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CASE REPORT

The Treatment of Fetal Supraventricular Tachycardia: A Case Report and Literature Review

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Abstract: Fetal supraventricular tachycardia (SVT), as the most common fetal tachyarrhythmia, can lead to serious complications such as decreased cardiac output, ascites, pleural or pericardial effusion, skin edema, and even fetal hydrops in specific cases, marking the severe stage of fetal congestive heart failure. Hydrops significantly increases infant mortality. Therefore, timely treatment and restoration of normal fetal heart rate are essential for fetal survival and full-term delivery. Based on clinical cases, this article discusses the current situation of fetal SVT treatment and individual differences, as well as the strategy of fetal drug conservative treatment and the best time to terminate pregnancy. Here we report a case of supraventricular tachycardia diagnosed by fetal heart rate monitoring at 29 weeks and 2 days of gestation. The results of fetal heart ultrasound and Doppler monitoring showed that the fetus had the possibility of cardiac insufficiency and fetal intracranial hydrops, and the fetal heart rate returned to normal after oral treatment with digoxin and sotalol. The pregnancy was terminated by cesarean section at 36 weeks and 5 days of gestation because of recurrent supraventricular tachycardia, reverse a wave of venous catheter and oligohydramnios. The newborn weighed 3035 grams. No arrhythmia was found in the follow-up of pregnant women and newborns. Fetal supraventricular tachycardia does not necessarily terminate pregnancy. In fact, most of these fetuses have a good prognosis after conservative treatment in utero. The treatment plan should take into account the gestational age, the specific conditions of the fetus and the mother. The purpose of this article is to review the diagnosis and treatment process of this case and provide valuable reference for clinical practice.

Keywords: fetal arrhythmia, fetal supraventricular tachycardia, pregnancy, timing of termination of pregnancy, case report

Introduction

In clinical practice, the common fetal tachycardia are sinus tachycardia (ST), supraventricular tachycardia (SVT), ventricular tachycardia (VT) and atrial flutter (AF). SVT is the most common, accounting for 70% −75%; the main causes of SVT include fetal chromosomal abnormalities, structural heart disease or other defects, 30% of SVT can be combined with the above cardiac abnormalities, and the incidence of fetal hydrops is as high as 30%-40%.[1] The causes of fetal supraventricular tachycardia also include fetal hypoxia, electrolyte disorders in pregnant women, fetal congenital heart disease and so on. However, in most fetus with SVT, the reason tachycardia develops is unknown.[2] The discovery of fetal SVT does not necessarily mean termination of pregnancy, and most fetuses with SVT have a good prognosis after intrauterine treatment. The treatment plan should take into account the gestational age, the specific condition of the fetus and the health status of the mother. In this process, the conservative treatment of drugs should undergo rigorous multiple assessments and continuous follow-up to accurately determine and adjust the initial treatment strategy and drug dosage.[3] At the same time, pay attention to pharmacokinetic factors and closely monitor the possible adverse reactions of the mother. Termination of pregnancy should be considered as soon as the arrhythmia condition does not improve or the fetus is at risk.

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Case Introduction

A 28-year-old female during her 29 weeks gestation routine prenatal examination, she felt that the fetal movement was frequent and there were no other discomfort symptoms. Fetal heart rate monitoring showed that the fetal heart rate was 257 beats per minute, and the diagnosis of fetal arrhythmia and tachycardia was clear. Fetal echocardiography showed fetal tachycardia (Figure 1), reversal of ductus venosus a-wave (Figure 2), umbilical vein fluctuation sign (Figure 3), a small amount of pericardial effusion in the left ventricular wall (Figure 4), a small amount of mitral regurgitation (Figure 5), and fetal intracranial hydrops (The ultrasound report describes fetal intracranial periventricular hyperechogenicity, with no images retained). She was hospitalized for "fetal arrhythmia, fetal tachycardia, possible fetal heart failure, and suspected fetal distress". The patient was immediately given symptomatic treatment such as oxygen inhalation, 5g impact and 10g (1–2g/H) intravenous infusion of magnesium sulfate, 0.25 mg digoxin, three times a day, 80mg sotalol tablets, twice a day, and 10 mg intravenous dexamethasone to promote fetal lung maturation at the first day of hospitalization. We had completed the rheumatological and immunological tests and no abnormalities were detected.

It was considered that fetal tachycardia for a long time will lead to fetal heart failure, fetal hydrops, intrauterine distress and fetal death. But if the pregnancy is terminated at 29 weeks gestation due to fetal tachycardia, the neonate will be transferred to NICU (Neonatal Intensive Care Unit) after birth, the cost is huge, and there is the possibility of neonatal cerebral palsy and death. We opted for pharmacological treatment. The frequency of supraventricular tachycardia gradually decreased within 3 days after take digoxin and sotalol. No supraventricular tachycardia was found in the fetal heart rate monitoring and fetal heart ultrasound on the 4th and 5th days. It was considered that the fetus's condition was stable, and the patient was discharged on the 5th day after admission. After discharge, the patients took digoxin

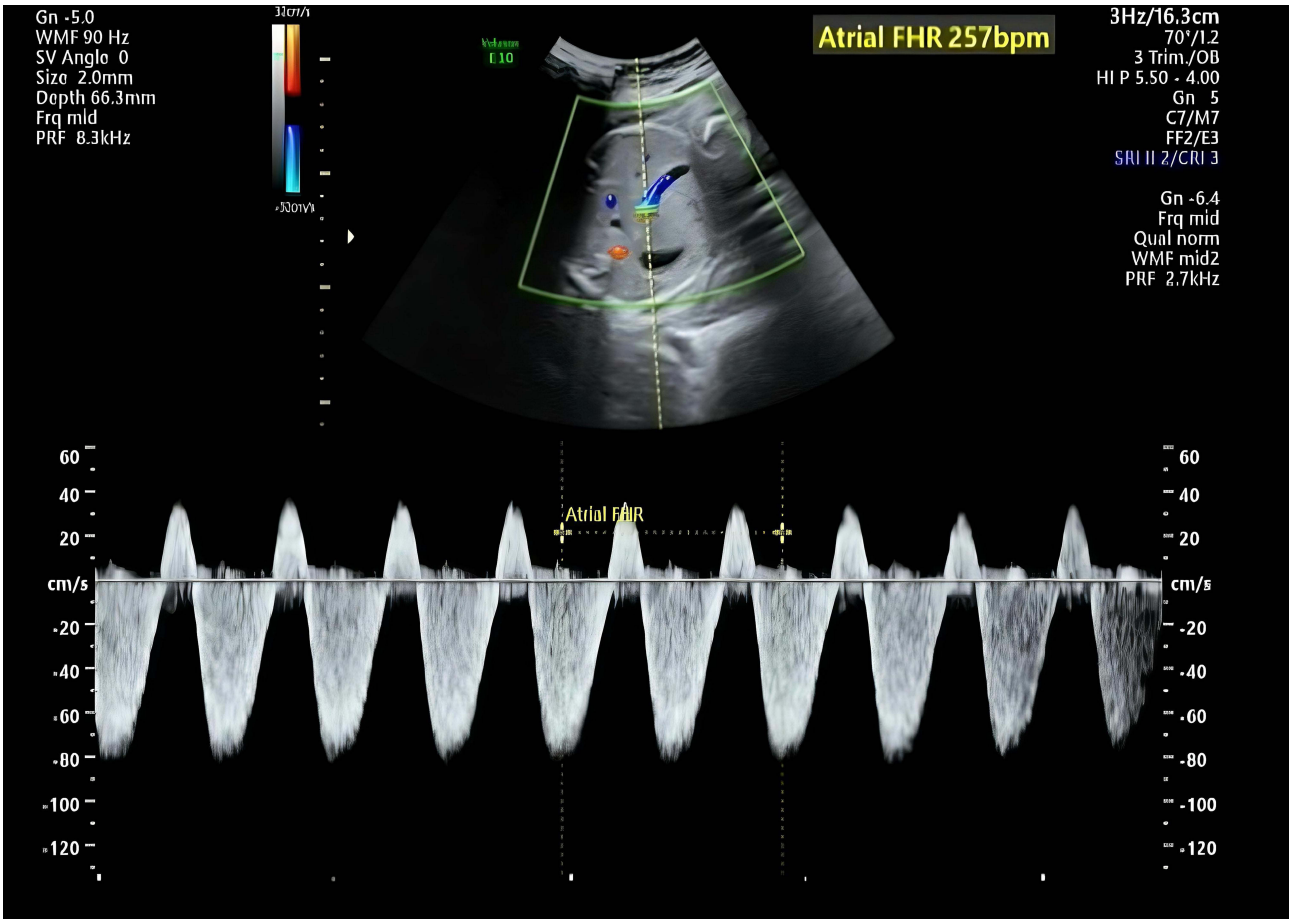

Figure 1 Fetal tachycardia.

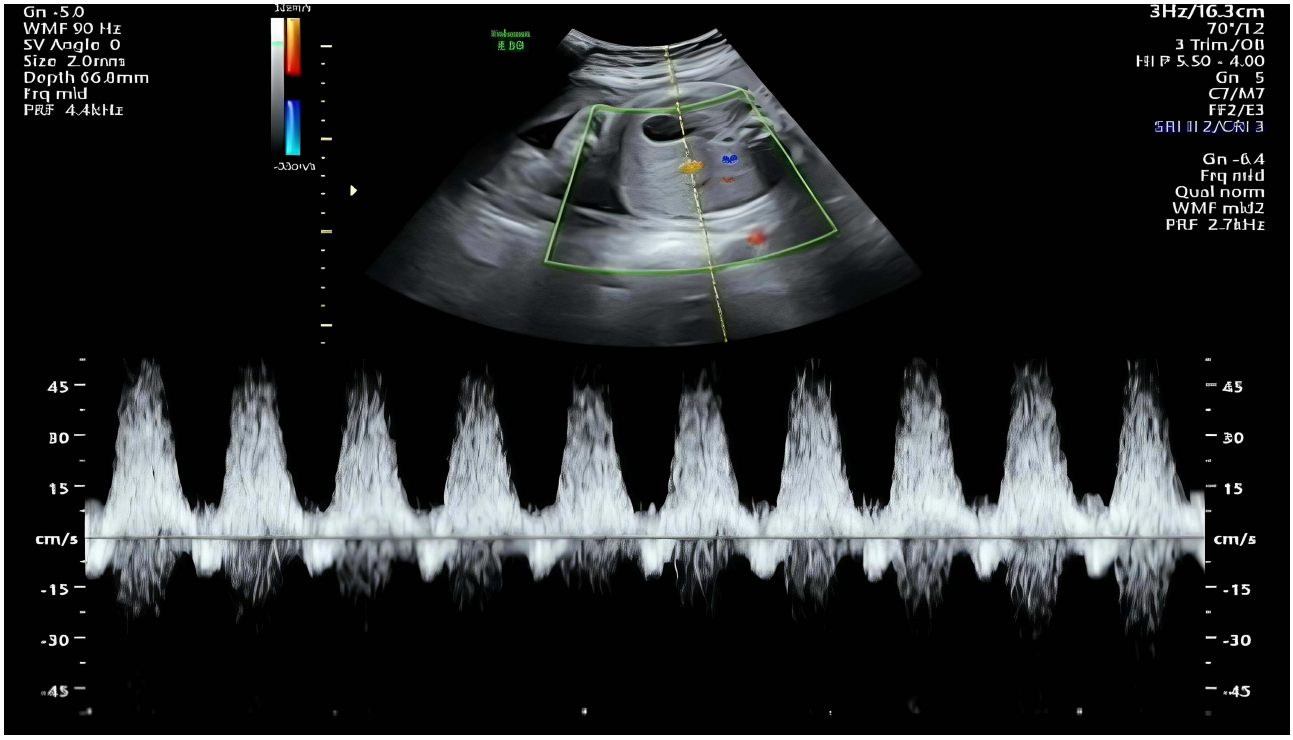

Figure 2 The ductus venosus (DA): a wave was reversed.

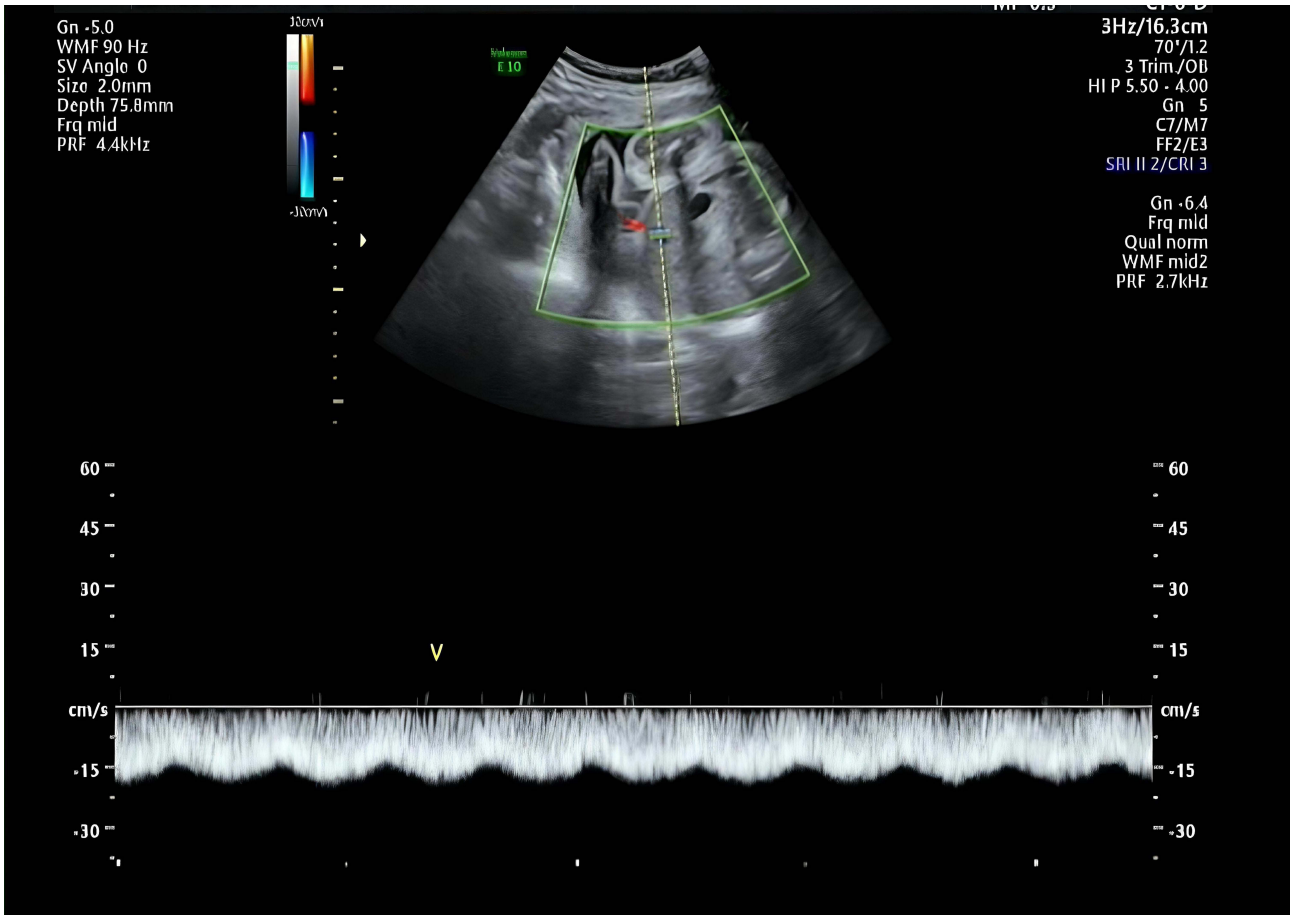

Figure 3 Umbilical vein fluctuation sign.

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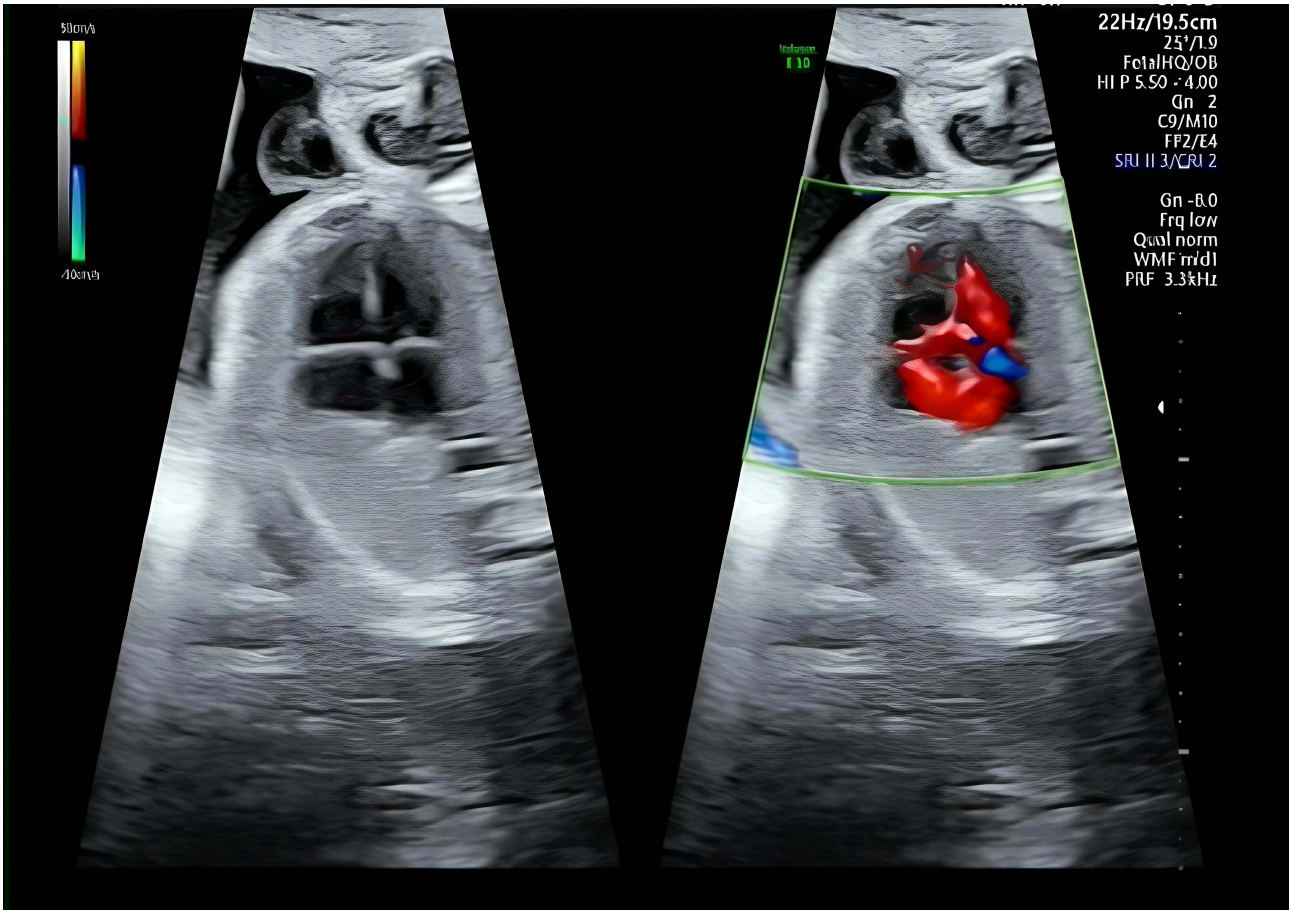

Figure 4 A small amount of pericardial effusion in the left ventricular wall.

0.25 mg twice daily and sotalol 80mg twice daily for symptomatic treatment. At home, the fetal heart rate was monitored daily, and the blood concentration of digoxin was monitored regularly, during which the drug was gradually reduced. After 33 weeks of gestation, the patient stopped taking sotalol and digoxin according to medical supervision.

The patient monitored the fetal heart rate daily at home. At 36+2 weeks gestation, an FHR (Fetal Heart Rate) of 250 beats/min was detected. The patient administered digoxin 0.25 mg, resulting in a decrease of the FHR to 130 bpm within 3 hours. At 36+5 weeks gestation, the patient observed the FHR elevated to 260 beats/min, accompanied by reduced fetal movements, prompting subsequent presentation to the hospital. Fetal echocardiography performed after visiting our hospital showed that the atrial FHR was 243 beats/min with M wave (Figure 6), and reduced pulsatility and resistance indices in the MCA (Middle Cerebral Artery) (Figure 7); the DA (ductus venosus): a wave was reversed (Figure 8); amniotic fluid index: 50mm; the right atrium and right ventricle were enlarged. Fetal cardiac dysfunction and fetal hypoxia were diagnosed by fetal echocardiography. A cesarean section was performed at the 36+5 weeks gestation, delivering a live neonate weighing 3035g with clear amniotic fluid, accompanied by umbilical artery blood gas analysis demonstrating a pH of 7.2. Intraoperative neonatology consultation was requested, the neonate's heart rate was 152 times/minute, and the rhythm was regular. After the operation, the mother and neonate were transferred back to the postpartum unit for further observation. After 4 days of hospitalization, the mother recovered well; the neonate's vital signs were stable, the heart sounds were strong, the rhythm was regular, the sucking was strong, the urinary and bowel functions were normal. The neonate was discharged with his mother. This study was approved by the ethics review committee of the Second Hospital of Dalian Medical University, and public written informed consent was obtained from patients.

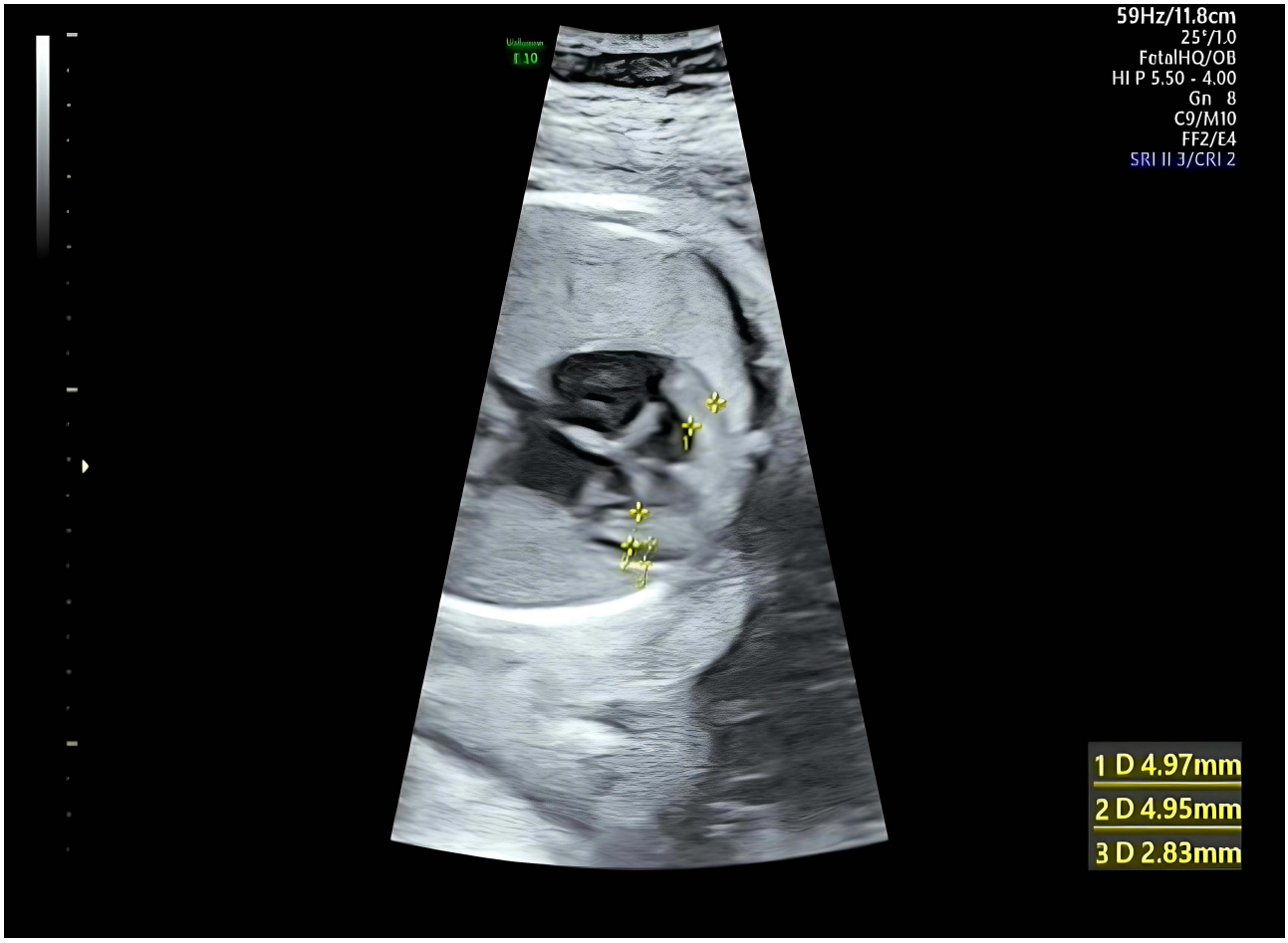

Figure 5 A small amount of mitral regurgitation.

Discussion

SVT was defined as FHR 1:1 atrioventricular activity exceeding 200 BPM. It is the most common type of fetal tachycardia, accounting for 60% to 90% of fetal tachyarrhythmias, with a prevalence of 1/1000 to 1/25,000.[4] Some confounders may be maternal, such as excessive caffeine intake, smoking, and illicit drug abuse. Fetal factors, especially congenital defects such as diaphragmatic hernias, result in frequent fetal atrial premature beats, which may progress to tachyarrhythmias. Fetal supraventricular tachycardia is more common at 30 to 32 weeks of gestation, and its mechanism is mostly atrioventricular reentry. Its characteristics include: 1. Small changes in heart rate (240–290 beats/min); 2. Usually 1:1 conduction; 3. Sudden arrest; 4. Effective for conventional antiarrhythmic drugs (such as digoxin).[1]

The common diagnostic methods of fetal arrhythmia are fetal heart auscultation, fetal heart rate monitoring and fetal echocardiography. Fetal echocardiography is an accurate method for the diagnosis of fetal arrhythmias.[5,6] If arrhythmia is found, it should be carefully identified whether it is paroxysmal or persistent. M-mode ultrasound and Doppler ultrasound should be used to identify the nature of premature beats. For persistent and recurrent arrhythmia, cardiac color Doppler ultrasound should be further performed.[7] When the atrioventricular and ventriculoatrial intervals are measured from simultaneous superior vena cava inflow and aortic outflow Doppler tracings, short ventriculoatrial tachycardia such as atrioventricular reentry tachycardia can be differentiated from the long ventriculoatrial tachycardias such as atrial ectopic tachycardia, permanent junctional reciprocating tachycardia, and sinus tachycardia.[1] Fetal heart rate monitoring is another effective diagnostic method. Although fetal tachycardia mostly occurs in the third trimester, Copel et al[8] the study found that about 23.2% of fetuses had tachycardia at gestational age ≤ 24 weeks. Because fetal heart rate monitoring is of little significance in the early stage of pregnancy, if fetal tachycardia persists, fetal echocardiography

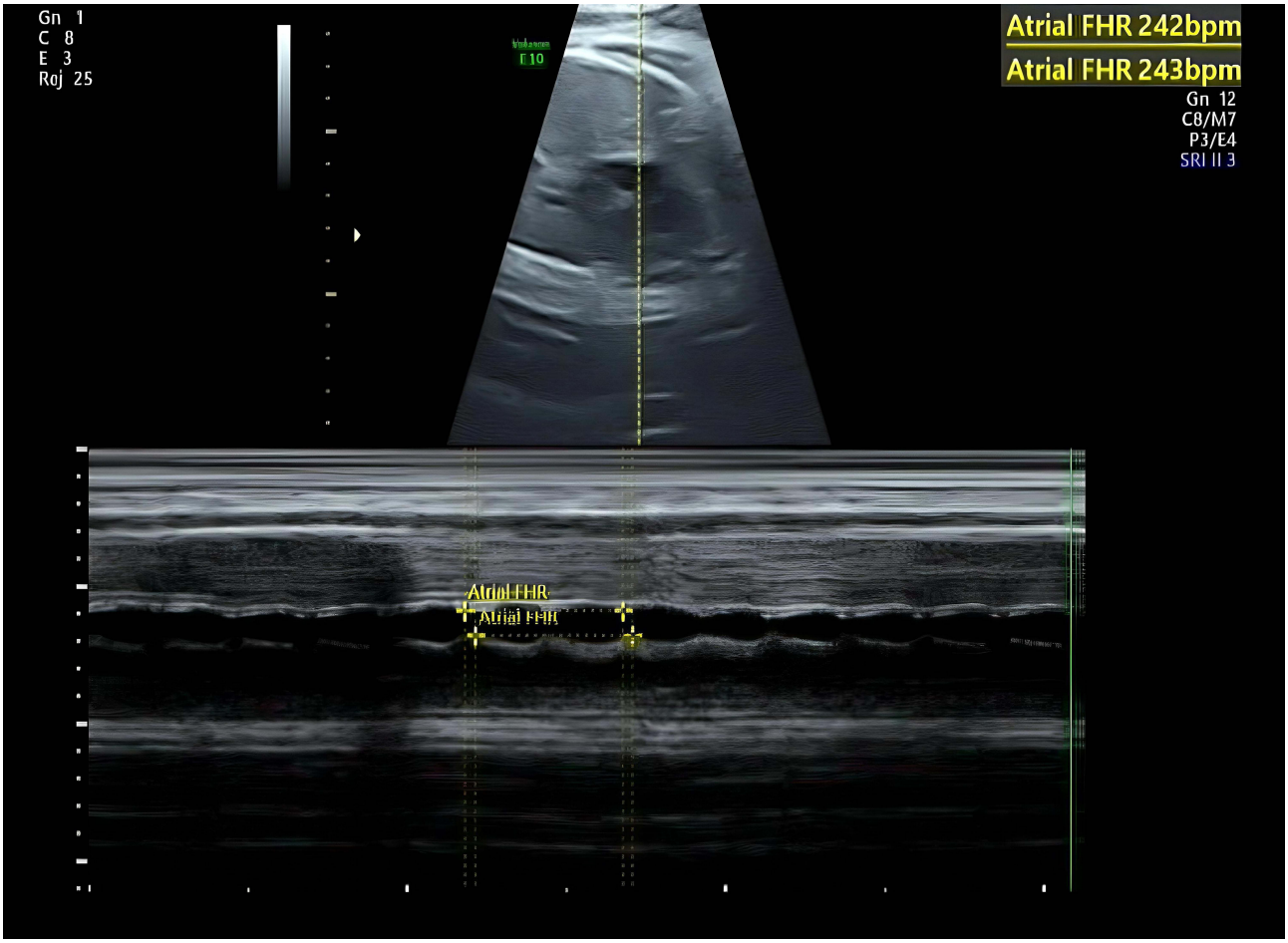

Figure 6 Atrial FHR was 243 beats/min with M wave.

should be performed. Fetal ECG monitoring is also a safe option if this is not possible. However, the discovery of fetal arrhythmias does not necessarily mean termination of pregnancy, and most fetuses with arrhythmias have a good prognosis after intrauterine treatment. The treatment plan should take into account the gestational age, the specific situation of the fetus and the health status of the mother.[9]

Although preterm birth and postpartum treatment avoid the uncertainty of placental transfer by drugs, our previous experience and other investigators have shown that fetuses treated in this manner have a high mortality rate and a high rate of complications.[10] For fetuses presenting with isolated tachycardia, transplacental pharmacotherapy is prioritized as the primary intervention, accompanied by ongoing echocardiographic surveillance of cardiac function and Doppler assessment of placental perfusion dynamics. In cases progressing to fetal heart failure or hydrops, early delivery is favored at gestational ages ≥34 weeks, whereas at <34 weeks, management requires careful weighing of the success probability of intrauterine therapy against the risks of prematurity-associated complications.[11]

In 2014, the American Heart Association (AHA) recommended digoxin and sotalol as first-line drugs in the treatment of fetal cardiovascular disease.[12] Our patient underwent treatment in 2023, but the 2024 AHA guidelines have updated pharmacological recommendations. Therapy of SVT without fetal hydrops: flecainide monotherapy, If unsuccessful, add digoxin or switch to sotalol (with or without digoxin). Persistent SVT: switch to flecainide combined with sotalol or initiate amiodarone monotherapy. Therapy of SVT with fetal hydrops: Combination of flecainide + digoxin. If unsuccessful switch to sotalol + digoxin or flecainide + sotalol. Refractory cases: use amiodarone (with or without digoxin) or direct intramuscular drug administration to the fetus (eg, antiarrhythmic agents delivered via fetal intramuscular injection).[1]

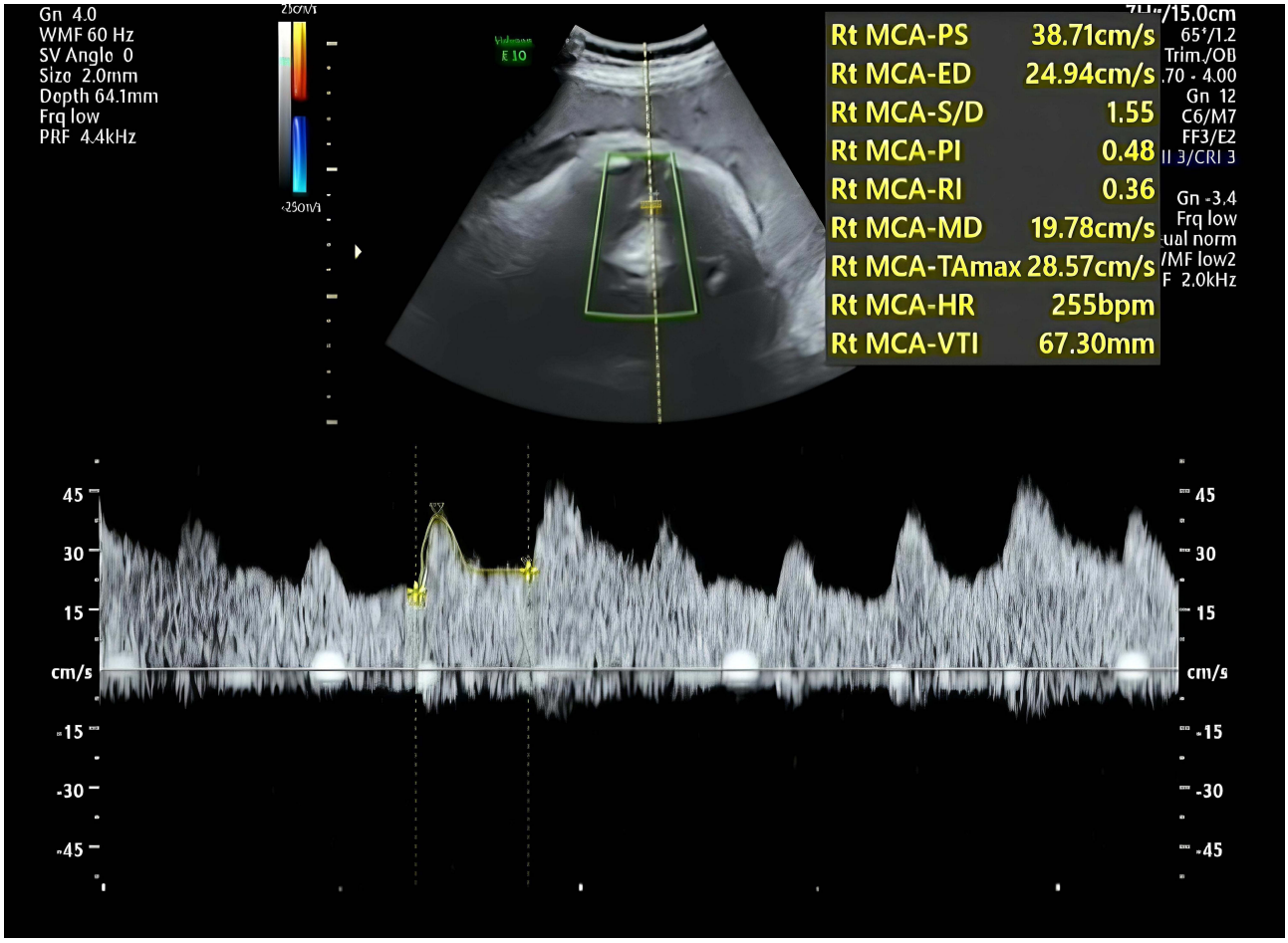

Figure 7 Reduced pulsatility and resistance indices in the MCA.

In a meta-analysis by Hill et al,[13] flecainide was superior to digoxin in converting fetal SVT. In fetuses presenting with hydrops, this benefit was even more notable First-line monotherapy with flecainide was more efficient than digoxin for treating atrioventricular reentry tachycardia.[14] Digoxin crosses the hydropic placenta poorly, which is why it is not routinely used as first-line therapy in hydropic pregnancies.[15]

Other intrauterine treatment methods can not only indirectly transport into the fetus through the placenta through maternal circulation, but also directly treat arrhythmia with fetal hydrops through fetal muscle, umbilical vein and intraperitoneal injection. Parilla et al reported a successful case of treating supraventricular tachycardia with hydrops fetalis by combining maternal administration of digoxin and intramuscular injection of digoxin to the fetus.[16] However, considering that direct administration of digoxin may cause different degrees of trauma to the fetus, this method has not been widely used in clinical practice.[17]

At 29 + 2 weeks after menopause, the patient underwent ultrasound examination, which showed that the fetus was at risk of heart failure and could face a serious condition of fetal distress. In view of the critical condition of the fetus, the continuation of the pregnancy may increase the risk of fetal distress and even intrauterine death, so we considered termination of pregnancy. However, after in-depth evaluation, we found that the patient's condition had stabilized with conservative medical treatment. At the same time, the family members of the patients also expressed a strong desire for conservative treatment. Based on the above factors, we decided to adopt conservative drug treatment and provide patients with symptomatic and supportive treatment for 5 days. During this period, the intrauterine condition of the fetus gradually stabilized, and no obvious abnormalities were found in fetal heart rate monitoring and fetal heart ultrasound examination, indicating that the condition has been effectively controlled. After discharge, the patient was advised to

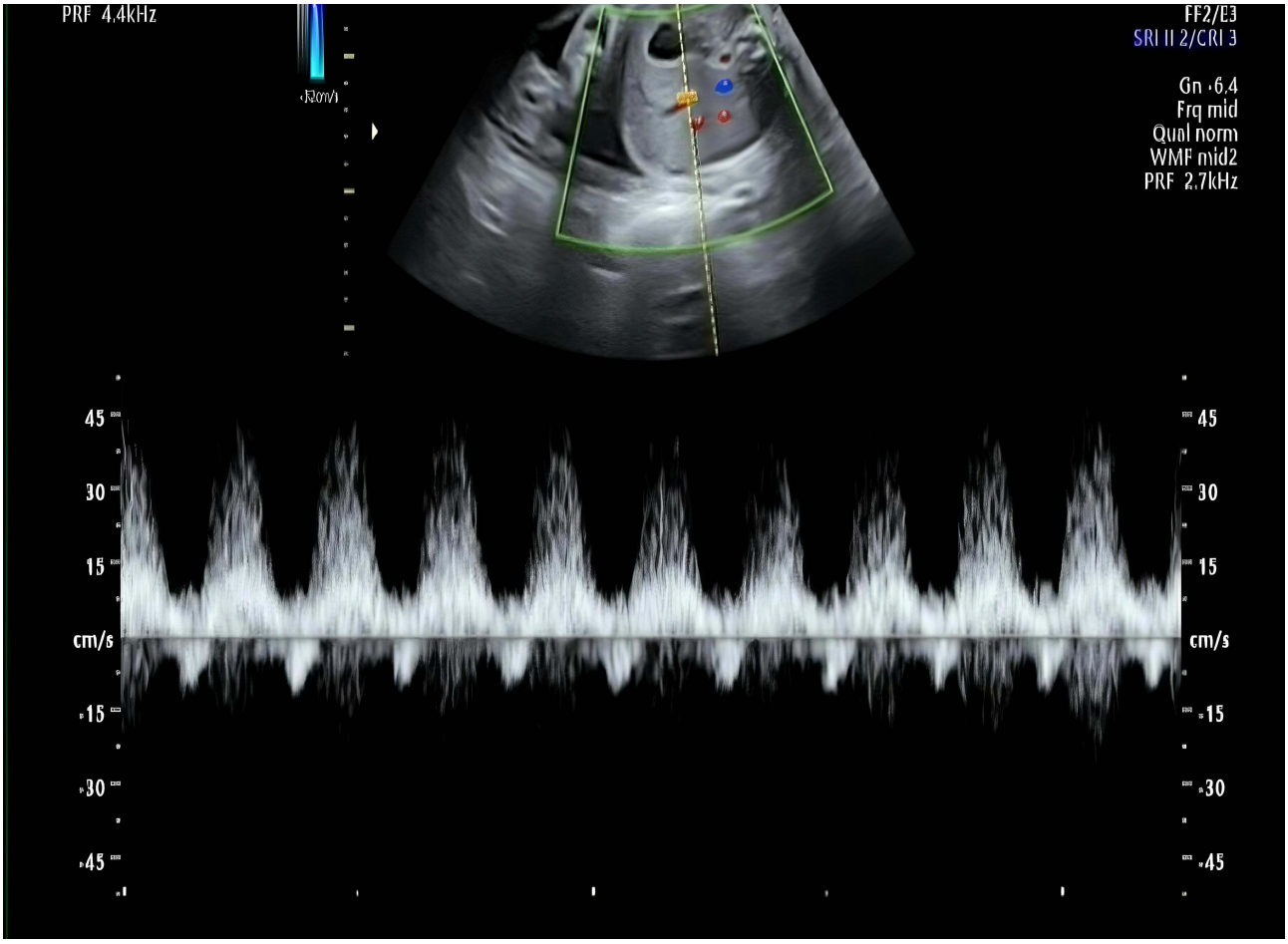

Figure 8 The DA (ductus venosus): a wave was reversed.

continue oral digoxin (0.25 mg, twice a day) and sotalol tablets (80 mg, twice a day) for maintenance treatment. After discharge, the dosage was gradually reduced until the drug was stopped. When the pregnancy reached 36 + 5 weeks, the fetal supraventricular tachycardia could not be relieved and the fetal movement was reduced again, the pregnancy was terminated in time. After the termination of pregnancy, both the mother and the neonate maintained stable conditions with a favorable prognosis and positive follow-up care outcomes.

This treatment is worth learning from: in the face of the emergency situation of fetal arrhythmia and abnormal ultrasound indicators, we did not hastily choose the treatment plan for termination of pregnancy, but according to the gestational age, whether the intrauterine condition of the fetus deteriorates, whether it is accompanied by obstetric complications, the ability of neonatal treatment, and the attitude of patients to the fetus. Individualized treatment plan was formulated. However, when the fetal lung has reached a mature stage, we have considered the progress of the patient's disease and the real-time condition of the fetus in the uterus, and conducted a comprehensive and in-depth discussion with reference to the latest medical literature and treatment guidelines. After fully weighing the advantages and disadvantages of intrauterine intervention and termination of pregnancy, we decided not to take intrauterine drug treatment, but prefer to recommend termination of pregnancy. It is worth noting that although fetal arrhythmia is not an absolute contraindication for vaginal delivery, the indications for cesarean section can be appropriately relaxed when the fetus is accompanied by hydrops or cardiac dysfunction and the family members have a positive attitude towards the treatment of the fetus.

Although the fetus in this case has no organic disease, the long-term intermittent fast heart rate of the fetus increases the risk of fetal heart failure, intrauterine distress, and even intrauterine death. Although the symptoms have been alleviated after systematic treatment and no obvious complications have been found after termination of pregnancy,

serious adverse reactions may occur in the fetus, even if they are well controlled in the uterus, they may recur within 2 weeks after birth. Therefore, clinically, when we encounter similar cases again, we should still intervene quickly and continue to follow up closely. This measure is of great positive significance for maintaining the health of pregnant women and fetuses. Although medical treatment of SVT is generally safe for the mother, careful and thorough daily clinical monitoring is essential to ensure that treatment is both safe and effective. At present, the treatment of fetal arrhythmia has not yet formed a clear and standardized system, which requires us to continue in-depth exploration and continuous optimization in medical practice.

In the face of the relative lack of clinical experience in this field, we should rely on abundant literature and solid theoretical basis to avoid relying solely on the means of termination of pregnancy. On the contrary, we should dare to explore and try diversified and individualized treatment programs, tailor-made the most suitable treatment measures for each patient's unique situation, in order to achieve good maternal and infant outcomes.

This study has two primary limitations: first, due to the absence of fetal chromosomal microarray analysis (CMA) or whole-exome sequencing (WES), potential influences of hereditary arrhythmia syndromes (eg, long QT syndrome, Timothy syndrome) on fetal supraventricular tachycardia cannot be excluded, which may lead to an incomplete etiological interpretation. Second, although no arrhythmia was detected at neonatal discharge, the lack of systematic long-term follow-up (including 6- and 12-month electrocardiographic monitoring and cardiac ultrasonography reevaluation) hinders assessment of the long-term effects of pharmacological interventions on the cardiac conduction system and potential recurrence risks. These limitations constrain the clinical applicability of the study's conclusions in genetic counseling and prognostic assessment.

Data Sharing Statement

All available information is included in the manuscript.

Declarations

The case report has been reviewed and approved by the Ethics Committee of The Second Hospital of Dalian Medical University for publication.

Acknowledgments

Written informed consent was obtained from the mother for publication of this case report and any accompanying clinical data. Parental consent was additionally provided on behalf of the newborn infant. All information has been anonymized to protect patient privacy, with removal of identifiers including names, hospital numbers, and specific dates. This case report was conducted in compliance with the Declaration of Helsinki.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

Xue Cui and Nuowei Ji should be considered co-first authors for this study. The authors declare no conflicts of interest in this work.

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