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Hydrops Fetalis and Mirror Syndrome Secondary to Rh-D Alloimmunization, Associated with Oligohydramnious: A Case Report

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ABSTRACT

One of the most common causes of fetal anemia is red cell alloimmunization. The most common routes of maternal sensitization are via blood transfusion or fetomaternal hemorrhage. Antibodies can cross the placenta during pregnancies in alloimmunized women and, if the fetus is positive for these specific erythrocyte surface antigens, result in hemolysis of fetal erythrocytes and anemia. This in turn, can lead to potentially disastrous consequence for the fetus, such as Hydrops Fetalis (HF), a high-output cardiac failure syndrome. The standard treatment in fetuses with anemia is intrauterine transfusion (IUT). Mirror Syndrome (MS) is a rare condition in pregnancy, in which maternal edema in pregnancy is seen in association with severe fetal and/or placental hydrops. The pathogenesis, although not well established, mimics trophoblastic damage and maternal vascular endothelial dysfunction, as is also observed in preeclampsia, and hence, the two conditions may have a similar clinical presentation [1]. The clinical manifestations of this disease are complex. It is easily underdiagnosed and timely intervention is needed to prevent fetal and maternal morbidity. MS can be reversible when the underlying factors are identified and modified [2, 3]. If correction of the underlying fetal abnormality is not possible, the consensual treatment is to deliver the hydropic fetus and placenta, with improvement of the maternal condition shortly thereafter. We report a case of HF and MS secondary to Rh-D alloimmunization that did not respond to IUT.

Introduction

Hydrops Fetalis (HF) is the excessive fluid accumulation in two or more fetal tissues, which results in gross edema, occurrence of ascites, pleural and pericardial effusions. In addition, an increased amount of amniotic fluid (polyhydramnious) and an enlarged placenta (placentomegaly) may be observed, although these findings are not part of the formal diagnostic criteria for HF. In etiology, it has been divided into immune HF (IHF) [4] and nonimmune HF (NIHF) [5]. It is a serious condition indicating a bad prognosis of affected fetuses. IHF is a fetal hydrops resulting from the passage of maternal antibodies into the fetal compartment causing fetal anemia and subsequent hydrops. This disease process can affect both the

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fetus and neonate and is better labeled as hemolytic disease of the fetus and newborn (HDFN). Until the latter half of the 20th century, it was believed to be due to Rhesus (Rh) blood group isoimmunization of the fetus. More recent recognition of factors other than isoimmune hemolytic disease that can cause or be associated with FH, led to the use of the term nonimmune hydrops to identify those cases in which the fetal disorder was caused by factors other than isoimmunization [6]. Hemolytic disease of the newborn, secondary to rhesus alloimmunization was once a major contributor to perinatal morbidity and mortality. Today, rhesus immune globulin has markedly decreased the prevalence of this disease. It is less than three cases occur in every 1000 live births [7]. Universal use of prophylactic anti-D immune globulin has reduced the need for IUT dramatically; however, the procedure continues to be an essential modality for treatment of severe fetal anemia from a variety of causes, such as RhD alloimmunization. MS is a rare complication of fetal hydrops appearing as triple edema (fetal, placental as well as maternal) [8], in which the mother mirrors the hydropic fetus. This syndrome was described firstly in 1892 by the Scottish obstetricians John William Ballantyne [9]. The exact incidence of MS worldwide is unknown. Most of the currently available data in the literature are provided in sporadic case reports that have been published in the literature [10]. The pathophysiologic mechanism of this rare syndrome remains to be elucidated, although a recent report suggested that a functional alteration in the placenta similar to that noted in preeclampsia may be involved [11]. Fetal prognosis is poor when MS develops. As described by S.Allarakia et al. and T.Braun et al., the condition results in intrauterine fetal death in over 50% of cases [12, 13].

Case presentation

A 26-year-old Afghan woman (gravida 6, parity 5, live 2, Intra Uterine Fetal Death (IUFD) 3, at 27 weeks gestational age) was referred to our center due to HF. Her pregnancy was in spontaneous conception. Her past medical and drug history was unremarkable. She had a history of two normal term deliveries six and nine years ago. Her blood type was A negative, and her husband's blood type was O positive. The blood type of the first and second children was positive. The third, fourth and fifth pregnancy was terminated at the gestational age of 32, 22 and 20 weeks due to IUFD secondary to HF, respectively. All deliveries were vaginal. In the first and second pregnancies, she did not take any prenatal care, did not inject anti-D immunoglobulin, and her deliveries have been at home. Her Indirect Coombs Test (ICT) was positive in third and fourth pregnancy at the time of delivery, but she did not have any pregnancy care. Fetal assessments (NT and anomaly ultrasound, echocardiography and karyotype) were normal in this pregnancy. Moreover, parental karyotype and hemoglobin electrophoresis were both normal. In this pregnancy, antibody titer and serial Middle Cerebral Artery Peak Systolic Velocity (MCA PSV) ultrasound were performed, but unfortunately, the mother was not referred in time. The last MCA PSV in ultrasound was reported to be normal at 25 weeks gestational age. In addition, the last antibody titer was at 22 weeks gestational age that was higher than critical titer (titer: 1/128). When mother was referred to our center, she had an ultrasound the day before, which reported HF. At the time of admission, the mother's vital signs were stable. In general physical examination, she had generalized edema especially in lower extremities. An ultrasound was repeated in our center which showed high MCA PSV (> 2 MOM), HF (ascites, generalized subcutaneous edema and pericardial effusion) (Figs. 1, 2, 3) and dilated cardiomyopathy (Fig. 4). The volume of Amniotic Fluid (AF) was low (oligohydramnious, AF Index: 5 centimeter) and placenta was thick (thickness: 6.26 cm) (Fig. 5). No reason for oligohydramnious could be found, as rupture of membrane, fetal growth restriction, or genitourinary abnormalities. On the first day, the laboratory test results were as follows: Hemoglobin (Hgb): 10 g/dl, Hematocrit (Hct.): 29.6, Platelet: 156,000 per microliter, Creatinine (Cr): 0.9, AST: 91, ALT: 86, ALP: 811, LDH: 552 and Urine protein (UP): 2+. 90 cc packed red blood cell was transfused due to severe fetal anemia (Hgb: 2.9 g/dl, reticulocyte count: 47% and corrected reticulocyte count: 9%), and fetal Hgb was increased to 7.4 g/dl. Two days after the first IUT, the IUT was performed again, which Hgb was 6 g/dl before IUT, and 12.6 g/dl after 120 cc blood transfusion. Dramatically, amniotic fluid volume increased after IUT (AFI: 10 cm one day after first IUT and AFI: 15 cm one day after second IUT). Three days after the last IUT, the MS did not improve despite treatment of fetal anemia. Due to decreased platelets to 90,000 per microliter, increased liver enzymes (AST: 250, ALT: 280) and proteinuria (Urine protein 3+), she became a candidate for termination of pregnancy with diagnosis of MS. At this time, there was no evidence of hypertension and hemoconcentration, and the mother had no symptoms of headache, nausea, vomiting, epigastric pain and blurred vision. Termination of pregnancy was performed with oxytocin induction, and finally at 28 weeks gestational age, a girl baby weighing 1200 gram was born (vaginal delivery) with Apgar scores 1 and 4 at one and five minutes, respectively. The baby's Arterial Blood Gas (ABG) showed PH: 7.21,

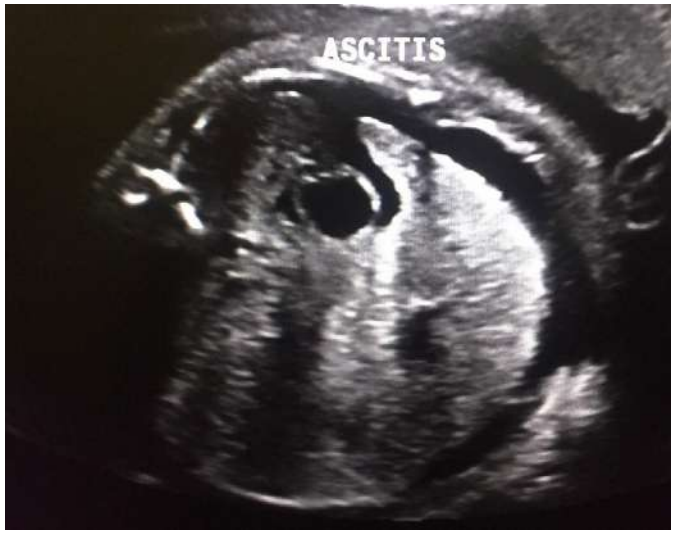

Fig. 1. Axial view shows ascites.

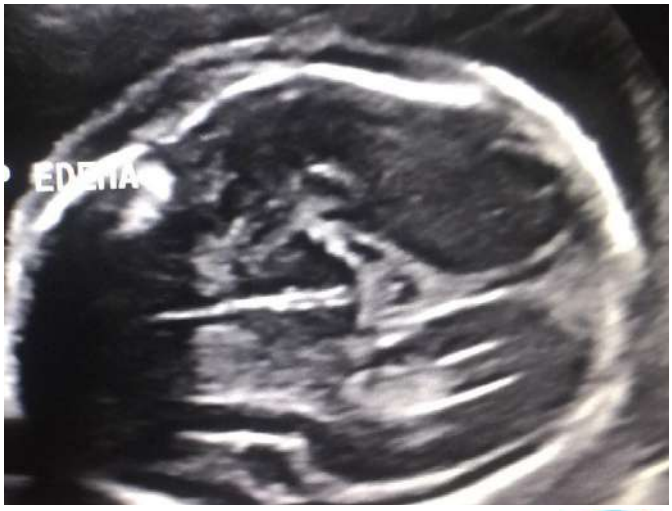

Fig. 2. Axial view shows scalp edema.

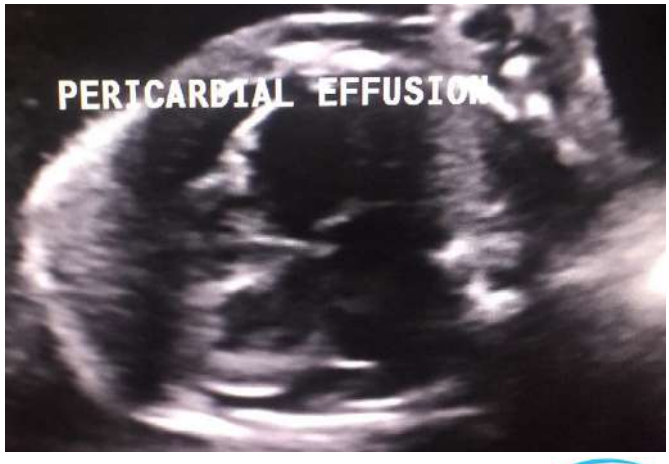

Fig. 3. Axial view shows pericardial effusion.

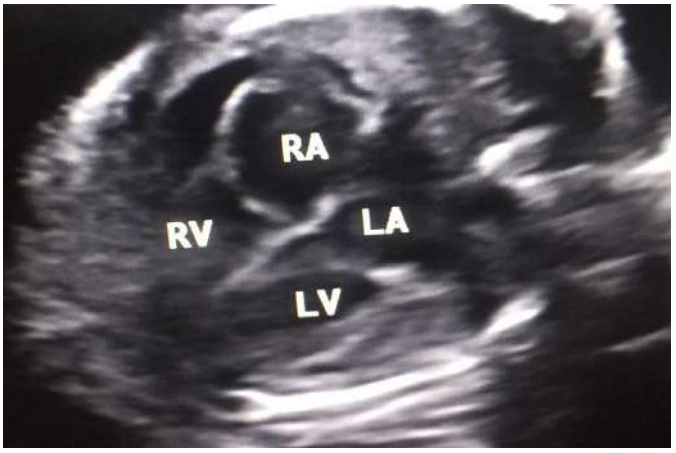

Fig. 4. Axial view shows dilated cardiomyopathy. (RA: Right Atrium, RV: Right Ventricle, LA: Left Atrium, LV: Left Ventricle).

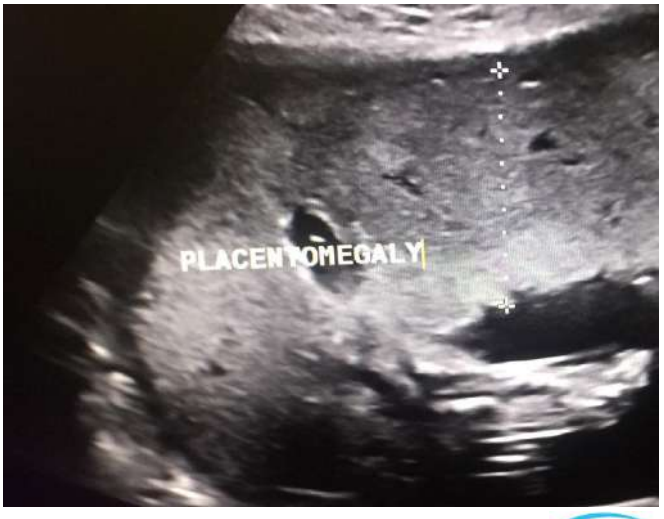

Fig. 5. Placentomegaly.

PCO2: 41, HCO3: 16.4 and Base Excess (BE): -10.4. On general examination of the baby, there was clear subcutaneous edema and hepatosplenomegaly. Her Hgb was 10 g/dl after birth. The baby died several hours after birth due to prematurity and FH. With normalized platelet and decreased liver enzymes, the mother was discharged in good general condition four days after delivery.

Discussion

Maternal alloimmunization, also known as isoimmunization, occurs when a woman's immune system is sensitized to foreign erythrocyte surface antigens, stimulating the production of immuno-globulin G (IgG) antibodies (14). Immune hydrops is most often a complication of a severe form of Rh incompatibility, which can be prevented. This is a condition in which mother who has Rh negative blood type makes antibodies to her baby's Rh positive blood cells, and the antibodies cross the placenta. IHF is much less common today since the invention of a medication known as Rh immunoglobulin (RhoGAM). Despite the development and implementation of anti- D immune globulin prophylaxis, HDFN due to maternal RhD alloimmunization continues to occur worldwide. This medication is given to pregnant women at the risk of Rh incompatibility to prevent complications. Once sensitization occurs, rhesus immune globulin is no longer effective. Furthermore, evaluation for the presence of maternal anti-D antibody should be

undertaken at the first prenatal visit [15]. In cases of HF, accumulation of interstitial fluid occurs because the production of interstitial fluid greatly exceeds the lymphatic return. Although the pathogenesis is not clearly understood, HF appears to be multifactorial due to mechanisms that produce elevated central venous pressure, impair lymphatic return and increased capillary leakage. Moreover, HF is not itself a disease, but an ultrasound marker of other fetal complications. It is defined as an abnormal collection of fluid in at least two different fetal organ spaces. Hydrops was defined as mild when there was a distinct rim of ascites, with or without pericardial effusion, and as severe when there was an abundant amount of fluid collection, usually ascites, with skin edema. HF was severe in our case. Pregnant women with HF may experience the following symptoms if the fetus has HF: polyhydramnious and abnormally thick placenta. Our case had thick placenta but oligohydramnious. In developed countries, most cases of HF will be diagnosed prior to delivery because of the routine use of antenatal ultrasound [16]. An attempt to determine the etiology of hydrops should be made at the time of diagnosis, since several etiologies can be confirmed or excluded based upon ultrasound findings. The cause of hydrops can be determined prenatally or postnatally in 60 to 85 percent of cases. The presence of hydrops is a poor prognostic indicator for perinatal survival. Management and intervention are affected by the underlying disease process and the gestational age at detection. Fetal anemia, fetal arrhythmias and complications of monochorionic twin pregnancy are amenable in utero intervention. Maternal-fetal medicine specialists and neonatologists should be involved in the management of these pregnancies. Close surveillance of maternal status is important because of the increased risks of MS. MS (Ballantyne's syndrome) is a rare, potentially life-threatening obstetric complication characterized by the development of maternal edema, hypertension and proteinuria in association with fetal hydrops. The pathogenesis of MS remains unclear and may be misdiagnosed as preeclampsia. The maternal condition mirrors the edema present in the fetus and/or the placenta [17]. This entity was first described in association with rhesus-immunization, although MS is most commonly associated with non-immune fetal hydrops (NIFH) of unknown etiology [18]. The pathophysiological mechanism behind the syndrome remains unknown; however, it has been suggested that the hydropic placenta is the likely source, as the correction of fetal hydrops (and, hence, placenta hydrops) or termination of the pregnancy (and removal of the placenta) resolves the syndrome [19], as in our case. Besides, MS is referred to as pseudotoxaemia. About half of the patients with MS develop hypertension and proteinuria, which is consistent with the clinical diagnosis of preeclampsia. The case of the current study had proteinuria without hypertension. The worsening of the imbalance between angiogenic and anti-angiogenic factors is the likely cause of progression towards toxaemia of pregnancy [20]. Recent reports suggest that an increased amount of the placental factor sFlt-1 and decreased PlGF expression is strongly associated with MS [11, 21]. However, the same pattern of changes was also noted in patients with preeclampsia [11]. To solve this puzzle, De Olivera et al. [22], in 2011, demonstrated that the ratio of sFlt-1/PlGF of >85 favors a diagnosis of preeclampsia. He hypothesized that MS patients have higher PlGF levels due to an increased placental mass; therefore, exhibit lower ratios. The incidence of MS peaks during the late second trimester of pregnancy, with a mean gestational age at the time of diagnosis of 27 weeks+_ 30 days, as our case (range of gestational age: 16-39 weeks) [12]. Maternal findings in a decreasing order of frequency in MS are as following: weight gain and edema (84%), hypertension (60.1%), anemia or low hematocrit (51.3%), dyspnea or pulmonary edema (30%), elevated uric acid/creatinine (20.3%), elevated liver enzymes (19.4%), oliguria (15%), headache (12.3%), visual symptoms (8.8%), low platelet count (8.8%), nausea and vomiting (5.2%). Fetal findings in a decreasing order of frequency are as following: HF (94.7%), placental edema (62.8%), polyhydramnious (33.6%), anomaly (17.6 %), organomegaly (16.8%), tumor (15%), oligohydramnious (13.2%) [12]. In a systematic review by Allarakia et al. [12], in approximately half of the cases, maternal and fetal findings were diagnosed simultaneously. However, in 36.28%, fetal findings were identified earlier. There was no difference in fetal mortality between cases in whom fetal and maternal findings were diagnosed simultaneously and those who were diagnosed on different dates. Thus, the sequence of presentation has limited prognostic value. Perinatal associated comorbidities in a decreasing order of frequency are as following: fetal anemia (16.8%), Rh-alloimmunization (16.8%), multiple gestation pregnancy (16.8%), twin-to-twin transfusion (13.2%), parvovirus B19 (12.3%), cytomegalovirus (2.7%), coxsackie virus (0.8%), metabolic disorder (0.8%) [12]. The infusion of red blood cells into the fetus is one of the most successful in utero therapeutic procedures. Observational studies have clearly demonstrated that IUT of the severely anemic fetus improves survival. The association of edema, oliguria and hemodilution might be the characteristic of MS, as opposed to preeclampsia with low hematocrit [23]. It should be declared that our case had hemodilution. The earlier onset during pregnancy is an important factor that differentiates MS from preeclampsia, as our case [2]. Overlap between MS and preeclampsia can occur, but it is unusual for MS to present as late as

preeclampsia. Additionally, liver function tests and the platelet count usually remain unaffected in MS [24], but like in preeclampsia, laboratory findings may include proteinuria, low platelet count and elevation of creatinine, hepatic enzymes and uric acid levels [25]. The case of the study had low platelet and elevated liver enzymes. It is reasonable to think that MS is one of the variants of preeclampsia with some similar findings. If the symptoms of MS continue, the termination of pregnancy is recommended to prevent maternal mortality and morbidity [26], as in our case. Fetal prognosis in MS is usually worse than in preeclampsia, resulting in many cases in intrauterine fetal demise [10], and being 56% the currently reported rate of intrauterine fetal death in MS [20]. Yeom et al. [27], reported no difference in the incidence of polyhydramnious or placental thickness between cases of neonatal survival and those with neonatal death. In MS, interventions to correct the FH related to anemia were significantly associated with improved fetal survival [28]. When the underlying fetal insult is corrected, we can expect a slow but sustained recovery of maternal condition that may require an adequate intensive support [29]. When FH is irreversible, induction or termination of the pregnancy may be the only choice to ensure the safety of the mother, as in our case. Vaginal delivery may be preferred, but complications as maternal pulmonary edema or deterioration of the fetal condition can lead to an emergent cesarean section [3, 18, 30]. Our case had vaginal delivery. A complete reversal of maternal symptoms usually occurs following delivery or termination [20], as in our case. MS does not usually present with oligohydramnious [31]. In a systematic review by Allarakia et al. [12], polyhydramnious was detected in 33.6% of fetuses and oligohydramnious in 13.2% of them. In this case, we were faced with oligohydramnious. The pathogenesis of oligohydramnious in MS is not explained in the literatures due to the small number of cases and the rarity of the disease. This may be due to decreased placental function secondary to its edema. Usually fetal anemia secondary to alloimmunization is chronic and is not cause of acute prerenal kidney failure. Prerenal kidney failure is one of the causes of oligohydramnious. Acute anemia (hypovolemic shock) such as massive fetomaternal hemorrhage can be an acute cause of fetal renal impairment. Shahar et al. [32], described three cases in which fetomaternal hemorrhage caused hypovolemic shock at birth. All cases had severe anemia. Fetuses with hydrops usually have polyhydramnious. In our case, fluid volume increased dramatically after IUT, this may be due to increased placental function or fetal kidney function. As a result, more studies are needed to investigate the causes of oligohydramnious in MS in the future.

Conclusion

MS is serious for the mother and baby, and in utero treatment of hydropic fetus can resolve the maternal MS. Therefore, early diagnosis and intervention in HF is essential in cases that can be treated in utero. The case of this study was an example of a HF and MS that was referred in the late stage. For this reason, the course of MS did not improve despite IUT. Another reason could be that MS is a variant of preeclampsia, and pregnancy termination is the only treatment if clinical symptoms or laboratory abnormalities persist.

Ethical Considerations

Compliance with ethical guidelines

There were no ethical considerations to be considered in this research.

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Authors' contributions

All authors equally contributed in preparing this article.

Conflict of interest

Authors declare that there is no conflict of interest.

References

[1] Mathias CR, Rizvi C. The diagnosis conundrum of maternal mirror syndrome progression to preeclampsia- A case report. Case Reports in Women's Health 23(2019) e00122. https://doi.org/10.1016/j.crwh.2019.e00122 PMID:31193796]

[2] Brandao AM, Domingues AP, Fonseca EM, Miranda TM, Moura JP. Mirror syndrome after fetoscopic laser treatment-a case report. Rev Bras Ginecol Obstet 2016; 38(11): 576-579. https://doi.org/10.1055/s-0036-1593895[PMID:28008591]

[3] Li H, Gu WR. Mirror syndrome associated with heart failure in a pregnant woman: a case report. Int J Clin Exp Med 2015; 8(9): 16132-16136.

[4] Gautier E, Benachi A, Giovangrandi Y, et al. Fetal RhD genotyping by maternal serum analysis: A two-year experience. Am J Obstet Gynecol 2005; 192:666-669. https://doi.org/10.1016/j.ajog.2004.10.632 [PMID:15746656]

[5] Sohan K, Carroll SG, De La Fuente S, et al. Analysis of outcome in hydrops fetalis in relation to gestational age at diagnosis, cause and treatment. Acta Obstet Gynecol Scand 2001; 80:726-730. https://doi.org/10.1034/j.1600-0412.2001.080008726.x [PMID:11531615]

[6] Hamdan AH. Pediatric hydrops fetalis: Background, Pathophysiology, Etiology. Pediatrics: Cardiac Disease and Critical Care 2017. https://emedicine.medscape.com/article/974571-overview#:~:text=The%20basic%20mechanism%20for%20the,or%20decreased%20plasma%20osmotic%20pressure.

[7] McLean LK, Hedriana HL, Lanouette JM, Hanss C, Haesslein. A retrospective review of isoimmunised pregnancies managed by middle cerebral artery peak systolic velocity. American journal of Obstetrics and Gynecology 2004; 190: 1732-1738. https://doi.org/10.1016/j.ajog.2004.02.060 [PMID:15284784]

[8] Ballantyne KIH. Triple edema. Am J Obstet Gynecol 1971; 110: 115-120. https://doi.org/10.1016/0002-9378(71)90226-2

[9] Ballantyne JW. The disease and deformities of the fetus. Edinburg: Oliver & Boyd; 1892.

[10] Braun T, Brauer M, Fuchs I, Czernik C, Dudenhausen JW, Henrich W, Sarioglu N. Mirror syndrome: a systematic review of fetal associated conditions, maternal presentation and perinatal outcome. Fetal Diagn Ther 2010; 27(4): 191-203. https://doi.org/10.1159/000305096 [PMID:20357423]

[11] Goa S, Mimura K, Kakigano A, Tomimatsu T. Normalisation of angiogenic imbalance after intrauterine transfusion for mirror syndrome caused by parvovirus B19. Fetal Diag Ther 2013; 34: 176-179. https://doi.org/10.1159/000348778 [PMID:23711762]

[12] Allarakia S, Khayat HA, Karami MM, et al. Characteristics and management of mirror syndrome : a systematic review (1956-2016). J Perinat. Med. 2017; 45(9):1013-1021. https://doi.org/10.1515/jpm-2016-0422 [PMID:28315852]

[13] Braun T, Brauer M, Fuchs I, Czernik C, Dudenhausen JW, Henrich W, Sarioglu N. Mirror syndrome: a systematic review of fetal associated conditions, maternal presentation and perinatal outcome. Fetal Diagn Ther 2010; 27: 191-203. https://doi.org/10.1159/000305096 [PMID:20357423]

[14] Baur AR, Springel EH. Erythrocyte alloimmunization in pregnancy. Obstetrics and Gynecology 2018. https://emedicine.medscape.com/article/273995-overview#:~:text=Maternal%20alloimmunization%2C%20also%20known%20as,immunoglobulin%20G%20(IgG)%20antibodies.

[15] Moise KJ. Management of rhesus alloimmunization in pregnancy. Obstetrics and Gynecology 2002; 100(3): 600-611. https://doi.org/10.1016/S0029-7844(02)02180-4

[16] Wafelman LS, Pollock BH, Kreutzer J, Richards DS, Hutchison AA. Nonimmune hydrops fetalis: fetal and neonatal outcome during 1983-1992. Biol Neonate 1999; 75(2): 73. https://doi.org/10.1159/000014080 [PMID:9852356]

[17] Jones T, Miesnik SR. Mirror syndrome: a rare, potentially life-threatening obstetric complications. Journal of Obstetric, Gynecology & Neonatal Nursing 2015; 44(1): S85. https://doi.org/10.1111/1552-6909.12594

[18] Society for Maternal-Fetal Medicine. (SMFM), Norton ME, Chauhan SP, Dashe JS. Society for Maternal-Fetal Medicine (SFMF) clinical guidlinre#7: non-immune fetal hydrops. Am J Obstet Gynecol 2015; 212(2): 127-139. https://doi.org/10.1016/j.ajog.2014.12.018 [PMID:25557883]

[19] Silvia F NP, Karen CF. Significant clinical manifestations in Ballantyne syndrome, after a case report and literature review: recognizing preeclampsia as a differential diagnosis. Case Rep. Obstet. Gynecol 2019; 2019 (7 pages). https://doi.org/10.1155/2019/2013506 PMID:30949371

[20] Espinoza J, Romero R, Nien JK, Kusanovic JP, Richani K, Gomez R, et al. A role of the anti-angiogenic factor sVEGFR-1 in the mirror syndrome (Bullantyne syndrome). J Matern Fetal Neonatal Med 2006; 19(10): 607-6143. https://doi.org/10.1080/14767050600922677 [PMID:17118734]

[21] Prefumo F, Pagani G, Fratelli N, Benigni A, Frusca T. Increased concentrations of antoangiogenic factors in mirror syndrome complicating twin-to-twin transfusion syndrome. Prenat Diagn 2010; 30: 378-379. https://doi.org/10.1002/pd.2461 [PMID:20127899]

[22] De Oliveira L, Sass N, Boute T, Moron AF. SFlt-1 and PlGF levels in a patient with morror syndrome related to cytomegalovirus infection. Euro J Obstet Gynecol Repr Bio 2011; 158: 366-367. https://doi.org/10.1016/j.ejogrb.2011.04.049 [PMID:21621898]

[23] Carbillon L, Oury JF, Guerin JM, Azancot A, Blot P. Clinical biological features of Ballantyne syndrome and the role of placental hydrops. Obstet Gynecol Surv 1997; 52(5): 310-314. https://doi.org/10.1097/00006254-199705000-00023 [PMID:9140132]

[24] Iciek R, Brazert M, Klejewski A, Pietryga M, Brazert J. Ballantyne syndrome (mirror syndrome) associated with severe non-immune fetal hydrops-a case report. Ginekol Pol 2015; 86(9): 706-711. https://doi.org/10.17772/gp/57845 [PMID:26665574]

[25] Giacobbe A, Grasso R, Interdonato ML, Lagana AS, Valentina G, Triolo O, Mancuso A. An unusual form of mirror syndrome: a case report. J Matern Fetal Neonatal Med 2013; 26(3): 313-315. https://doi.org/10.3109/14767058.2012.722734 [PMID:22957971]

[26] Rahimi-Sharbaff F, Saeidi N. Successful management of a complicated mirror syndrome: a case report. Case Reports in Clinical Practice 2019; 4(2): 61-63. https://doi.org/10.18502/crcp.v4i2.1709

[27] Yeom W, Paik ES, An JJ, et al. Clinical characteristics and perinatal outcome of fetal hydrops. Obstet Gynecol Sci 2015; 58: 90-97. https://doi.org/10.5468/ogs.2015.58.2.90 [PMID:25798421]

[28] Eiland S, Cvetanovska E, Bjerre AH, Nyholm, Sundberg, Norgaard LN. Mirror syndrome is a rare complication in pregnancy, characterized by edema and hydrops fetalis. Ugeskr Laeger 2017; 10(15): 179. [PMID: 28416051]

[29] Chimenea A, Gracia-Diaz L, Calderon AM, De Las Heras MM, Antinolo G. Resolusion of maternal mirror syndrome after successful fetal intrauterine therapy: a case series. BMC Pregnancy and Chilbirth 2018; 18:85. https://doi.org/10.1186/s12884-018-1718-0 [PMID:29625560]

[30] Tayler E, DeSimone C. Anesthetic management of maternal mirror syndrome. Int J Obstet Anesth 2014; 23(4): 386-389.https://doi.org/10.1016/j.ijoa.2014.05.003 [PMID:25066819]

[31] Vidaeff AC, Pschirrer ER, Mastrobatissta JM, Glistrap LC 3rd, Ramin SM. Mirror syndrome: a case report. J Repord Med 2002; 47: 770-774. [PMID: 12380459]

[32] Shahar E, Birenbaum E, Inbar D, Brish M. Hypovolemic shock at birth due to extensive fetomaternal hemorrhage. Isr J Med Sci 1981; 17(6): 441-444. https://doi.org/10.1097/00006254-198206000-00019