UDC 618.3:612.6

I.V. Lakhno¹, I.M. Sykal¹, S.M. Korovai², V.M. Korotych², A.E. Tkachov² Maternal and fetal arrhythmia as a sign of hemodynamic deterioration: a case report

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Ukrainian Journal Health of Woman. 2024. 1(170): 87-89; doi: 10.15574/HW.2024.170.87

For citation: Lakhno IV, Sykal IM, Korovai SM, Korotych VM, Tkachov AE. (2024). Maternal and fetal arrhythmia as a sign of hemodynamic deterioration: a case report. Ukrainian Journal Health of Woman. 1(170): 87-89; doi: 10.15574/HW.2024.170.87.

The co-existing maternal (MA) and fetal arrythmia (FA) are associated with maternal goiter disease, chorioamnionitis, or Ballantyne's syndrome. **The aim** of the study — to determine the involvement of maternal arrhythmia and fetal arrhythmia in the pathogenic scenario of hemodynamic deterioration in Ballantyne's syndrome.

Clinical case. It is presented the case of sustained several weeks of MA and FA. A pregnant woman aged 36 years was admitted to the division of maternal and fetal medicine at 34 weeks of gestation. She was gravida 4 and para 3. She had complaints of rapid heartbeat, left-side chest discomfort, and lower extremities edema. The diagnosis of maternal sinus tachycardia was supported via electrocardiography. The indices of fetal, umbilical, and uteroplacental hemodynamics detected via Doppler ultrasound were appropriate. However, fetal heart rate was 209 beats/min. The transplacental attack of oral sotalol 80 mg thrice daily was prescribed. But maternal and fetal tachycardia persisted to stay. The tricuspid regurgitation was detected via Doppler ultrasound next day. The fetus was hydropic. The male baby of 2400 g, 46 cm length, 31 cm head circumference, and Apgar score 3→5 was delivered via caesarean. The newborn was discharged in 21 days. He was admitted again in one month for rehabilitation. Maternal heart rate reduced to 72 beats/min and edema regressed in three days after birth.

Conclusions. MA and FA before fetal hydrops are supposed to be the early signs of mirror syndrome. This speculation needs further investigation.

The research was carried out in accordance with the principles of the Helsinki Declaration. The informed consent of the patient was obtained for conducting the studies.

No conflict of interests was declared by the authors.

Keywords: maternal arrhythmia, fetal arrhythmia, Ballantyne's syndrome, transplacental treatment.

Аритмія матері та плода як можлива ознака синдрому Баллантайна: клінічний випадок І.В. Лахно¹, І.М. Сикал¹, С.М. Коровай², В.М. Коротич², А.Е. Ткачов²

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Супутня аритмія матері та плода пов'язана з тиреотоксикозом, хоріоамніонітом або синдромом Баллантайна.

Мета — визначення участі аритмії матері та аритмії плода в патогенетичному сценарії погіршення гемодинаміки при синдромі Баллантайна.

Клінічний випадок. Наведено випадок тривалої кількатижневої аритмії в матері та плода. Вагітну віком 36 років із IV вагітністю та III пологами госпіталізовано до відділення екстрагенітальної патології в 34 тижні вагітності. Вона мала скарги на прискорене серцебиття, неприємні відчуття у грудній клітці зліва, набряки нижніх кінцівок. Діагноз синусової тахікардії матері підтверджено за допомогою електрокардіографії. Показники матково-плацентарної та плодово-пуповинної гемодинаміки, визначені за допомогою ультразвукової допплерометрії, були нормативними. Проте частота серцевих скорочень плода становила 209 уд./хв. Призначено трансплацентарну атаку соталолу перорально по 80 мг 3 рази на добу. Але тахікардія в матері та плода зберігалася. Трикуспідальну регургітацію виявлено за допомогою ультразвукової допплерографії наступної доби. Плід мав початкові ознаки набряку. Шляхом кесаревого розтину вилучено живого недоношеного хлопчика масою тіла 2400 г, довжиною 46 см, окружністю голови 31 см, оцінкою за шкалою Апгар 3→5 балів. ЧСС матері знизилася до 72 уд./хв, а набряки регресували за три доби після народження. Новонародженого виписано на 21-шу добу. Через місяць його знову госпіталізовано на реабілітацію.

Висновки. Аритмія матері та плода може бути ранньою ознакою дзеркального синдрому. Це припущення потребує подальшого дослідження.

Дослідження виконано відповідно до принципів Гельсінської декларації. На проведення досліджень отримано інформовану згоду па-

Автори заявляють про відсутність конфлікту інтересів.

Ключові слова: аритмія матері, аритмія плода, синдром Баллантайна, трансплацентарне лікування.

Introduction

Maternal arrhythmia (MA) is known to be associated with gestational hypervolemia and abnormal autonomic resetting [1]. Inappropriate sinus tachycardia is a frequent pattern of maternal cardiac rhythm disturbances. Tachycardia could be transient or persistent and

requires pharmacologic interventions. Since the origin and pathogenesis of MA are still disputable, the efficiency of β -blockers is not absolute [8].

Fetal arrhythmia (FA) could be a satellite of congenital heart disease or be functional by nature. There are several groups among all cases of FA: irregular rhythms, tachyarrhythmia and bradyarrhythmia. FA is a reason for fetal compromise,

hydrops fetalis, and antenatal death [5]. The basic antiarrhythmic drugs for transplacental therapy are digoxin, sotalol, flecainide, and amiodarone. Fetal non-immune hydrops, congestive heart failure, and tricuspid regurgitation are factors for reduced efficiency of transplacental treatment in FA. Sotalol was reported as the best agent for transplacental antiarrhythmic treatment in hydropic fetuses [2]. However, the therapeutic strategy in FA depends on gestational age and fetal maturity. Vaginal or cesarean delivery is preferable without prior transplacental therapy in case of gestational term over 37 weeks [10].

The co-existing MA and FA may have a mutual origin. Such cardiac disturbances in the mother and fetus are associated with maternal goiter disease, chorioamnionitis, or Ballantyne's syndrome [7]. MA has been recently suggested as an early sign of hemodynamic failure in mirror syndrome. Therefore, all cases of fetal non-immune hydrops should be monitored for MA [3].

The *aim* of the study — to determine the involvement of MA and FA in the pathogenic scenario of hemodynamic deterioration in Ballantyne's syndrome.

The research was carried out in accordance with the principles of the Helsinki Declaration. The informed consent of the patient was obtained for conducting the studies.

Clinical case

It is presented the case of sustained several weeks of MA and FA. A pregnant woman aged 36 years was admitted to the division of maternal and fetal medicine at 34 weeks of gestation. She was gravida 4 and para 3. She had complaints of rapid heartbeat, left-side chest discomfort, and

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Fig. 1. Fetal tachycardia via Doppler ultrasound

lower extremities edema. Her weight gain for this pregnancy was 19 kg. Maternal sinus tachycardia was detected 3 weeks ago and metoprolol 50 mg twice daily was prescribed. Fetal supraventricular tachycardia was diagnosed at the same time. She had two deliveries at term and one early pregnancy loss. Her general anamnesis were unremarkable. She had regular antenatal visits with her general practitioner in the suburban area of Kharkiv. She had a blood pressure of 120/80, a pulse of 122 per minute, and a normal body temperature at the admission ward. She had no prodromes suggestive of any gestational complications. Laboratory findings have not revealed any pathologies (hemoglobin, leukocytes, platelet count, serum aspartate aminotransaminase, serum alanine aminotransaminase, serum urea, creatinine concentration, serum coagulation profile, and urinalysis indices values were normal). The diagnosis of maternal sinus tachycardia was supported via electrocardiography. The indices of fetal, umbilical, and uteroplacental hemodynamics detected via Doppler ultrasound were appropriate. However, fetal heart rate was 209 beats/min (Figure 1). The chorioamnionitis and maternal thyrotoxicosis were ruled out.

The transplacental attack of oral sotalol 80 mg thrice daily was prescribed. But maternal and fetal tachycardia persisted to stay. The tricuspid regurgitation was detected via Doppler ultrasound next day (Figure 2). It was a sign of fetal deterioration. The fetus was hydropic. It has mild edema of the subcutaneous tissue. The male baby of 2400 g, 46 cm length, 31 cm head circumference, and Apgar score 3→5 was delivered via caesarean. The neonatal supraventricular tachycardia was found via electrocardiography. The baby passed



Fig. 2. Tricuspid regurgitation in the process of transplacental treatment

to the neonatal resuscitation unit. The cardiac rhythm was restored with amiodarone infusion 5 mg/kg over 4 hours and then oral amiodarone 5 mg/kg twice a day for 10 days and then 5 mg/kg once daily. The newborn was discharged in 21 days. He was admitted again in one month for rehabilitation. Maternal heart rate reduced to 72 beats/min and edema regressed in three days after birth.

Discussion

It is presented a case of Ballantyne's syndrome that mirrored maternal and fetal cardiac failure. Fetal hydrops and maternal edema were basic signs of hemodynamic deterioration [3,7]. However, the presence of both MA and FA was rather a rare combination for mirror syndrome [9].

The rate of Ballantayne's syndrome is very low. The guidelines or protocol on this syndrome are not available. However, several authors expressed an opinion that pre-eclampsia could mimic mirror syndrome [6]. Maternal edema is a sign of increased vascular permeability in pre-eclamptic patients. The absence of arterial hypertension and proteinuria contributed to ruling out pre-eclampsia in this case. Sustained FA and non-immune fetal hydrops are associated with

structural cardiac abnormalities in the majority of hydropic fetuses. It has not found any fetal malformations in this case study. Viral infection is also involved in the pathogenesis of fetal hydrops [7]. Maternal and neonatal tests for TORCH infection were negative.

The fetal prognosis in Ballantayne's syndrome is poor. The reason for an antenatal fetal death is volemic overload and heart failure with an increased cardiac output. Several cases of successful intrauterine treatment of FA in mirror syndrome are known [10]. However, the chances for cardiac rhythm restoration in hydropic fetuses are low [4]. It has not managed with sotalol transplacental attack but performed a cesarean section immediately after detection of fetal deterioration.

Conclusions

MA and FA before fetal hydrops are supposed to be the early signs of mirror syndrome. This speculation needs further investigation.

It is assumed that co-existing MA and FA could be the presentation of Ballantayne's syndrome. Early detection of its signs is the way to ameliorate fetal outcomes.

No conflict of interests was declared by the authors.

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