

International Journal of Clinical Obstetrics and Gynaecology

ISSN (P): 2522-6614
ISSN (E): 2522-6622
© Gynaecology Journal
www.gynaecologyjournal.com
2023; 7(5): 01-05
Received: 01-06-2023
Accepted: 09-07-2023

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Development of Purtscher like retinopathy after preeclampsia-the blurry caesarean: A case report

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DOI: <https://doi.org/10.33545/gynae.2023.v7.i5a.1370>

Abstract

Pre-Eclampsia is a prime cause of maternal and perinatal morbidity, characterized by development of hypertension and proteinuria that can lead to multi organ failure.

We report one such case of a 23-year-old primigravida at 33 weeks and 5 days of gestational age, diagnosed to have gestational hypertension since 32 weeks and on oral medications for the same came to hospital with complaints of intermittent headache and swelling of lower limbs for past 2 weeks. On examination she had elevated BP of 160/110 mm Hg, she was admitted in labour room and was started on (Zuspan regimen) Magnesium sulphate. She started to develop blurring of vision for which ophthalmology opinion was sought, her fundus picture was normal at that time. Persistently elevated BP necessitated emergency caesarean section. Patient delivered a preterm low birth weight baby of 1.490 kgs. Post caesarean patient had worsening of vision, hence repeat ophthalmology opinion was taken which showed features of early papilledema. Fundus Fluorescein Angiography done showed multiple cotton wool spots and microleaks from capillaries confirming a diagnosis of Purtscher Retinopathy and advised for a trial of steroids. IV Methylprednisolone 1gm for 3 cycles and followed by Tablet. Prednisolone 60 mg once daily. Patient is in close follow up with the ophthalmologist.

Keywords: Preeclampsia, purtscher's retinopathy, papilledema, chronic hypertension

Introduction

Hypertensive disorders include preeclampsia, gestational hypertension, and chronic hypertension can complicate up to 10 percent of pregnancies. As a group, they are one member of the deadly triad-along with haemorrhage and infection—that contributes greatly to maternal morbidity.

Preeclampsia affects 3 to 7 % of pregnant women, which is new-onset or worsening of existing hypertension with proteinuria after 20 weeks gestation. Eclampsia is unexplained generalized seizures in patients with preeclampsia, diagnosed by measuring blood pressure and urine protein and by further tests to evaluate for end-organ damage (eg, pulmonary edema, impaired liver or kidney function). Treatment is usually with IV magnesium sulphate and delivery at term or earlier for maternal or fetal complications.

Postpartum Eclampsia occurs often within the first 4 days but sometimes up to 6 weeks of postpartum. Untreated preeclampsia is present for a variable time, then can suddenly progress to eclampsia, which occurs in 1/200 patients with preeclampsia. Untreated eclampsia is usually fatal. Pathophysiology is because of improper vascular remodelling of maternal spiral arteries. Also, severe Preeclampsia may cause organ damage like acute pancreatitis due to the micro vascular changes, ocular pathology like Purtscher's retinopathy which is a rare condition that is associated with complement-activating systemic diseases. After pancreatic injury or inflammation, proteases such as trypsin activate the complement system and can potentially cause coagulation and leukoembolization of retinal precapillary. Preeclampsia foretells raised rates of cardiovascular and metabolic disease in later life, which could be reason for subsequent lifestyle education and intervention.

Purtscher's retinopathy is usually associated with trauma, acute pancreatitis, vasculitis, lupus, and bone fractures. It was rarely described postpartum in patients with preeclampsia as well as associated with HELLP syndrome (haemolysis elevated liver enzymes low platelets) Due to our inability to accurately predict the development of pre-eclampsia, the mainstay remains the treatment of the symptoms rather than prevention.

Case report

A 23-year-old, Primigravida at 33 weeks and 5 days of gestational age, diagnosed to have Gestational Hypertension since 32 weeks and on maximum dose antihypertensives reported to the hospital with complaints of intermittent headache and bilateral lower limb swelling for 2 weeks. On examination she had elevated blood pressure of 160/110mmHg, she was admitted in labour room and was started on intravenous Labetalol 10mg followed by Zuspan regimen of Magnesium sulphate along with higher dose of antihypertensive (Labetalol 200mg thrice and Nifedipine 20mg thrice.). Blood investigations done showed normal lab values. Along with Vitals, Urine output and Knee jerk were monitored thoroughly. Ultrasound obstetrics showed fetal growth restriction and mild placental insufficiency. (Table 1) During the course of admission patient started to develop blurring of vision, for which ophthalmology opinion was obtained and found to have normal fundus. Despite all conservative management, her blood pressure was persistently elevated hence, she underwent emergency caesarean section and delivered a preterm low birth weight, girl baby of 1.490 kgs. Post caesarean patient complaints of poor vision, repeat ophthalmology fundus examination revealed features of early papilloedema following which MRI Brain +MR Venogram was

done which showed small focus of calcification in left parasagittal parietal lobe. EEG was normal. All blood investigations were repeated and found to have deranged pattern: Decreasing trend of platelets (1.79lakhs/cumm to 90,000/cumm to 76,000/cumm), Haemoglobin (11.4g/dl to 10.7g/dl to 9.6g/dl and increasing in LDH (361 IU/L to 669 IU/L) and Fibrinogen (336 IU/L to 482 IU/L) all the parameters pertaining to Partial HELLP Syndrome.(Table 2) Blurring of vision worsened, hence ophthalmology review was taken, who suspected Purtscher's Retinopathy as fundus examination showed retinal white areas around the optic nerve head (Fig.1). Fundus Fluorescein Angiography showed multiple microleaks from retinal capillaries. (Fig.2) Since Purtscher's Retinopathy is associated with acute pancreatitis, hence Serum Amylase and Lipase were done which were elevated to 576 IU/L and 332 IU/L respectively. Acute pancreatitis was confirmed with ultrasound abdomen. Bilateral eye fundus photography confirmed to be Purtscher Retinopathy and advised for a trial of steroids. IV Methylprednisolone 1gm was started once daily for 3 cycles and then switched to Tablet. Prednisolone 60mg once daily. On the last visit patient's vision was 6/18 in Right eye with sparing of fovea and 6/60 in Left eye with involvement of fovea.

Table 1: Clinical examination on admission

Blood Pressure	Bilateral Pitting Peel Edema Grade II 160/100 mm Hg
Per Abdomen	30-32weeks Gravid Uterus Cephalic, Fetal heart sound present
Lab Investigations	Hemoglobin:11.4g/dl Platelets:1.79lakhs/cumm LDH: 361 IU/L Fibrinogen: 336 IU/L Urine Albumin: 3+
Ophthalmology Fundus Examination	No evidence of retinopathy
USG Obstetrics scan	Growth Scan showed IUGR and mild placental insufficiency

Table 2: Post Caesarean Status:- Partial HELLP

Lab investigations	Hemoglobin:9.5g/dl Platelets:76,000/cumm LDH: 669 IU/L Fibrinogen: 482 IU/L Urine Albumin: Present RFT - Creatinine = 1.1
Fundus Examination	Early Papilloedema. Bilateral Fundus Photography showed Purtscher Retinopathy
Fundus Fluorescein Angiography	Multiple cotton wool spots and microleaks from capillaries
MRI Brain +MR Venogram	Small focus of calcification in left parasagittal parietal lobe.





Fig 1: Fundus photograph demonstrates marked peripapillary whitening showing ischemic areas. Discs and vessels appear undisturbed. Notice flame-shaped haemorrhages in both eyes



Fig 2: Right (left) and left (right) eyes. Venous phase fluorescein angiogram of both eyes demonstrates marked capillary nonperfusion with mild leakage of retinal vessels and staining of optic nerve head.

Discussion

Hypertension is defined as Blood pressure $>140/90$ mm Hg on 2 occasions 4 hours apart. Chronic Hypertension is when hypertension is diagnosed in a female of gestational age less than 20 weeks. Her BP will continue to remain elevated even after delivery. Gestational Hypertension is when elevated BP is detected after 20 weeks of gestation and BP falls back to normal within 12 weeks of delivery. Preeclampsia is defined as new-onset hypertension after 20 weeks of gestation and proteinuria and/or evidence of end-organ compromise, including CNS symptoms (headache and/or visual changes), pulmonary edema, thrombocytopenia, renal insufficiency, or liver dysfunction [1]. Pre-eclampsia can be classified into 3 based on the onset – Early onset is at 20-34 weeks of gestational age, Preterm or Late onset

is at ≥ 34 weeks till 37 weeks of gestational age and Term Onset is at >37 weeks of gestational age. Chronic Hypertension with Superimposed Pre-eclampsia is when a female with chronic hypertension conceives and suddenly at 20 weeks of gestational age any of the following develop have Uncontrollably elevated BP, New onset proteinuria or signs of end organ damage. HELLP syndrome is on the severe spectrum of preeclampsia with severe systemic signs and is not specifically characterized as a separate entity by the American College of Obstetrics and Gynaecologists (ACOG). Eclampsia is diagnosed with new-onset of generalized seizures in a woman with preeclampsia [1]. Pre-eclampsia stands as a prime cause of maternal and perinatal mortality and morbidity [2]. Normally, vascular remodelling occurs in pregnancy where the

maternal spiral artery opens into the intervillous space. There are two phases of vascular remodelling: 1st phase is completed by 12 weeks and 2nd phase completed by 18-20 weeks. The extra villous cytotrophoblast, mainly the endovascular trophoblast replaces the lining of maternal spiral artery converting it from high resistance to low resistance vessels. This leads to an increased blood flow through the maternal spiral artery, thereby increasing volume of blood reaching intervillous spaces which maintains uteroplacental flow. In Pregnancy Induced Hypertension, there is abnormal vascular remodelling and abnormal placentation. The 2nd phase of vascular remodelling doesn't occur causing incomplete cytotrophoblast invasion. Vessels remain narrow hence causing increased resistance to blood flow, decreasing the blood volume, thereby causing decreased uteroplacental flow and as a result decreased fetal blood supply. Multiple risk factors predispose a female to develop pre-eclampsia such as nulliparity, new paternity, molar pregnancy, age of female <18 years or >40 years, previous history of pre-eclampsia, female with chronic hypertension, multiple pregnancy and diabetic mother.

There are a few predictors of Pregnancy Induced Hypertension which are: Increased SFLT1, Increased serum endoglin, decreased VEGF, decreased placental growth factor, increased Systolic /Diastolic flow ratio in Umbilical Artery, Uterine artery doppler showing: 1) Persistence of Diastolic notch beyond 22-24 weeks and 2) Increased Pulsatility index or increased resistance ≥ 95 percentile. Due to our inability to accurately predict the development of pre-eclampsia, the mainstay remains the treatment of the symptoms rather than prevention.

Prevention of Pregnancy Induced Hypertension can be done by Low dose aspirin, calcium supplementation in females with low calcium, weight loss prior to pregnancy in obese females [3].

Definitive treatment of pre-eclampsia is by the removal of placenta/termination of pregnancy via induction of labour. The gestational age at which termination of pregnancy is done is based on the condition of the pregnancy.

The first line drugs for managing pregnancy induced hypertension are: IV Labetalol, IV Hydralazine and Oral Nifedipine. Magnesium Sulphate is given to prevent convulsions by either Zuspan or Pritchard Regimen. Therapeutic range of MgSO₄ is 4-7 mEq/L. [4]. Patient is monitored for signs of magnesium toxicity. First sign of toxicity is the absence of knee jerk occurring when MgSO₄ levels are > 10mEq/L. Other signs are diaphoresis, flushing and slurring of speech. At 12mEq/L respiratory depression occurs, at 15mEq/L respiratory paralysis occurs and at ≥ 24 mEq/L cardiac arrest occurs.

Rarely, due to the micro vascular changes acute pancreatitis can occur due to pre-eclampsia. Preeclampsia/eclampsia associated retinopathy is characterized by retinal arteriolar narrowing due to systemic hypertension and ischemia that may cause damage to the retinal and choroidal vasculature and to the retinal pigmented epithelium (RPE) [5]. This ischemic state may commonly manifest as 1) Reduced arteriolar calibre and arteriovenous ratio, 2) Retinal haemorrhages, 3) Edema, 4) Cotton wool spots secondary to arteriolar damage, 5) Choroidal dysfunction with secondary RPE damage, 6) Serous retinal detachment, 7) Retinal pigment epitheliopathy, 8) Vitreous haemorrhage. Other potential findings include 1) Peripheral retinal neovascularization, 2) Choroidal neovascularization, 3) Macular edema, 4) Macular ischemia, 5) Tear of the retinal pigment epithelium, 6) Bilateral Purtscher's like- retinopathy, 7) Retinal arterial and venous occlusion, and 8) Disseminated intravascular coagulopathy (DIC).

Purtscher's retinopathy is a rare condition that is associated with

complement-activating systemic diseases such as acute pancreatitis. The pathogenesis of Purtscher retinopathy is probably due to leuкоembolization, which results in arterial blockage and subsequent ischemia of the microvascular bed. There exists another possible source of emboli- fat embolization following enzymatic digestion in the pancreas, leading to elevations in serum lipase [6]. Once in the precapillary arteriolar vasculature, the fat emboli induce complement activation which promotes leukocytic aggregation that can become large enough to occlude retinal vessels [8]. After pancreatic injury or inflammation, proteases such as trypsin activate the complement system and can potentially cause coagulation and leuкоembolization of retinal precapillary [9].

In a study conducted in UK by Ashish Agarwal and Martin McKibbin, without treatment, 50% of eyes improved by at least 2 Snellen lines at final follow-up and 23% improved by at least 4 Snellen lines [10]. Although there are some case reports of successful treatment using high-dose steroids in IV form, the major treatment modality is expectancy.

Conclusion

Hypertensive disorders are seen in 10% of all pregnancies and include gestational hypertension, preeclampsia, eclampsia, and HELLP syndrome. Preeclampsia/eclampsia and HELLP syndrome can present with retinal involvement. Serous retinal detachment can be found in 1-2% of the cases with preeclampsia/eclampsia. Preeclampsia/eclampsia associated retinopathy refers to the retinal pathologies seen in the hypertensive disorders of pregnancy. The case highlights the adverse effects pre eclampsia can have on the mother as well as on the baby.

In the given case we can see one of the rarer manifestations of severe pre eclampsia in the form of acute pancreatitis developing due to the microvascular changes which further lead on to the development of Purtscher Retinopathy. Symptomatic management of pre-eclampsia must be done at the earliest to avoid maternal blood pressure from soaring too high. Imminent signs of eclampsia must be looked out for and pregnancy must be terminated by delivery preferably cesarean section based on the severity of the pre eclampsia.

Conflict of Interest

Not available

Financial Support

Not available

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How to Cite This Article

Dr. Abhay Shaileshbhai Patel, Dr. Athawale Tanmay Suneel, Dr. Jignesh Singh, Dr. Sudhanshu Chitravanshi. Functional outcome of posterior cruciate ligament substituted total knee arthroplasty. *International Journal of Clinical Obstetrics and Gynaecology*. Yy;vol(issue):pp. DOI: <https://doi.org/>

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