Obstetrics and Gynaecology Cases - Reviews

CASE REPORT

Eclampsia at 20 Weeks of Gestation: A Case Report

Mary Sue Makin[*]

Daeyang Luke Hospital in Lilongwe, Malawi

*Corresponding author: *Mary Sue Makin, Daeyang Luke Hospital in Lilongwe, Malawi*

Check for updates

Abstract

A 23-year-old primigravida with unknown last menstrual period and 20 weeks gestation by ultrasound presented to Daeyang Luke Hospital in Lilongwe, Malawi with a history of headache, fever, vomiting and new-onset of convulsions. At the time of her admission the full blood count instrument at our hospital was out of service. A rapid blood test for malaria was positive. After an initial blood pressure of 164/127 and 3+ proteinuria on urinary dipstick the diagnosis of eclampsia was made. She was given magnesium sulfate by intravenous protocol and labor was induced with vaginal misoprostol. She delivered a nonviable premature infant after six hours. This case is presented because of the rarity of eclampsia at 20 weeks gestation [1] and to discuss some of the recent advances in the pathogenesis of preeclampsia especially as it pertains to early developmental changes in the maternofetal junctional zone.

Introduction

Hypertensive disorders of pregnancy cause 14% of all maternal deaths globally, approximately 42,000 each year. 99% of these deaths occur in low resource settings [2]. According to WHO estimates, the incidence of preeclampsia in developing countries (2.8% of live births) is seven times higher than in developed countries (0.4% of live births). Eclampsia increases the risk of maternal death both in developed countries (0.4% of live births) and in developing countries (15%) [3-7]. Adequate obstetrical care in developing countries must include clinicians and facilities who are well versed and well prepared to care for preeclamptic and eclamptic patients.

The precise etiopathogenesis of preeclampsia is still a subject of extensive research, but most researchers believe it is multifactorial. Globally about 80% of all preeclamptic pregnancies occur after 34 weeks and are classified as "late onset". These pregnancies have nor-mally grown babies and no changes in blood flow in the umbilical arteries. Early onset preeclampsia makes up the rest of the affected pregnancies and is characterized by an inadequate and incomplete trophoblast invasion of maternal spiral arteries and clear signs of fetal growth restriction [8].

The pathogenesis of preeclampsia is complex with numerous genetic, immunologic and environmental factors interacting. Recently Hladunewich, et al. described preeclampsia as a two-stage process. The first stage is one of abnormal placental development occurring in the first trimester of pregnancy. This abnormal development results in placental insufficiency and the release of excessive amounts of placental materials into the maternal circulation. The second stage, the maternal syndrome, results from the failure of cytotrophoblast remodeling of uterine spiral arteries. This cytotrophoblastic failure is thought to release secreted factors that enter the maternal circulation resulting in the signs and symptoms of preeclampsia [7,8].

Extensive angiogenesis is necessary in human placentation to establish a suitable network for the supply of oxygen and nutrients to the fetus. Developing placentas secrete a variety of pro-and antiangiogenic factors. Placental angiogenesis has been found to be defective in preeclampsia as evidenced by the failure of the cytotrophoblasts to convert from an epithelial to an endothelial phenotype, based on cell surface marker studies [8-11].

In 2003 Maynard hypothesized that placental ischemia is an early event in preeclamptic pregnancies, leading to placental production of a soluble factor or factors that cause maternal endothelial dysfunction. He found that excess circulating sFlt1 secreted by the

ClinMed
INTERNATIONAL LIBRARY

Makin. Obstet Gynecol Cases Rev 2021, 8:201

• Page 1 of 3 •

placenta in preeclampsia led to endothelial dysfunction, hypertension and proteinuria. He also found that rising levels of sFlt1 were accompanied by falling levels of vascular endothelial growth factor (VEGF), a signal protein produced by cells that stimulate angiogenesis, and falling levels of placenta growth factor (PlGF), a potent angiogenic factor of the VEGF family [12,13].

During implantation fetal trophoblast cells and maternal uterine tissues, which include endometrium and myometrium, come into intimate contact with each other. This results in a zone made up of fetal and maternal cells which can be called the maternofetal junctional zone. At the time of delivery part of the junctional zone adheres to the placenta and forms the bottom of the intervillous space, the basal plate. The remaining part of the junctional zone adheres to the uterine wall after delivery and makes up the placental bed [14,15].

The placental bed is that part of the decidua and adjoining myometrium which lies below the placenta. The function of this area is to maintain an adequate blood supply to the intervillous space of the placenta. Early in pregnancy spiral arteries from the placental bed are invaded by cytotrophoblasts that break down arterial endothelium, internal elastic lamina and the muscular layer of the vessel. These physiologic changes convert the vessels from muscular end arteries to wide mouthed sinusoids. A 5-10-fold dilation of the spiral arteries occurs resulting in a decrease in vascular resistance and an increase in blood flow to the fetus [14-16].

In normal pregnancies the physiologic changes described above involve the myometrial segment of the spiral arteries and these changes are completed by 20 weeks. In preeclampsia and intrauterine growth restriction the changes in the spiral arteries are limited to the decidual segment of the placental bed and the process does not extend into the myometrium [12].

Preeclampsia is classically described as the presence of hypertension and proteinuria in a previously normotensive pregnant woman after the 20th week of pregnancy. This diagnosis is also applied in the absence of proteinuria if there is evidence of target organ damage [11].

The diagnosis of eclampsia should always be considered when convulsions occur in a pregnant woman after the 20th week of gestation. Occurrence of eclampsia before 20 weeks is rare and a molar pregnancy should be considered. During management of a presumed eclamptic patient if convulsions continue to occur even after therapeutic dosages of magnesium sulfate have be administered, a cerebrovascular accident must be considered. Other conditions to be considered include expansive brain injury, toxic and metabolic encephalopathies, reversible cerebral vasoconstriction syndrome, thrombotic thrombocytopenic purpura, and central nervous system infection [14].

Convulsions without neurological deficits can be caused by metabolic abnormalities such as hypocalcemia, hyponatremia and hypoglycemia. Drug or alcohol withdrawal, infection (meningitis, encephalitis, sepsis) or recent head trauma should be considered. Disorders such as thrombotic thrombocytopenic purpura and uremic hemolytic syndrome can be triggered by pregnancy. A prior diagnosis of epilepsy should be ruled out [1,14].

Case Description

A 23-year-old primigravida presented to the hospital with a history of headache, fever, vomiting and four episodes of convulsions the day of admission. Her mother described tonic-clonic movements lasting one to two minutes. She had no previous history of hypertension, convulsions or epilepsy. Admission blood pressure was 184/134, pulse 111, temperature 36.4 °C. A full blood count was not available at our hospital on admission, but the rapid test for malaria was positive and urine protein was 3+ on dipstick. Ultrasound revealed a viable intrauterine pregnancy with estimated gestational age 20 weeks. A lumbar puncture was done with benign results.

She was started on intravenous artesunate, intravenous magnesium sulfate and hydralazine per hospital protocols. Induction of labor with vaginal misoprostol was begun four hours after admission with the diagnosis of eclampsia at 20 weeks gestation. Twelve hours after induction she delivered a nonviable fetus and had an evacuation of retained products of conception in the theatre. Over the next 48 hours her blood pressures fluctuated between 150 and 174 systolic and 94 and 124 diastolic. She was discharged home on nifedipine 10 mg PO BD on her second postpartum day. Her blood pressure was 130/80 two weeks postpartum on no antihypertensives.

Conclusions

This case presentation is remarkable for the early gestational age of 20 weeks at which the convulsions occurred. 91% of all cases of eclampsia develop at or after 28 weeks gestation. 7.5% occur between 21 and 28 weeks. Only 1.5% occurs at 20 weeks or earlier [7].

This patient arrived at our hospital late in the afternoon at a time when our instrument for doing full blood counts was not functioning. With the elevated blood pressure and 3+ proteinuria the diagnosis of eclampsia was secure. The possibility of cerebral malaria was also considered. A lumbar puncture was performed to conclusively rule out meningitis. Because of the impossibility of survival for the fetus at this gestational age in a developing country like Malawi, the decision to induce labor was made. Even though we had no access to a full blood count, liver or kidney function tests, or cerebral imaging, the situation was considered critical to begin the induction of labor. Fortunately, the patient had an uneventful vaginal delivery and returned to a normal blood pressure by two weeks after the delivery.

References

1. Sibai B (2005) Diagnosis, prevention and management of eclampsia. Obstet Gynecol 105: 402-410.

2. Nakano K, Tsunemi T, Akasaka J, Shigemitsu A, Naruse K, et al. (2017) A case of very early onset eclampsia, placental abruption and intrauterine fetal death. Hypertens Res Pregnancy 5: 17-19.

3. Tanaka M, Tsujimoto Y, Goto K, Kumahara K, Onishi S, et al. (2015) Preeclampsia before 20 weeks of gestation: A case report and review of the literature. CEN Case Rep 4: 55-60.

4. Vousden N, Lawley E, Seed PT, Gidiri MF, Goudar S, et al. (2019) Incidence of eclampsia and related complications across 10 low- and middle-resource geographical regions: Secondary analysis of a cluster randomised controlled trial. PLoS Med 16: e1002775.

5. Meazaw M, Chojenta C, Muluneh M, Loxton D (2020) Systematic and meta-analysis of factors associated with preeclampsia and eclampsia in sub-Sahara Africa. PLoS One 15: e0237600.

6. World Health Organization (WHO) (2019) Trends in Maternal Mortality 2000 to 2017. Estimates by WHO, UNICEF, UNFPA, World Bank Group and the United Nations Population Division, Geneva, Switzerland.

7. Lilungulu A, Bintabara D, Mujungu S, Chiwanga E, Chetto P, et al. (2020) Incidence and predictors of maternal and perinatal mortality among women with severe maternal outcomes: A tanzanian facility-based survey for improving maternal and newborn care. Obstet Gynecol Int 2020: 5390903.

8. Huppertz B (2008) Placental origins of preeclampsia: Challenging the current hypothesis. Hypertension 51: 970-975.

9. Hladunewich M, Karumanchi S, Lafayette R (2007) Pathophysiology of the clinical manifestations of preeclampsia. Clin J Am Soc Nephrol 2: 543-549.

10. Zhou Y, Damsky C, Fisher S (1997) Preeclampsia is associated with failure of human cytotrophoblasts to mimic a vascular adhesion phenotype. J Clin Invest 99: 2139-2151.

11. Peracoli J (2019) Pre-eclampsia/eclampsia. Rev Bras Ginecol Obstet 41: 318-322.

12. Maynard S, Min J, Merchan J, Lim KH, Li J, et al. (2003) Excess placental soluble fms-like tyrosine kinase 1 (sFltl1) may contribute to endothelial dysfunction, hypertension and preeclampsia. J Clin Invest 111: 649-658.

13. Valenzuela F, Perez-Sepulveda A, Torres M, Correa P, Repetto G, et al. (2012) Pathogenesis of preeclampsia: The genetic component. J Pregnancy 2012: 632732.

14. Kaufmann P, Black S, Huppertz B (2003) Endovascular trophoblastic invasion: Implications for the pathogenesis of intrauterine growth restriction and preeclampsia. Biol Reprod 69: 1-7.

15. Nobis P, Gautam H, Kathar K, Goswami P, Sharma J (2020) Spiral artery morphology in relation to hypertension in pregnancy and birthweight by placental bed biopsy: A retrospective analysis. Ann Clin Lab.

16. Hennessy A, Makris A (2011) Preeclamptic nephropathy. Nephrology 16: 134-143.

Makin. Obstet Gynecol Cases Rev 2021, 8:201

• Page 3 of 3 •