Fetus is Mature after 32 Weeks of Pregnancy

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Abstract
Aim: Fetal maturity was estimated by various methods in 164 monochorionic diamniotic (MD) twins.

Methods: Twin-to-tin transfusion syndrome (TTTS) was prevented by intensive maternal rest and tocolysis until fetal adrenal cortex maturity, which was estimated in the initiation mechanism of uterine contraction, gray level histogram width (GLHW) of fetal lung and liver ratio multiplied by gestational weeks was used to estimate fetal lung maturity.

Results and discussion: Fetal adrenal-cortical steroid, reacted stresses after maturation, which caused Braxton-Hicks uterine contraction after 30 gestational weeks, where ultrasonic cardio-thorax ratio enlarged to 0.4 or more, then the MD twins received Caesarean delivery to prevent TTTS in 32 ± 2.2 gestational weeks where coefficient of variation (CV) of weeks was as small as 7%. Thus, fetal maturity was estimated to be 32 gestational weeks. As GLHW ratio of fetal lung and liver multiplied by gestational weeks was >29 in mature fetal lung, the index was 31 in 32 gestational weeks, and no problem was found in the iMD twins born in 32 ± 2.2 gestational weeks.

Conclusion: Human fetus is mature in 32 gestational weeks.

Keywords: Fetal maturity; Pregnancy; Uterine contraction; Gestational weeks

Methods

We would like to confirm fetal maturation in the management of 164 MD twins born with Cesarean delivery after the maternal rest, tocolysis and detailed ultrasound monitoring, developed no established TTTS in the MD twins, while the existence of mild blood transfer to the recipient twin from the donor, was confirmed by the enlarged ultrasonic cardio-thorax ratio.

Fetal lung gray level histogram width (GLHW), an ultrasonic tissue characterization, was measured in fetal lung and liver to estimate the lung maturity and the fetal age was studied in transient Braxton-Hicks uterine contraction, which determined the Caesarean delivery at 32 ± 2.2 gestational weeks (Figure 1) [1].

Results

Fetal lung GLHW was 35 and that of fetal liver was 36 in 32 weeks of pregnancy [2]. Fetal lung was mature, because the lung/liver ratio of GLHW multiplied by pregnancy weeks was 31 which was larger than 29 in 32 weeks of pregnancy, when the twins received Caesarean delivery to avoid established TTTS after the first blood transfer to recipient from the donor, which was suspected to be caused by a Braxton Hicks uterine contraction [3]. Although there was very mild reactive cardiac function change in the recipient only within 2 days in the NICU, the infants were healthy and no failure afterwards.

Novel treatment of MD twins to avoid the TTTS was beneficial because the MD twins received Caesarean delivery (CD) after ultrasonic imaging of cardio-thoracic ratio (CTR) larger than 0.4, which was resulted by the inflow of blood into the recipient heart in the uncontrollable uterine contraction, which was uncontrollable by maternal rest and tocolysis in around 32 gestational weeks, which would be Braxton-Hicks contraction caused by prostaglandin developed at placental site by the emission of fetal adrenal corticosteroids. In summary, the uncontrollable uterine contraction might be a Braxton Hicks contraction, which was caused by fetal reaction to the stress around 32 weeks, where it will be a physiologic and endocrine reaction of a mature fetus to the stress. Therefore, it was uncontrollable by the tocolysis with terbutaline.

Discussion

The 82 mothers of 164 MD twins who received the treatments with maternal rest and tocolysis followed by Caesarean delivery around 32
weeks of pregnancy. Why there was no TTTS in 28 and 24 weeks of pregnancy? The early TTTS might develop by blood transfer in no ultrasound monitoring in their pregnancy, and controllable contraction was suppressed by maternal rest and tocolysis in our novel treatment. The 32 weeks of pregnancy will be important fetal age in our present study, namely, it may indicate fetal maturity, as the 32 weeks of pregnancy is the stage when transient Braxton-Hicks’ contraction initiates before the appearance of labor contraction. According to approved theory, the fetus excretes adrenal corticosteroids into umbilical blood which reached placenta, stimulating local decidua to secrete prostaglandin developing the initial uterine contraction at placental site as the pace-maker of whole uterine contraction, namely, the Braxton-Hicks contraction may show the fetal maturity in the production of protective adrenal corticosteroid against stresses. If so, the authors would keep the pregnancy until the stage of the functional maturity of twins to resist stresses. The Braxton-Hicks contraction will enhance the first blood transfer to the recipient from the donor, and enlarged the recipient heart in MD twin pregnancy. Thus, the twins were guarded by fetal functional maturity from the surgery of cesarean delivery, where the Caesarean delivery would not damage twins. Also there was no problem in the infants after the Caesarean delivery. The Braxton-Hicks contraction may promote the blood transfer, but there was the interval to prepare the Caesarean delivery. Also, the Braxton-Hicks contraction would be uncontrollable by the tocolysis, because it was physiologic and endocrinologic.

**Conclusion**

Fetal functional maturity will develop at around 32 weeks of pregnancy, which was estimated by feal lung maturity and Braxton-Hicks uterine contraction.

**References**

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