International Journal of Science and Research (IJSR)
ISSN: 2319-7064
SJIF (2020): 7.803

A Case Report of Postpartum Preeclampsia, Eclampsia and HELLP Syndrome in a Para 1+0 Now

Mwakio Patterson, MBChB, BSc. Anat, HDRH

Senior Practitioner and Reproductive Medicine Specialist, Head of department of Obstetrics and Gynecology at St. Joseph Shelter of Hope Mission Hospital, Voi Kenya. Member of Society for Maternal - Fetal Medicine (SMFM) Global health committee and Member of Department of Human Anatomy, UCU School of Medicine, Kampala Uganda, reviewer on international journal of gynecology and obstetrics (IJGO)

Abstract: ***Pre - eclampsia is pregnancy specific syndrome that is characterized by hypertension accompanied by proteinuria, developing after the 20th week of gestation in an otherwise normotensive patient. It is a significant cause of maternal mortality and morbidity. According to WHO report of 2019, Pre - eclampsia was the 3rd cause of mortality contributing about 14% of maternal mortality globally. This syndrome complicates about 2 - 5% of pregnancy, according to WHO report of 2019. The prevalence of Pre - eclampsia is estimated to be 10% in Africa with a mortality rate of 4 in every 1000 births in Ethiopia, Alemayehu belay et at 2016. The prevalence of pre - eclampsia in Kenya is estimated to range between 5.6 to 6.5%, but the number is expected to be higher in rural areas, Charity Ndwiga et al 2017.***

Keywords: Pre-eclampsia, Eclampsia, Proteinuria, Postpartum, HELLP Syndrome, Mortality, Morbidity, Trophoblastic tissues, Magnesium sulphate, Hydralazine

1. Introduction

Pre - eclampsia is a multisystem pregnancy specific disorder characterized by hypertension accompanied, occurring after the 20th week of gestation in an otherwise normotensive patient. Proteinuria can be measured based on quantitative analysis as concentration of greater or equal to 0.3g/dl of protein in a 24hr urine collection (Sibai, 2005). It is important to note that, in the presence of hypertension and biochemical and/or clinical evidence of end organ damage, diagnosis of Pre - eclampsia can be made **Without** proteinuria (ACOG 2013).

Risk factors

1) Previous history of Pre - eclampsia
2) Preexisting hypertension i. e., Chronic Hypertension
3) Renal diseases
4) Change in male partner
5) African women
6) Phospholipid syndrome

Pathogenesis

Pre - eclampsia develops when there is a defective placental development i. e., poor trophoblastic proliferation. This leads to poor development of spiral arteries which are tortious and elongated. The result is placental hypoperfusion which leads to placental release of active oxygen radicals. These radicals then cause widespread vascular dysfunction leading to prostacyclin: Thromboxane A2. With high concentration of Thromboxane A2 and Endothelin, this leads to vasoconstriction and hence hypertension. Destruction of vascular endothelium and exposure of collagen fibers may lead to thrombocytes aggregates and hence consumptive coagulopathy.

Diagnosis: Diagnosis of pre - eclampsia depends on the presence of hypertension accompanied by proteinuria or other diagnostic parameters.

Blood pressure: Systolic blood pressure (SBP) of 140mmHg or more and/or Diastolic blood pressure (DBP) of 90mmHg or more, taken in 2 occasions at least 4hrs apart after 20 weeks of gestation. In the presence of SBP of 160mmHg or more and DBP of 110mmHg, diagnosis can be made within minutes to facilitate timely administration of antihypertensives.

Proteinuria: 0.3g or more per 24hrs urine collection or protein: creatinine ratio of 0.3 or Dipstick reading of 2+.

Other diagnostic criteria
Thrombocytopenia of < 100, 000/microliter
Renal insufficiency with serum creatinine levels of ≥1.1mg/dl
Pulmonary oedema
Impaired hepatic functions with elevated liver transaminases, or
New onset of headache associated with blurred vision.

Complications

Eclampsia: This is the onset of grand mal seizures attributed to the disorder. This phenomenon develops due to brain vascular spasms leading to abnormal firing of action potentials hence the development of seizures. In one study by Nicola et al in 2017, found that, out of 2, 692 women with eclampsia, 6.9% died due to its complications.

HELLP Syndrome: This is a fetal complication of pre - eclampsia characterized by Hemolysis, Elevated liver enzymes and Low platelets. It is a life - threatening complication, risking disseminated intravascular coagulopathy which is almost always a death sentence. This condition is usually classified into 3 classes based on Mississippi classification system for HELLP syndrome. In one study by Abdulkadir Turgut and his colleagues, reported

maternal mortality and perinatal mortality rate of HELLP syndrome to range from 0.24% and 6.6 - 60%, respectively. This is a statistically significant figure.

Other complications include renal shut down, abroptio placenta, pulmonary and brain oedema and hypovolemic shock among others.

Management of Pre - eclampsia

Management of pre - eclampsia is based on 5 principles of management; I. Control of hypertension, ii. Prophylaxis against Eclampsia, iii. Check for biochemical and/or clinical evidence of end organ damage, iv. Fetus surveillance, and delivery. Use of antihypertensive when SBP is ≤150mmHg or DBP≤99mmHg is controversial but the author is advising proper use of antihypertensives such as Labetalol, Nifedipine, Methyldopa or oral hydralazine depending on the availability and ability of the patient to purchase. SBP ≥160mmHg and/or DBP≥110mmHg may require immediate hospital admission, control of BP, fetus heart tracing, administration of antenatal steroids and Magnesium sulphate for prophylaxis against eclampsia - eclampsia diagnosed ≥34 weeks should be followed by delivery as soon as possible (NICE, 2018)

2. Case Report

Patient X, 29 years old para 1+ 0 now, had a spontaneous vertex delivery (SVD) assisted by a traditional birth attendant (TBA) on their way to the hospital. She attended 6 antenatal visits, the first at 12 weeks and the last one 3 days prior to delivery (at 39/40+ gestation by date). She has been normotensive throughout her antenatal and the last BP was 128/76mmHg.

She arrived to the hospital about 40 minutes after delivery, clinical assessment to the mother and the infant was done. Patient was clinically stable, not pale and afebrile was 126/89mmHg, pulse rate 92/minute, SPO2 99% and respiratory rate 18/breast were bilaterally soft and active, uterus well involuted at 22/52, there was minimal lochia loss, no perineal, vaginal wall or cervical tear. There were minimal intrauterine clots which were evacuated and she was given Oxytocin intramuscular 10 i. u and allowed to breastfeed at the end of assessment at 9: 45am. At 12: 00noon she reported to the nurse that she developed headache and visual disturbance and she was unable to breastfeed. BP was taken which was 175/115mmHg, pulse of 81/m and SPO2 96% and the doctor was alerted immediately. At 12: 15pm patient developed a grand mal seizure for about 1 - minute, standard protocol was followed i. e. A, B, C and Intravenous hydralazine 10mg was administered slowly and blood samples taken for Complete blood count (CBC), Liver function test (LFT), Urea, Electrolytes &Creatinine (UECs) and Urine. Foley catheter was fixed and the patient responded well but a tongue bite was noted at this time. Results were out in few minutes showed Thrombocytes count of 27, 000/microliter, Creatinine was 1.05mg/dl, AST was at 346U/L, ALT 239U/L and Urinalysis showed few pus cells.

Patient was started on Magnesium sulphate immediately, 4g of 20% solution was given as a loading dose then 1g/hr. BP was taken at this point which was 172/110mg and the second dose of IV Hydralazine 10mg slowly was given. Instructions to monitor magnesium sulphate toxicity were given to the nurses i. e., Monitoring of input/output, patellar tendon reflex as well as BP every 20minutes. Few minutes later, patient had another episode of grand mal seizure which lasted about 1 minute.2g of Magnesium sulphate was given immediately and maintenance dose increased to 2g/hr. The seizure stopped, monitoring continued and hydralazine was then prescribed 10mg iv slowly every 8hrs. At 4: 30pm patient was stable 154/99mmHg, normal urine output/R and patellar reflex. Tongue bite was then repaired and a nurse attached for close monitoring.

Day 2

1) There was no history of another seizure overnight.
2) Patient was clinically stable.
3) BP 154/101, P/R 87/m, SPO2 99% and R/R 18/m.
4) GCS was 15/15.
5) Hydralazine iv 10mg every 8hrs.
6) Nifedipine 20mg every 12hrs was added.
7) MgSO4 was to continue to complete 24hrs.
8) IV Dexamethasone 10mg every 12hrs.
9) Investigations were repeated: CBC Thrombocyte count was 32, 000/microliters 301 U/L, ALT 236U/L & Cr 1.01mg/dl.
10) Able to breastfeed.

Day 3

1) Patient continued to improve clinically.
2) BP 134/97mmHg, P/R 85/m, R/R 18/m and SPO2 99%
3) IV Hydralazine was stopped.
4) P. O Hydralazine 25mg every 8hrs.
5) P. O Nifedipine 20mg every 12hrs continued.
6) MgSO4 was stopped.
7) There was no sign of MgSO4 toxicity.
8) IV Dexamethasone 10mg every 12hrs to continue.
9) CBC Thrombocytes was 58, 000/microliters, AST167 U/L, ALT 194 U/L and Cr.0.96mg/dl.
10) Close monitoring continued.
11) Able to breastfeed.

Day 4

1) Clinically stable patient.
2) BP 126/84mmHg, P/R 92/m, R/R 18/m and SPO2 99%
3) P. O Hydralazine was stopped.
4) P. O Nifedipine 20mg every 12hrs continued.
5) IV Dexamethasone was stopped.
6) CBC Thrombocytes count 94, 000/microliters 82 U/L, ALT 115 U/L and Cr.0.94mg/dl.
7) Able to breastfeed.

Day 5

1) Clinically stable patient.
2) BP 124/85mmHg, P/R 94/m, R/R 17/m and SPO2 99%
3) P. O Nifedipine 20mg stopped.
4) CBC Thrombocytes count 134, 000/microliters 40 U/L, ALT 78 U/L
5) Able to breastfeed.

Day 6

1) Clinically stable patient.
2) BP 123/87mmHg, PR 91/m, R/R 18/m and SPO2 99%

3) P. O Nifedipine 20mg every 12hrs to continue
4) CBC Thrombocytes count 152.000/microliters 34 U/L, ALT 51 U/L
5) Able to breastfeed and she was discharged to come back for review after 7 days.

Review on the 7th day after discharge, patient clinically stable, BP 121/72mmHg, CBC Thrombocytes count 198, 000/microliters 24 U/L and ALT 42 U/L.

3. Conclusion

Pre - eclampsia/Eclampsia commonly manifest during antepartum period but can also present during post - partum period. Traditional diagnostic criteria are the presence of hypertension accompanied by proteinuria after 20 weeks of gestation, however, presence of proteinuria is not a must criteria. In the presence of hypertension and other ≥1diagnostic criteria is sufficient. Pre - eclampsia/Eclampsia carries high mortality and morbidity risks but with prompt active management, the condition tends to respond well. Meticulous physical examination and investigations are important to diagnose complications early and institute early management.

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