Strategies to Reduce Infantile Cerebral Palsy

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Abstract

Aim: To prevent cerebral palsy by avoiding fetal brain damage.

Methods: 1. Emergency early delivery timing is before the loss of FHR variability, which is such severe fetal brain damage as anencephaly, 2. FGR caused by the fibrin deposit in placental intervillous space should be soluted to prevent severe hypoxia, 3. Preterm brain periventricular echo density (PVE) should be rejected to prevent PVL and CP, 4. Neonatal hypoxic ischemic encephalopathy (HIE) should be prevented by emergency C-section in sudden continuous bradycardia, and Developed HIE is treated by hypothermia. Anti-glutamate drug should be established, 5. Neonatal respiratory distress syndrome (RDS) is predicted by GLHW ultrasonic tissue characterization to treat by steroid in the fetus and artificial surfactant in neonate, 6. Preeclampsia is treated by anti-sympathicotinic therapy to prevent placental infarction, hypoxia and CP, 7. Developed CP is treated by suitable stem cell therapy.

Results: Effect to reduce infantile CP is shown in some strategies, while most of them should be established in the future.

Keywords: CP; Fetus; New-born; Preterm birth; Brain damage; Hypoxia; Ultrasound; PVL; PVE; NRFS; Fetal monitoring; FGR; Fibrin deposit; Loss of acceleration; Loss of variability

Introduction

The cerebral palsy (CP) is an infantile motility disability caused by various congenital or acquired brain damages. Its symptoms are paraplegia, quadriplegia, hemiplegia, spastic plegia, athetosis, ataxia and chorea, mostly without intelligential damage. Although maternal and perinatal infants' mortalities were dramatically reduced in the past 100 years in Japan [1], world CP production rate has not been changed even after introduction of detailed fetal monitoring, despite some studies on fetal heart rate monitoring reported the reduction of CP in Japan [2,3]. Strategies to prevent CP will differ according to the situation and developing process of CP.

Methods, Results and Discussion

Rapid delivert in cases before the loss of fetal heart rate long term variability

Although it was recommended to perform rapid delivery after the loss of fetal heart rate (FHR) long term variability, the strategy will reduce fetal death, but the CP prevention will be unable, because the FHR variability develops by the loss of fetal brain response to minor fetal movements in the most severe hypoxia after the loss of FHR acceleration in, which was the same as the complete loss of variability and acceleration against fetal movement in ACG of fetal anencephaly [4], namely, fetal state after the loss of variability will be the same as anencephaly; that is total loss of brain function, thus, the CP will not reduce. Therefore, early delivery with C-section should be done before the loss of variability. The developmental process was studied by the analysis of ACG, which recorded FHR and fetal movements, where the loss of FHR variability will be caused by the most severe hypoxic fetal brain damages, which may cause infantile CP (Figure 1).

Figure 1A: Severe fetal brain damage shown by the loss of FHR baseline variability and that of acceleration in the ACG of a heavy late decelerations, who died due to brain hemorrhage at 3 months after birth. Figure 1A is the same as anencephalic FHR; Figure 1B: The loss of FHR baseline variability and acceleration in the ACG of anencephalic fetus.

Therefore, the authors recommend to perform the rapid delivery with C-section before the loss of variability [5-9].

The strategy to deliver the fetus before the loss of variability will be the artificial births in cases of the loss of acceleration, which precedes the loss of variability, in the decreased long term variability, severe FHR changes but preserving variability, or in high hypoxia index, which may be tentatively 20-24 points, which was lower than the index in cases of the loss of variability, that was 25-26 [6]. Hypoxia index is (the sum of fetal bradycardia duration [min] × 100)/the lowest FHR in...
the bradycardia lower than 110 ppm. Further studies are required on the hypoxia index.

Since the loss of variability caused by the brain damage was as severe as anencephaly, thus it may develop at least one in 5,000 deliveries, therefore, new management will reduce 200 CP cases in 1,000,000 births in a year in Japan, which will be 5% or more of CP cases developed in Japan in a year, if the CP development rate is 0.2% (2/1,000) of births. The assumption may be adopted to the world deliveries.

The solution of plcental intervillous fibrin deposit

Fibrin deposits in intervillous space damage active transfer of fetal nutritive material from maternal blood to the fetus initially developing fetal growth restriction (FGR), and it progresses to damage passive villous transfer of oxygen in advanced FGR producing fetal hypoxia and fetal asphyxia then fetal death in cardioiopyin antigen positive pregnancy. The fibrin deposit is diagnosed by the high tissue characterization value of gray-level histogram width (GLHW), and treated with 5000 iu heparin administration to the pregnant woman, where GLHW reduces, estimated fetal weight increases and fetal asphyxia was treated [5] (Figures 2 and 3).

Management of preterm neonates of periventricular echo density (PVE)

Infantile brain damage was frequent in preterm neonates [7]. The 18% of preterm neonates, who preserved PVE until preterm birth, which was high echogenicity of periventricular tissue in B-mode image (Figure 4), which showed larger gray-level histogram width (GLHW) value than normal brain in tissue characterization, and developed neonatal PVL and CP, which was around 0.2% of total birth, while the PVE vanished preterm birth or full term births did not develop neonatal PVL and CP [10].

Therefore, the strategies will (1) Pharmacological tocolysis of preterm labour until full term births. or (2) Sedation of preterm labour until full term birth by anaesthesia or paralysis of uterus-brain nerve, which is the part of positive feed-back loop to produce regular preterm labour contraction [11] or (3) Ultrasonic study of preterm infant’s brain immediately after preterm births to detect the PVE before
developing PVL and the PVE will be treated until its disappearance by treatments with growth factor, hydrocortisone, erythropoietin etc., while their effect has not been established (Figure 4).

Figure 4: The fetus of the figure died by placental infarction (necrosis) in severe preeclampsia, which was caused by the hypoxia due to the restriction of uterine arterial blood flow from sympathicotonic arterial constriction of preeclampsia, and it would develop CP in viable case. Anti-sympathicotonic treatment will prevent fetal damage and CP in preeclampsia.

Emergency C-section is performed prevent neonatal hypoxic ischemic encephalopathy (HIE), which causes neonatal brain damage. The most effective treatment to prevent neonatal brain damage and CP is hypothermia at present including local hypothermia of neonatal head or general body hypothermia. Free radical scavenger or anti-glutamate has been studied in the HIE treatment [12].

Since neonatal respiratory distress syndrome (RDS) may further cause neonatal brain damage due to hypoxia, the RDS prediction in antenatal stage may support neonatal surfactant therapy after the birth. Non-invasive ultrasonic tissue characterization detected fetal lung immaturity, and 96% of neonatal RDS is predicted by the GLHW ratio of fetal lung and liver multiplied by gestational weeks, of which value was less than 29, when neonatal RDS was predicted [13]. The technique may detect the effect of steroid therapy by repeated studies, because the technique is fully non-invasive if compared to the fetal lung immaturity diagnosis with the analysis of amniotic fluid obtained by amniocentesis. Repeated steroid therapy is possible, if fetal lung immaturity is diagnosed by the tissue characterization.

Preeclampsia is characterized by increased sympathicotony, which causes arterial stricture then the reduction of arterial blood supply [14], which may cause various ischemic damages in maternal organs, where there was a case of fetal death due to placental infarction (necrosis) caused by the hypoxia based on the restricted uterine arterial blood supply to the placenta due to sympathicotonic arterial constriction in the preeclampsia (Figure 5). The hypoxia may cause fetal brain damage and CP too. Anti-sympathicotonic treatment will reduce fetal damage and CP in preeclampsia.

Umbilical cord blood stem cell therapy was reported to be favorable to neurological damage [15]. Further improved stem cell therapy will be applied to the CP patient in the future.

References