STA55. Pathophysiology of preeclampsia from the view point of immunology

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Introduction: The pathogenesis of preeclampsia remains largely unknown. However, many researches support the immune maladaptation hypothesis. Epidemiological findings show that first pregnancy, the use of barrier contraceptive method or short cohabitation, oocyte donation cases and obesity are the risks for preeclampsia. These factors seem to be independent.

Objective: We tried to explain the relationship between the epidemiological risks for preeclampsia and immune maladaptation.

Method: We have studied the immune system in preeclamptic cases and oocyte donation cases using flow cytometry and immunohistochemical examination.

Results: Human pregnancy represents a semiallograft to the maternal host, therefore tolerance system is required. In oocyte donation cases, all the MHC of the fetus are allograft to maternal therefore more strict tolerance system is needed. Nevertheless, our study showed the number of regulatory T (Treg) cells which induce tolerance were scare at fetomaternal interface. Moreover, macrophage, T cell and NK cells that play important roles for vascular remodeling were also scarce in placental bed biopsy samples. Importantly, vascular remodeling was inadequate in oocyte donation cases regardless of the presence or absence of preeclampsia. These findings suggest that macrophage, T cell and NK cells play an important role for vascular remodeling of spiral artery and poor placentation is present in oocyte donation pregnancy. We also showed that effector Treg cells decreased and exhausted Treg cells increased in peripheral blood of preeclampsia. Expression of Bcl-2 in Treg cells was decreased and expression of Bax in Treg cells was increased suggesting that Treg cells in preeclampsia are more likely to die by apoptosis.

In our mice model, seminal plasma plays an important role for induction of paternal antigen specific Treg in the uterus. This finding explains barrier contraceptive method or short cohabitation are the risk of preeclampsia.

We also showed the positive relationship between BMI and the frequencies of cytotoxic T cells and cytotoxic NK cells.

Conclusion: These findings support that immune maladaptation is one of the mechanisms of preeclampsia.

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PL56. Long-term consequences of preeclampsia Annetine Staff (University of Oslo, Oslo, Norway)

Introduction: There is a clearly documented increased risk of long-term maternal cardiovascular disease after pregnancy complications such as preeclampsia, premature birth and fetal growth restriction. The risk is highest in pregnancies with both maternal and fetal manifestations of abnormal placentation. We lack today however the mechanistic understanding of the association. We also lack the evidence-based recommendations on how and how frequent to follow up these women at risk, both pre- and postmenopausally.

Objective and methods: The talk will briefly review the epidemiological associations, with emphasis on cardiovascular disease (CVD) after preeclampsia. In addition, the talk will review common risk factors for preeclampsia and CVD, as women developing preeclampsia may have risk factors in common with older persons developing CVD. Additionally, the talk will suggest how preeclampsia and other

placentally-mediated disorders may themselves contribute to an augmented cardiovascular burden that may directly or indirectly affect long-term vascular health.

Results: Further understanding of the process underlying maternal and placental mediators of preeclampsia may help cast light on development of cardiovascular disease later in life. Further research is needed to ascertain whether specifically targeted group of women with such pregnancy complications benefit from prevention strategies, such as with oral statins or aspirin, similarly to other population groups at risk, and thereby improve long-term maternal health.

Conclusion: This talk will suggest how longitudinal pregnancy cohorts and biobanks across the world may improve the understanding of CVD in parous women. Options for such studies from pregnancy biobanks within the Global CoLaboratory research network are presented.

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PL59. The impact of classification of hypertensive disorders of pregnancy based on the ACOG 2013 and ISSHP 2014 criteria János Rigó Jr., Bálint Alasztics, Anikó Árokszállási, Noémi Dobó, Mária Prosszer, József Gábor Joó, Attila Molvarec (Semmelweis University, 1st Department of Obstetrics and Gynaecology, Budapest, Hungary)

Introduction: A standardized classification of hypertensive disorders of pregnancy would be highly needed to achieve the comparability of the studies and the adequate management of the patients. In the last two years, the American College of Obstetricians and Gynecologists (ACOG), as well as the International Society for the Study of Hypertension in Pregnancy (ISSHP) revised the classification of hypertensive disorders in pregnancy.

Objectives: In Hungary, the ACOG 2002 criteria were used in the last decade to classify hypertensive disorders during pregnancy. This study aimed to determine the impact of the ACOG 2013 and ISSHP 2014 criteria on the occurrence of different forms of hypertensive disorders, as well as on perinatal outcome compared to the ACOG 2002 criteria.

Methods: All pregnant women with hypertensive disorders and singleton pregnancies who delivered in the 1st Dept. of Ob-Gyn. at the Semmelweis University between 1 Jan 2012 and 31 Dec 2014 (n = 755) were enrolled in this study. We determined the prevalence of different forms of hypertensive disorders according to the ACOG 2002, ACOG 2013 and ISSHP 2014 criteria. We also examined the reasons for re-classification of chronic hypertensive (CHT) and gestational hypertensive (GHT) patients to the preeclampsia (PE) group, as well as its impact on the perinatal outcome.

Results: The extended definition of PE according to ACOG 2013 and ISSHP 2014 classifications raised the incidence of PE by 8,2% and 17,2% compared to the ACOG 2002 classification.

The most frequent cause of re-classification of GHT and CHT patients to the PE group were abnormal laboratory findings in 35% of cases according to ISSHP 2014 classification and subjective symptoms in 36% of cases according to ACOG 2013 classification.

The median values of fetal birth weight were significantly lower in women with GHT (3330 g (IQR: 2985–3750 g) vs. 2735g (IQR: 2125–3375 g), p < 0.001) and CHT (3330 g (IQR: 2980–3650 g) vs. 2605 g (IQR: 1720–3350 g), p < 0.001) who were re-classified to the PE group compared to those who remained in the original group. There was no statistically significant difference between the median values of gestational age at delivery and birth weight in PE patients based on ACOG 2002 and ISSHP 2014 classifications: 36 weeks (IQR: 32–38 weeks) vs. 36 weeks (IQR: 33–38 weeks), 2450 g (IQR: 1540–3180 g) vs. 2530 g (IQR:1650–3260 g).