Atypical presentation of Severe PET in late pregnancy, Early detection methods of PET: Two case reports and literature review

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Introduction
Preeclampsia is a dynamic process that is part of a spectrum of conditions known as the hypertensive disorders of pregnancy. A multisystem disorder usually associated with raised blood pressure and proteinuria commonly results in serious complications of 2-8% of pregnancies. The endothelial dysfunction that arises from placental sources leads to the end-organ phenomenon, symptoms experienced, and holds the key to early detection. Although outcome is often good, preeclampsia can be devastating and life threatening for both mother and baby particularly in developing countries. In recent years, there have been significant advances on detecting the prevalence of preeclampsia, both early and late onset, as early as 11-13 weeks.

Severe preeclampsia is illustrated by features of severe end-organ damage, with proteinuria, CNS dysfunction, visual disturbances, and severe blood pressure elevation. It has been demonstrated that the earlier the onset of severe preeclampsia, the higher the perinatal mortality rate [1]. Not all cases of preeclampsia are easy to detect. Atypical cases develop before 20 weeks, beyond 48 hours postpartum or present with some of the signs and symptoms of preeclampsia without the usual hypertension or proteinuria [2]. Thus early detection and prediction algorithms become even more valuable.

Assessing risk factors early along with screening methods will lead to better prediction of preeclampsia. For example, mothers with OC are at increased risk of developing gestation diabetes and preeclampsia [3]. Obstetric cholestasis (OC) of pregnancy is a common liver disorder and presents with pruritus and elevated bile acids. It has been related to a high frequency of abnormal intra-partum fetal heart rate, amniotic fluid meconium, prematurity, and perinatal mortality. The triad of early detection of preeclampsia is focused on identifying risk factors with thorough maternal history, biophysical, and biochemical parameters. We would like to present two interesting cases of severe atypical preeclampsia, which we have managed recently in our unit to address the severity of PET complications and the necessity for early detection and management.

Case 1:
This patient was a 22 year old lady G4 P3+5 who developed severe atypical late-onset preeclampsia at 37+4 weeks. A preeclamptic risk factor present in this patient was heterozygosity for Factor V Leiden. This patient was stabilized with magnesium sulphate (MgSO4) prophylaxis for which she developed toxicity. This necessitated intensive care management and close observation, and she was delivered by cesarean section at 37+6 weeks. Her pregnancy followed an uncomplicated course up until 33 +6 when there was decreased fetal movement. CTG in the Maternal Assessment Unit was normal at 3640. At 37/40, blood pressure was elevated to 130/90 along with decreased fetal movement and PET bloods were sent. At 37 +4 our patient developed headache, visual disturbances of flashing lights, swollen hands and feet, chest tightness, proteinuria, and blood pressure was elevated to 160/105. The outcome ended favorably for this lady and the baby was born in a good condition. However the progression to severe preeclampsia, management with MgSO4, and ultimately toxicity could have been avoided with early detection and prophylactic management.

Case 2:
This patient was a 41 year old lady G1 P1 +1. She had developed Obstetric Cholestasis early at 28+5 weeks, proteinuria at 30+5 weeks. At 34 weeks she had elevated LFTs. By 36 weeks she had decreased GFR. At 37 weeks she was diagnosed with severe atypical late-onset preeclampsia and the decision was made to stabilize her with MgSO4 prophylaxis. She presented with severely elevated MAP, high proteinuria, epigastric pain, flashing lights, headache and edema. The decision was made for immediate induction and she delivered at 37+4 weeks by instrumental delivery. Although certain risk factors were assessed, OC was diagnosed and treatment with ursodeoxycholic acid was initiated early at 28 weeks, the patient unfortunately still progressed to severe preeclampsia.

Discussion:
These two patients presented with severe atypical PET necessitating rigorous treatment with MgSO4 and immediate delivery. All efforts at early detection and management should be incorporated into practice to avoid such adverse outcomes. It has been demonstrated that preeclampsia can be detected at 11-13 weeks with a high degree of sensitivity [5]. As preeclampsia is a dynamic process, so too are the detection methods. There is no single test, rather the clinical picture helps physicians detect early risks. Effective screening for PE can be provided by a combination of maternal characteristics and obstetric history, uterine artery PI and maternal serum PAPP-A at 11 + 0 to 13 + 6 weeks gestation [5]. One common laboratory screening test is the PreeclampsiaScreen (OC) [9]. The detection rates at 11-13 weeks are far more sensitive for early onset PE (symptomatic before 34 weeks gestation), than late onset PE (symptomatic after 37 weeks) [4]. Akolekar et al. in 2011 found that the detection rate of preeclampsia in the first trimester by a combination of several markers (PAPP-A, PP13, inhibin A, activin A, Mndoglin, PTX3, P-selectin, blood pressure, Dopplersonography, and history) is increased significantly to a detection rate of 91% for early-onset PE and 60% for late-onset PE[5].

There are many risk factors associate with preeclampsia, our patients had several of them. Cholestasis and clotting disorders such as Factor V Leiden are associated with increased risk of preeclampsia [6]. Assessing risk factors early on in pregnancy is vital. The pathological mechanisms causing hepatic impairment in women with preeclampsia may predispose to cholestasis [5], leading to abnormal liver function and complaints of pruritis, which may be the patient only presenting symptom to alert the physician. Risk assessment of common pathologies such as OC may help lead to the early detection by alerting you to perform screening, allowing for prophylactic management of preeclampsia. Once an early diagnosis of preeclampsia is made, 100mg OD aspirin started before 16 weeks was linked with a significant reduction in the incidence of severe preeclampsia (RR 0.10; 95% CI 0.01 to 0.74)[7].

Conclusion:
The results of severe preeclampsia to both mother and baby can be devastating. With laboratory capabilities of preeclampsia detection at 11-13 weeks, it would be desirable in the future to integrate preeclampsia risk calculation to the regular prenatal care in first trimester. The relationship of costs and benefit must be further explored. We support first trimester detection of preeclampsia and further research of its benefit and early management.

References:

http://www.blackwellpublishers.com/advmt/advmt/532452/online/0/1/1862006/advmt532452.htm

Image 1 Image 2 Image 3 Image 4 Image 5 Image 6