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EDITORIAL

Why hypertension is good new and preeclampsia bad news-demonstrating the failure of prevention

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Abstract

Hypertensive disorders in pregnancy continue to be an intriguing and potentially lethal complication in humans and some other primates. In a simplistic way the current hypothesis is that the genesis of preeclampsia starts at 12 to 14 wk gestation with failure of trophoblast invasion in the spiral arteries, resulting in some degree of hypoxemia in the placenta. The hypoperfused placental tissue starts to secrete variable amounts of angiogenic and antiangiogenic factors which eventually cause endothelial damage all over the pregnant women's body with one of the many signs of preeclampsia as the clinical endpoint. For some incomprehensible reason a major interest has existed for decades concerning the early prediction of preeclampsia, most commonly tested using uterine artery Doppler (the earlier the better) and various serum markers, alone or in combination. Any new model for detection has been welcomed enthusiastically, although nothing has changed in the outcome of women presenting with preeclampsia.

Key words: Hypertensive disorder; Pregnancy; Preeclampsia

Hypertensive disorders in pregnancy continue to be an intriguing and potentially lethal complication in humans and some other primates[1]. In a simplistic way the current hypothesis is that the genesis of preeclampsia starts at 12 to 14 wk gestation with failure of trophoblast invasion in the spiral arteries, resulting in some degree of hypoxemia in the placenta. The hypoperfused placental tissue starts to secrete variable amounts of angiogenic and antiangiogenic factors which eventually cause endothelial damage all over the pregnant women's body with one of the many signs of preeclampsia as the clinical endpoint[2]. For some incomprehensible reason a major interest has existed for decades concerning the early prediction of preeclampsia, most commonly tested by uterine artery Doppler (the earlier the better) and various serum markers, alone or in combination. Any new model for detection has been welcomed enthusiastically, although nothing has changed in the outcome of women presenting with preeclampsia. But is the news really so bad?

We and others have demonstrated that actual birth weight in cases of gestational hypertension, which do not develop into preeclampsia, tends to be higher than in normotensive women[3,4]. One possible explanation for this is that the originally hypoperfused placenta is highly successful in increasing its blood supply by secreting angiogenic factors. Isolated gestational hypertension is good news and it is only when the rescue mechanism activated by the placenta fails, that preeclampsia will develop with

all the well-known detrimental effects including fetal growth retardation, preterm birth, fetal death, and possibly maternal convulsions and death.

If some kind of endothelial damage makes the difference, then almost anything that has a vascular effect (and most drugs, nutritional supplements and physical exercise have been shown to influence blood vessels) can be (and has been) tried as a measure to prevent the development of preeclampsia. Possibilites range from calcium to calcium blockers, from aspirine to NO, any vitamin or food supplement such as arginine, from bed rest to physical training.Both individual studies and meta-analyses are often conflicting. For example, Trivedi *et al*[5] recently concluded that low dose aspirin resulted in a significant reduction of preeclampsia in high risk patients, while another meta-analysis which was published almost simultaneously on the same subject by Rossi *et al*[6] concluded that low dose aspirin does not lead to less preeclampsia in high risk women. At least we all agree that aspirin should not be given to low risk women. Not only does this demonstrate that a disturbing level of subjectivity makes our current model and methods for evidence based medicine fail in providing clinicians a tool to guide their management, but it also shows that we simply have no idea what is really happening in pre-eclampsia. The really bad news is that when one critically considers all preventive measures that have ever been proposed, we are left with nothing to offer our patients except "promising news", as promising as it has been for the last 30 years.

As long as we do not understand why trophoblast invasion fails, and why not all preeclamptic placentae show failed trophoblast invasion, we will continue to be unable to differentiate between the good news of a compensatory hypertension and the bad news of the overshooting reaction that we call preeclampsia.

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