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One Case of Stillbirth Caused by Maternal Fetal Transfusion Syndrome and Literature Review

Fenjiao Li[1], Yuli Cen[1], Xian Chen[1], Haihua Ma[1], Qiuping Wang[2*]

[1]Department of Obstetrics, Tianlin County People's Hospital, Baise, China
[2]Department of Obstetrics, Shenzhen Guangming District People's Hospital, Shenzhen, China
Email: *58839530@qq.com

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Abstract

Fetomaternal hemorrhage (FMH) is an obstetric emergency characterized by the pathological transfer of fetal erythrocytes across the placental barrier into the maternal circulation. FMH can lead to severe complications, including fetal anemia, non-immune hydrops, and perinatal mortality. The pathogenesis involves multifactorial interactions, such as placental barrier dysfunction, hemodynamic abnormalities, and vascular endothelial injury. Due to its insidious clinical presentation and limitations in current diagnostic modalities, FMH is prone to missed or delayed diagnosis. This study presents a case of intrauterine fetal demise secondary to FMH and conducts a narrative literature review to establish an early warning indicator system, refine risk assessment models, and propose evidence-based individualized intervention strategies. The objectives are to enhance clinical recognition, standardize diagnostic and therapeutic protocols, and ultimately improve perinatal outcomes.

Keywords

Fetomaternal Hemorrhage, Pathogenesis, Diagnostic Techniques, Therapeutic Management

1. Introduction

Fetal maternal hemorrhage (FMH) is defined as the pathological transfer of fetal blood into the maternal circulation through the placental villus space during pregnancy or delivery, representing an abnormal condition of fetal maternal blood exchange [1]. Although a small amount of fetal blood entering the maternal blood stream during pregnancy or delivery is usually asymptomatic, FMH occurs when fetal blood loss reaches a critical threshold (about 30 ml), which may lead to fetal anemia, non-immune edema, and intrauterine death [2], and the incidence of this

volume is estimated to be 0.033% - 0.1% [3]. FMH accounts for more than 10% of perinatal mortality and 3% - 5% of unexplained stillbirths [4]. Survivors face an elevated risk of neurological sequelae and potential maternal complications, such as hemolytic transfusion reactions [5] [6]. The pathogenesis involves dynamic dysfunction of the placental barrier, which is characterized by loss of syncytiotrophoblast integrity, abnormal shear stress at the villus interface, and upregulation of proinflammatory cytokines (such as tnf-α, IL-6), which together disrupt the maternal fetal hemodynamic gradient [7]. Despite the progress in prenatal diagnosis, early detection still faces challenges due to non-specific symptoms (such as reduced fetal movement) and limited access to professional tests (such as flow cytometry) in resource limited settings. Current research gaps include standardized risk stratification tools and cost-effective primary care screening programs [4]. Clinical management is challenged by its asymptomatic onset and lack of validated biomarkers, often delaying diagnosis until irreversible fetal damage occurs. Current limitations still exist in early warning systems, high-risk stratification, and standardized prevention programs. This study presents a case of stillbirth secondary to FMH and a comprehensive narrative review of the global literature, aiming to (1) describe early diagnosis strategies, (2) establish a risk assessment framework, and 3. propose personalized treatment interventions. By synthesizing the available evidence, we sought to improve clinical understanding and optimize perinatal outcomes.

2. Case Information

A 31-year-old pregnant woman was admitted to the hospital due to amniotic fluid leakage after natural rupture of the fetal membrane at 39 weeks of pregnancy. This pregnancy is an embryo transfer. No abnormalities were found during prenatal checkups. OGTT examination for 26+ pregnant women: blood glucose level at 2 hours postpartum was 12.57 mmol/L; Provide dietary and exercise guidance to ensure good blood sugar control. The accident occurred during a prenatal examination at 37 weeks of pregnancy: fetal heart rate monitoring showed no response on NST, and the pregnant woman refused to be hospitalized. On the second day, the NST was rechecked and there was still no acceleration. It is recommended to be hospitalized again. The pregnant woman did not accept it and left on her own. Until the 39th week of pregnancy, the pregnant woman experienced irregular tightness in the lower abdomen and natural rupture of the membranes, resulting in amniotic fluid leakage. The patient was admitted to the hospital for delivery. At admission, fetal heart rate monitoring showed no response to NST (**Figure 1**). Oxygen therapy and intravenous injection of sodium lactate Ringer's solution were given to improve the intrauterine condition of the fetus, but were ineffective. It is recommended to perform a cesarean section immediately. However, the pregnant woman firmly refused the surgery. After repeated persuasion was ineffective, ultrasound re-examination showed that the maximum depth of the sheep pond measured was 17 mm, and the amniotic fluid index was 35 mm. Notify pregnant women

of oligohydramnios and abnormal fetal monitoring, and recommend immediate surgery. The pregnant woman and her husband agreed. An emergency cesarean section was performed 30 minutes later, with a preoperative fetal heart rate of 110 beats per minute. However, when the fetus was delivered, there was no heartbeat, breathing, muscle tone, or reflex, abnormal pale skin, no blood color throughout the body, and an Apgar score of 0-0-0. Immediately intubate, perform positive pressure ventilation and chest compressions, insert a catheter into the umbilical vein, and inject 0.5 ml of adrenaline through the umbilical vein. After 30 minutes of continuous resuscitation treatment, the Apgar score remained at 0. The birth weight of the fetus was 3070 g, and the examination showed swelling of the umbilical cord and grade I contamination of the amniotic fluid, with a volume of 100 ml. Umbilical cord blood results: HGB 25.00 g/L, RBC 0.74×10^{12}/L, HCT 8.80%, PLT 46.00×10^{9}/L (**Figure 2**). CT scans of the head, chest, and abdomen indicate: (1) Changes in neonatal hypoxic-ischemic encephalopathy (HIE). (2) Swelling of subcutaneous soft tissue at the top of the left forehead. (3) Bilateral congenital pulmonary dysplasia accompanied by atelectasis, pulmonary dampness, and pneumothorax. The family members will personally handle the autopsy. During the surgery, the appearance of the placenta was pale, and pathological examination reported local bleeding between the villi and increased cellulose deposition in the villi spaces; Localized calcification of the umbilical cord (**Figure 3**). No maternal alpha fetoprotein was detected before and after surgery (not performed in a county-level hospital). Diagnosis: (1) Fetal maternal transfusion syndrome; (2) stillbirth: stillbirth (**Table 1**).

Table 1. Timeline of critical events.

Gestational Week	Event	Clinical Decision
37	Non-reactive NST	Recommended hospitalization
39	Premature rupture of membranes	Immediate cesarean refused
39 + 1	Persistent NST abnormality	Emergency cesarean performed
Postpartum	Neonatal HGB 25 g/L	stillbirth

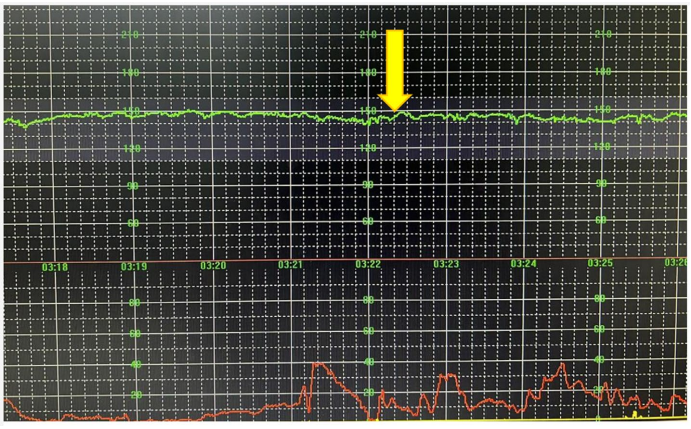

Figure 1. Yellow arrow point: Fetal heart rate monitoring: NST unresponsive.

project	result	Reference indicators
NEU#	2.91	0.6-0.75
LYM#	3.29	2.4-9.5
MON#	7.37	0.15-1.56
EOS#	0.12	0.07-1.02
BAS#	0.84	0-1
HGB	25	110-160
RBC	0.74	5.2-6.4
HCT	8.8	28-52

Figure 2. Fetal blood routine indicators after birth, yellow arrow point HGB: 25 g/L.

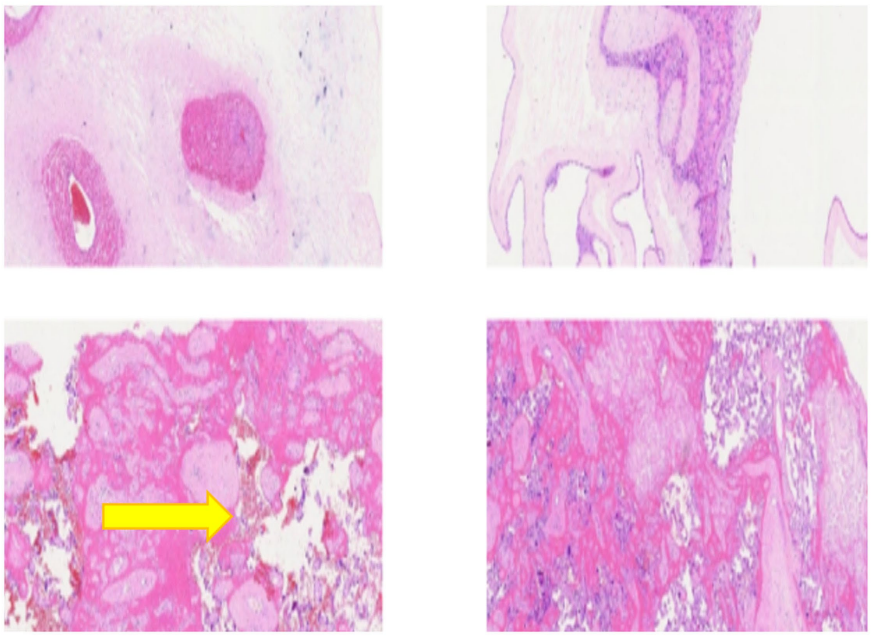

Figure 3. Yellow arrow point: pathological examination reported local bleeding between the villi and increased cellulose deposition in the villi spaces; Localized calcification of the umbilical cord.

Ethical Statement: Written informed consent was obtained from the patient and her family members for the publication of this case report and accompanying images. The consent documentation has been archived in accordance with institutional ethical guidelines.

3. Discussion

3.1. Definition and Pathogenesis

FMH is one of the less common diseases in obstetrics. The disease was first reported by Chown in 1954: a case of severe neonatal anemia caused by a large amount of maternal fetal blood transfusion. There are many clinical studies on this disease abroad, and clinical research on this disease began in the 1960s [2]. However, there

are currently no large-scale reports in China. Until now, it is believed that the pathogenesis of this disease is due to the pressure difference between the umbilical artery and the chorionic villi, which causes a small amount of fetal blood to slowly or rapidly enter the maternal blood circulation, and can occur during prenatal amniocentesis, choriocarcinoma, or strong uterine contractions during delivery [3]. However, the vast majority of FMH cases have unknown causes, and Giacoia [4] found that 82% of the 134 FMH cases had no cause. The high-risk factors for FMH have been extensively studied and found to be related to maternal factors (severe preeclampsia, placental abruption, intrauterine infection), fetal abnormalities (edema, twin blood transfusion, monochorionic twins), obstetric therapeutic procedures (chorionic villus monitoring, amniocentesis, etc.), and increased fetal intrauterine activity in late pregnancy.

3.2. Performance During Labor

The clinical manifestations and fetal outcomes of FMH are related to blood loss. During pregnancy or childbirth, fetal blood intermittently enters the maternal blood circulation through the villi. As a physiological phenomenon, due to the higher pressure between the mother and placenta than between the fetus and placenta, 95% of pregnant women may experience 0.05 - 0.1 ml of fetal red blood cells entering the mother during pregnancy. A blood volume of less than 15 ml entering the mother usually does not cause severe symptoms of fetal maternal blood transfusion [8]. When the blood loss exceeds 30ml, it develops into severe FMH, which can cause serious complications including intrauterine stillbirth, fetal anemia, and neonatal respiratory distress syndrome. It is worth noting that hypovolemic shock caused by a large amount of FMH can increase the perinatal mortality rate to 33% - 50% [9]. The most common clinical manifestations of FMH patients are reduced fetal movement, decreased or absent fetal strength. Fetal heart rate monitoring often manifests as baseline variability and late deceleration, with a typical waveform being a sine curve. Some scholars refer to the reduction or disappearance of fetal movement, fetal edema, and the sinusoidal waveform of fetal heart rate monitoring curve as the FMH triad. Reduced fetal movement may be an early signal, while fetal edema and sinusoidal curves are late manifestations of FMH [10]. However, it is extremely rare in clinical practice to have a typical triad at the same time. Therefore, when there is unexplained fetal movement reduction, fetal death in the uterus, fetal heart rate sine wave, ultrasound indicating fetal edema, postpartum neonatal anemia, etc., it should be highly suspected that FMH has occurred, and early intervention should be taken to reduce the occurrence of adverse outcomes.

3.3. Auxiliary Examination

At present, in addition to considering the clinical manifestations of patients, the diagnosis of FMH also needs to be combined with some auxiliary examinations. Common auxiliary examinations include the following.

① Red blood cell acid elution test method (Kleihauer Betke test)

The KB experimental method is currently the most commonly used screening tool for clinical diagnosis of FMH. The principle is based on the difference in stability between fetal hemoglobin (HbF) and adult hemoglobin (HbA) in acidic environments. HbF has stronger acid resistance. When the maternal peripheral blood smear is treated with acidic buffer, fetal red blood cells containing HbF show characteristic "ghost cells" after eosin staining. The amount of fetal blood loss can be estimated by counting under a microscope. However, this method has the following limitations: the experimental process takes a long time, usually requiring 2 - 3 hours; The quality of smear preparation (such as pH control, smear thickness) and the subjectivity of cell counting can affect the accuracy of the results; Some pathological conditions, such as hereditary hemoglobinopathies (including sickle cell anemia, beta thalassemia, etc.), have high HbF levels themselves, leading to false positive results [11]. Therefore, in clinical applications, a comprehensive judgment should be made based on the patient's medical history and other examination results.

② Flow cytometry detection method

Flow cytometry uses specific antibodies (anti HbF antibodies) to label fetal red blood cells in maternal peripheral blood, and quantitative analysis is performed using a flow cytometer. This technology has the advantages of high sensitivity, high accuracy, good repeatability, and stable and reliable results. However, due to the expensive testing equipment and the need for professional technical personnel, it is currently mainly used in scientific research and the diagnosis of special cases, and has not been widely applied in clinical practice [12].

③ Detection of peak systolic velocity of middle cerebral artery (MCA-PSV)

MCA-PSV indirectly evaluates the degree of fetal anemia by measuring the systolic peak flow velocity of the fetal middle cerebral artery through Doppler ultrasound. When the MCA-PSV value is ≥ 1.5 times the median multiple (MOM), it suggests that the fetus may have moderate to severe anemia. It has the advantages of non invasiveness and high sensitivity, and is suitable for monitoring the condition and evaluating the treatment effect of FMH. However, Stefanovic *et al.* [12] pointed out that MCA-PSV is more suitable for the diagnosis of acute FMH and is not recommended as a routine screening method. Piva *et al.* [13] further showed that only about 1% of individuals in the general pregnant population would detect FMH through MCA-PSV testing. Therefore, its indications should be strictly controlled in clinical applications.

④ Maternal serum alpha fetoprotein (AFP) detection

AFP, as an important fetal protein, is mainly synthesized in the fetal liver and yolk sac, and is closely related to placental barrier function. The maternal serum AFP level shows characteristic changes during pregnancy: it can be detected in early pregnancy, linearly increases from 14 - 20 weeks, and gradually decreases after 20 weeks. Liu Ying [14] found that the combined detection of HbF and AFP levels in maternal blood can significantly improve the accuracy of FMH diagnosis, with higher sensitivity and specificity than single indicator detection. However,

there are limitations in the application of FMH diagnosis, such as: (1) various pathological conditions (such as neural tube defects, multiple pregnancies, placental abnormalities, etc.) can lead to elevated AFP levels; (2) Individual differences are significant and there is no clear reference range; (3) The timing of detection has a significant impact on the results. Therefore, AFP testing should be used as an auxiliary diagnostic tool and combined with other examination results for comprehensive judgment.

⑤ Pathological examination

The cause of AFP is not clear, but research has shown that it is related to the placental barrier. Pathological examination of the placenta after delivery mostly reveals obvious lesions, such as bleeding and infarction [15]. However, this examination can only be performed after delivery and cannot be used as a preventive or screening method.

3.4. Treatment

The treatment of FMH should be tailored to the gestational age, severity of the condition, and the rescue capabilities of local hospitals, mainly including prenatal intrauterine blood transfusion and postpartum neonatal treatment.Indications and Implementation of Intrauterine Transfusion (IUT) [16]. For those with gestational age less than 32 weeks, intrauterine blood transfusion therapy is used to prolong gestational age while promoting fetal lung maturation. The indications for implementing IUT are as follows: (a)MCA-PSV ≥ 1.5 MOM; (b) Fetal hemoglobin ≤ 0.65 MOM; (c) If the fetal hematocrit (Hct) is less than 30%, after excluding other obstetric indications for emergency termination of pregnancy, IUT is recommended. This technology uses umbilical vein puncture and irradiation to remove white blood cells and concentrate red blood cells, which can significantly increase fetal hemoglobin concentration (average increase of 4.2 ± 0.8 g/dL 24 hours after surgery) and Hct level (average increase of 12.3 ± 3.1%), while reducing MCA-PSV by about 35% - 45%, effectively delaying disease progression and prolonging pregnancy cycle [17]. In clinical practice, about 72% of cases require ≥ 2 sequential IUT to maintain fetal blood parameters within safe thresholds [18]. Before implementing IUT, a multidisciplinary consultation is required, with a focus on evaluating: (a) the location of the placenta and the feasibility of the umbilical cord puncture route; (b) Fetal biophysical score (BPP ≥ 6 points); (c) Maternal coagulation function status. The postoperative monitoring system should include: 1 - 2 daily fetal movement counts combined with stress free tests (NST); 2 - 3 ultrasound evaluations per week (including blood flow parameters such as MCA-PSV, umbilical artery PI, and venous catheter alpha wave); Measure maternal anti-D antibody titers every 48 hours (for Rh negative pregnant women). Special attention should be paid to the potential risks of repetitive IUT: studies have shown that ≥ 3 blood transfusion procedures can increase fetal mortality to 8.7%, mainly attributed to chorioamnionitis and complications related to umbilical cord puncture [11].

3.5. Decision Timing for Terminating Pregnancy

For those with gestational weeks ≥ 32 - 34 weeks and MCA-PSV > 1.5 MOM, it is recommended to undergo a planned cesarean section after completing glucocorticoid induced fetal lung maturation (recommended betamethasone 12 mg im q24 h x 2 doses). Adequate blood preparation is required before surgery (it is recommended to prepare 10 - 15 ml/kg of O-Rh negative irradiated red blood cells), and a neonatal resuscitation team should be formed to implement delayed umbilical cord ligation (DCC) and targeted transfusion therapy. It is worth noting that for acute FMH accompanied by fetal hemodynamic decompensation (such as loss of alpha waves in the venous catheter or disappearance of end diastolic blood flow in the umbilical artery), pregnancy should be terminated immediately regardless of gestational age [19]. And to improve the HbF of newborns, it is recommended to squeeze the blood in the umbilical cord into the newborn's body through umbilical cord compression before cutting off the umbilical cord to increase the effective circulating blood volume of the newborn, improve the blood perfusion of various organs, and reduce the risk of anemia in newborns.

3.6. Regional Medical Resource Allocation

For medical institutions that do not have the conditions for implementing IUT, a standardized referral process should be established: ① For gestational weeks < 24 weeks, it is recommended to refer to a maternal fetal medical center within 72 hours; ② 24 - 28 weeks of gestation: Initiate remote multidisciplinary consultation and synchronously stabilize the fetal condition before transportation (including maternal hyperbaric oxygen therapy); ③ Pregnancy weeks > 28 weeks: Choose on-site delivery or transportation according to the transportation time window, and provide a neonatal transportation incubator and portable blood gas analysis equipment.

4. Conclusions

FMH has an insidious onset and as a potential critical complication of pregnancy, its clinical symptoms are non-specific. Once it occurs, the consequences can be severe. In clinical practice, pregnant women should be cautious of the occurrence of fetal movement reduction, abnormal fetal heart monitoring, and pale skin in newborns during delivery, considering the possibility of FMH. In terms of diagnosis, auxiliary examinations should be carried out based on the hospital's own conditions, and those who do not have the conditions should be referred in a timely manner. Treatment should be selected based on gestational age and condition, mainly including intrauterine blood transfusion and termination of pregnancy. At present, clinical understanding of this disease is still limited, especially in primary hospitals where there may have been no exposure to FMH cases. To analyze the high risk factors of patients in this article, including diabetes after embryo transfer, pregnancy, premature rupture of membranes, and maternal fetal blood group incompatibility (maternal type A RH+, fetal type O RH+). Whether the pathogen-

esis is related to the appeal high risk factors cannot be verified, but it has not attracted enough attention. At 37 weeks of pregnancy, during prenatal examination, it was found that the fetal heart rate monitoring was flat, without acceleration, and there was little fetal movement. Ultrasound examination did not indicate fetal edema or abnormal umbilical cord conditions. After waiting for 14 days, due to threatened labor, the fetal membrane ruptured and was admitted to the hospital. After admission, the baseline variation of fetal monitoring was flat, and it was recommended to terminate the surgery. However, the pregnant woman insisted on vaginal delivery, and intrauterine resuscitation such as oxygen supplementation was ineffective. Follow-up ultrasound showed oligohydramnios and turbidity, and the pregnant woman agreed to the surgery. However, the best surgical time was missed, resulting in adverse consequences of stillbirth.

Case-Literature Synthesis this case aligns with established reports of delayed FMH diagnosis due to atypical presentation (e.g. absent fetal edema or sinusoidal heart rate) [10]. However, the absence of MCA-PSV monitoring diverges from current guidelines recommending Doppler screening for high-risk pregnancies [12]. Notably, the patient's refusal of cesarean despite abnormal NST underscores socioeconomic and cultural barriers to timely intervention in rural settings, a phenomenon documented in low-resource obstetrics [20]. Clinical Implications Based on this case analysis and literature synthesis, we advocate for: Mandatory KB testing in county hospitals for pregnancies with reduced fetal movement (≥24 weeks), as missed KB screening contributed to diagnostic delays in 68% of FMH cases [12]; Telemedicine consultations to bridge expertise gaps, with studies showing a 40% reduction in diagnostic delays when tertiary centers provide real-time guidance [16]; Culturally tailored patient education programs to improve compliance, addressing documented distrust in surgical interventions among rural populations [21].

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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