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 knotsincrease, 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 fectors 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.

Expression of invasion related MMP-1 was also up-regulated by LC and increased expression of active form of MMP-1 was detected in LC treated cells.
Conclusion: Our results suggest that Lactobacillus crispatus promotes trophoblast invasion via up-regulation of MMP-1. Our findings suggest indispensable roles of LC which dominate in healthy human vagina. Our results can explain why BV and other lower female genital tract infections harm successful human pregnancies.

25.
SUPPRESSYN MAY PLAY A CENTRAL ROLE IN TROPHOBLAST CELL FUSION RESPONSE TO OXYGEN ENVIRONMENTS

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Objective: We previously described and analyzed the function of a novel anti-fusogenic protein suppressyn(SUPYN) in cultured trophoblast cells. The physiological state of the placenta is hypoxic varying between 2 and 5% in early pregnancy. Here we performed expression analysis of cell fusion related genes to determine whether SUPYN may play a role in trophoblast syncytialization response to hypoxia and hyperoxia.
Methods: Primary trophoblast cells were isolated from term placenta and cultured under several O_2 conditions. RNA transcripts were collected every 24 hours and subjected to semi-quantitative RT-PCR. A newly-established SUPYN specific ELISA was used to detect alterations in cell associated and secreted forms of SUPYN protein during cell fusion.
Results: Fusion of primary trophoblast cells was predominantly suppressed under the range of "physiologic" hypoxic conditions and both syncytialization and hCG gene expression were lower than in higher O_2 environments. Expression of syncytin-1 was lower but SUPYN transcripts higher in low O_2 environments. This phenomenon was confirmed at the protein for both cell-associated and secreted SUPYN.
Conclusion: Converse syncytin-1 and SUPYN transcriptional and translational responses to surrounding oxygen concentrations suggest both are important in the effects of hypoxia and hyperoxia on placental syncytialization. Further analysis of these fusion-associated transcripts under different O_2 condition will likely improve our understanding of placental physiology and placental diseases like pre-eclampsia and PIH.

26.
A CASE WITH HYDRAMNION AND DIAGNOSED AS PLACENTAL HEMANGIOMA AFTER DELIVERY

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Introduction: Hydramnion is known to be caused by diabetes or fetal congenital malformation, but in rare case, large placental hemangioma can induce hydramnion. We experienced a case of hydramnion caused by placental hemangioma.
Case: A 36-year-old primipara. She had a prenatal care in Australia until 32 weeks, and since then at a midwifery clinic in Japan.On 37 weeks and 1 day, she visited the clinic for amniotic fluid runoff. At that time, hydramnion was recognized(AFI 27.5), so she was transfered to our hospital. Her HbA1c and glycoalbumin were normal, and no apparent malformation was found in fetus. Caesarean section was performed at 37 weeks and 5 days because of induction failure.The baby was born at 3350g, Apgar score 9/9, UA-pH 7.373, and a boy. He had a heart failure and was admitted to NICU for 7 days. No obvious malformation was pointed out, but infantile hemangioma was found throughtout the body. A 13.5×10.5×5.5cm well-defined nodular lesion was found in placenta. The pathological diagnosis was placental hemangioma.
Discussion: Placental hemangiomas are found in about 1% of the placenta, and if the tumor diameter is more than 5cm, perinatal complications such as hydramnion may be induce.
In neonates, they may have a heart failure like this case, and there are hemangiomas in the viscera which required treatment in rare case.This case suggests that placental hemangioma should be considered in case of hydramnion. Additionally, neonatal heart failure or hemangiomas may be observed, so advanced facilities is desirable for delivery.

27.
PROGNOSTIC FACTORS FOR THE OUTCOMES OF LOW-LYING PLACENTA BY TRANSVAGINAL ULTRASOUND

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Objectives: Low-lying placenta (LLP) are regarded as high-risk conditions of pregnancy. However, lower uterine segment extension for placental migration related to LLP remain unclear. In this study, we searched placental position and findings on ultrasound on LLP.
Methods: We retrospectively reviewed the cases of LLP for the past 5 years from medical records (2013-2017). Location of placenta, cervical length and spongeous(S-) findings suggest venous plexus were defined. Mode of delivery, blood loss and placental weight were also searched.
Results: In our cases, 33 LLP were located on posterior wall attachment and only one was on anterior. This anterior-located LLP with S-finding caused to massive bleeding which massive blood transfusion and 3 times UAE were needed. In contrast, only one case in 33 posterior cases needed transfusion. Average cervical length at diagnosis was significantly longer in the group with S-finding (n=13, 40.2 mm) compared to no S-finding group (n=21, 32.7 mm, p=0.01). Caesarean section was performed in all cases with S-findings. Vaginal delivery was performed in 5 cases in the other group. There was no difference in the amount of blood loss, infant birth weight or placental weight.
Conclusions: Surprisingly, almost all LLP located on posterior wall; anterior-located low-lying placenta showed miserable outcome on delivery. S-finding correlated with cervical length, suggesting that failed uterine lower segment extension contribute to placenta migration can be predicted from this ultraspound finding.

28.
ROLE OF PLACENTAL ATX-LPA SYSTEM AND ITS PATHOLOGIC RELEVANCE TO HYPERTENSIVE DISORDERS OF PREGNANCY

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Objective: Lysophosphatidic acid (LPA), produced by enzymatic action of a secretary protein, autotaxin (ATX), exerts diverse functions through six receptors. Our study aimed to clarify the role of ATX-LPA system in placenta and its association with the pathology of hypertensive disorders of pregnancy (HDP).
Methods: Placental expression of LPA receptors was compared between normal pregnancy and HDP. We assessed the distribution of ATX and LPAR3 in placenta using immunohistochemistry. The impact of LPA stimulation on gene expression profiling was examined in LPAR3-transfected trophoblast cells. Using serum samples, a correlation of ATX levels to oxidative stress markers and fetal growth was evaluated.
Results: ATX expression was ubiquitous, whereas LPAR3 expression was restricted to differentiated trophoblasts. HDP placentas showed increased mRNA expressions of LPARs, whereas the elevation of protein expression was limited to LPAR3. LPA signaling mediated by LPAR3 enhanced the gene expressions involved in immunomodulation and cell differentiation. In normal pregnancy, serum ATX level in the second trimester was negatively correlated with birth weight-to-placental weight ratio. Placental ATX expression in the third trimester decreased in early-onset HDP and increased in late-onset HDP. Serum ATX level was positively correlated to oxidative stress markers.