PIH + IUGR. Student's *t*-test was utilized to reveal the concordance between fetal renal vascularisation in normal and complicated cases.

Results: The mean values of fetal renal vascularisation decreased in complicated cases.Significant difference ($p\langle 0.01$) could be observed between pregnancies complicated by PIH ($VI_{mean\pm SD}$:5.15 ± 0.59%, $FI_{mean\pm SD}$:35.75 ± 3.77, $VFI_{mean\pm SD}$:1 ± 0.27), PIH + GDM ($VI_{mean\pm SD}$:2 ± 0.32%, $FI_{mean\pm SD}$:29 ± 02.8, $VFI_{mean\pm SD}$:0.5 ± 0.12) and PIH + IUGR ($VI_{mean\pm SD}$:2.3 ± 0.57%, $FI_{mean\pm SD}$:31.6 ± 6.8, $VFI_{mean\pm SD}$:0.7 ± 0.17).The mean values (±SD) of fetal renal volume were significantly less in pregnancies complicated by PIH + GDM, and more in PIH + IUGR pregnancies compared to PIH cases (mean values (±SD): V_{PIH} :15 ± 7 ml, $V_{PIH + GDM}$:13 ± 4 ml, $V_{PIH + IUGR}$:8.5 ± 2.1 ml ($p < 0.05$). There were no significant differences ($p < 0.01$) between the right and left renal vascularisation indices.

Conclusion: Fetal renal vascularisation indices were significantly diminished in GDM and IUGR cases. Fetal downregulated vascularisation had influence on fetal renal development, thus on fetal renal volumes. PIH has a harmful effect not only on maternal, but also on fetal morbidity.

doi:10.1016/j.preghy.2015.07.083

O134. Polycystic ovary syndrome as a risk factor of pregnancy induced hypertension – Review of the literature

Szabolcs Várbíró [a], Eszter Horváth [b], Nándor Ács [a] ([a] Semmelweis University, 2nd Department of Obstetrics & Gynecology, Budapest, Hungary, [b] Institute of Human Physiology and Clinical Experimental Research, Semmelweis University, Budapest, Hungary)

Introduction: Polycystic ovary syndrome (PCO) seems to be a special form of metabolic syndrome that occurs in fertile women. It is accompanied with an increased risk for obesity, lipid- and glucose metabolism disorders, and hypertension. Beyond the hyperandrogenic state, other mechanisms, such as oxidative-nitrosative stress or vitamin D deficiency, might also have key roles in the pathogenesis of PCO and its consequences. These parameters are also involved in the development of preeclampsia. The apparent common characteristics of the two diseases might suggest their interrelation.

Methods: In this review, we list the common risk factors of polycystic ovary syndrome and preeclampsia, try to calculate the risk of preeclampsia in patients with polycystic ovary syndrome and its possible therapeutic consequences.

Results: Most of the authors agree, that presence of polycystic ovary syndrome elevates the risk of preeclampsia, however, some of them still debated it. Vitamin D deficiency and increased oxidative-nitrosative stress that can be observed in PCOS may be at least partly responsible for the increased probability of preeclampsia development among these patients. Upon the literature we summarize the optimal preconceptional management of patients suffering from polycystic ovary syndrome for lowering the risk of preeclampsia.

Conclusion: As PCOS is accompanied by an increased risk for adverse pregnancy outcomes such as preeclampsia, these women are patients of special obstetrical care.

Acknowledgements: OTKA-PD 113022, Eszter M. Horváth is supported by the János Bolyai Hungarian State Research Fellowship, Szabolcs Várbíró is supported by the Research Grant of the Hungarian Society of Hypertension (2015–16).

doi:10.1016/j.preghy.2015.07.084

O135. HELLP??? – A case

Eva-Christine Weiss, Gordana Tomasch, Uwe Lang, Wolfgang Schöll (Medical University of Graz, Department of Obstetrics & Gynecology, Graz, Austria)

Introduction: HELLP affects about 1% of all pregnancies and up to 20% of pregnant women with severe preeclampsia/eclampsia. Clinical symptoms are typically associated with right upper epigastric pain, headache and visual disturbance. In about 15–20% patients present without hypertension and/or proteinuria.

Objectives: After delivery symptoms and lab tests of HELLP resolve gradually within 3–4 days, in case of persistent disease awareness for other thrombotic micorangiopathies has to be raised.

Methods: We present a case of a 26 year old primigravida, who presented in her 35+1 week of gestational age in our labour ward with epigastric pain and starting labour. Further clinical signs (mild hypertension and proteinuria) and her massively deranged lab results suggested severe HELLP with haemolytic anaemia and profound thrombocytopenia (9G/l). After ordering a thrombelastogram (TEG) and short preparation with cortison and blood products an uneventful cesarean section was performed. A healthy boy was born (2480 g/47 cm length; Apgar 7/8/10; umbilical a/v pH 7.29/7.30), but unfortunately his mother's recovery took an unexpected course, continuing lab tests suggested a thrombotic microangiopathy (TTP/aHUS) and accordingly therapy was started, but nevertheless she died on day 13 after delivery.

Results: We discuss her disease, management and differential diagnosis in review of the literature.

Conclusions: In pregnant patients with severe thrombocytopenia and microangiopathic haemolytic anaemia and other symptoms suggesting severe HELLP other thrombotic microangiopathies have to be excluded.

doi:10.1016/j.preghy.2015.07.085

P33. Decreasing of placental progesterone induced blocking factor expression and spiral artery remodeling disturbance in mice preeclampsia model

Manggala Pasca Wardhana [a], Budi Wicaksono [a], Erry Gumilar Dachlan [a], Muhammad Ilham Aldika Akbar [a], Ernawati [a], Agus Sulistyono [a], Aditiawarman [a], Hermanto Tri Juwono [a], Widjiati [b] ([a] Airlangga University, Department of Obstetrics and Gynecology, Surabaya, Indonesia , [b] Airlangga University, Medical Veterinary Faculty, Surabaya, Indonesia)

Introduction: Preeclampsia is a major cause of morbidity and mortality in our developing country. The biggest obstacle in the high prevalence of preeclampsia and its management is due to lack of knowledge in preeclampsia pathologic mechanism. Progesterone is an important pregnancy hormone that has immunomodulatory effect by inducing progesterone induced blocking factor (PIBF) release, due to binding of progesterone with its receptor in lymphocyte. PIBF inhibits cytolytic NK cells activity and inducing T-helper 2 cytokine dominant while on contrary, preeclampsia has a condition of T-helper 1 cytokine dominant and increase of NK cells cytolytic activity. This was the first study to evaluated PIBF expression directly on the placenta when the pathogenesis of preeclampsia occurred after completion of vascular remodeling in animal models.

Objective: The objectives of this study were to compare placental PIBF expression, spiral artery wall thickness as characteristic of vascular remodeling disturbance and investigated correlation between both in mice preeclampsia model.

Methods: This experimental study used 32 pregnant *mus musculus* mice and randomly divided to normal group and preeclampsia model group. Our preeclampsia model was created by injecting anti-Qa2 (anti HLA-G) 10 ng from 1st until 4th day of pregnancy, to reduced HLA-G expression. Termination of both groups were

performed after completion of spiral artery remodeling in mice by day 16th of pregnancy followed by immunohistochemistry examination for PIBF expression using *immunoreactive score* and spiral artery wall thickness measurement. Ethical clearance was taken from Medical Veterinary Faculty ethics commission. Difference and correlation were analysed using SPSS 20. Probability values <0.05 were considered statistically significant.

Result: In the result, PIBF were expressed in trophoblast, decidual and lymphocyte cells in placental tissue. The placental PIBF expression on preeclampsia model (2.01 ± 1.14) was significantly reduced ($p \pm 0.01$) compared with control (3.99 ± 2.53). There was a significant increase of spiral artery wall thickness ($p \pm 0.001$) in preeclampsia model (20.70 ± 5.93 micrometers) compared with control (9.98 ± 3.36 micrometers). There was a significant association ($p \pm 0.001$) with negative correlation (rho/Pearson correlation = −0.623) between placental PIBF expression and the spiral artery wall thickness.

Conclusion: We concluded a decreased expression of placental PIBF and the increase of spiral artery wall thickness as a characteristic of spiral artery remodeling disturbance in mice model of preeclampsia, with negative correlation between placental PIBF expression and spiral artery wall thickness.

doi:10.1016/j.preghy.2015.07.086

P34. Role of high-mobility group A1 protein in trophoblast invasion

Yuka Uchikura [a], Keiichi Matsubara [a], Yuko Matsubara [a], Miki Mori [b] ([a] Ehime University, Department of Obstetrics and Gynecology, Toon, Japan, [b] Ehime Prefectural Central Hospital, Department of Obstetrics and Gynecology, Matsuyama, Japan)

Objectives: Early in pregnancy, abundant neovascularization at the implantation site is important for normal placentation. For placental development and reduction of uterine vascular resistance, cytotrophoblasts must migrate to the decidua and the uterine myometrium, with subsequent invasion of the spiral arteries resulting in the spiral artery remodeling. Especially, extravillous trophoblasts (EVT) invade into the uterine decidual spiral arterioles and regulate the remodeling of these vessels for fetal blood supply. While, disturbed arterial remodeling can lead to the serious complications such as preeclampsia and fetal growth restriction. High-mobility group A1 protein (HMGA1) is known to play an important role in the proliferation of trophoblast; however, no specific function of HMGA1 has been reported for trophoblast invasion. The aim of this study was to evaluate the effect of HMGA1 on trophoblast invasion.

Methods: We investigated HMGA1 expressions in cytotrophoblast derived from preeclampsia model mouse, CD40-L mouse, using immunofluorescence analysis. Wound healing cell migration and matrigel invasion test was also performed using HTR-8/SVneo cells transfected with HMGA1 plasmid.

Results: HMGA1 was expressed in nucleus of trophoblasts derived from control mouse; in contrast, cytoplasmic expression was observed in CD40-L mouse. HMGA1 plasmid stimulated cell migration and invasion in HTR/SVneo cells; however, extranuclear translocation of HMGA1 suppressed cell migration and invasion.

Conclusions: Cellular localization disorder of HMGA1 resulted in a lowering of its invasions and migration. Transfer of HMGA1 in cytotrophoblast is crucial for the trophoblast invasion into decidua in preeclampsia.

doi:10.1016/j.preghy.2015.07.087

P35. Novel interaction of placental caveolin-1 expression with markers of oxidative stress and the renin-angiotensin system (RAS) in pre-eclampsia

Hiten Mistry [a], Anna Czajka [b], Marta Hentschke [c], Carlos Poli-de-Figueiredo [c], Bartira Pinheiro da Costa [c], Fiona Broughton Pipkin [d], Lesia Kurlak* [d] ([a] University of Bern, Department of Nephrology, Hypertension and Clinical Pharmacology, Berne, Switzerland, [b] King's College London, Diabetes Research Group, London, UK, [c] PUCRS, Laboratory of Nephrology, Porto Alegre, Brazil, [d] University of Nottingham, Department of Obstetrics & Gynecology, Nottingham, UK)

Introduction: Caveolin-1 (cav-1) is one of the major protein components of caveolae required for the coordination of some signalling pathways, including that of angiotensin II (AngII) with its Type 1 receptor (AT1R). We have previously reported decreased cav-1 and increased AT1R protein expression in placentae from women with pre-eclampsia (PE). Within the PE placenta, an increase in AngII acting via AT1R may lead to increased vasoconstriction, but possible functional effects of altered cav-1, and interactions with components of RAS and markers of oxidative stress have not been investigated.

Objective: To establish mechanistic interactions of placental cav-1 with RAS components and surrogate markers of oxidative stress and antioxidant concentrations.

Methods: Immunohistochemistry was performed on paraffin-embedded serial placental sections from 24 normotensive (NC) and 19 women with PE, using antibodies to cav-1, prorenin receptor (PRR), angiotensinogen (AGT), AT1R, AngII type 2 receptor (AT2R) and eNOS. Protein expression was semi-quantitatively assessed and also analysed with respect to previously measured maternal plasma TBARS (Thiobarbituric acid-reactive substances) concentration and placental glutathione peroxidase (GPx) activity.

Results: Positive correlations were observed between placental expression of cav-1 and the AT2R ($P = 0.003$) and PRR ($P < 0.0001$) in NC; these were lost in PE ($P > 0.7$, $P > 0.5$). However, cav-1 was inversely correlated with eNOS expression in both NC and PE placentae ($P < 0.001$, $P = 0.003$). No significant correlations were observed with AGT or AT1R. A negative correlation was observed between maternal TBARS and cav-1 protein ($P = 0.003$), and between placental GPx activity and cav-1 protein ($P = 0.027$) only in the PE placentae.

Conclusions: A complex cascade links activation of the AT1R with NADPH oxidase activation, generation of reactive oxygen species (ROS), the uncoupling of eNOS and subsequent modification of redox-sensitive caveolar proteins in vascular smooth muscle outside pregnancy. Our findings suggest that this cascade is altered in hypertensive pregnancy, in a direction which would lead to enhanced local vasoconstriction.

doi:10.1016/j.preghy.2015.07.088

P36. Identifying a novel link between preeclampsia and chronic hypertension in the MTHFR-gene using the population based Norwegian HUNT Study

Liv Cecilie Thomsen [a], Nina McCarthy [b], Phillip Melton [b], Gemma Cadby [b], Rigmor Austgulen [c], Eric Moses [b], Line Bjørge [d], Ann-Charlotte Iversen [c] ([a] Norwegian University of Science and Technology (NTNU), Centre of Molecular Inflammation Research, Bergen, Norway, [b] University of Western Australia, Centre for Genetic Origins of Health and Disease, Perth, Australia, [c] Norwegian University of Science and Technology (NTNU), Centre of Molecular Inflammation Research, Trondheim, Norway, [d] Haukeland University Hospital, Department of Obstetrics & Gynecology, Bergen, Norway)