/20	Elevated D-dimer (1.64, NL is 0–0.5 mg/mL)	Not available	Not available	Not available
Mee *et al*., 2017	1	30 YO, 28 weeks	Bilateral CRVO	20/40, 20/40	NVE	Right eye treated with PRP	No, VA worsened	20/63
Gonzalvo *et al*., 2000	1	30 YO, 28 weeks	CRVO in right eye	CF, 20/200	Severe pre-eclampsia and HELLP	Not available	Symptoms worsened after 10 days after delivery	20/125

Table 1 (*continued*)

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Authors	Number of patients	Demographics	Retinal findings (CRVO, BRVO, CRAO, BRAO, CSR, etc.)	Vision in impacted eye	Ocular complications (CME, NVE, etc.) or remarkable systemic findings	Treatment	Resolution with delivery	Final vision
Bahar *et al*., 2022	1	36 YO, 36 weeks	CRVO with CLRAO	CF	Not available	LMWH 40 mg subcutaneously daily for 7 days	No, VA worsened	10/125
Zhang and Reddy, 2017	1	~30s YO (exact age unspecified), 20 weeks	Bilateral CRVO	20/50, 20/40	Protein S level decreased (61%), heterozygous for factor V Leiden deficiency	Not available	Patient lost to follow-up	Patient lost to follow-up
Al-Mujaini *et al*., 2008	3	Patient 1: 27 YO, 7 months	CSCR	20/200	Not available	No, spontaneous resolution of CSCR	Yes	20/20
		Patient 2: 28 YO, 7 months	CSCR	20/200	Not available	No, spontaneous resolution of CSCR	Yes	20/20
		Patient 3: 25 YO, 6 months	CSCR	20/180	Not available	No, spontaneous resolution of CSCR	Yes	20/20
Al-Mujaini and Wali, 2014	2	31 YO, 6 months	CSCR	20/25	RPE detachment	No, spontaneous resolution of CSCR	No	20/200
Maggio *et al*., 2015	1	35 YO	CSCR	20/20, 20/200	No	Not available	Yes	Worsened BCVA due to atrophy
Chakraborti *et al*., 2014	1	30 YO, third trimester	Bilateral CSCR	20/25, CF	Serous macular detachment	Not available	No	20/200
Hirji *et al*., 2010	1	37 YO, 5 days post delivery	CSCR	20/80	Not available	Not available	No	20/200
Normalina and Alias, 1998	1	39 YO, 30 weeks	CSCR	20/320, 20/200	Not available	Not available	Yes	Exact vision not disclosed
Lopez-Yang and Garcia, 2015	1	40 YO, 6 months pregnant	CSCR	20/30, 20/20	Not available	Single dose of 0.05 cc intravitreal bevacizumab in right eye on presentation	No	20/30, 20/20
Motulsky *et al*., 2018	1	32 YO, 31 weeks	CRVO	20/25, 20/200	Not available	Single dose of 4 mg triamcinolone acetonide intravitreal injection in left eye	No	20/20-2

Table 1 (*continued*)

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Pole *et al*., 2020	1	34 YO, 32 weeks	CSCR	20/30, 20/20	Serous neurosensory detachment	Not available	No	20/20
Komoto *et al*., 2019	1	31 YO, post C-section delivery at 37 weeks	PIH	HM, HM	Bilateral retinal detachment associated with SRFM	Not available	No, presented after delivery	20/25, 20/20
Van Rysselberge *et al*., 2022	1	25 YO, 5 months	HELLP	20/200, 20/20	Serous retinal detachments	Beta blockers and $MgSO_4$ protocol for HTN management, emergent C-section	No	20/20, 20/20
Tsui and Kolomeyer, 2022	1	36 YO, 4 days post emergent C-section	HELLP	Listed as bilateral blindness	Bilateral Purtscher-like retinopathy	Not available	Not available	Not available
Haque *et al*., 2021	1	23 YO, 2 days post emergent C-section	HELLP	Not available	Bilateral serous retinal detachment	Not available	Not available	Complete recovery
Lin *et al*., 2012	1	20 YO, 2 days post emergent C-section	HELLP	20/100, 20/200	Bilateral serous retinal detachment	Not available	Presented after delivery	20/25, 20/25
Velazquez-Villoria *et al*., 2019	1	36 YO, does not list start of symptoms	HELLP	HM, HM	Pigment epithelial detachments	Not available	Not available	20/25, 20/25
Jayaraj *et al*., 2020	1	29 YO, 4 hours after C-section at 37 weeks	Pre-eclampsia	CF, CF	Bilateral serous retinal detachment	Treatment for high blood pressure	Presented after delivery	20/30, 20/30
Raposo *et al*., 2020	1	37 YO, POD 1 at 36 weeks	Pre-eclampsia	Listed as decreased vision in both eyes	Bilateral serous retinal detachment	Enoxaparin and nifedipine	Presented after delivery	Recovery to baseline
Santoro *et al*., 2021	1	39 YO, 34 weeks 1 hour post delivery	Pre-eclampsia without HTN	20/400, 20/400	Bilateral serous retinal detachment	Treatment was deferred	Presented after delivery	20/20, 20/20
Gupta and Sheidow, 2019	1	33 YO, post-partum period	Pre-eclampsia	20/400, 20/200	Bilateral serous retinal detachment	Not available	Presented after delivery	Not available

Table 1 (*continued*)

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Authors	Number of patients	Demographics	Retinal findings (CRVO, BRVO, CRAO, BRAO, CSR, etc.)	Vision in impacted eye	Ocular complications (CME, NVE, etc.) or remarkable systemic findings	Treatment	Resolution with delivery	Final vision
Raval and Das, 2019	1	20 YO, 2 days post C-section	Severe pre-eclampsia	CF, CF	Diagnosis of bilateral Purtscher-like retinopathy with gross macular ischemia in preeclampsia secondary to antiphospholipid syndrome	Tapering dose of oral corticosteroid for 4 weeks	Presented after delivery	20/200, CF
Zamora *et al.*, 2022	1	37 YO, 34 weeks, post delivery	HELLP	20/50, 20/50	Bilateral serous retinal detachment	Diuretics for HTN	Presented after delivery	20/200, 20/200
Leff *et al.*, 1990	1	33 YO, 7 months	HELLP	VA was 20/30 RE, 20/100 LE	Vitreous hemorrhage, presumed NVE	Not available	Yes	20/30
Lanzetta, 1999	1	Age unspecified, post placental abruption and IUD at 34 weeks	Eclampsia and DIC	Not available	RPE rip	Not available	Presented after IUD	20/20, 20/20
Kim and Choi, 2000	1	28 YO, 16 weeks, post dilatation and curettage for elective abortion	BRAO secondary to amniotic fluid embolism	Listed as diminished, HM	Not available	Not available	No	HM
Hoines and Buettner, 1989	1	30 YO, 40 weeks post C-section	DIC secondary to placental abruption	Not available	Bilateral serous retinal detachment	Not available	Not available	Not available
Stewart *et al.*, 2007	1	25 YO, 38.5 weeks	HELLP syndrome	CF, CF	Bilateral Purtscher-like retinopathy	Not available	No	Permanent vision loss
Kini *et al.*, 2021	1	26 YO, post-partum	HELLP syndrome	20/50, 20/50	Not available	Not available	Not available	Not available
Patel *et al.*, 2005	1	40 YO, post-partum	PIH	CF, CF	DIC	Not available	Not available	20/200, 20/120

In cases where both eyes were impacted, the right eye VA is listed before the left eye. CRVO, central retinal vein occlusion; BRVO, branch retinal vein occlusion; CRAO, central retinal artery occlusion; BRAO, branch retinal artery occlusion; CSR, central serous retinopathy; CME, cystoid macular edema; NVE, neovascularization elsewhere; YO, years old; CLRAO, cilioretinal artery occlusion; PO, per os (taken by mouth); PFO, patent foramen ovale; OCT, optical coherence tomography; PAMM, paracentral acute middle maculopathy; AVM, arteriovenous malformation; BID, 2 times a day; VA, visual acuity; NL, normal; PRP, panretinal photocoagulation; CF, counting fingers; HELLP, hemolysis, elevated liver enzymes, and low platelets; LMWH, low-molecular-weight heparin; CSCR, central serous chorioretinopathy; RPE, retinal pigment epithelium; BCVA, best-corrected visual acuity; PIH, pregnancy-induced hypertension; HM, hand motion; SRFM, subretinal fibrin-like material; HTN, hypertension; RE, right eye; LE, left eye; IUD, intrauterine demise; DIC, disseminated intravascular coagulation.

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References

1. Abu-Raya B, Michalski C, Sadarangani M, et al. Maternal Immunological Adaptation During Normal Pregnancy. Front Immunol 2020;11:575197.
2. Mirshahi A, Feltgen N, Hansen LL, et al. Retinal vascular occlusions: an interdisciplinary challenge. Dtsch Arztebl Int 2008;105:474-9.
3. Soma-Pillay P, Nelson-Piercy C, Tolppanen H, et al. Physiological changes in pregnancy. Cardiovasc J Afr 2016;27:89-94.
4. Kourtis AP, Read JS, Jamieson DJ. Pregnancy and infection. N Engl J Med 2014;370:2211-8.
5. Jürgens L, Yaici R, Schnitzler CM, et al. Retinal vascular occlusion in pregnancy: three case reports and a review of the literature. J Med Case Rep 2022;16:167.
6. Maurya RP, Srivastav T, Chaudhary S, et al. Retinal vascular disorders during pregnancy: An observational study. Indian Journal of Obstetrics and Gynecology Research 2018;5:282-6.
7. Park SJ, Choi NK, Seo KH, et al. Retinal vein occlusion and pregnancy, pre-eclampsia, and eclampsia: the results from a nationwide, population-based study using the national claim database. PLoS One 2015;10:e0120067.
8. Errera MH, Kohly RP, da Cruz L. Pregnancy-associated retinal diseases and their management. Surv Ophthalmol 2013;58:127-42.
9. Noma H, Funatsu H, Yamasaki M, et al. Pathogenesis of macular edema with branch retinal vein occlusion and intraocular levels of vascular endothelial growth factor and interleukin-6. Am J Ophthalmol 2005;140:256-61.
10. Li W, Ma M, Hu B, et al. Spontaneous resolution of bilateral central retinal vein occlusion with cystoid macular oedema during pregnancy. Acta Ophthalmol 2019;97:e482-3.
11. Kurtz WS, Glueck CJ, Hutchins RK, et al. Retinal artery and vein thrombotic occlusion during pregnancy: markers for familial thrombophilia and adverse pregnancy outcomes. Clin Ophthalmol 2016;10:935-8.
12. Ozdamar Y, Gurlevik U, Acaroglu G, et al. Combined cilioretinal artery and partial central retinal vein occlusion in the postpartum period. Retin Cases Brief Rep 2010;4:99-101.
13. Gonzalvo FJ, Abecia E, Pinilla I, et al. Central retinal vein occlusion and HELLP syndrome. Acta Ophthalmol Scand 2000;78:596-8.
14. Zhang AY, Reddy AK. Bilateral Decreased Vision During Pregnancy. JAMA Ophthalmol 2017;135:807-8.
15. Wester ST, Murray TG. Retinal arteriovenous malformation presenting with retinal vein occlusion during pregnancy. Retin Cases Brief Rep 2010;4:112-5.
16. Pokroy R, Raskin E, Masarwa D. Central retinal vein occlusion with severe macular edema in early pregnancy. Retin Cases Brief Rep 2022;16:547-9.
17. Motulsky E, Zheng F, Liu G, et al. Swept-Source OCT Angiographic Imaging of a Central Retinal Vein Occlusion During Pregnancy. Ophthalmic Surg Lasers Imaging Retina 2018;49:206-8.
18. Bahar MM, Ghalandarpoor-Attar SN, Shabani A, et al. Idiopathic combined retinal vessels occlusion in a pregnant woman: a case report. J Med Case Rep 2022;16:191.
19. Loh MA, Abirami S, Chong MF. Bilateral central retinal vein occlusion in a pregnant lady. Med J Malaysia

2017;72:130-2.
20. Park YJ, Park KH, Woo SJ. Clinical Features of Pregnancy-associated Retinal and Choroidal Diseases Causing Acute Visual Disturbance. Korean J Ophthalmol 2017;31:320-7.
21. Askim AJ, Thrane AS, Giaever P, et al. Branch retinal artery occlusion as a clinical manifestation of hereditary haemorrhagic telangiectasia (Osler-Weber-Rendu syndrome) in pregnancy. Acta Ophthalmol 2018;96:197-9.
22. Daruich A, Matet A, Dirani A, et al. Central serous chorioretinopathy: Recent findings and new physiopathology hypothesis. Prog Retin Eye Res 2015;48:82-118.
23. Pole C, Gaw Stephanie SL, Tsui Irena I. Utility of optical coherence tomography angiography in pregnancy-associated central serous chorioretinopathy. Am J Ophthalmol Case Rep 2020;20:100979.
24. Sunness JS, Haller JA, Fine SL. Central serous chorioretinopathy and pregnancy. Arch Ophthalmol 1993;111:360-4.
25. Morikawa M, Cho K, Kojima T, et al. Risk factors for central serous chorioretinopathy in pregnant Japanese women. J Obstet Gynaecol Res 2017;43:866-72.
26. Chakraborti C, Samanta SK, Faiduddin K, et al. Bilateral central serous chorio-retinopathy in pregnancy presenting with severe visual loss. Nepal J Ophthalmol 2014;6:220-3.
27. Perkins SL, Kim JE, Pollack JS, et al. Clinical characteristics of central serous chorioretinopathy in women. Ophthalmology 2002;109:262-6.
28. Gawecki M, Groszewska E. Central serous chorioretinopathy during pregnancy complicated by systemic hypertension - a case report. Klin Oczna 2016;117:256-9.
29. Zeeman GG. Neurologic complications of pre-eclampsia. Semin Perinatol 2009;33:166-72.
30. Roos NM, Wiegman MJ, Jansonius NM, et al. Visual disturbances in (pre)eclampsia. Obstet Gynecol Surv 2012;67:242-50.
31. Raposo JTBV, Melo BCDS, Maciel NFBB, et al. Serous Retinal Detachment in Pre-eclampsia: Case Report and Literature Review. Rev Bras Ginecol Obstet 2020;42:772-3.
32. Jayaraj S, Samanta R, Puthalath AS, et al. Pre-eclampsia associated bilateral serous retinal detachment. BMJ Case Rep 2020;13:e238358.
33. Kotlyar A, Arepalli S, Rachitskaya AV, et al. Bilateral Purtscher-like retinopathy with profound macular ischemia and vision loss in severe pre-eclampsia. J Clin Gynecol Obstet 2016;5:97-9.
34. Weinstein L. Syndrome of hemolysis, elevated liver enzymes, and low platelet count: a severe consequence of hypertension in pregnancy. Am J Obstet Gynecol 1982;142:159-67.
35. Stewart MW, Brazis PW, Guier CP, et al. Purtscher-like retinopathy in a patient with HELLP syndrome. Am J Ophthalmol 2007;143:886-7.
36. Haque OI, Waris A, Rizvi SA. Bilateral serous retinal detachment: an unusual complication of HELLP syndrome. BMJ Case Rep 2021;14:e233282.
37. Kini AT, Tabba S, Mitchell T, et al. Simultaneous Bilateral Serous Retinal Detachments and Cortical Visual Loss in the PRES HELLP Syndrome. J Neuroophthalmol 2021;41:e60-3.
38. Levi M. Disseminated intravascular coagulation (DIC) in pregnancy and the peri-partum period. Thromb Res 2009;123 Suppl 2:S63-4.
39. Patel N, Riordan-Eva P, Chong V. Persistent visual loss after retinochoroidal infarction in pregnancy-induced hypertension and disseminated intravascular coagulation. J Neuroophthalmol 2005;25:128-30.
40. Kramer MS, Rouleau J, Baskett TF, et al. Amniotic-fluid embolism and medical induction of labour: a retrospective, population-based cohort study. Lancet 2006;368:1444-8.
41. Fong A, Chau CT, Pan D, et al. Amniotic fluid embolism: antepartum, intrapartum and demographic factors. J Matern Fetal Neonatal Med 2015;28:793-8.
42. Kim IT, Choi JB. Occlusions of branch retinal arterioles following amniotic fluid embolism. Ophthalmologica 2000;214:305-8.
43. Lopez-Yang CE, Garcia CA. Persistent unilateral central serous chorioretinopathy in a breastfeeding woman. BMJ Case Rep 2015;2015:bcr2014206719.

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