

Decidual degeneration - 11 cases

DCH - 5 cases

Sever CAM - 4 cases

Abruptio placentae - 2 cases

Delivery of all 37 cases was 22 to 40 gestational weeks

**Conclusions:** There are many premature deliveries which present hypoxia leading to ventricular hemorrhage and PVL. These brain damages can lead to CP. Through placental pathology we found bad circulation between the placenta and the baby. We can definitely conclude that placental pathology can contribute to our understanding of CP prediction and protection.

## 21.

### FINDINGS REGARDING THE HIGH RATES OF CASES OF DECIDUAL PATHOLOGIC LESIONS AT 34 GESTATIONAL WEEKS OR EARLIER DEVELOPING INTO HYPERTENSION DISORDER OF PREGNANCY (HDP)

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**Object:** I examined the placentas in cases of Hypertension Disorder of Pregnancy (HDP) at 34 gestational weeks or earlier in order to help discover causes and treatment

**Method:** I examined the placentas of 118 cases of HDP at 34 gestational weeks or earlier - defined by standard diagnosis - by microscope.

**Results:** Out of 118 cases, atherosclerosis, thrombosis, and decidual degeneration was found in 98 cases –83.1 %

Ischemic villi was found in 90 cases –76.3%

Villous vessels abnormality was found in 40 cases - 33.9%

Severe Chorioamnionitis(CAM) could not be found

**Conclusions:** Problems with decidual vessels from the mother to the placenta at 34 gestational weeks and earlier leads to a high rate of HDP.

According to the Amsterdam Conference 2014, the definition of MVM is bad circulation leading to ischemic villi, syncytial knots increase, and the development of fibrin deposition. This definition of MVM applies to bad maternal circulation affecting the placenta.

We also found FVM which resulted from MVM, as the degeneration of the terminal villi led to occlusion in the central villous vessels. This developed into other parts of the terminal villi.

According to the Amsterdam conference, MVM and FVM are different phenomena. However this study shows that the two are connected.

Also in this study I found no sign of CAM. I suspect the reason is immunological relations.

## 22.

### EXTRAPLACENTAL INTER-TWIN ANASTOMOTIC VESSELS IN MONOCHORIONIC DIAMNIOTIC TWINS

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**Objective:** Inter-twin anastomotic vessels that were observed on the outside of placental parenchyma could contribute to residual anastomotic vessels during fetoscopic laser photocoagulation (FLP). The objective of this study was to elucidate the prevalence of extraplacental inter-twin anastomotic vessels and to examine the relative factors.

**Methods:** We conducted retrospective study of MD twin cases whose placentas were inspected postnatally. Cases that underwent FLP or with fetal demise were excluded. An anastomotic vessel which was located out of placental parenchyma by macroscopic inspection was defined as extraplacental inter-twin anastomotic vessels. The prevalence of extraplacental inter-twin anastomotic vessels was examined and association with factors including umbilical cord insertion was analyzed with Fisher's exact test.

**Results:** One-hundred and twenty eight cases were included. The prevalence of extraplacental inter-twin anastomotic vessels was 5.5% (seven cases). The relative factor of extraplacental inter-twin anastomotic vessels was velamentous cord insertion of both fetuses (P=0.035). Extraplacental

inter-twin anastomotic vessels were found in two of six cases with VCI of both fetuses.

**Conclusion:** Extraplacental inter-twin anastomotic vessels were found in 5% of MD twins, therefore it might be recommended to observe placenta during FLP in light of anastomotic vessels that were difficult to be found. It might be useful for the prediction of extraplacental inter-twin anastomotic vessels to understand umbilical cord insertion preoperatively.

## 23.

### ELEVATION OF ANGIOGENIC FACTORS IN PREGNANCY WITH MIRROR SYNDROME CAUSED BY FETAL CARDIAC FAILURE

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**Introduction:** Mirror syndrome (MS) is characterized by the combination of maternal generalized edema, fetal hydrops and placental hypertrophy. Elevation of soluble Flt-1 or soluble Endoglin in preeclampsia is also reported to be related to the onset of MS.

We experienced a MS case due to fetal cardiac failure with elevation of angiogenic factors.

**Case:** A 27-year-old woman, gravida1, para0, had prenatal care in a local clinic. At 26 weeks of gestation, small amount of fetal ascites was observed. At 28 weeks of gestation, she had dyspnea with weight gain and fetal ascites apparently increased. She was transferred and admitted in our hospital. Fetal ultrasound examination revealed ascites, pericardial fluid ascites, subcutaneous edema, and placental hypertrophy. Hypokinetic biventricular movement and the thinning of the myocardium were detected in the fetus, suggesting the cardiac dysfunction.

She had an emergency cesarean section on the same admission day. The placenta was edematous and weighed 460 g. In microscopic examination, stromal edema was observed in the majority of the villi.

Elevation of soluble Flt-1 (7580pg/ml) and soluble Endoglin (25.4 ng/ml) was detected in the maternal serum on the admission day.

**Discussion:** We experienced a MS case due to fetal cardiac failure. A rapid progress of fetal cardiac failure and hydrops makes placenta blood flow disrupted. Disruption of placental blood is related to elevation of angiogenic factors and clinical presentation such as maternal edema. The angiogenic factors may be a key of MS pathogenesis.

## 24.

### LACTOBACILLUS CRISPATUS PROMOTES TROPHOBLAST INVASION

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Human vaginal cavity has a unique microbiome dominated by Lactobacilli. Recent reports show non pregnant uterine cavities and placentae also possess unique microbiome dominated by Lactobacilli. Their reduction frequently observed in the patients with bacterial vaginosis (BV) as well as uterine infection such as endometritis and chorioamnionitis are great concern for reproductive health. In the presented study, we examined tissue-bacterial interactions of immortalized human trophoblasts and Lactobacilli in order to analyze their roles on trophoblast functions.

**Methods:** HTR-8/SVneo cells were plated on Matrigel chambers with or without Lactobacillus crispatus (LC), Lactobacillus acidophilus (LA), Escherichia.coli and Staphylococcus aureus. The invasive activity was directly evaluated using the microscopy and the time-lapse imaging using by the IncuCyte® system. Genes induced by Lactobacilli were screened by microarray and then verified with real time PCR and ELISA.

**Results:** Matrigel invasion of HTR-8/SVneo cells was up-regulated significantly by LC while others were not significantly promotive or cytotoxic.