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Severe preeclampsia in a non-proteinuric patient

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Abstract

We present a 57year old now Para 2+0 lady who conceived through assisted reproductive technology and subsequently presented to us at 12weeks of gestation for antenatal care. Her antenatal period was uneventful until the 32nd week of gestation when she suddenly developed hypertension without any proteinuria. On admission, renal and liver function tests were found to be grossly deranged. She was stabilized with anti-hypertensives and given a course of steroids for fetal lung maturity. She subsequently had an emergency lower segment caesarean section at 33 weeks of gestation and was delivered of a live male baby weighing 1.8 kilograms. A high index of suspicion for pre-eclampsia is very important in the management of patients with hypertension in pregnancy, even in the absence of proteinuria.

Keywords: Pregnancy induced hypertension, proteinuria, assisted reproductive technology, severe preeclampsia.

Introduction

Preeclampsia is a dangerous and potentially deadly disease common in pregnancy. In Nigeria, the incidence of preeclampsia ranges from 2-16.7%.[1] This incidence is higher than in the developed countries where incidences of 2-10% are being reported.[1] Also, the risk of death from this condition in low-income countries, which can progress to eclampsia, can be up to 33 times that of high-income countries.[2] Preeclampsia can be super imposed on a patient with chronic hypertension or occur de novo in pregnancy after the 20th week of gestation. It has until recently been characterized by hypertension and proteinuria in pregnancy.[3] However, recent research has revealed that proteinuria is not necessary for the diagnosis of preeclampsia and it is now advocated that in the presence of hypertension, every pregnant patient should be investigated for end organ damage.[4,5]

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The patient suddenly developed hypertension at 32 weeks of gestation without any accompanying proteinuria. However, there was end organ damage that was detected biochemically and radiologically as the liver and the kidneys were affected, which signified the presence of severe preeclampsia.

Case report

A 57-year-old lady presented at a private medical facility for antenatal care. She had conceived by assisted reproductive technology and had an embryo transfer and was 12 weeks and 4days pregnant at presentation. There was no family history of hypertension. Her booking blood pressure was 110/70 mmHg and her body mass index was 26.2 kilogram per square meter. She had had an uneventful delivery at term of a female baby 12years prior to presentation in a private facility to the same man, her husband.

Two weeks later at a gestational age of 14weeks 4days, she had a cervical stitch inserted under saddle block by the McDonald's technique.

Subsequent antenatal care at two weekly intervals

remained uneventful until 32weeks of gestation, when her blood pressure was found to be 160/110mmHg. She had no symptoms and symphysio-fundal height was compatible with gestational age. There was no proteinuria.

She was admitted and started on antihypertensives, antenatal steroids, twice-daily blood pressure measurement, daily weighing and daily urinalysis. A fluid/urinary output measurement was requested and remained satisfactory. A request was made to the laboratory for full blood count with platelets, liver function tests and electrolytes, urea, creatinine and uric acid. An obstetric ultrasound scan was also requested for, and two units of blood were to be cross matched and reserved for her.

The blood pressure stabilized at 130/80 mmHg by the next day and two days later the results of the laboratory investigations were retrieved and showed:

E/U/C (mmol/L): Creatinine: 183(53-115), Uric acid (female): 0.56 (0.18-0.30)

Liver function test (mmol/L): Aspartate Transaminase (AST): 114 (0-12), Alanine Transaminase (ALT): 43 (0-12), Alanine Phosphatase (ALP): 165 (9-35)

Full blood count: Hb: 12g/dL and the platelet count: 145x109/L (100-300); All other parameters were within normal limits.

The ultrasound sound scan revealed a male fetus with an estimated fetal weight of 2.1 kg and a biophysical profile of 10/10.

The next day, she remained free of symptoms, with a blood pressure ranging from 130/80 to 140/85mmHg.The urine remained negative for protein. She was reviewed by the nephrologist who requested an abdominal ultrasound scan as well as a repeat of the E/U/C and uric acid tests.

A repeat of the creatinine revealed a value of 163 mmol/L and a uric acid of 0.42mmol/L. The liver enzymes AST was 36 mmol/L, ALT 21 mmol/L. A clotting profile revealed parameters essentially within normal limits. The renal ultrasound scan showed evidence of acute renal injury.

The findings and implications were further explained to the patient and the husband and they were counseled for delivery by emergency caesarean section which they consented. She was subsequently delivered by caesarean section under spinal anaesthesia of a live male baby weighing 1.8kg at 33weeks of gestation. Apgar scores were 8 and 9 at 1 and 5 minutes respectively. Her immediate post operative condition was satisfactory.

A day post operation, she started graded oral fluids and she was encouraged to be ambulatory. Her condition remained stable but her blood pressure was 150/100 on the second day for which she was recommenced on anti-hypertensive drugs. Her repeat tests on the second post operation day were: creatinine 205 mmol/L, liver function tests AST 88 mmol/L, ALT 78 mmol/L and ALP 376 (1-270) mmol/L. Subsequent repeat tests showed a gradual decline of these parameters until by the 7th day post operation when the creatinine level was 91mmol/L and the liver enzymes were minimally elevated. Her blood pressure was 130/80mmHg. Her baby was healthy

She was discharged with referrals to the cardiologist and the nephrologist for further control and assessment of her cardiac and renal functions. At postnatal checkup, her investigations were normal and repeat renal scans showed no abnormality and her blood pressure was 110/70mmHg. Her baby was thriving.

Discussion

Preeclampsia/eclampsia has been found to be leading cause of severe maternal outcome in Nigeria.[6] Not too long ago, pregnancy induced hypertension was found to be the mildest form of hypertensive disorders which involved a continuum of hypertension, preeclampsia and eclampsia. In this spectrum, preeclampsia was diagnosed only in the presence of proteinuria in association with pregnancy induced hypertension.[7]

However, recently, it has been discovered that proteinuria does not have to be present in the diagnosis of preeclampsia.[4,5] Preeclampsia can be diagnosed when one of the following conditions is present in addition to hypertension: proteinuria; evidence of end organ affectation like renal insufficiency; hepatic impairment characterized by two times above normal of the value of transaminases; neurological complications; evidence of haematological complications eg thrombocytopenia or haemolysis; or evidence of utero - placental insufficiency.[5]

Our patient's antenatal care was uneventful until the occurrence of sudden hypertension at 32 weeks of

gestation without any proteinuria. A high index of suspicion motivated a search for end organ involvement which would implicate preeclampsia. This revealed high values of creatinine, uric acid and liver transaminases.

The high index of suspicion for this patient was born out of several risk factors that she had for preeclampsia. Advanced maternal age has been implicated as a risk factor for preeclampsia. A prolonged interval between the last pregnancy and the current pregnancy has also been found to increase the risk of preeclampsia. Indeed, it has been found that a 10-year gap in pregnancies confers the same risk factor for preeclampsia as for the nullipara.[8] In addition, assisted conception, especially with donor gametes, is associated with an increased risk of preeclampsia.[9] Our patient had all of these risk factors. Other known risk factors for preeclampsia which were absent in our patient include: previous preeclampsia, diabetes mellitus, chronic hypertension, BMI>30-35kg/m^2, chronic renal disease, and multiple gestation.[5]

A very high index of suspicion is necessary in the diagnosis of preeclampsia. This is even more important in the presence of pregnancy induced hypertension without proteinuria. This will eliminate a false sense of security in the presence of only pregnancy hypertension. Indeed, it has been suggested that every pregnant woman who has hypertension should be investigated for end-organ insufficiency.[4] This is especially necessary in this environment where the poor health infrastructure worsens the prognosis of this disease for both mother and fetus. Only then can we prevent the development of very serious adverse sequaelae including eclampsia, which is the most common cause of maternal mortality in our region.[10]

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