Serous Exudative Retinal Detachment in Pregnancy-induced Hypertensive Patients in Paholpolpayuhasena Hospital (Case report)

Wichai Reungwilaisup, M.D.

ABSTRACT : Pregnancy-induced hypertension (PIH) is condition known in the other names as toxemia of pregnancy, (pre)eclampsia and edematous, proteinuric and hypertensive (EPH) gestosis. The purposes of this report are to describe 2 unusual cases of serous exudative retinal detachment (SERD) in PIH and to determine the possible relationships between SERD, renal complications and PIH

In this series SERD, a characteristic of choroidal ischemia, was associated with HELLP syndrome, acute renal failure, and dyspnea, and was reabsorbed while the blood pressure was still high in the first patient, and was occurring with neither high blood pressure nor labor stress in the second patient. These suggest that choroidal ischemia is not directly caused by hypertension (HT). But they may share the same common mechanisms which may have versatilely ischemic choroido endothelio-coagulopathic, hepatohematopathogenic and angiotension-increasing regulatory effects with higher affinity to microvasculature in the kidneys and peripheral vessels than in the eyes or other organs. Hence, loss of proteins from the kidneys, swelling, and HT are more common features found in PIH than other manifestations. **Thai J Ophthalmol 2005 ; July-December : 19(2) : 195-203.**

Keywords : *Pregnancy induced hypertension, (PIH) Serous exudative retinal detachment, hemolysis, elevated liver enzymes and low platelets (HELLP) syndrom*

Introduction

Pregnancy-induced hypertension (PIH) is the condition referred to in the other names as toxemia of pregnancy, (pre) eclampsia and edematous, proteinuric and hypertensive (EPH) gestosis. Sometimes, these terms are classified as the sub-group of each others (i.e. (pre) eclampsia as the sub-group of PIH, and PIH as the sub-group of EPH gestosis, etc.). There are several ophthalmologic conditions reported in literature occurring associated with PIH, including retinopathy, serous exudative retinal detachment (SERD), cortical blindness, homonymous hemi-anopia, transient myopia, vitreous hemorrhage, retinal arterial occlusion, ischemic optic neuropathy, optic neuritis, ischemic papillophlebitis, peripheral neovascularization, and abnormal eye movements.[1-14] SERD is rare but serious condition. Some authors consider SERD as an indication for termination of pregnancy in PIH patients in order to preserve the mothers' vision[15-17] with the controversy about the fetal prognosis.[17-18] Nowadays, the pathogenic mechanism of SERD in PIH is partially understood because of its rarity and transientness. Choroidal ischemia is currently believed to

Department of Ophthalmology, Paholpolpayuhasena Hospital

be a cause of SERD.[15,19-33] However, the relation ship between choroidal ischemia and hypertension (HT) occurring in PIH patients is still undetermined.

Though recently, a case of thrombophilia associated with SERD in PIH was reported,[34] more cases of hemolysis, elevated liver enzymes and low platelets (HELLP) syndrome and renal failure in PIH were published. Some of them were associated with SERD.[10-1,35-6] Their relationship will be discussed further.

We reported 2 unusual cases of SERD in PIH the possible relationship between SERD, HELLP syndrome, renal complications and PIH was described. The first patient was associated with HELLP syndrome, acute renal failure and dyspnea which tended to be pulmonary edema. Her SERD resolved while her blood pressure was still high. The other case developed SERD before the delivery time while her blood pressure was normal.

Case 1

A 21-year-old Chinese-Thai woman, gravida II, abortion I, no hypertensive background, referred by an obstetrician, to the eye department of Paholpolpayuhasena hospital on April 28th, 2005, 4 days after the delivery at 38 weeks' gestation due to severe PIH, 3 days after the beginning of transient renal shut down, pulmonary edema and 1 episode of convulsion.

During prenatal period, she had more than 39 kg weight gained. She complained of headache, epigastric tightness, and mild blurring of vision in both eyes for 2 days before the admission to the obstetrics department of Paholpolpayuhasena hospital in the night of April 23th, 2005.

At admission time, her blood pressure was 210/150 mm Hg. Pretibial pitting edema with +4 proteinuria were also detected. The delivery by cesarean section was completed in that night, after treatment with magnesium sulfate, nifedipine and intravascular fluid. One viable 3,250 g male baby was born with Apgar scores of 9 points at 1 min and 10 points at 5 min after birth. Twenty-two hours after the operation, staying in ICU with sustained high blood pressure (146/94-184/136 mmHg), and treated with the repeated doses of 5 mg nifedipine sublingually, she developed acute dyspnea, renal shutdown with urine output of less than 8 mL/hr and had a generalized convulsion shortly after. She also had anemia, thrombocytopenia, jaundice, and impaired liver function. She was diagnosed as HELLP syndrome with renal failure with severe PIH. Treatment included phenobarbital, nifedipine, aldomet, ranitidine, sodamint, and acetaminophen orally, furosemide, diazepam, dexamethasone, 1 unit of group B Rh D positive packed red blood cells, 2 units of fresh frozen plasma intravenously and intravenous fluid therapy. Her conditions improved within a few weeks, except for HT that required antihypertensive medicine for a couple of months. Finally, all the medication could be tailed off.

Pertinent laboratory studies on different intervals performed at Paholpolpayuhasena hospital were shown in table 1.

On ophthalmologic examination, her vision was 20/400 in the right eye and 20/800 in the left eye, with normal ocular tension and normal external and anterior eye segment examinations in both eyes. The pupils measured 4 mm in size and reacted to light sluggishly. In-

Table 1 Pertinent laboratory studies of case 1 on different intervals performed at Paholpolpayuhasena hospital

	April 24th, 2005*	**April 26th, 2005**	**April 28th, 2005**	**April 29th, 2005**	**May 2nd, 2005**	**May 6th, 2005**
Hemoglobin (g/dL)	14.2	10.0	8.6	8.2		
Hematocrit (%)	42.1	28.7	24.9	23.6		
White blood cell count (/mL)**	13,300	28,000	25,300	16,400		
Platelet count (/mL)	45,000	64,000	134,000	174,000		
BUN (mg/dL)	31.3	79.1	113.3	111.1	77.9	38.0
Creatinine (mg/dL)	2.2	5.5	7.6	7.3	5.5	2.8
Urate (mg/dL)	7.5					
Na (mmol/L)	130.3	132.9	133.4	134.1	137.0	
K (mmol/L)	5.8	5.3	5.1	4.7	5.1	
Cl (mmol/L)	106.3	102.0	99.3	101.5	103.4	
HCO_3 (mmol/L)	13.2	12.9	16.4	17.3	17.8	
LDH (U/L)	4,632	2,021		766		
SGOT (U/L)	1,472	111				
SGPT (U/L)	274	92				
Alkaline phosphatase (U/L)		141				
Total bilirubin (mg/dL)		7.5				
Direct bilirubin (mg/dL)		4.0				
Serum albumin (g/dL)		2.1				
Serum globulin (g/dL)		2.5				
PT (s)		10.4				
PTT (s)		21.6				

*Dark brown serum was also noted.

**All were associated with predominated polymorphonuclear cells.

direct ophthalmoscopy of the fundi revealed bilateral large bullous SERD, sparing only a small area on the upper part of the maculae and some of the upper part of the peripheral retina with clear sub-retinal fluid. There was some pale yellow-white material in the lower peripheral retina. There was no retinal tear detected. No hemorrhage or retinal vasculopathy found in both eyes. She was given diclofenac topically, prednisolone and multivitamins orally. By the second postpartum week, her blood pressure was still high (160/100 mm Hg), her vision improved partially to 20/70 in her right eye and 20/40 in her left eye. Her SERD resolved with minimal hyperpigmentation remaining in both maculae. On the latest examination, 1 year after the delivery, her best corrected visual acuity was 20/20 in both eyes. The hyperpigmentation was unremarkable.

Case 2

A 30-year-old Thai woman, gravida III, para I, abortion I, arrived at the eye department of Paholpolpayuhasena hospital on May 2nd, 2005 at 36 weeks' gestation. She complained of blurred vision in both eyes for 1 week. She also had headaches, fainting episodes, pitting edema, +4 proteinuria, and more than 23 kg weight gain. She was excised a benign fibrocystic mass from her right breast a few years ago, with no recurrence of the mass. She had no hypertensive history and didn't have any labor pain at the time she arrived.

During prenatal period, her blood pressure was always normal (100/70-120/90 mm Hg). She once experienced threatened abortion at 7 weeks' gestation, which was self-improving after home-rest. No other serious problem was detected, except for some paresthesia of fingers without significant sensational impairment from 25-33 weeks' gestation.

On examination, her vital signs were normal. Her blood pressure was 100/70 mm Hg. She weighed 78 kg. with pretibial pitting edema in both legs. Her best corrected visual acuity was counting finger at 1 m in both eyes. She had normal ocular tension readings, normal external and anterior eye segment examinations. The pupils measured 4 mm in size and reacted to light actively. Indirect ophthalmoscopy of the fundi revealed bilateral large cyst-like bullous retinal elevations with clear subretinal fluid in both maculae. No exudate, hemorrhage or retinal vasculopathy was found in either eye. There was also some ascetic fluid detected. Shortly after her eye-examination, a blood pressure remeasurement was done. It was 140/70 mm Hg. She was hospitalized for closed observation.

After admission, her blood pressure started to rise up to 180/140 mm Hg, and the diagnosis of severe PIH was made. She underwent cesarean section with tubal resection 5 hours later, after treatment with magnesium sulfate, nifedipine and intravascular fluid. One viable 2000 g female baby was delivered with good Apgar scores (10 points at 1 and 5 min).

By the first postpartum day, her blood pressure declined to 150/107 mm Hg. She felt no headache and had better sight, but her visual acuity was still counting finger at 1 m. No convulsion occurred. Two days later, her blood pressure increased up to 192/127 mm Hg. She had worse vision. Indirect ophthalmoscopic reexamination of her fundi showed SERD, involving all the area of previous lesion with more extension peripherally in

both eyes. Sub-retinal fluid was clear. Optic discs were mild swelling with few peripapillary hemorrhages. No retinal tear was detected. She was given diclofenac topically and supplementary multivitamins orally. Her best corrected visual acuity improved partially to 20/70 in her right eye by the second postpartum week and 20/100 in her left eye by the eighth postpartum week. Her SERD resolved, with some remaining mottling hyperpigmentation in both maculae.

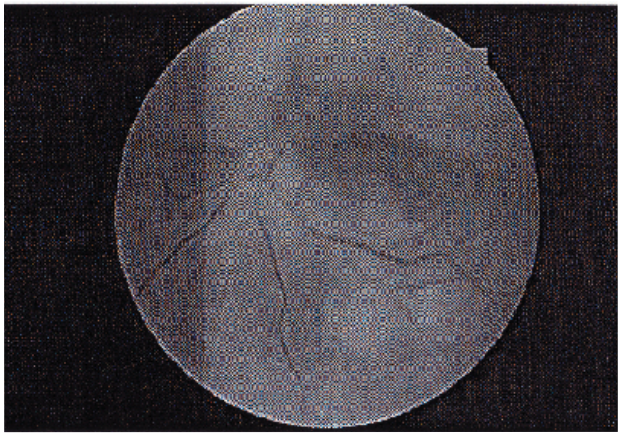

Fig. 1 Exudative retinal detachment in the right eye. PIH patients with SERD usually have bilateral bullous retinal detachment, without retinal break. Sometimes they have cyst-like SERD and may be confused with central serous choroidoretinopathy. (Courtesy of Dr. Apichart Singalavanija, Department of Ophthalmology, Siriraj Hospital)

Discussion

Several types of ophthalmic complication in PIH patients have been reported. These include the one associated with hypertensive retinopathy (HTR) such as changes of retinal vessels, exudates, and hemorrhages,[12,22,35-7] the one related to extra-ocular edematous nervous tissue such as cortical blindness, optic neuropathy,[1,3-6] and the one associated with choroidal ischemia such as SERD and its consequences. SERD is usually bilateral and bullous in appearance (as in figure 1) and some residual macular hyper-pigmentation and/or Elschnig spots are frequently reported. But sometimes it may be cyst-like appearance.

Previously, it was believed that ophthalmologic complications of PIH were the direct consequences of high blood pressure since most of the early publications revealed the association of HTR with patients who had ophthalmologic symptoms. However, current reports showed that PIH patients with no hypertensive history tended to have choroidal ischemic related complications rather than HTR.[15,38-39]

Clinical evidences from many reports of SERD in PIH patients demonstrated change in fundus fluorecein angiography, indocyanine green angiography and multifocal electroretinography, showed the choroidal ischemia as the cause of SERD.[15,19-23,40-1] But its relationship to HT is still unclear. Because most SERD patients usually presented to ophthalmologist after or at the same time PIH occurred,[14-5,22-3,32-3,36-7,42-3] it seemed that the very high blood pressure occurring in PIH patients was responsible for the vasospasm and choroidal ischemia.

In 1999, Poppe[44] et al reported the case of a 22-year-old, et al termed pregnant woman with blurred vision from SERD occurring a few hours after the normal delivery. Her blood pressure was 150/90 mm Hg with little proteinuria detected. They stated that the retinal detachment of a pregnant woman did not have to be proceeded by symptoms of toxemia of pregnancy and the period of delivery might accelerate and release mechanisms da-

maging choriocapillaris, which caused the flow of fluid from choroidal vessels to sub-retinal space.[44]

In this series, the first patient had her SERD resolved while her blood pressure was still high (160/100 mm Hg). This implicated the un-correlation between the re-absorption of sub-retinal fluid and the return within the normal range of blood pressure. In the second case, the patient had SERD while her blood pressure was normal. This suggested that choroidal ischemia was not directly caused by hypertension, even though they may share the same common mechanisms. Since HELLP syndrome possessed some characteristics of immunopathogenic condition such as microangiopathic hemolytic anemia and thrombocytopenia, was discovered in PIH patients, it was possible that those common mechanisms should involve the immune system. Furthermore, the common mechanisms may have versatilely ischemic choroidoendotheliocoagulopathic, hepatohematopathogenic, and angiotension-increasing regulatory effects, and they have tendency to possess higher affinity to microvasculature in the kidneys and peripheral vessels than in the eyes or other organs. Hence, loss of protein from the kidney, swelling, and hypertension were much more common than other manifestations in PIH patients.

To date, there is no concensus on standard treatment for SERD. Though SERD has some tendency to resolve itself after the termination of pregnancy, many medications have been also used, such as corticosteroids, antihypertensive agents, nutrients and osmotic agents. In 1998, Kokot reported the case of a 31-year-old pregnant woman with low serum protein and albumin who regained her bilateral retinal detachment reattached after parenteral administration of albumin. This suggested that protein and albumin loss in PIH also aggravated SERD and the parenteral albumin supplement might be helpful in SERD with PIH patients.[45]

Nevertheless, SERD in the second patient became larger after her blood pressure had increased. Therefore, hypertension could possibly cause more fluid leakage from choroidal vasculature to sub-retinal space after the development of choroidal ischemia. To reduce the chance of severe SERD in PIH patients, if the fetal circulation is not trouble-fully compromised, it may be beneficial to keep the maternal blood pressure within the normal range as soon as possible.

In summary, thanks for the more knowledge about SERD, PIH and all the related complications and their relationship we learned and for plenty of developments in the patients' care of such conditions we improved in the past centuries, both patients partially regained their vision back in some weeks after the delivery. But a lot much more about SERD, PIH and all the related complications' natures, preventions and treatments are what we have to study further in order to limit more of or to get rid of their perdition.

Acknowledgements

The auther would like to thank Dr. Jutalai Tanterdtham for her advice and Dr. Apichart Singalavanija for his suggestions in writing this article and for providing his valuable photographs since there was no fundus camera available in Paholpolpayuhasena hospital by the time the patients arrived.

Sincere thanks are extended to Dr. Prayongsri

Kampraphan and Dr. Orawan Tangtongpet, obstetrists who kindly supported the 2 cases, and to Dr. Chaowaree Reungwilaisup and Melissa A. Taavola for their lovely assistance in grammatical proofs.

REFERENCES

1. Sommerville-Lange LB. A case of permanent blindness due to toxemia of pregnancy. Br J Ophthalmol 1950 ; 34 : 431-4.
2. Schroter F. A rare case of severe pregnancy toxicosis with transitory myopia, exudative ablatio retinae and ascites. Geburtshilfe Frauenheilkd 1967 ; 27(3) : 298-301.
3. Beal MF, Chapman PH. Cortical blindness and homonymous hemianopia in the post partum period. JAMA 1980 ; 244 : 2085-7.
4. Beck RW, Gamel JW, Willcourt RJ, et al. Acute ischemic optic neuropathy in severe preeclampsia. Am J Ophthalmol 1980 ; 90 : 342-6.
5. Beeson JH, Duda EE. Computed axial tomography scan demonstration of cerebral edema in eclampsia preceeded by blindness. Obstet Gynecol 1982 ; 60 : 529-32.
6. Monteiro MLR, Hoyt WF, Imes RK. Puerperal cerebral blindness. Arch Neurol 1984 ; 41 : 1300-1.
7. Moodley J, Pillay M, Pillay R. Temporary blindness and eclampsia. A report of 2 cases. S Afr Med J 1985 ; 26 ; 68(9) : 677-8.
8. Jaffe G, Schatz H. Ocular manifestations of preeclampsia. Am J Ophthalmol 1987 ; 15 ; 103(3 Pt 1) : 309-15.
9. Brancato R, Menchini U, Bandello F. Proliferative retinopathy and toxemia of pregnancy. Ann Ophthalmol 1987 ; 19 :182-3.
10. Burke JP, Whyte I, MacEwen CJ. Bilateral serous retinal detachments in the H.E.L.L.P. syndrome. Acta Ophthalmol 1989 ; 67 : 322-4.
11. Leff SR, Yarian DL, Masciulli L, et al. Vitreous hemorrhage as a complication of HELLP syndrome. Br J Ophthalmol 1990 ; 74 : 498.
12. Iida T, Hagimura N, Otani T, et al. Choroidal vascular lesions in serous retinal detachment viewed with indocyanine green angiography. Nippon Ganka Gakkai Zasshi 1996 ; 100(10) : 817-24.
13. Roberts D, Haslett E, Hickey-Dwyer M, et al. Eclampsia complicated by bilateral retinal detachments and abnormal eye movements. Lancet 1998 ; 14 ; 351(9105) : 803-4.
14. Waziri-Erameh MJ, Omoti AE, Edema OT. Bilateral total loss of vision following eclampsia a case report. Afr J Reprod Health 2003 ; 7(2) : 106-8.
15. Bos AM, van Loon AJ, Ameln JG. Serous retinal detachment in preeclampsia. Ned Tijdschr Geneeskd 1999 ; 27 ; 143(48) : 2430-2.
16. Dornan KJ, Mallek DR, Wittmann BK. The sequelae of serous retinal detachment in preeclampsia. Obstet Gynecol 1982 ; 60(5) : 657-63.
17. Haddad A, Faleh R, Abdi A, et al. Serous retinal detachment in severe pre-eclampsia. Two case reports. Tunis Med 2002 ; 80(8) : 500-4.
18. Sihota R, Bose S, Paul AH. The neonatal fundus in maternal toxemia. J Pediatr Ophthalmol Strabismus 1989 ; 26(6) : 281-4.
19. Martin VA. Disseminated intravascular coagulopathy. Trans Ophthalmol Soc U K 1978 ; 98(4) : 506-7.
20. Fastenberg DM, Fetkenhour CL, Choromokos E, et al. Choroidal vascular changes in toxemia of pregnancy. Am J Ophthalmol 1980 ; 89(3) : 362-8.
21. Mabie WC, Ober RR. Fluorescein angiography in toxaemia of pregnancy. Br J Ophthalmol 1980 ; 64(9) : 666-71.
22. McEvoy M, Runciman J, Edmonds DK, et al. Bilateral retinal detachment in association with preeclampsia. Aust N Z J Obstet Gynaecol 1981 ; 21(4) : 246-7.
23. Sathish S, Arnold JJ. Bilateral choroidal ischaemia and serous retinal detachment in preeclampsia. Clin Experiment Ophthalmol 2000 ; 28(5) : 387-90.
24. Oliver M, Uchenik D. Bilateral exudative retinal detachment in eclampsia without hypertensive retinopathy. Am J Ophthalmol 1980 ; 90(6) : 792-6.
25. Gaudric A, Binaghi M, Coscas G. Acute choriocapillaris occlusion and Elschnig's spots during a toxemia of pregnancy. J Fr Ophtalmol 1981 ; 4(3) :223-9.
26. Combe JC, Boiron M, Lecoq PJ. Acute occlusion of the choriocapillaris in a case of pregnancy toxemia. Bull Soc Ophtalmol Fr 1982 ; 82(12) : 1557-63.
27. Gass DM, Pautler SE. Toxemia of pregnancy pigment epitheliopathy masquerading as a heredomacular dystrophy. Trans Am Ophthalmol Soc 1985 ; 83 : 114-30.
28. Kinyoun JL, Kalina RE. Visual loss from choroidal ischemia. Am J Ophthalmol 1986 15; 101(6) : 650-6.
29. Valluri S, Adelberg DA, Curtis RS, et al. Diagnostic indocyanine green angiography in preeclampsia. Am J Ophthalmol 1996 ; 122(5) : 672-7.
30. Kwok AK, Li JZ, Lai TY, et al. Multifocal electroretinographic and angiographic changes in preeclampsia. Br J Ophthalmol 2001 ; 85(1) : 111-2.
31. Ramaesh K, Nagendran S, Saunders DC. Choroidal ischaemia and serous retinal detachment in toxaemia

of pregnancy. Eye 1999 ; 13 (Pt 6) : 795-6.
32. Lee C, Hsu TY, Ou CY, et al. Retinal detachment in postpartum preeclampsia and eclampsia : report of two cases. Changgeng Yi Xue Za Zhi 1999 ; 22(3) : 520-4.
33. Prado RS, Figueiredo EL, Magalhaes TV. Retinal detachment in preeclampsia. Arq Bras Cardiol 2002 ; 79(2) : 183-6.
34. Maslovitz S, Lessing JB, Kupferminc MJ. Bilateral retinal detachment in preeclamptic women with thrombophilia. Int J Gynaecol Obstet 2005 ; 91(1) : 65-6.
35. Sibai BM, Ramadan MK, Usta I, et al. Maternal morbidity and mortality in 442 pregnancies with hemolysis, elevated liver enzymes, and low platelets (HELLP syndrome). Am J Obstet Gynecol 1993 ; 169(4) : 1000-6.
36. Sanchez Vicente JL, Ruiz Aragon J, Nanwani K, et al. Retinal detachment in preeclampsia and HELLP Syndrome. Arch Soc Esp Oftalmol 2003 ; 78(6) : 335-8.
37. Riss B, Skorpik C, Riss P, et al. Severe changes in the fundus oculi in pregnancy toxemia. 3 cases. Wien Klin Wochenschr 1983 ; 14 ; 95(19) : 692-5.
38. Saito Y, Omoto T, Kidoguchi K, et al. The relationship between ophthalmoscopic changes and classification of toxemia in toxemia of pregnancy. Acta Soc Ophthalmol Jpn 1990 ; 94 : 870-4.
39. Schreyer P, Tzadok J, Sherman DJ, et al. Fluorescein angiography in hypertensive pregnancies. Int J Gynecol Obstet 1990 ; 34 : 127-32
40. Valluri S, Adelberg DA, Curtis RS, et al. Diagnostic indocyanine green angiography in preeclampsia. Am J Ophthalmol 1996 ; 122(5) : 672-7.
41. Milkowski S. Bilateral retinal detachment in the course of pregnancy toxemias. Wiad Lek 1972 ; 1 ; 25(23) : 2167-71.
42. Chatwani A, Oyer R, Wong S. Postpartum retinal detachment. A case report. J Reprod Med 1989 ; 34(10) : 842-4.
43. Singalavanija A, Dangosintr N, Namatra C. Retinal detachment in toxemia of pregnancy. J Med Assoc Thai 1989 ; 72(10) : 597-600.
44. Poppe E, Sajkowska M, Bernacka I. Bilateral retinal detachment in toxemia of pregnancy. Klin Oczna 1999 ; 101 (3) : 209-11.
45. Kokot W, Raczynska K, Mielnik J. The effect of total protein and albumin levels in serum on retinal detachment due to EPH-gestosis. Klin Oczna 1998 ; 100(3) : 167-9.

Serous Exudative Retinal Detachment ในผู้ป่วย Pregnancy-induced Hypertension ในโรงพยาบาลพหลพลพยุหเสนา (รายงานผู้ป่วย)

วิชัย เรืองวิไลทรัพย์ พ.บ.

บทคัดย่อ รายงานผู้ป่วย serous exudative retinal detachment (SERD) ซึ่งมีความแปลก 2 ราย ที่เกิดร่วมกับ pregnancy-induced hypertension (หรือชื่ออื่นคือ toxemia of pregnancy, (pre)eclampsia และ edematous, proteinuric and hypertensive (EPH) gestosis) ซึ่งผู้ป่วยรายแรกมีภาวะ hemolysis, elevated liver enzymes and low platelets (HELLP) syndrome ไตวาย และมีอาการหอบเหนื่อยแทรกซ้อน และต่อมา SERD ในผู้ป่วยรายนี้หายไปในขณะที่ความดันโลหิตยังสูงอยู่ ส่วนผู้ป่วยรายที่ 2 เกิด SERD ขึ้นตั้งแต่ขณะที่ความดันโลหิตยังเป็นปกติและไม่มีการเจ็บครรภ์คลอด โดยได้วิเคราะห์ความสัมพันธ์ที่เป็นไปได้ของภาวะ serous exudative retinal detachment, HELLP syndrome, renal complications กับ pregnancy-induced hypertension เอาไว้ด้วย

ปรากฏการณ์ข้างต้น เชื่อว่าน่าจะเกิดจากการที่ภาวะความดันโลหิตสูงไม่ได้เป็นสาเหตุโดยตรงที่ทำให้เกิด choroidal ischemia หากแต่เป็นภาวะที่พบได้ร่วมกันโดยมีกลไกการเกิดโรคร่วมอันเดียวกันโดยกลไกนี้ มี *versatilely ischemic choroidoendotheliocoagulopathic, hepatohematopathogenic and angiotension-increasing regulatory effects* และมีความโน้มเอียงที่จะส่งผลต่อระบบหลอดเลือดในไตและระบบหลอดเลือดส่วนปลายมากกว่าระบบหลอดเลือดในดวงตาและที่อวัยวะอื่นๆ ในร่างกาย จึงทำให้การสูญเสียโปรตีนทางปัสสาวะ, การบวมน้ำ และความดันโลหิตสูง เป็นลักษณะที่พบได้บ่อยในผู้ป่วย PIH **จักษุเวชสาร 2548 ; กรกฎาคม-ธันวาคม : 19(2) : 195-203.**

กลุ่มงานจักษุวิทยา โรงพยาบาลพหลพลพยุหเสนา