

Prenatal Diagnosis of Fetal Seizure: A Case Report

A 35-yr-old woman carrying a 17-week-old fetus presented with right hydronephrosis and a single umbilical artery. Karyotyping was normal and targeted ultrasonography showed an otherwise normal fetus. After 28 weeks of gestation, the mother felt rapid, repetitive fetal movement and an ultrasound at 30 weeks of gestation revealed tonic clonic movements of the fetal trunk and extremities. At 36 weeks of gestation, an emergency repeat Cesarean section was performed because of a premature rupture of the membranes and a male infant weighing 4,295 gm was delivered. After birth, the infant continued to have movements suggestive of a generalized tonic clonic seizure. Brain computed tomography and magnetic resonance imaging revealed normal structures and an electroencephalography showed generalized suppression. Treatment with phenobarbital resulted in substantial improvement in the number of seizure episodes, however fine seizure-like movement continued in both of the hands, feet and in the tongue until the five-month follow-up. This is the first Korean report of a fetal seizure being diagnosed during the prenatal period.

Key Words : *Fetal Seizure; Ultrasonography*

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INTRODUCTION

Since the first report of a fetal seizure by Badr El Din, there have been several reports of fetal seizures that are witnessed during ultrasonography or perceived by the mother (1-3). Most cases are due to congenital anomalies, mainly in the central nervous system, and have a poor prognosis (2).

Normal fetal movement is well documented by ultrasonography. Abnormal forceful, jerky, and periodic fetal movement can be associated with a fetal seizure. The seizures occur repeatedly, usually involving the whole fetal body, and at a frequency that varies from two movements/second in clonic convulsions to several times/minute in lightning convulsions (2, 3).

We have observed seizure-like fetal movement starting at 30 weeks of gestation, and continuing after birth until the five-month follow-up. This is the first report describing the prenatal diagnosis of a fetal seizure by ultrasonography in Korea.

CASE REPORT

A 35-yr-old multigravida (2-0-1-2) presented for routine prenatal care at our hospital. Her past medical and obstetrical history was uneventful except that her previous babies were both large (3,900 g and 4,200 g). At 17 weeks of ges-

tation, amniocentesis was performed for karyotyping owing to advanced maternal age and the karyotype result was normal. At this time, we identified right hydronephrosis and a single umbilical artery. The ultrasonography showed otherwise normal organ images and the fetal weight and amniotic fluid were adequate for the gestational age. The patient visited our clinic for a scheduled targeted ultrasonography at 21 weeks of gestation, which showed no abnormal findings except for the hydronephrosis and single umbilical artery.

At 30 weeks of gestation, we performed a follow-up ultrasonography during which we observed abnormal fetal movement. The fetus showed rapid, repetitive fine movement of the whole body lasting 30-60 sec with intervals between motions lasting 1-5 min. The abnormal movement was also detected by the hand of examiner. The movement ceased intermittently, but the movement-free time was less than 5 min. The mother had also felt this movement since two weeks prior. We tentatively diagnosed it as a fetal seizure. A non-stress test (NST) was performed and showed a reactivity with a baseline of 130-140 bpm. The parents were counseled on the meaning of abnormal fetal movement, including congenital seizure with poor prognosis and the parents determined to continue the pregnancy.

At 36 weeks of gestation, the mother went into preterm labor with a premature rupture of the membranes. Seizure-like fetal movement was still observed by ultrasonography. An emergency repeat Cesarean section was performed and a

male infant weighing 4,295 g was delivered. The Apgar score were 4 and 5 at 1 and 5 min, respectively. There were no abnormal findings in the gross appearance but the neonate was hypotonic and cyanotic with generalized seizure-like movements. Initial respiration and crying were poor. Immediate intubation was performed and mechanical ventilation commenced. The baby continued to have a generalized tonic clonic seizure associated with subtle seizures and phenobarbital administration was started. The seizure activities persisted until the serum phenobarbital concentration exceeded 20 $\mu\text{g}/\text{mL}$.

The initial arterial blood gas analysis (ABGA) revealed a pH of 7.211, a PCO_2 of 53 mmHg, a PO_2 of 41 mmHg and an O_2 saturation of 65%, which was improved after mechanical ventilation. The initial glucose level was 21, which was normalized with fluid infusion, and work up for the diabetes reveals no abnormality. A postnatal brain computed tomography and magnetic resonance imaging showed no abnormal findings. An electroencephalography (EEG) showed generalized suppression activity with random sporadic sharp waves and spikes in multiple areas. Genetic and viral examinations were negative. Pathologic examination of the placenta showed no abnormal findings. An abdominal sonogram showed severe right hydronephrosis due to ureteropelvic junction obstruction and a nephrostomy was performed at postnatal day 3.

Extubation was performed at postnatal day 9 and there was substantial improvement in the number of seizure episodes, however fine seizure-like movement was still observed in both hands and feet, as well as in the tongue. The baby continued to have intermittent seizures, even with medication, and showed developmental delay and lack of neck control at the five-month follow-up examination.

DISCUSSION

Seizures occur in 0.5% of term infants and 22.2% of preterm infants, though fetal seizures are very rare (4-7). Neonatal seizures are considered important predictors of neurologic diseases and the prognosis depends on several factors (8). Congenital anomalies, asphyxia, tonic convulsions, abnormal EEG, and seizures within the first day of life carry a worse prognosis than late-onset seizures (8). *In utero* seizures may be associated with the poorest outcome (9).

With increasing evidence that a large proportion of neonatal neurological dysfunction originates in the antepartum period, it is to be expected that the behavioral presentation of that dysfunction might also occur before birth (10, 11). Whatever the specific mechanism of injury, neonatal seizures represent important mileposts for assessing risk for later sequelae in children, ranging from epilepsy to developmental disorders.

There have been some reports on the prenatal diagnosis of seizure-like fetal movement detected by real time ultrasonog-

raphy or by the pregnant mother (2, 3). In most of these cases, the seizure activity presented as obvious, rapid myoclonic jerking of the fetal extremities (2, 3). Some cases had structural central nervous system malformation, genetic syndromes associated with restriction of limb movement, or severe growth restriction (2, 12). The most common cause of fetal seizure is a congenital anomaly, mainly of the central nervous system (2).

No specific criteria have been established to assist in the prenatal diagnosis of fetal seizures. Skupski et al. reported two characteristics of the seizure-like movements: 1) a repetitive, episodic movement with a duration consistent with a seizure episode, and 2) a regularity of movement at a frequency consistent with that of seizure activity (12).

Fetal movement represents neural activity and is an excellent means of assessing fetal nervous system dysfunctions as well as postnatal function because there is a continuum of neural function from prenatal to postnatal life (13). Therefore, it is reasonable to assume that neural dysfunction in the fetus will be present after birth.

In this case, fetal seizure activity was first perceived by the mother at 28 weeks of gestation, and was then well documented by ultrasonography. And the *in utero* abnormal movement continued after birth. Reactive NST and postnatal ABGA suggested that the fetal seizure was not associated with prenatal hypoxia or acidosis. Also, the postnatal brain imaging study revealed no abnormal findings. Regardless of the cause, the outcome for the infant appears severe.

Previously-reported cases and our own case suggest that fetal seizure has a poor prognosis (2). However, it is noteworthy that reported cases are likely to represent the severe end of the spectrum, and there might be a broad spectrum of cases of *in utero* seizures that have a more favorable prognosis (3). Careful investigation of fetal movement is necessary to predict the prognosis and help in informed decision-making. Also, fetal movement monitoring can provide clues about the causal pathways for neonatal seizures that begin before birth.

The use of ultrasonography to diagnose abnormal fetal movements, including fetal seizures, can better prepare the parents and clinicians for delivery of a neurologically-impaired neonate. Further, the ultrasonography can provide evidence that abnormal behavior predates the birth procedure, which will decrease the legal risk for the obstetrician. It is also essential to perform detailed imaging studies of the brain in the presence of fetal seizures to understand the etiology and to predict the risk of recurrence.

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