Placenta and Reproductive Medicine

ORIGINAL ARTICLE

Emergent serious condition of the neonates associated with cerebral palsy in Japan

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Abstract

Objective: To analyze the emergent serious conditions contributing to cerebral palsy (CP) of neonates without a history of the necessity of neonatal resuscitation within 5 minutes after delivery in Japan. **Methods:** To examine the clinical courses regarding CP cases, we reviewed the summary reports of antenatal- and peripartum CP cases published on the home page of the Japan Obstetric Compensation System for Cerebral Palsy (JOCSC) launched in 2009. **Results:** Between January 2009 and February 2016, in Japan there were 51 cases analyzed as CP due to emergent serious condition of the neonates without a history of necessity of neonatal resuscitation within 5 minutes after delivery at the Japan Obstetric Compensation System for CP. The main pathological conditions described as the cause of CP were as follows: the concept of brief resolved unexplained events/apparent life-threatening event (*n* = 15), neonatal Group B streptococcus infection (*n* = 12), herpes simplex virus infection (*n* = 6), neonatal hypoglycemia (*n* = 3), and neonatal hyperkalemia (*n* = 3). **Conclusions:** Based on the current cases, we should note that neonates are very unstable, especially in the early period after delivery.

Key words: cerebral palsy, The Japan Obstetric Compensation System for Cerebral Palsy, emergent serious condition of neonates

INTRODUCTION

The Japan Obstetric Compensation System for Cerebral Palsy (JOCSC) was launched in January 2009 to compensate for the economic burden of children with severe cerebral palsy (CP, severe hemiplegia or moderate quadriplegia requiring nursing) associated with the delivery and to conduct an analysis of factors contributing to CP, with the goal of improving the quality of health care.[1] Under the system, the compensation is now paid to the parents of the child when severe CP develops due to delivery regardless of the responsibility of the medical staff. In Japan, almost all childbirth facilities participate in the casualty insurance under which the operating organization is the contractor and pay the insurance premiums accordingly. If a delivery is approved to be eligible for the compensation by the operating organization, insurance money will be paid to the expectant mother (child) as compensation payment from the insurance company (Figure 1).

The JOCSC has systematically organized and accumulated individual case information, and has compiled an annual report every year that proposes recurrence prevention measures based on the findings.[2] For all the cases approved to be eligible for the compensation, the operating organization has analyzed the cause from a medical point of view based on the information described in the medical records and other documents submitted from the childbirth facilities and information from parents. Specifically, the "Cause Analysis Committee/Cause Analysis Committee Sub-committee" composed of obstetricians, pediatricians, midwives, lawyers, experts, *etc.*, has analyzed the cause, prepares a Cause Analysis Report for each case, and delivers it to the relevant childbirth facility, the child and his/her parents. To prevent the recurrence of similar cases, the JOCSC

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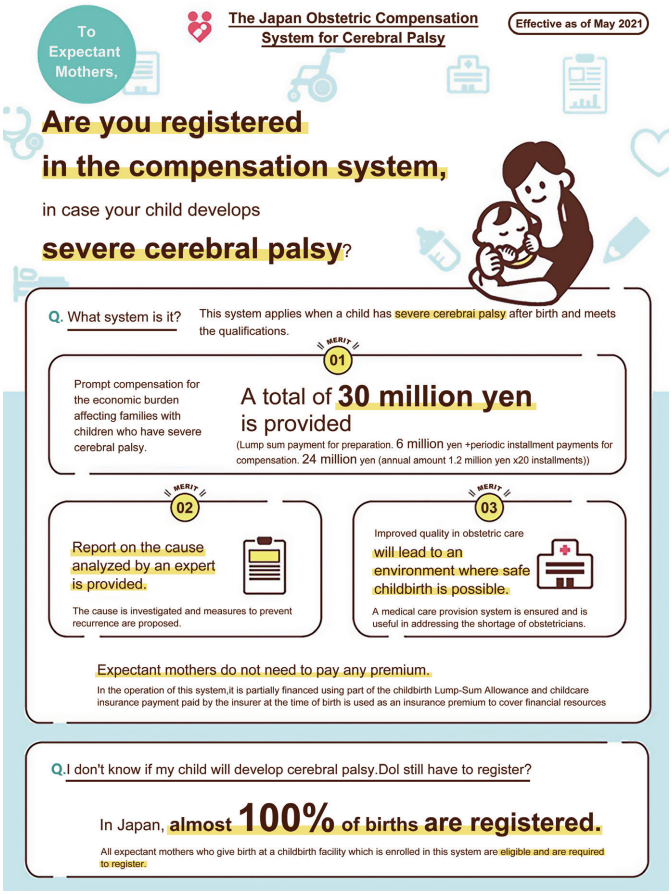

Figure 1. Leaflet of the Japan Obstetric Compensation System for Cerebral Palsy (JOCSC).[1]

has provided the annual report to the public, delivery institutions, related academic societies/organizations, government agencies, *etc.*

In the sixth annual report of the JOCSC published in 2016, an enlightenment to tighten the newborn management in the early postnatal period has been posted.[2] The report presented the cases of CP due to neonatal events.[2] The JOCSC originally had covered "severe CP associated with delivery after 33 weeks of gestation without congenital anomalies" only; however, the indemnity eligible has been expanded to support more miserable neonates and families.[1]

Based on these backgrounds, we reviewed here the cases of CP due to emergent serious condition of neonates without a history of necessity of neonatal resuscitation within 5 min after delivery.

MATERIALS AND METHODS

The protocol of this study was approved by the board of directors of the Japan Association of Obstetricians and Gynecologists.

To re-examine the clinical courses regarding CP cases until February 2016, we reviewed the summary reports of antenatal- and peripartum CP cases published on the home page of the JOCSC launched in 2009. This is a free to access resource, and the cause analysis reports (summary reports) of the patients can be accessed here: http://www.sanka-hp.jcqhc.or.jp/documents/analysis/index.html.[3] By the end of February 2016, 817 deliveries had been registered as the indemnity eligible for the JOCSC. Of these, 51 (6.2%) had been analyzed as cases of CP due to emergent serious condition of neonates without a history of necessity of neonatal resuscitation within 5 min after delivery.[3,4] All of them were evaluated as normal newborns immediately after delivery; however, their conditions changed suddenly and eventually developed severe CP. The main pathological conditions described as the cause of CP were as follows: (1) the concept of brief resolved unexplained events (BRUE)/apparent life-threatening event (ALTE) (n = 15), (2) neonatal Group B streptococcus (GBS) infection (n = 12), (3) herpes simplex virus (HSV) infection (n = 6), (4) neonatal hypoglycemia (n = 6), and (5) neonatal hyperkalemia (n = 3).[4] Of the remaining 9 cases, 3 cases had cerebral infarction, 2 cases had neonatal hyperbilirubinemia, 2 cases had neonatal infection other than GBS infection, and the others were one case each (neonatal hypothermia, Ohtahara syndrome [= early infantile epileptic encephalopathy with burst-

Table 1: Clinical overview of 15 neonates with BRUE/ALTE enrolled for the Japan Obstetric Compensation System for Cerebral Palsy

Case	Parity	Gestational age at delivery (w)	Delivery mode	Neonatal birth weight (g)	Neonatal asphyxia	Onset time (after delivery)	Situation at the time of onset	Discoverer of anomalous neonatal condition
1	Multiparity	38	Normal delivery	2,685	No	50 min	STS	Medical staff
2	Nulliparity	39	Normal delivery	3,092	No	1 h 40 min	STS	Mother
3	Nulliparity	40	Normal delivery	3,695	No	1 h 40 min	Co-sleeping	Mother
4	Nulliparity	37	Normal delivery	2,855	No	1 h 50 min	STS	Medical staff
5	Nulliparity	40	Normal delivery	3,342	No	2 h	STS	Unknown
6	Nulliparity	40	Normal delivery	3,454	No	6 h	Hospital room	Medical staff
7	Nulliparity	41	Normal delivery	3,200	No	8 h	Co-sleeping	Medical staff
8	Nulliparity	38	Normal delivery	2,938	No	11 h 30 min	Co-sleeping	Unknown
9	Multiparity	38	Elective cesarean delivery	2,704	No	11 h 50 min	Co-sleeping	Unknown
10	Nulliparity	38	Normal delivery	2,634	No	14 h	Hospital room	Mother
11	Nulliparity	39	Vacuum -extraction	2,550	No	16 h	Co-sleeping	Medical staff
12	Multiparity	40	Normal delivery	3,456	No	1 d	Hospital room	Medical staff
13	Multiparity	37	Elective cesarean delivery	2,645	No	2 d	Co-sleeping	Medical staff
14	Nulliparity	40	Normal delivery	3,325	No	2 d	Co-sleeping	Medical staff
15	Nulliparity	39	Normal delivery	3,186	No	4 d	Co-sleeping	Mother

STS: skin-to-skin contact; BRUE: brief resolved unexplained events; ALTE: apparent life-threatening event.

suppression] and metabolic disease). We reviewed here the 5 pathogeneses leading to the occurrence of CP.

RESULTS

BRUE/ALTE

BRUE have been concerning episodes of short duration (typically < 1 min) characterized by a change in breathing, consciousness, muscle tone (hyper- or hypotonia), and/or skin color (cyanosis or pallor).[5,6] The episodes occur in a normal-appearing infant in the first year of life, self-resolve, and have no readily identifiable explanation for the cause of the event. Previously called ALTE, the term BRUE was defined by the American Academy of Pediatrics (AAP) in 2016.[3,5–7] The new concept may reflect the transient nature of the event and lack of a clear cause and the diagnosis of BRUE can only be made when there is no explanation for a qualifying event after an appropriate history and physical examination. Therefore, the 15 cases of the current study may include the cases with the concept of ALTE and/or near-miss sudden infant death syndrome (SIDS).

Table 1 shows the 15 cases of BRUE/ALTE. Five cases of them (33%) had onset within 2 h after delivery and 12 cases (80%) had onset within 1 d after delivery. Those with breathing stopped were found by the nurses or parents. Four cases of them (27%) had episodes during early skin-to-skin contact (STS), and 8 cases (53%) had them during co-sleeping (bed-sharing) with their mothers. The doctors immediately rushed to resuscitate them; however, sequelae were left in their brains. The concept of ALTE was diagnosed because the underlying disease leading to apnea could not be diagnosed.

In 2021, Ohki *et al*.[8] performed a questionnaire survey on the management of low-risk neonates and incidence of unexpected death, ALTE from 2008 to 2009 in Japan, and 118 (0.02%) incidents were reported. Of these, 15 (13%) died and 28 (24%) had sequelae. Thirty-two percent of them had the episodes suddenly within 2 h after delivery and 74% had the episodes suddenly within 24 h after delivery, which was almost the same as the prevalence of this study. In addition, these episodes were not associated with the implementation of early STS. In other words, even neonates who have no problems are unstable in the early postnatal period and may have miserable episodes suddenly.

Co-sleeping (bed-sharing) has been discouraged in recent decades because it may increase the risk of suffocation and/or sudden infant death syndrome, especially among smokers. In the present cases, the fatal neonatal event occurred although the mothers were non-smokers.[9]

The other important points of these studies in Japan are that the main discoverers of the neonatal sudden episodes were medical staff such as midwives and nurses who visited the room.[4,8] It should be noted that mothers, especially primiparas women, sometimes cannot notice

the neonatal episodes. In other words, it is necessary to give specific guidance to the mothers about the signs of abnormalities in the neonates. In an early observation in Japan by Takatsu *et al*.,[10] 25 of 47 cases (53%) of SIDS occurred during bed-sharing. In addition, newborns receiving rooming-in care have been also reported to be a high-risk group for the delayed detection of near death, especially for primigravida mothers who are inexperienced in baby care.[11,12] In addition, health professionals should educate families about the risks of accidental asphyxiation with shared-sleeping arrangements.

Neonatal GBS infection

GBS (or Streptococcus agalactiae) can be transferred during delivery to neonates from mothers who are colonized with GBS in the birth canal.[13,14] GBS can cause sepsis and meningitis in newborns. In Japan, a specimen for GBS cultivation as a screening test for all pregnant women has been usually obtained from the introitus of both the vagina and the anus at 35–37 weeks of gestation according to the Japanese Guidelines for Obstetrical Practice.[15] In our recent study, about 17% of Japanese pregnant women have been reported to be colonized with GBS in birth canal.[16]

Neonatal GBS infections are classified into early-onset type (onset within 6 d after delivery) and late-onset type (onset after 7 d after delivery).[12,13] The former type has been pointed out to be mainly a birth canal infection during vaginal delivery; however, there have been some cases of negative GBS screening tests during pregnancy. In some previous studies, more than half of early-onset GBS disease has been reported to occur in neonates born to women with negative GBS screening tests.[17,18] On the other hand, to date there have not been any preventive methods for the latter type of neonatal GBS infection. Therefore, further studies such as real-time PCR assay for predicting colonization of GBS at labor may be needed.[19]

Table 2 shows the 12 cases of neonatal GBS infection resulted in CP. There were 8 and 4 cases of early and late-onset type GBS infection, respectively. The early-onset type developed with rapidly progressing respiratory disorders, while the late-onset type started with mild symptoms such as decreased feeding and gradually progressed. In all cases, the Bacterial Sepsis Workup was performed at the time of visit to emergency medical institutions, and a diagnosis of bacterial infection was performed promptly; however, the neurological prognoses were not completely improved.

In 63% (5/8) of early-onset type and 25% of late-onset type infection cases (1/4), the results of the GBS screening test were negative. On the other hand, 50% (4/8 cases) of

Table 2: Clinical overview of 12 neonates with GBS infection enrolled for the Japan Obstetric Compensation System for Cerebral Palsy

Case	Gestational age at delivery (w)	Delivery mode	Results of GBS culture during pregnancy (gestational age at culture, w)	Neonatal birth weight (g)	Neonatal asphyxia	Onset time (after delivery)	Early or late onset	Other points
1	40	Vaginal delivery	Negative (35)	3,390	No	6 h	Early	–
2	37	Vaginal delivery	Positive (8), Negative (32)	2,978	No	6 h	Early	–
3	39	Vaginal delivery	Negative (33)	3,236	No	6 h	Early	–
4	36	Vaginal delivery	Positive (12)	2,006	No	12 h	Early	–
5	38	Vaginal delivery	Positive (31), Negative (36)	2,800	No	1 d	Early	Antibacterial use during pregnancy
6	39	Vaginal delivery	Negative (35)	3,682	No	2 d	Early	–
7	39	Vaginal delivery	Negative (19)	3,354	No	4 d	Unknown	–
8	36	Vaginal delivery	Negative (13, 27, 29, 35)	2,668	No	5 d	Unknown	–
9	36	Cesarean delivery	Not implemented	2,192	No	11 d	Unknown	–
10	39	Vaginal delivery	Positive (36)	3,336	No	12 d	Unknown	Antibacterial use during pregnancy
11	38	Vaginal delivery	Positive (36)	2,752	No	17 d	Unknown	–
12	38	Cesarean delivery	Negative (36)	2,686	No	18 d	Unknown	–

GBS: Group B streptococcus.

Table 3: Clinical overview of 6 neonates with HSV infection enrolled for the Japan Obstetric Compensation System for Cerebral Palsy

Case	Gestational age at delivery (w)	Delivery mode	Neonatal birth weight (g)	Neonatal asphyxia	Onset time (after delivery)	Maternal past history of HSV infection	Maternal symptom of HSV infection	Route of infection
1	39	Cesarean delivery	2,750	Yes	At delivery	No	No	In utero
2	39	Vaginal delivery	3,920	No	9 d	No	No	Birth canal
3	38	Vaginal delivery	3,120	No	9 d	No	No	Birth canal
4	40	Vaginal delivery	3,155	No	12 d	No	No	Unknown
5	36	Vaginal delivery	2,838	No	13 d	No	No	Unknown
6	36	Cesarean delivery	2,206	No	19 d	No	No	Unknown

HSV: herpes simplex virus.

Table 4: Clinical overview of 6 neonates with hypoglycemia enrolled for the Japan Obstetric Compensation System for Cerebral Palsy

Case	Gestational age at delivery (w)	Delivery mide	Neonatal birth weight (g)	Neonatal asphyxia	Onset time (after delivery)	Maternal (gestational) diabetes	Disease diagnosis
1	35	Cesarean delivery	2,615	No	1 d	No	Unknown
2	41	Cesarean delivery	2,600	No	2 d	No	Hyperinsulinemia
3	37	Normal delivery	3,640	No	2 d	No	Hyperinsulinemia
4	38	Normal delivery	3,450	No	3 d	No	Hyperinsulinemia
5	41	Vacuum-extraction	3,552	No	3 d	No	Hyperinsulinemia
6	42	Vacuum-extraction	2,998	No	Unknown	No	Hyperinsulinemia

the early-onset type and 25% (1/4 cases) of the late-onset type infection cases were managed without complying with the clinical practice guidelines including the use of antibacterial agents during delivery published in the USA or Japan. Based on these cases and previous studies,[17,18] it may not be possible to prevent 50% of the early-onset type infection even if the current guidelines are followed closely. In addition, for the prevention of late-onset type infection, it is necessary to examine the establishment of preventive methods through repeated epidemiological investigations.

HSV infection

Neonatal HSV infections are uncommon. However, the morbidity and mortality associated with the infection are not low.[20,21] Neonatal HSV infections develop after an incubation period of 2 to 10 d, but are characterized by a lack of specific initial symptoms. If appropriate treatment is given from an early stage, the prognosis will not be worse; otherwise, the rate of neonatal death or neurological sequelae will be high. In addition, it has been reported that only about 30% of mothers with genital herpes infections had genital herpes findings.[21,22] Most neonates with HSV disease have been reported to be born to mothers without a history or symptom of HSV infection.[22]

As shown in Table 3, none of the 6 cases with CP due to HSV infection showed any findings suggestive of HSV infection in the mother. Therefore, it took 2–4 d for the neonates to be referred to the neonatologists after the onset with the symptom of somehow strange except Case 1 with neonatal asphyxia.

For neonatal HSV infection, it is known that initiation of long-term antiviral suppressive therapy at an early stage of the disease can lead to significant improvement in morbidity;[21] however, the initial action might be delayed due to the absence of findings of HSV infection in the mother. The other 5 cases were referred to neonatal specialists after they became severe.

Based on the current cases, we have to keep in mind that neonatal infectious diseases may develop even if the mother does not have any infectious symptoms. And it needs to be educated that it is important for mothers to feel 'somehow weird' to their children.[1]

Neonatal hypoglycemia

Hypoglycemia is the most common metabolic disturbance occurring in the neonatal period.[23,24] Prolonged and/or symptomatic hypoglycemia may correlate with long-term neurodevelopmental deficits. However, a clear definition of neonatal hypoglycemia has been lacking because current screening guidelines and management algorithms had been

based on limited evidence. In addition, we do not routinely measure blood glucose concentrations in healthy infants without risk factors for hypoglycemia.

As shown in Table 4, there were some cases of CP due to neonatal (persistent) hyperinsulinemia hypoglycemia. The disease can cause cerebral damage mainly in the occipital lobe due to impaired excessive secretion of insulin. Approximately one-third of the cases develops in macrosomia. In addition, epidemiologically it has been characterized by being more common in macrosomia without (gestational) diabetes in the mother. In the cases in Table 4, the initial symptoms of the neonates seemed to be somehow strange such as less crying and less movement. The timings of blood glucose measurement of them were presumed to be delayed because the mother had no diabetes and the neonate was not macrosomia.

Neonatal hyperkalemia

In the current cases, there were 3 neonates with hyperkalemia leading to CP. Neonates are usually in a relative hyperkalemia state until the first urination, and they will develop arrhythmias when serum K level exceeds 7 mEq/L.[4,25,26] Their symptoms are manifested by sudden lethal arrhythmia such as ventricular fibrillation.

Neonatal Hyperkalemia has been suggested to be mainly caused by the long-time use of magnesium sulfate with and without ritodrine as tocolysis until just before the delivery, because the half-life of blood magnesium concentration in newborns is 40 h or more. According to a Japanese nationwide retrospective cohort study performed in 2014,[27] the occurrence of neonatal hyperkalemia was associated with concomitant usage of ritodrine and magnesium sulfate compared with no usage. The details of the mechanism leading to the disease are still unknown; however, avoiding long-term tocolysis until just before delivery has been mentioned as a preventive procedure. Therefore, in Japan there are now in the process of changing from long-term tocolysis to short-term tocolysis as in Western countries. Otherwise, for newborns whose mothers received tocolytic agents until just before the delivery, observation using a continuous heart rate monitor or frequent check of serum potassium levels is now recommended irrespective of the symptom.[4]

DISCUSSION

Based on the current cases with CP, we should note that neonates are very unstable, especially in the early period after delivery.[4] It cannot be ruled out that these cases may not have been able to prevent the onset of CP by only following the current guidelines.[15] Even under these situations, we need to take steps to reduce the incidence of CP as much as possible. Of course, it goes without saying that the management thoroughly following the details of the guidelines is a prerequisite. And what more can be done is also important. The mother sometimes cannot be a discoverer of the anomalous condition of the neonate, while she sometimes can become an observer of the anomalous condition of the neonate. And then, it is important for us to keep in mind that we should always check the neonate directly if we have a mother's complaint.

In addition, there were some conspicuous cases in which the CP could not be prevented by medical treatment by guidelines such as neonatal GBS infections with negative maternal GBS culture, neonatal herpes infections with no history or symptoms in the mother, and neonatal hypoglycemia without maternal diabetes or macrosomia.[15] Based on these cases, we should not deny the current guidelines, and we should understand the incidence of CP has been within the small level because we had practiced neonatal care according to the guidelines.[4,15] Our perinatal management should be carried out on the premise at a minimum we should implement what is described in the guidelines.

In addition, we need to enlighten our mothers and family doctors as follows: even if you can not explain the symptoms specifically, if you feel that the neonate is not feeling well as usual, please consult a perinatal specialist.

Conflict of Interest

Shunji Suzuki is an Editorial Board Member of the journal. The article was subject to the journal's standard procedures, with peer review handled independently of this member and his research group.

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