**Unusual Case Report of Pre-Eclampsia with Severe Features and Painful Massive Bilateral Pedal Oedema Upto the Groin and Vulva**

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**Citation:** Onuminya DS. Unusual Case Report of Pre-Eclampsia with Severe Features and Painful Massive Bilateral Pedal Oedema Upto the Groin and Vulva. Gynecol Reprod Health. 2023; 7(1): 1-4.

**ABSTRACT**

The aim of this case report is to elucidate the unusual presentations and outcomes of sudden onset of pre-eclampsia with severe features and massive progressive painful bilateral pedal oedema up to the groin and vulva in an unbooked 23-year-old primigravida. The blood pressure was significantly elevated (170/130 mmHg) on presentation with associated significant proteinuria (+++) and elevated serum creatinine. Obstetric scan done on presentation confirmed a live active intrauterine fetus at 26 weeks of gestation. She was managed conservatively with antihypertensive and was delivered of a live male neonate with good Apgar scores following induction of labour at 28 weeks of gestation. Both mother and baby were discharged home in good conditions.

**Keywords**
Pregnancy, Pre-eclampsia, Oedema, Management Outcome.

**Introduction**

Pre-eclampsia is an idiopathic disorder of pregnancy characterised by proteinuric hypertension [1-4]. Pre-eclampsia is a major public health problem worldwide, but worst in the developing countries including Nigeria with increased risk of maternal and perinatal morbidities and mortalities [5-9]. The increased risk of maternal and perinatal complications associated with pre-eclampsia in developing countries are due to lack of prenatal care, lack of access to hospital care, poverty and inappropriate diagnosis and management of pre-eclampsia [7-9]. There are four subclassification of pre-eclampsia: early-onset, preterm, late-onset and term pre-eclampsia. Early onset pre-eclampsia is associated with a much higher risk of short- and long-term maternal and perinatal morbidity and mortality [2]. At the time of diagnosis, the initial objective is the assessment of disease severity. Severe hypertension with systolic blood pressure of equal or greater than 160mmHg and/or diastolic blood pressure of equal or greater than 110mmHg, thrombocytopenia of less than 100,000 per microliter, liver transaminase above twice the normal values, haemolysis-elevated liver enzymes-low platelet count (HELLP) syndrome, renal failure, persistent epigastric or right hypochondriac pain, visual symptoms and acute pulmonary oedema are severity criteria [10]. In the pathophysiology of pre-eclampsia, cytotrophoblast fails to remodel spiral arteries, leading to hypoperfusion and ischemia of the placenta. The fetal consequence is growth restriction. On the maternal side, the ischemic placenta releases factors that provoke a generalized maternal endothelial dysfunction, which is in turn responsible for clinical features and complications of pre-eclampsia [2,10]. A new onset of hypertension, proteinuria and feet Oedema arising after 20 weeks of gestation in a previously normotensive woman are typical features of pre-eclampsia. The bilateral pedal oedema of pre-eclampsia is usually insidious in onset and painless. However, the pedal oedema in the index case was sudden in onset, painful, massive and extending to the groin and vulva.

The patient responded to the anti-hypertensive drug therapy and eventually had induction of labour with a spontaneous vaginal delivery of a live male neonate that weighed 1.4kg with good Apgar scores. The vulva oedema resolved completely a week postpartum. Both mother and baby were discharged home in good condition.
two weeks after delivery. Despite a better understanding of pre-eclampsic pathogenic mechanisms and maternal haemodynamic alterations during pre-eclampsia, the only curative treatment remains placenta and fetus delivery. The aim of this case report is to highlight the unusual presentations, and outcomes of sudden onset of pre-eclampsia in a primigravida with massive progressive painful bilateral pedal oedema.

**Case Report**

An unbooked 23-year-old primigravida presented in the emergency unit of the hospital with 2 days history of massive progressive painful bilateral lower limb swelling extending to the groin and vulva areas. She thought the vulva involvement was a boil but suddenly increased in size and she could not walk because of the pain. There was no history of trauma to the vulva. No history of associated fever or itching. The pregnancy was desired and spontaneous in an intramarital relationship. She was not a known hypertensive or diabetic. No prior history of renal problem or disease. There was no known drug allergy. Patient could not remember her last menstrual period, but the obstetric scan done at presentation put her gestational age at 26 weeks.

At presentation, she was in painful distress but not febrile and temperature was 37°C. She was not clinically pale, not jaundiced but had bilateral painful pitting oedema extending to the groin and shining oedematous labia majora and minora. She was not in respiratory distress and the chest was clinically clear.

The pulse rate was 78 beats/minute and blood pressure were 170/130 mmHg.

The abdomen was uniformly enlarged with gravid uterus and it moved with respiration. There was no area of tenderness. The kidneys, spleen, and liver were not palpably enlarged. The Simphysio-fundal height was 27cm and this was compatible with gestational age of 26 weeks, which was determined with the aid of obstetric scan at presentation. There was no palpable uterine contraction.

The vaginal examination revealed a massive shiny vulva oedema and digital examination could not be done due to severe tenderness. The pad removed was stained with serosanguinous discharge following a fine needle aspiration of the vulva swelling by the referring doctor.

Urinalysis done showed significant proteinuria (3 pluses of protein observed), the Packed Cell Volume (PCV) was 36%, total white blood cells count was essentially normal. The Liver and Renal function test results were normal except for elevated creatinine of 217 micromols per litre as against the normal range of 45 - 84 micromols per litre. The emergency obstetric scan done showed a live fetus at 26 weeks gestation, cephalic and fetal heart rate was 145 beats/minute.

Based on clinical findings and investigation done, the diagnosis of pre-eclampsia with severe features was made. The clinical pictures of the vulva before and after delivery are shown in Figures 1 & 2 below.
She was treated with intravenous hydralazine 10mg slowly stat; Magnesium sulfate loading dose of 14g administered (4g intravenously and 5g intramuscularly in each buttock) which was completed within 24 hours to prevent convulsion while waiting for surgery; Alpha Methyl Dopa tablets 500mg 8 hourly and Nifedipine tablets 20mg 12 hourly; Intravenous Augmentin 1.2g 12 hourly and Metronidazole 500mg 8 hourly (the presumptive antibiotic therapy was given for five days to prevent infections); and Intravenous pentazocine 30mg 6 hourly were commenced. The patient was admitted into maternity ward. Urethral catheter was passed to monitor urinary output. The patient was worked up for delivery after stabilisation. The Anaesthetist assessed her clinical status as ASA III, and declined emergency caesarean section because the patient was considered unfit under any form of anaesthesia. Her blood pressure was 140/100 mmHg after 24 hours of treatment, but her massive painful pedal oedema persisted. Dexamethasone course was given to hasten fetal lung maturity.

The conservative management continued for a week on admission. Her blood pressure was 130/90mmHg hence Nifedipine was discontinued, however the massive oedema persisted.

Second week into admission, the patient’s condition remains same except for the good control of the blood pressure. The patient was counselled on the need to consider the induction of labour using Misoprostol and she obliged. Then, 50µg of Misoprostol was inserted using only the index finger to push into the posterior fornix after 30mg of intravenous pentazocine. She eventually delivered a live male fetus with good Apgar scores of 9 in one minute and 10 in five minutes; and an unusual fetal weight of 1.4kg at 28 weeks of gestation. The baby was admitted into the special baby care unit and was managed in the incubator for two weeks. The mother improved rapidly following the delivery of the baby. The massive oedema resolved completely a week after delivery. Both mother and baby were discharged home in good conditions after two weeks of delivery on request owing to financial constraints. Baby's weight on discharge was also unusual, 1.6kg.

The blood pressure of mother on discharge was 120/80mmHg and she was given a week appointment for postnatal care.

Discussion

Pre-eclampsia is an idiopathic disorder of pregnancy characterised by proteinuric hypertension. Estimate of 63,000 women die worldwide each year because of pre-eclampsia and its complications with 95% of these occurring in developing countries [1].

In UK, pre-eclampsia is the second largest cause of both direct maternal death and perinatal loss, responsible for the death of 6-9 women annually [2] and over 175 babies [3]. The prevalence of pre-eclampsia in Nigeria ranges from 2 - 16.7 % with approximately 37,000 women dying from pre - eclampsia annually [9].

More than 10% of women will develop pre-eclampsia in their first pregnancy, although the overwhelming majority of these will have successful pregnancy outcomes, the condition can give rise to severe multisystem complications including cerebral haemorrhage, hepatic and renal dysfunction and respiratory compromise [2].

Pre-eclampsia complicates 3-5% of all pregnancies and it is characterised by placental and maternal vascular dysfunction that may lead to adverse outcomes such as severe hypertension, stroke, seizure, renal and hepatic injury, haemolysis - elevated liver enzymes - low platelet count (HELLP) syndrome, haemorrhage, fetal growth restriction or even death [2,4,8,10]. These complications were minimal in the index case both for mother and neonate. The birth weight of 1.4Kg is quite unusual and big for the gestational age and pre-eclampsia where fetal growth restriction is anticipated. Furthermore, the gain of 200 grams weight in this neonate within two weeks of birth is also unusual, but this was the finding following extensive expressed breast milk feeding within the period. This is not in keeping with the usual initial weight loss in neonates after birth. Though, mother and baby were discharged home 14 days after delivery in good conditions, on request because of financial constraints, it is usually desirable to observe them for a reasonable length of time before discharge especially in cases with severe features as in the index case.

The commonest presentation of pre-eclampsia is proteinuric hypertension, however, the index patient presented with progressive painful lower limb oedema extending to the groin with associated painful vulva oedema. The only abnormal finding on blood investigations was significant elevated creatinine of 217 micromols per liter, which reverted to normal range a week after delivery. The clinical picture of the dramatic resolution of oedema a week after delivery is as shown in Fig. 2. This is in keeping with the fact that Pre-eclampsia always resolves a short time after the baby is born [10]. There are several causes of oedema in pregnancy, these include physiologic and pathogenic oedema.

Physiologic oedema in pregnancy occurs when body fluids increase in the third trimester of pregnancy. This results from hormone-induced sodium retention. Fluid may also accumulate in the tissue due to increased blood flow and pressure of the growing uterus on the pelvic veins and inferior vena cava during recumbency, obstructing outflow from both femoral veins. These physiologic changes in pregnancy are not usually associated with elevated blood pressure and are also hallmark of good placenta functions.

Pathologic causes of oedema are less common but often dangerous. They may include pre-eclampsia, deep venous thrombosis (DVT), cellulitis, cardiogenic and renal disorders. DVT is more common during pregnancy because pregnancy is a hypercoagulable state, and pregnant women may be less mobile. Pre-eclampsia results from pregnancy -induced hypertension; however, not all women with Pre-eclampsia develop oedema. Extensive cellulitis, which usually causes focal erythema, may resemble general oedema. Therefore, evaluation of patients with lower-extremity oedema during pregnancy as in the index case aims to exclude Pre-eclampsia, DVT, cellulitis, peripartum cardiomyopathy,
physiologic oedema in pregnancy and other causes of oedema. Some fluid accumulation during pregnancy is normal, particularly during the third trimester. It is called physiologic oedema [11]. Fluid accumulates during pregnancy because the adrenal glands produce more of the hormones that make body retain fluid (aldosterone and cortisol). During pregnancy total body water increases by 6 to 8 liters, 4 to 5 liters of which are extracellular, of which at least 2 to 3 liters are interstitial [11]. At some stage in pregnancy, 8 out of 10 women have demonstrable clinical oedema. It is worth of note that in pregnancy there is alteration in local Starling forces whereby there is a moderate fall in interstitial fluid colloid osmotic pressure and a rise in capillary hydrostatic pressure, as well as changes in hydration of connective tissue ground substance. Oedema in pregnancy therefore is not a reliable criterion for diagnosis of pre-eclampsia. It is also worth of note that volume expansion therapy in pregnancy runs the risk of pulmonary or cerebral oedema, particularly in the immediate puerperium and the role of diuretics in obstetric practice should be restricted to the management of pulmonary oedema in pre-eclampsia. Vulva oedema and erythematous oedema associated with DVT are rare but dangerous complications of pregnancy [11,12].

The features seen in the index patient is in favour of pre-eclampsia with severe features characterised by significant elevated blood pressure on presentation and significant proteinuria. The significant proteinuric hypertension resolved after delivery even before the patient was discharged. However deep venous thrombosis is not completely ruled out because of the sudden painful onset of the oedema. More so, pregnancy is a significant predisposing factor to DVT and painful bilateral lower limb swellings are symptomatic of DVT in the inferior vena cava.

In conclusion, although painful oedema hardly occur in pre-eclampsia, it should always be first considered in pregnant women with severe proteinuric hypertension. The cure to this condition is the delivery of the fetus and its placenta even when patient is considered unfit for routine emergency caesarean section as observed in the index case with both mother and baby delivered and discharged home in good conditions. Such patient requires a multi-disciplinary follow up to avert complications.

Informed consent was obtained from the patient to use the clinical pictures in Figure. 1 & 2 for this publication.

References
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