Taiwanese Journal of Obstetrics & Gynecology 54 (2015) 459–460

Contents lists available at ScienceDirect

Taiwanese Journal of Obstetrics & Gynecology

journal homepage: www.tjog-online.com

Research Letter

Important clinical information from successful treatment of a case with isolated severe oligohydramnios and deficient fetal growth late in the second trimester

Fu-Nan Cho [a,*], San-Nung Chen [a], Ju-Yueh Li [a], Yu-Hsiang Chang [b], James R. Carey [c], Wen-Shiung Liou [a]

[a] Department of Obstetrics and Gynecology, Kaohsiung Veterans General Hospital, Kaohsiung, Taiwan
[b] Department of Pediatrics, Kaohsiung Veterans General Hospital, Kaohsiung, Taiwan
[c] Department of Applied Chemistry, National University of Kaohsiung, Kaohsiung, Taiwan

ARTICLE INFO

Article history:
Accepted 10 June 2014

A 31-year-old primigravid woman presented at 27 weeks of gestation with complaints of lack of amniotic fluid (AF) for 2 days due to unknown causes. The patient's last prenatal examination at another hospital 1 month prior was unremarkable except for a growth-restricted fetus that had been dated using the first-trimester sonography. The patient had no underlying medical or obstetric conditions other than long working hours, poor sleep, and a dislike of drinking water. The patient denied taking any medication and did not have any sign of leakage of AF. In addition, the patient did not suffer from fever, anemia, leukocytosis, or abdominal pain.

Ultrasound examination revealed severe oligohydramnios with an amniotic fluid index (AFI) < 0.5 cm (Figure 1) and 4 weeks of symmetrically deficient fetal growth. The patient underwent amnioinfusion with 600 cc of isotonic warm saline. Further study by sonography showed normal bilateral kidneys with no other fetal anomalies. Color Doppler revealed normal umbilical venous flow but a significant brain sparing effect with a pulsatility index (PI) of 0.82 on the middle cerebral artery (MCA) and 1.41 on the umbilical artery (UA; Figure 2). Cardiotocography demonstrated normal fetal heart beats and no uterine contraction. The patient was hydrated intravenously with 1500 cc of isotonic saline and was administered 2 L/min oxygen via nasal cannula for 1 day. The patient was advised to keep hydrated. Sonography on the following day revealed an AFI of 7.6 cm, normal ductus venosus flow and a dramatic improvement of the brain sparing effect with a PI of 1.05 on the MCA and 0.69 on the UA. The absence of both oligohydramnios and brain sparing effect was revealed using a sonography performed after 7 days treatment and also on follow-up sonograms conducted once a week.

Spontaneous rupture of amniotic membrane with a clear watery discharge was noted at 36 weeks. The cardiotocography revealed normal variability with no deceleration. Oxytocin was then given to enhance the uterine contraction. Unfortunately, episodes of repeated late deceleration and smooth short-term variability appeared 8 hours later coinciding with a cervical dilation of 2.5 cm. Emergency cesarean section was then performed. Placental abruption with 100 cc blood clots was noted during the operation. A thin umbilical cord was inserted to the peripheral placenta. Pathology studies confirmed placental infarction in 1/5 of the placenta (Figure 3). A male newborn, weighing 1734 g, was delivered with Apgar scores of 2, 4, and 7 at 1 minute, 5 minutes, and 10 minutes, respectively. The newborn, 16 months old at the time of this writing, eventually developed well without further complication.

The AF is isotonic with plasma during early pregnancy due to bidirectional diffusion through the fetal skin and becomes hypotonic after complete keratinization of the fetal skin at 25 weeks [1,2]. During the second half of pregnancy, the AF is derived from excretion of fetal urine and lungs. Removal of AF mainly depends on fetal swallowing (up to 250 cc/kg/d) and intramembranous absorption (up to 500 cc/d) via microvessels on the fetal surface of the placenta and umbilical cord. An osmotic gradient between the hypotonic AF and isotonic fetal plasma favors the intramembranous pathway [2–4]. Oligohydramnios may be attributed to fetal anomalies, placental lesion, rupture of fetal membrane, drugs, and uteroplacental insufficiency. If no evident causes are identified prenatally, the condition is identified as an isolated oligohydramnios.

Severe oligohydramnios associated with a growth-restricted fetus and significant brain sparing effect in the late second trimester or early third trimester is often a complicated situation

* Corresponding author. Department of Obstetrics and Gynecology, Kaohsiung Veterans General Hospital, 386 Ta-Chung First Road, Kaohsiung 81362, Taiwan.
E-mail address: fncho@isca.vghks.gov.tw (F.-N. Cho).

http://dx.doi.org/10.1016/j.tjog.2014.06.006

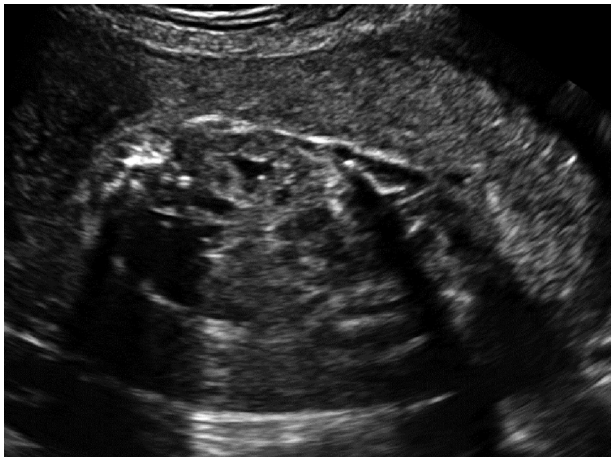

Figure 1. Severe oligohydramnios with an amniotic fluid index < 0.5 cm.

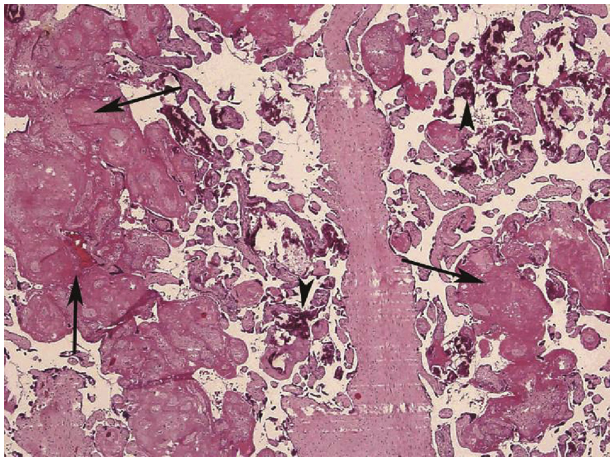

Figure 3. Histological examination using hematoxylin–eosin stain showing placental infarction with necrosis of villi (arrows) and calcification (arrowheads).

with very poor prognosis. Without timely adequate management, fetal hypoxia will progress to acidemia, thus resulting in heart failure and impaired brain function. Impairment of the left heart will cease the compensatory redistribution of blood supply to the brain, leading to the disappearance of the brain sparing effect. Right heart failure contributes to the appearance of increasing atrial reverse flow of the inferior vena cava, absent or reverse atrial flow of the ductus venosus, and pulsatile flow of the umbilical vein in sequence. In fetuses with early-onset fetal growth restriction and absent or reverse end-diastolic flow in the UA, poor perinatal outcomes were noted in fetuses with the disappearance of the brain sparing effect compared to the presence of brain sparing effect [5].

Interestingly, in this case a single amnioinfusion in combination with hydration and oxygen supply dramatically relieved the brain sparing effect in 1 day and persistently improved the isolated severe oligohydramnios. Amnioinfusion with an isotonic solution, immediately reducing the umbilical cord compression and lessening the intramembranous absorption of AF, may quickly improve the delivery of oxygen to the fetus and lead to the disappearance of the brain sparing effect. The cessation of redistribution of blood supply to the fetal brain allows more blood flow to the fetal kidneys, resulting in the improvement of the severe oligohydramnios.

A recent study has shown the promising effect of serial amnioinfusion in combination with tocolysis in cases with isolated oligohydramnios and severe growth restriction before 26 weeks of gestation [6]. Intravenous injection of 1500 cc per day of an isotonic solution for 6 days has been shown to have a beneficial effect on the isolated oligohyramnios during the third trimester [7]. Subsequent oral hydration is also helpful for maintenance of adequate AF volume. Adequate circulation volume together with oxygen supply may enhance the oxygenation of the uteroplacental area and lessen the progression of growth restriction.

In summary, isolated second-trimester oligohydramnios associated with severe growth restriction and significant brain sparing effect may be related to focal placental infarction and insufficient fluid supply. While amnioinfusion in combination with hydration and oxygenation supply can restore the AF volume, relieve the fetal distress, facilitate fetal growth, and prolong the gestation, obstetricians should beware of the potential risk of placental abruption and acute fetal asphyxia during the course of labor.

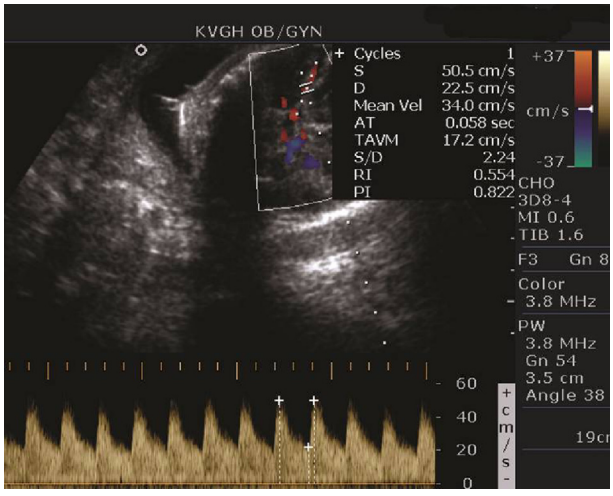

Figure 2. Significant brain sparing effect with a pulsatility index of 0.82 on the middle cerebral artery (and 1.41 on the umbilical artery; not shown) taken on the day of amnioinfusion.

Conflicts of interest

The authors have no conflicts of interest relevant to this article.

References

[1] Sherer DM. A review of amniotic fluid dynamics and the enigma of isolated oligohydramnios. Am J Perinatol 2002;19:253–66.
[2] Underwood MA, Gilbert WM, Sherman MP. Amniotic fluid: not just fetal urine anymore. J Perinatol 2005;25:341–8.
[3] Gilbert WM, Brace RA. The missing link in amniotic fluid volume regulation: intramembranous absorption. Obstet Gynecol 1989;74:748–54.
[4] Munn MB. Management of oligohydramnios in pregnancy. Obstet Gynecol Clin North Am 2011;38:387–95.
[5] Yeniel AÖ, Ergenoglu AM, Sanhal CY, Akdemir A, Akercan F, Kazandi M, et al. The disappearing brain-sparing effect in early-onset fetal growth restriction fetuses revisited. Fetal Diagn Ther 2014;36:166–72.
[6] Takahashi Y, Iwagaki S, Chiaki R, Iwasa T, Takenaka M, Kawabata I, et al. Amnioinfusion before 26 weeks' gestation for severe fetal growth restriction with oligohydramnios: preliminary pilot study. J Obstet Gynaecol Res 2014;40:677–85.
[7] Patrelli TS, Gizzo S, Cosmi E, Carpano MG, Di Gangi S, Pedrazzi G, et al. Maternal hydration therapy improves the quantity of amniotic fluid and the pregnancy outcome in third-trimester isolated oligohydramnios: a controlled randomized institutional trial. J Ultrasound Med 2012;31:239–44.