

Pre-eclampsia~News We Can Use!

By Tenaya Jackman

Overview

Preeclampsia occurs in 5-8% of all pregnancies in the United States. 75% of cases are mild. 25% of cases are severe. 10% occur in pregnancies less than 34 weeks gestation.

There has been a 40% increase in preeclampsia in the last decade due to multiple gestations/IVF, the obesity epidemic and older moms.

PIH is a term that is no longer recommended as it means different things to different people. There are three types of hypertension that can present in pregnancy.

Chronic hypertension is:

- present before pregnancy or before the 20th week in 3% of pregnancies.
- determined by 2 BP measurements taken at least 6 hrs apart, but no more than 7 days apart
- a systolic BP reading \geq 140
- **OR** a diastolic BP reading \geq 90.
- Although it may be first diagnosed in pregnancy it does not resolve by 12 weeks postpartum.

Gestational hypertension is:

- determined after 20 weeks in 6% of pregnancies
- determined by 2 BP measurements taken at least 6 hrs apart, but no more than 7 days apart
- a systolic BP reading \geq 140
- **OR** a diastolic BP reading \geq 90
- **without** proteinuria.
- Severe is 160/110 or above.

Both mild and severe hypertension require medical management including weekly labs and antenatal testing. Severe hypertension has greater morbidity than mild preeclampsia, including increased risk

for abruption, preterm delivery and small for gestational age babies. A high systolic blood pressure is a better indication of stroke risk than diastolic.

Preeclampsia is a syndrome characterized by:

- elevated blood pressure - 2 measurements taken 6 hrs apart, but no more than 7 days apart
- systolic BP reading \geq 140
- **OR** a diastolic BP reading \geq 90
- proteinuria – \geq 300 mg of protein in a 24 hour specimen.
- typically corresponds with \geq +1 urine dip stick
- appears in pregnancy after 20 weeks

What's new?

The cause of preeclampsia remains unknown. Vascular, environmental, immunological, and genetic factors all appear to play a role. The current understanding of what causes the problems seen in preeclampsia is that the cytotrophoblastic tissue of the placenta does not adequately migrate down the uterine spiral arteries and so they fail to develop into large channels. Instead, the vessels remain narrow, resulting in placental hypoperfusion, or decreased blood flow. Hypoperfusion becomes more pronounced as pregnancy progresses since the abnormal circulatory system of the uterus is unable to accommodate the normal rise in blood flow to the fetus/placenta with increasing gestational age. Decreased placental perfusion and resulting restriction in blood supply leads to overproduction of anti-angiogenic (against blood vessel growth) proteins, sFLT and endoglin, that control placental growth, around 26-28 weeks. This in turn damages the endothelium, the thin layer of cells that lines the inner surface of blood vessels.

All of the clinical features of preeclampsia can be explained as responses to generalized endothelial dysfunction - an imbalance between vasodilating and vasoconstricting substances in the inner lining of blood vessels that leads to irritation and spasm, increasing blood pressure and causing poor tissue perfusion in all organ systems. Headache, seizures, visual symptoms, epigastric pain, and fetal growth restriction are the result of endothelial dysfunction in the blood vessels of organs, such as the brain, liver, kidney, and placenta.

The only cure for preeclampsia is for the pregnancy to end. If the baby is preterm the goal of medical management is to slow or stop the progression of preeclampsia, prevent eclampsia and HELLP syndrome while giving the baby time to develop. The severity of the preeclampsia determines when induction is recommended at term.

A decreased platelet count and rising liver enzymes are used to increase the certainty of the diagnosis of preeclampsia and diagnose HELLP syndrome. An elevated hematocrit is also used in the evaluation of preeclampsia. Uric acid has not been found to be helpful.

No significant benefit has been found in:

- Vitamin C and E
- Calcium/magnesium
- Fish oil
- High protein diet

Although preeclampsia is not the result of malnutrition or inadequate protein, high protein diets promote cell growth, replace the protein lost in the urine and lower the risk for hypertension. Resting in bed on left side lie takes the pressure of the uterus off the inferior vena cava, facilitating venous return and increasing the amount of blood flowing through the kidneys. Blood pressure normally drops as a result and blood flow is enhanced to the placenta and baby.

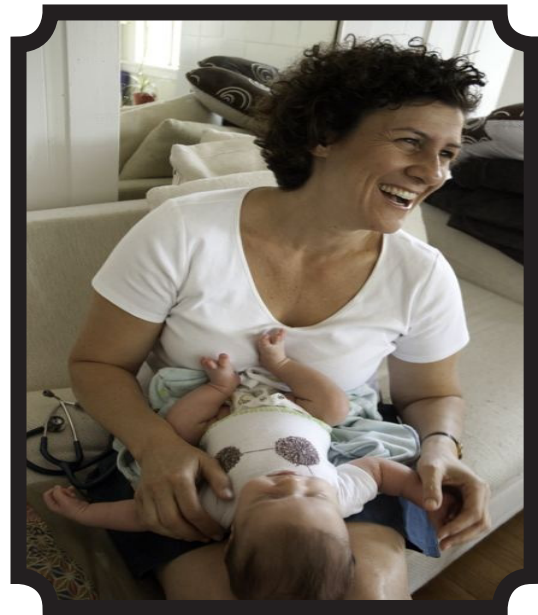
Summing up

This new evidence that preeclampsia results from decreased placental perfusion caused by abnormal uteroplacental vasculature overturns what many of us were taught and requires a reorientation to

this problem. It suggests that our role is in early identification of the problem and understanding that preeclampsia cannot currently be prevented. There is a NIH study underway involving 10,000 women looking for a prediction/ screening test and treatment.

References

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Tenaya Jackman became fascinated by midwifery while volunteering in rural health centers in Ghana, West Africa. She received a BA in Anthropology from the University of California, Santa Cruz and a Masters of Public Health from the UC Berkeley School of Public Health. She completed her midwifery training in 2000 at Maternidad La Luz, a birth center based, MEAC accredited program in El Paso, Texas. In 2002 she moved to Hawaii and began Hawaii Births, a midwifery and doula practice. She returned to the Bay Area in April of 2007 to practice midwifery. She is a member of the Bay Area Homebirth Collective and a board member of the California Association of Midwives.