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# Fetal magnetic resonance imaging (MRI) analysis of seven cases of obstetric mirror syndrome (OMS)

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**Summary**

*Objective:* The aim of this study was to analyze the fetal magnetic resonance imaging (MRI) findings of seven cases of obstetric mirror syndrome (OMS). *Materials and Methods:* The fetal MRI results of seven cases of clinically/pathologically confirmed OMS were retrospectively analyzed, including fetal hydrops, placental edema, association with related malformations, etc. *Results:* The seven OMS fetuses all displayed multi-site edema and two cases were associated with placental edema. Other associated malformations included fetal sacrococcygeal teratoma (SCT, 1/7), Rh-defect (D) caused immune hemolysis (1/7), fetal growth restriction (FGR, 2/7), twin-twin transfusion syndrome (TTTS, 2/7), and placenta chorioangioma (1/7). OMS can increase maternal and fetal morbidity and mortality, so early diagnosis of this disease could have important significance in providing the appropriate treatment protocol and improving the clinical outcomes. *Conclusions:* Fetal MRI was an important supplementary means of obstetric ultrasound, which could qualify fetal hydrops-associated malformations, characterize the nature of the tumor, and assess the situations of the heart and lung development of fetal edema simultaneously.

*Key words:* Obstetric mirror syndrome; Edema; Fetus; MRI.

## Introduction

Obstetric mirror syndrome (OMS), also known as the Ballantyne syndrome, refers to various degrees of edema in the maternal induced by fetal hydrops or placental edema and clinically characterized by progressive increase, and it could be associated with or without similar clinical manifestations of early preeclampsia as hypertension or proteinuria. In 1892, Ballantyne firstly reported the cases with gestational Rh (D)-isoimmunization-induced fetal and placental edema-related maternal edema and named it as OMS [1]. In China, the incidence of OMS is about 0.0154% [2]. Although its pathogenesis has not been made clear yet, OMS has similar pathophysiological changes as preeclampsia [3], which could increase maternal and fetal morbidity and mortality, and might include maternal severe pulmonary edema and heart failure, and the proportion of intrauterine death could be high [4-6]. Early diagnosis of OMS would be vital for timely treatment and good prognosis for mother and fetus [7, 8]. Currently, clinical reports of OMS is less than 100 cases; missed or misdiagnosis often happens due to lack of the awareness towards this disease, making prevention and treatment complex [9].

As an important supplementary means of obstetric ultrasonography, fetal MRI was firstly reported by Smith *et al.* in 1983 [10], and over the past decade, it has been more and more applied in obstetrics clinic, its complementary roles are particularly in the prenatal diagnosis of fetal central nervous system lesions [11, 12]. As for the diagnosis of OMS, fetal MRI would not be affected by excessive obesity or oligoamnios, could clearly display the abnormality of single or twins fetuses, and particularly could complement unspecified additional information that could not be confirmed by ultrasound, and also could be used in further evaluating fetal lung development [13]. So far, OMS-related fetal MRI analysis has not been reported. This study summarized the MRI results of seven OMS cases, aiming to further increase awareness towards this disease.

## Materials and Methods

The MRI findings of seven OMS cases hospitalized and treated in the present hospital from October 2013 to May 2015 were retrospectively analyzed. The pregnant women were aged 22-38 years, with a mean age of 29.00 ± 5.18 years. All of these seven pregnant women appeared with varying degrees of edema, hemodilution, and hypoproteinemia, among whom four were associated with hypertension, three with proteinuria, one with Rh blood type defect (Rh (D)), and high titer antibody (anti-CcEe512), one with hyperthyroidism and urinary glucose (+), and one with pulmonary edema. Among these seven cases, five cases were of singleton pregnancy, and two were of monochorionic diamniotic twin gestation (MCDA). Five cases were primipara and two were pluripara. Two cases were conceived by in vitro fertilization-embryo transfer (IVF-ET), and five were naturally conceived. The gestational ages were 22.6 ~32 weeks, with the mean as 27.8 weeks. This study was conducted in accordance with the declaration of Helsinki. This study was conducted with approval

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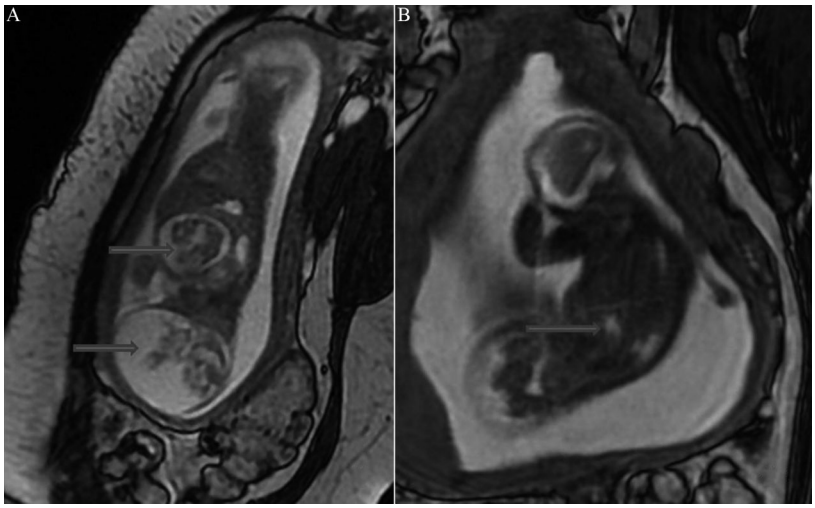


Figure 1. — A: Fetal sacrococcygeal teratoma (endogenous + exogenous) (arrow), seroperitoneum, and poor lung development. B: Fetus with sacrococcygeal teratoma (SCT) and bilateral hydronephrosis (arrow).

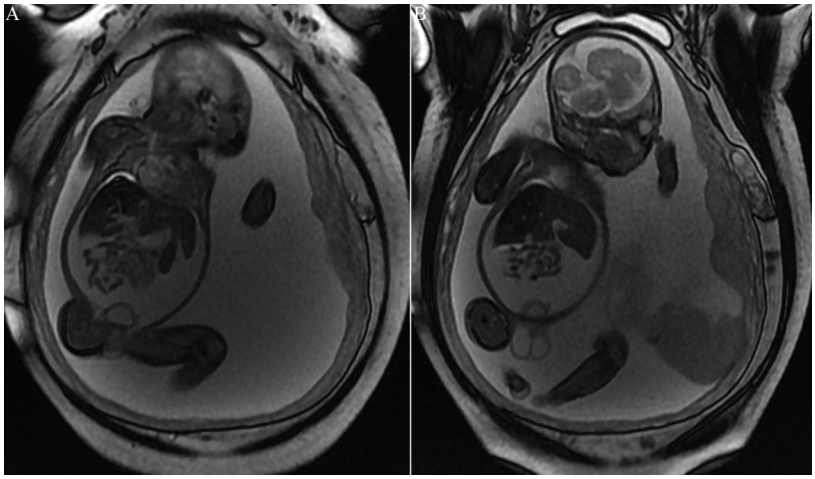


Figure 2. — A: Fetal autoimmune hemolytic anemia; FGR, polyhydramnios, fetal abdominal effusion, increased heart size, and hydropericardium. B: Fetal autoimmune hemolytic anemia, fetal abdominal effusion, poor lung development, reduced liver signal, and testicular hydrocele.

from the Ethics Committee of Tongji University. Written informed consent was obtained from all participants.

Fetal MRI was performed 24 hours after obstetric ultrasound using a 1.5 T optima MR 360 Fiber MR scanner. The pregnant women were placed in the supine position with foot-head direction by one 16-channel phase-array body surface coil, layer thickness 5~6 mm, interlayer spacing 0.5~1 mm, FOV 360×360~400 mm ×400 mm, and one- to two-time of motivation. The sequence of T2WI used 2D and 3D fast imaging employing steady state acquisition (2D/3D FIESTA), TR 4.0 ms, TE 1.5 ms, flip angle 55, matrix 224×224, scanning time of each layer 0.5~2.0 S and 10 ~ 20 S per scanning; the sequence of single shot fast spin echo (SSFSE) was used as TR l150~1350 ms, TE 42~90 ms, and flip angle 90. The sequence of T1WI used fast inversion recovery motion insensitive (FIRM), and the scanning range covered the entire uterus; no sedative and contrast agent was used during the examination process.

The images were co-analyzed by two radiologists with a job title higher than attending physician, large equipment manipulation license, and fetal MRI experience, and the results were to be jointly determined by them, including the location and extent of fetal edema, placenta edema and thickness, fetus-associated abnormalities, fetal lung development, etc. Fetal hydrops: the fetus appeared with fluid accumulation in at least two sites, or with separated serous cavity such as hydrothorax, ascites, pericardial effusion, whole body skin, and subcutaneous tissue edema; placental edema: MRI and general naked-eye observation revealed the placenta thickness greater than 5 cm and the placental pathology suggested villous edema; the clinical history was collected to understand the conditions of maternal edema: limited below knee was recorded as "+", extending up to thigh was recorded as "++", extending to vulva and abdominal wall was recorded as "+++", and overall edema or associated with ascites was recorded as "++++."

## Results

Among the nine fetuses in these seven OMS cases, seven fetuses had chest/seroperitoneum, six had pericardial effusion, three had scalp/whole body subcutaneous edema, one had bihydronephrosis, and one had hydrocele.

Among the seven OMS cases, two had significant placental thickening and edema, and one had placental adhesion. The mothers showed varying degrees of edema in these seven OMS cases, including: two cases with edema

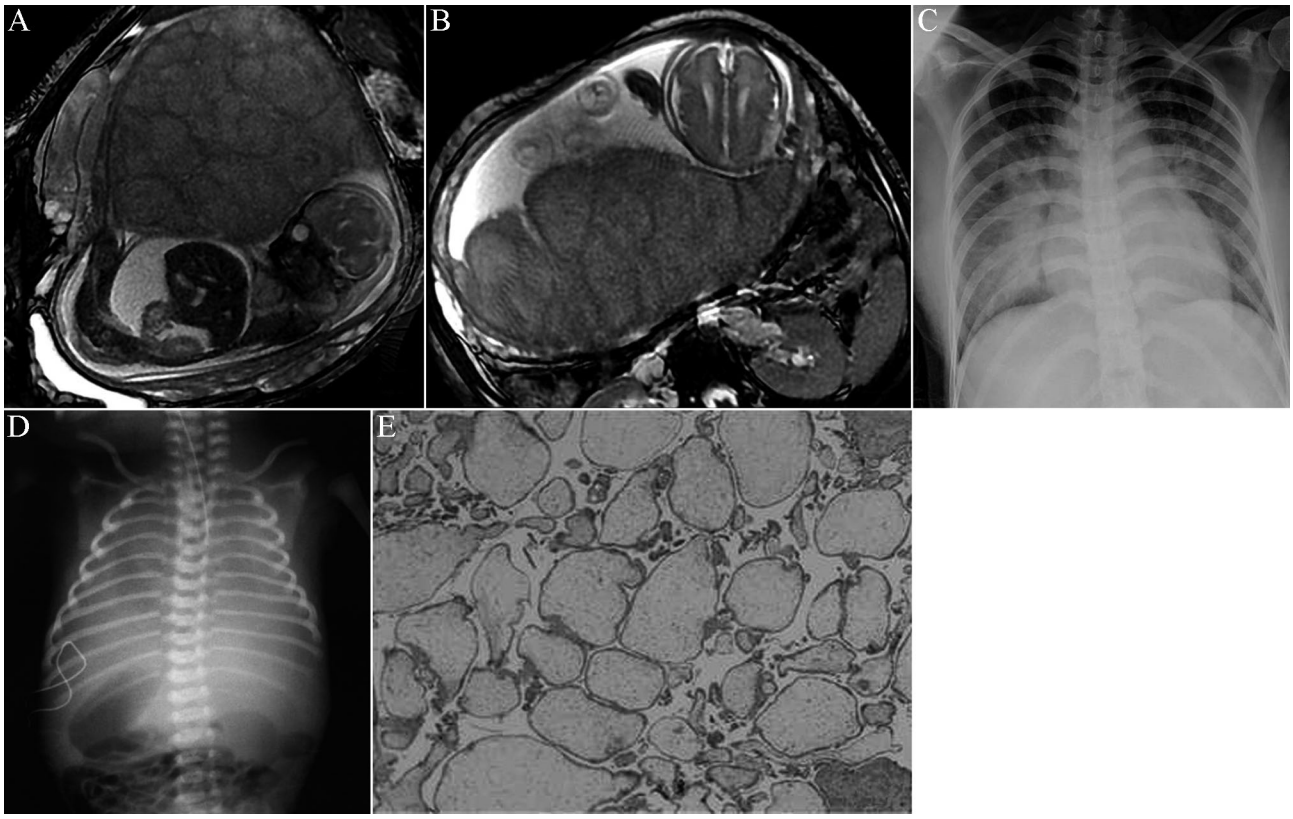


Figure 3. — A: GW 32 weeks, FGR; amniocentesis karyotype: 46, XN; the fetus displays systemic subcutaneous edema, ascites and pleural effusion, pericardial effusion, increased heart size, and poor lung development. B: The placenta is thickened by 117 mm. Intraoperative findings include significant uterine edema, pale color, poor uterine contractions after placenta delivery, and multiple active bleeding from the incision (postpartum hemorrhage). C: Prenatal maternal bedside anteroposterior chest radiography indicates bilateral pulmonary edema. D: Lower uterine-segment C-section plus breech-position midwifery delivered a baby girl, the puerperal radiography prompted neonatal respiratory distress syndrome (NRDS) level IV, ascites and pleural effusion, and the baby died half an hour later due to ineffective rescue. E: Manual dissection shows large placenta, weighing 1,000 grams with a thickness of ten cm, and partially adhesive. HE ×40 microscopy reveals placenta edema: increased villi, edema, balloon-like appearance, and the trophoblast cells on partial villous surface degenerated.

(+), two cases with edema (++), and three cases with edema (+++). At the same time, different degrees of hypertension, positive urinary protein, and hypoproteinemia were associated. One case appeared with prenatal pulmonary edema. All the maternal symptomatology spontaneously resolved after fetal hydrops was treated, or when the fetus died or was expulsed.

The associated malformations in these seven OMS cases included fetal sacrococcygeal teratoma, SCT (1/7) (Figure 1), Rh-defect (D) caused immune hemolytic edema (1/7) (Figure 2), FGR (2/7) (Figures 3 and 4), TTTS (2/7) (Figures 5 and 6), and placenta chorioangioma (1/7).

In addition to fetal hydrops, all these seven cases were associated with polyhydramnios, and among the nine fetuses, seven had fetal lung hypoplasia, one followed spontaneous intrauterine demise, three were aborted, one died after birth, and two were delivered at full-term with good physical health (Table 1).

Among these seven OMS cases, the one with placental chorioangioma was accurately diagnosed by MRI, while ultrasound diagnosed it to be placenta cyst; one case of intra- and extra-growing sacrococcygeal teratoma was correctly diagnosed by MRI, but ultrasonic suggested it to have an exogenous mixed sacral mass (unspecified). The one case of immune hemolytic edema, two FGR cases, and the two TTTS cases were all correctly diagnosed by ultrasound and MRI.

## Discussion

OMS is a group of pregnancy-associated rare diseases that would endanger maternal and fetal lives, and the number of cases reported is rare; meanwhile, intervention therapies are much more complex, so early diagnosis in the pregnancy stage would be crucial for timely clinical treatment and prognostic improvements. With extensive use of

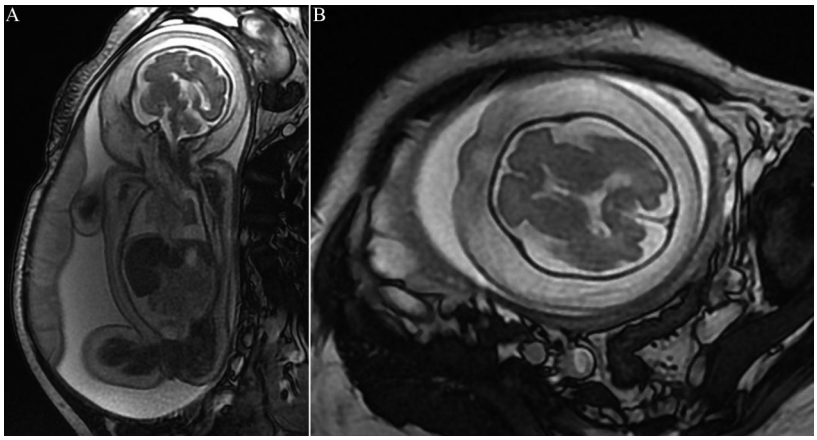


Figure 4. — A: G4P0, GW 27w+5 days, single birth, maternal edema (+++), and history of adverse pregnancy (overall edema and stillbirth). The fetus had FGR, polyhydramnios, extensive subcutaneous edema in trunk and limbs, ascites and pleural effusion, pericardial effusion, increased heart size, and reduced lung volume. B: Fetal scalp showing 20-mm edema; the fetus shows hemodilution, performed intrauterine blood transfusion, and died intrauterinely the next day. Autopsy showed the fetus with severe systemic edema, skin edema, ascites and pleural effusion, greater heart than gestational ages, lung hypoplasia, and severe placenta adhesions.

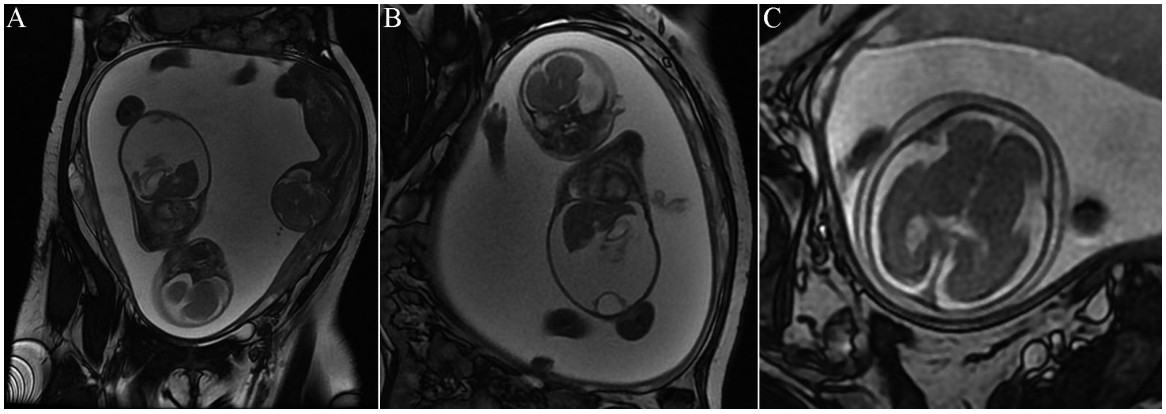


Figure 5. — A: 25-year-old pregnant woman, G2P0; GW 27w+6 days, monochorionic diamniotic twin (MCDA), TTTS IV; sIUGR in the blood-feeding fetus (left side), oligohydramnios and wall adherent, and poor bi-lung development by lower signal. The blood-reception fetus (right side) shows polyhydramnios. B: TTTS IV; the blood-reception fetus shows polyhydramnios, pericardial effusion, massive ascites, increased heart size, and decreased dual-lung signal. C: The blood-reception fetus showes eight-mm scalp edema. FLOC was ineffective and the patient returned to her local hospital for an abortion.

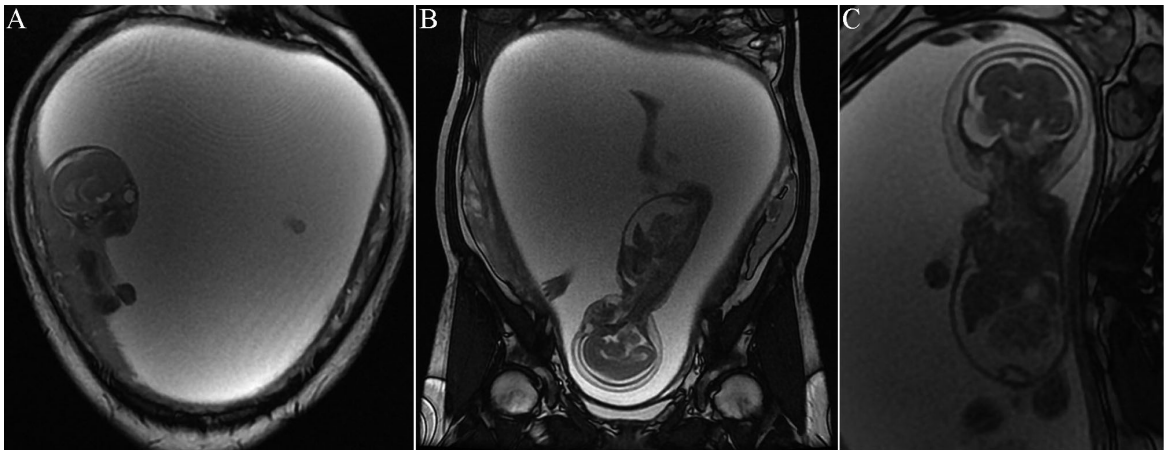


Figure 6. — A: GW 23w+3 days, MCDA, TTTS IV; the blood-feeding fetus shows sIUGR, attached to the right uterine wall. Amnioreduction and SRFA were performed to abort the blood-reception fetus, and the blood-feeding male fetus was born healthy at 39 weeks at full-term, weighing, 3,005 grams, with Apar score of 10 points. B: The blood-reception fetus shows polyhydramnios, scalp edema, excessive ascites, pericardial effusion, increased heart size, and poor lung development with decreased sinus. C: Amnioreduction and SRFA are performed to abort the blood-reception fetus.

Table 1. — *Analysis of 7 OMS cases.*

| No | GW | Maternal pregnancy and delivery history | Cause of fetal hydrops | Fetal MRI findings | Secondary maternal symptoms | Treatment outcomes | Pathological results |
|---|---|---|---|---|---|---|---|
| 1 | 22.6 | G3P0 | Fetal sacrococcygeal Teratoma | Slightly more amniotic fluid, fetal sacrococcygeal teratoma intra- and extra-growing, peritoneal fluid, pericardial effusion, bilateral hydronephrosis, poor lung development | Edema (++), hemodilution, hypoalbuminemia, proteinuria, hypertension | Rivanol induction of labor | Not performed autopsy |
| 2 | 32.3 | G4P2 | Fetal autoimmune hemolytic anemia; FGR | Polyhydramnios, fetal abdominal effusion, increased heart size, pericardial effusion, hydrocele of testis, poor lung development | Edema (+++), hemodilution, hypoalbuminemia, high blood pressure; Rh (D) and high-titer antibody (anti-CcEe512) | Back to Local hospital for Rivanol induction of labor | Not performed autopsy |
| 3 | 32 | G2P0 | Intrauterine growth restriction FGR; amniocentesis karyotype: 46, XN | Polyhydramnios, increased fetal right ventricle, whole body subcutaneous edema, lots of effusion in chest and abdominal cavity, pericardial effusion, poor lung development, umbilical cord edema; placenta thickened and edema by 117mm | Edema (+++), hypoalbuminemia, hemodilution. Prenatal bedside anteroposterior chest radiography: bipulmonary edema | Postnatal radiography prompted NRDS grade IV, ascites and pleural effusion, died | Severe systemic edema: skin edema, large chest and abdominal effusion, small pleural and pericardial effusion, scalp edema. Dual lung hypoplasia, increased right heart, brain choroid plexus vascular congestion, foot length less than gestational age. |
| 4 | 27.5 | G4P0 | Intrauterine growth restriction FGR | Polyhydramnios, fetal scalp edema by 20mm, wide subcutaneous edema in trunk and limbs, ascites and pleural effusion, pericardial effusion, increased heart size, reduced lung volume, and placenta adhesion. | Edema (+++), proteinuria, hypertension, hemodilution, hypoalbuminemia; history of adverse pregnancy (overall edema and stillbirth) | Intrauterine fetal blood transfusion, and the fetus died the next day, Rivanol induction of labor | The fetal body exhibited severe edema, skin edema, pleural effusion, the heart was greater than gestational age, dual-lung hypoplasia, and severe adhesion placenta |
| 5 | 27.6 | G2P0 | TTTS IV | monochorionic diamniotic twin (MCDA) pregnancy; the blood-reception fetus had excessive amniotic fluid, increased heart size, pericardial effusion, ascites, 8mm scalp edema, poor bi-lung development; placenta thickened by 63mm | Edema (++), hypoalbuminemia, hemodilution | Fetoscopic treatment was ineffective, and the patient was sent back to local hospital for Rivanol induction of labor | Not performed autopsy |
| 6 | 23.3 | G2P0 | TTTS IV | monochorionic diamniotic twin (MCDA) pregnancy; the blood-feeding fetus exhibited sIUGR; the blood-reception fetus had excessive amniotic fluid, scalp edema, ascites, pericardial effusion, increased heart size, and poor lung development | Edema (+), proteinuria, hypertension, hemodilution, hypoalbuminemia, hyperthyroidism during pregnancy, urinary glucose (+) | Amnioreduction with selective feticide of the hydropic fetus | A full-term baby boy was born at 39 weeks, 3005g, Apar score as 10 points. |
| 7 | 28.5 | G1P1 | placenta chorioangioma | More fetal amniotic fluid, minor peritoneal effusion; starting side of fetal placenta umbilical cord exhibited 77 * 68 * 45mm iso-height T1WI signal, and T2WI exhibited slightly-lower-than-amniotic-fluid high signal, associated with local nodular low signal, considered as chorioangioma. | Edema (+), hemodilution, hypoalbuminemia | symptomatic treatment maintenance pregnancy | A full-term baby girl was born at 41.1 weeks, 3383 g. Pathology: placenta chorioangioma associated with focal infarction |

fetal MRI, fetal medicine has obtained two sword-ultrasound and MRI, which have great help towards the diagnosis of OMS.

The mechanism of OMS still remains unknown, and it might be associated with the imbalance of angiogenin and the pathological status of anti-angiogenesis [14]. Among the causes of OMS, the factor of isoimmunization accounts for 29%, TTTS for 18%, viral infection for 16%, and fetal malformation and fetal/placental tumor for 37.5%. The embryonic mortality is 56%, but the related maternal symptoms normally resolved 4.8-13.5 days after the delivery [7]. OMS-related fetal/placental structural abnormalities include fetal congenital heart disease, fetal sacrococcygeal teratoma, placental chorionic hemangiomas, etc. Takahashi *et al.* [15] reported one case of OMS caused by fetal congenital mesoblastic nephroma (CMN), and the maternal symptoms of edema improved significantly after emergency cesarean section in the 28$^{th}$ gestational week. CMN caused maternal edema, but the fetus and the placenta did not appear with edema, and this clinical course might have prompted for the etiology of Ballantyne edema. Fetal leukemia-related OMS was also reported [16]. The etiological factors of the seven OMS cases in this study included one case of sacrococcygeal teratoma, one case of placental chorioangioma, one case of Rh (D) causing immune hemolysis, two cases of FGR, and two cases of TTTS.

OMS shows very similar symptoms as those in preeclampsia, and the difference lies in that OMS occurs frequently in early and severe fetal hydrops, which could develop in the first 16$^{th}$ week of pregnancy, while preeclampsia normally occurs later than 24 weeks of pregnancy. OMS is often accompanied with elevation of serum hCG, dilution anemia, polyhydramnios, large placenta, and villous edema. The perinatal fetal mortality of OMS is very high and cause-targeting treatments might generally improve fetal hydrops, such as fetal intrauterine transfusion therapy and fetoscopic laser occlusion (FLOC) [17]. Reversal of mirror syndrome after the fetal hydrops is corrected or the edema fetus is delivered. Some scholars also believed that it would still be a problem to consider whether additional intrauterine clinical intervention should have been performed to stop further deterioration of the fetal condition when progressive severe hydrops developed [18]. Fetal surgery generally does not dispose less than 26-week fetuses; therefore, non-edematous fetus now undergoes expectant treatment, maintaining the pregnancy to at least 30 weeks or more, to reduce perinatal mortality and to improve pregnancy outcomes.

The success in maintaining pregnancy and delivering baby in patients with OMS is rare. Chang *et al.* [19] reported one TTTS patient with 23+6 weeks pregnancy with OMS after amnioreduction and fetoscopic laser treatment, when fetal health was improved, maternal respiratory symptoms also improved, and this patient successfully delivered at 36 gestational weeks. In the present study, two patients ultimately maintained pregnancy and delivered, including one case of placental chorioangioma, in whom the fetal abdominal cavity showed a small amount of fluid, and the mother showed mild double-leg edema, hemodilution, and hypoproteinemia. After active symptomatic treatment, the baby was successfully delivered at 41.1 gestational weeks (full-term birth) with healthy conditions. The other TTTS patient, pregnant for 23.3 weeks, with a monochorionic diamniotic twin, showed selective intrauterine growth restriction (sIUGR) in the blood-feeding fetus, excessive amniotic fluid in the blood-reception fetus, as well as multisite of edema, poor heart and lung development, maternal edema (+), hemodilution, associated hyperthyroidism during pregnancy, urinary glucose (+); amnioreduction and selective radiofrequency fetal ablation (SRFA) were performed to abort the blood-receptive fetus, and the blood-feeding fetus was born healthy at 39 weeks. Among the other five cases in this study, the fetus in one case died after intrauterine blood transfusion, another died after birth, and the remaining three cases selected preterm delivery.

OMS is a disease with significant materno-fetal mortality and morbidity, so early diagnosis could be of significance in providing appropriate treatment protocols and improve clinical outcomes. MRI has no ionizing radiation, good soft tissue contrast, wide vision, and multi-planar imaging. Currently, fetal MRI inspection equipment mainly uses 1.5 T field-strength, and 3.0 T MRI examination has already been reported, which has higher soft tissue resolution and could much more clearly exhibit fetal anatomy and pathological abnormalities [20, 21]. In addition to conventional scanning sequences, such MRI spectroscopy imaging, diffusion tensor imaging, oxygen-dependent brain function imaging, and magnetic susceptibility weighted imaging could also be used for scientific researches [22].

Fetal MRI has fairly reliable diagnosis towards such acquired brain abnormalities as fetal ventricular dilatation, agenesis of the corpus callosum, posterior fossa abnormalities, neural tube closure insufficiency, abnormal neuronal migration, whole forebrain malformations, fetal brain hemorrhage, and brain tumors [12]. In addition, the diagnostic accuracy rate of fetal MRI towards urogenital system could be as high as 86.7%, and it could also correct ultrasonography. When OMS includes oligohydramnios, it would be difficult for ultrasound to clearly show the fetal structure, but fetal MRI could compensate its defects, hence it could be used a powerful complementary means of ultrasound [23]. MRI has obvious advantages towards maternal obesity, leiomyoma of uterus, twins or multiplets, fetal head into the pelvic cavity in the third trimester, and oligohydramnios in patients with OMS, and it could characterize anomalies associated with fetal hydrops, and assess fetal hydrops-resulted heart and lung developments. Among the nine fetuses in this study, seven had fetal lung hypoplasia, leading to adverse pregnancy outcomes. The application of fetal MRI lung-liver signal intensity ratio (LLSIR) to assess fetal lung

development has important roles towards the treatment of newborns after birth. Under normal pregnancy, fetal LLSIR shows positive linear correlation with gestational age. When fetal LLSIR is significantly reduced, it might suggest the presence of fetal pulmonary hypoplasia [13].

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