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Valvular heart disease during pregnancy: a clinical case and a literature review

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Background. As a result of improved diagnostic and reparative techniques, congenital heart diseases are becoming a significant problem for women of childbearing age. Nowadays, more pregnant women in the West are being diagnosed with an acquired heart disease because of the tendency to delay childbearing and increasing age-related risk of developing complications of hypertension, diabetes, obesity and other diseases. According to the Lithuanian Health Information Centre, the incidence of cardiovascular diseases in pregnancy is decreasing in Lithuania, from 1.4% in 2014 to 1% in 2016 (1). Heart diseases can aggravate maternal adaptive capabilities and complications that pose a threat to mother and foetus can occur. Management of such conditions presents a serious therapeutic challenge to multidisciplinary team. The aim of this article is to discuss the course of pregnancy and peculiarities of maternal and foetal care in a woman with hemodynamically significant heart disease.

Materials and methods. We present a clinical case of a 30-year-old nuliparous woman who was diagnosed with mitral valve disease with critical stenosis, grade II/III mitral valve insufficiency, moderate-severe pulmonary hypertension, heart failure stage C, and NYHA functional class II.

Results and conclusions. Pregnancy in conjunction with heart disease is a complicated condition that requires multidisciplinary prenatal care (consisting of an obstetrician gynaecologist, cardiologist, anaesthesiologist). Low molecular weight heparins should be the first choice medication for antithrombotic prophylaxis. Since pregnancy can aggravate a heart disease, preconception counselling and evaluation of the heart function are recommended.

Keywords: pregnancy, heart disease, valvular heart disease, mitral valve insufficiency, heart failure

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INTRODUCTION

As a result of improved diagnostic and reparative techniques, congenital heart diseases are becoming a significant problem for women of childbearing age (2). During pregnancy, woman undergoes many cardiovascular and hemodynamic changes:- expanded blood volume, increased cardiac output, heart rate and left ventricular end-diastolic pressure (preload) (3). The body of a healthy woman can adapt to these physiological changes. However, heart diseases can aggravate maternal adaptive capabilities and lead to complications such as cardiac decompensation or foetal death (4). Cardiovascular diseases in pregnant women are one of the most common non-obstetric causes of maternal and foetal mortality and morbidity (5).

A CLINICAL CASE

We present a clinical case of a 30-year-old nuliparous woman who was diagnosed with mitral valve disease with critical stenosis, grade II/III mitral valve insufficiency, moderate-severe pulmonary hypertension, heart failure stage C, NYHA functional class II, and acute respiratory failure.

Two years before the conception, the patient was diagnosed with congenital left iliac vein aplasia. While planning her first pregnancy, the woman made an appointment with a general practitioner (GP). After evaluating medical history, consultations by a vascular surgeon and a hematologist were recommended. Venous duplex scanning of lower extremities revealed chronic venous disease of *v. saphena magna sinistra*. Laboratory assessment was performed (APTT, SPA, fibrinogen, D-dimers, anti-cardiolipin(aCL) antibodies, different isotypes of immunoglobulins (IgG, IgA, IgM) of anti-β2-glycoprotein I autoantibodies) and the results were in normal range. No risk for venous thrombosis or any contraindications to pregnancy due to congenital venous aplasia were found.

Pregnancy monitoring was started at the 8th gestational week when the patient was admitted to the emergency room due to severe generalised weakness and dizziness. The symptoms started in early pregnancy and were gradually becoming stronger. Physical examination findings: vesicular breath sounds with no crackles, rhythmic heart activity, heart rate 62 bpm, clear heart sounds with a harsh systolic murmur in the mitral area (apex) and presystolic murmur in the 4th intercostal space on the left, arterial blood pressure 120/70 mmHg. Results of laboratory tests were in normal range. Electrocardiography (ECG) monitor registered sinus rhythm, PR interval 123 ms, QRS complex 103 ms, QTc interval 433 ms. QRS-T angle 79 degrees. Conclusion: enlargement of the left atrium. Echocardioscopy revealed fibrotic thickening of mitral valve (MV) cusps with no signs of calcification, immobile posterior cusp, doming of anterior cusp, symmetrical fusion of both MV commissures, MV opening in the centre of the valve, MV area 0.9 cm^2, regurgitant blood flow due to MV insufficiency (Fig. 1). Conclusions: fibrotic MV changes, severe MV stenosis, Wilkins score 4, grade II MV insufficiency, left atrial dilation (Fig. 2), pulmonary arterial hypertension.

After evaluating the patient's complaints, physical examination, laboratory, instrumental, and radiological assessment data, clinical diagnosis was made: complex MV disease with critical stenosis, grade II MV insufficiency, moderate pulmonary hypertension, heart failure stage C, NYHA functional class II. A cardiologist's consultation was recommended to the patient at the 12th week of pregnancy.

Two options of treatment were considered: (1) pregnancy termination due to maternal indications followed by heart surgery (valvuloplasty or prosthesis) and (2) percutaneous balloon valvuloplasty in conjunction with preserving pregnancy, which put the mother at a higher risk of various complications. Due to the patient's decision to continue with the pregnancy, conservative treatment was administered and the patient was prescribed Metoprolol tablets 12.5 mg/day in order to improve her heart function. Diuretics and anticoagulants were not prescribed. Medical surveillance by a general practitioner, an obstetrician-gynaecologist, and a cardiologist (every one or two weeks) was recommended. During multidisciplinary assessment of the patient's condition it was decided that if the patient's condition deteriorated and the heart failure progressed, the intervention would be carried out immediately.

Until the 31st week of pregnancy, the patient's health condition was normal. The patient

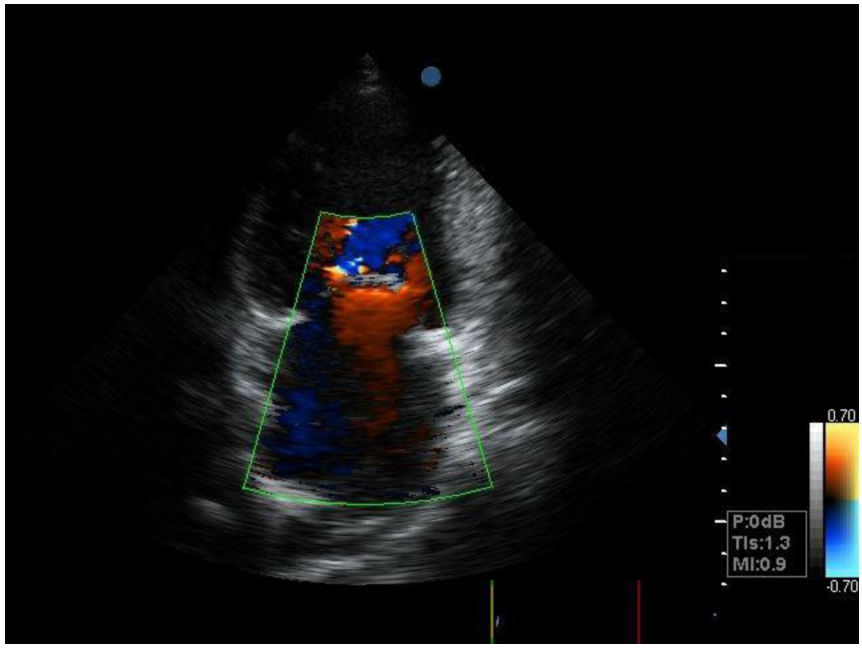

Fig. 1. Ultrasound scan showing regurgitant blood flow due to mitral valve insufficiency

was referred to Vilnius University Hospital Santaros Klinikos and hospitalised at the High Risk Pregnancy Department at the 31st week of pregnancy due to foetal distress. The only complaint the woman had was mild generalised weakness. During physical examination, vesicular breath sounds with no crackles, rhythmic heart activity, heart rate 59 bpm, clear heart sounds with a harsh systolic murmur in the mitral area (apex) and presystolic murmur in the 4th intercostal space on the left, arterial blood pressure 110/65 mmHg was found. Gynaecological examination showed no visible pathology, normal vaginal discharge was observed, and uterine cervix was closed. Ultrasound examination was made and it was found rhythmic foetal heart activity, foetal heart rate was140 bmp. There was a normal amount of amniotic fluid, no pathology of internal organs of the foetus was found. Foetal non-stress test (NST) was reactive. Laboratory test results were

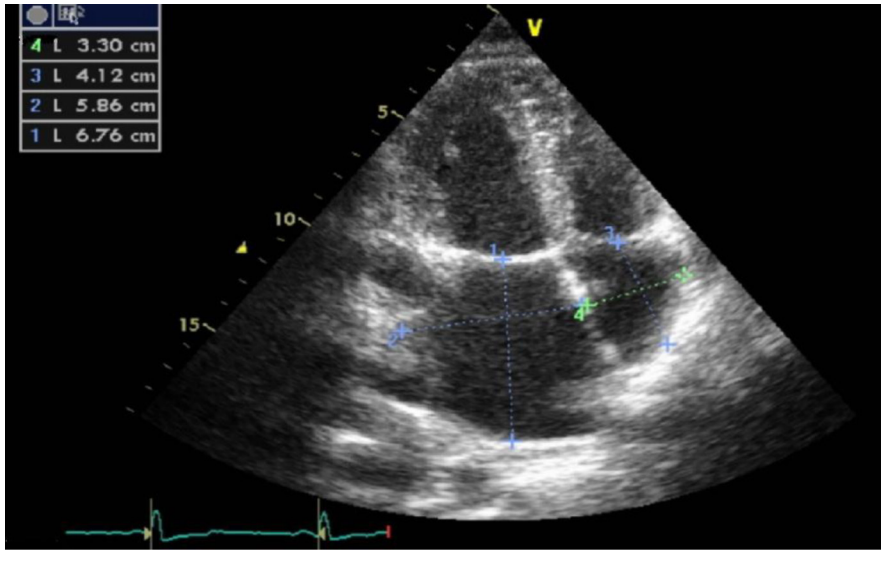

Fig. 2. Echocardioscopic view of the dilated left atrium

in normal range, except for D-dimers which were elevated (1270 mcg/l). The patient was consulted by a vascular surgeon and LMWH – Fraxiparin 0.3 ml/day subcutaneously was administered, and compression stockings (functional class II) were recommended in order to lower the risk of thrombotic events. In order to avoid excessive venous collateral damage, lower midline abdomen incision was recommended given there were indications for terminating the pregnancy.

At the 32nd week of gestation a follow-up sonography showed persistent foetal distress. Cardiologic assessment showed no dynamics in the condition of the woman's cardiovascular system: tolerance to physical effort remained unchanged and the patient's health condition was normal. Since the patient's cardiologic condition enabled her to continue the pregnancy, further management was adjusted considering the best outcome for the foetus. It was decided to discontinue cardiac medications. In order to design a personalised pregnancy care and optimal treatment tactics, multidisciplinary team discussion consisting of an obstetrician-gynaecologist, cardiologist, heart surgeon and anaesthesiologist was planned for the 34th gestational week.

Unfortunately, the patient's condition worsened at the 33rd gestational week: she experienced severe generalised weakness, anxiety, and shortness of breath accompanied by the sensation of substernal pressure when lying down. The patient's examination showed no regular uterine contractions. Foetal NST remained reactive, foetal heart sounds were clear and rhythmic, 135 bpm. In dynamics D-dimers increased from 1600 mcg/l to 2390 mcg/l in three days' period. The same tendency was seen with B type natriuretic peptide (BNP) which reached the level of 630.2 ng/l. Pulmonary oedema and pleural effusions were diagnosed after performing chest radiography (Fig. 3).

Echocardioscopic evaluation of MV showed no dynamics, although mild pericardial effusion (up to 1.0 cm near the right atrium) and a slight diastolic inversion of the free atrial wall were observed. Ultrasound imaging showed collection of fluid within the pleural space: 300 ml on the left and 650 ml on the right. Pharmacotherapy was recommenced and the patient was administered β-blocker (Metoprolol tablets 12.5 mg/day) and a minimal dose of loop diuretic (Furosemid tablets 20 mg/day).

Despite the treatment, the patient's condition gradually worsened (she complained of dyspnea during rest) and she was moved to the intensive care unit (ICU) due to respiratory failure. It was managed using oxygen therapy via a conventional face mask (oxygen flow 4 l/pm): sufficient spontaneous breathing, respiratory rate 17/min,

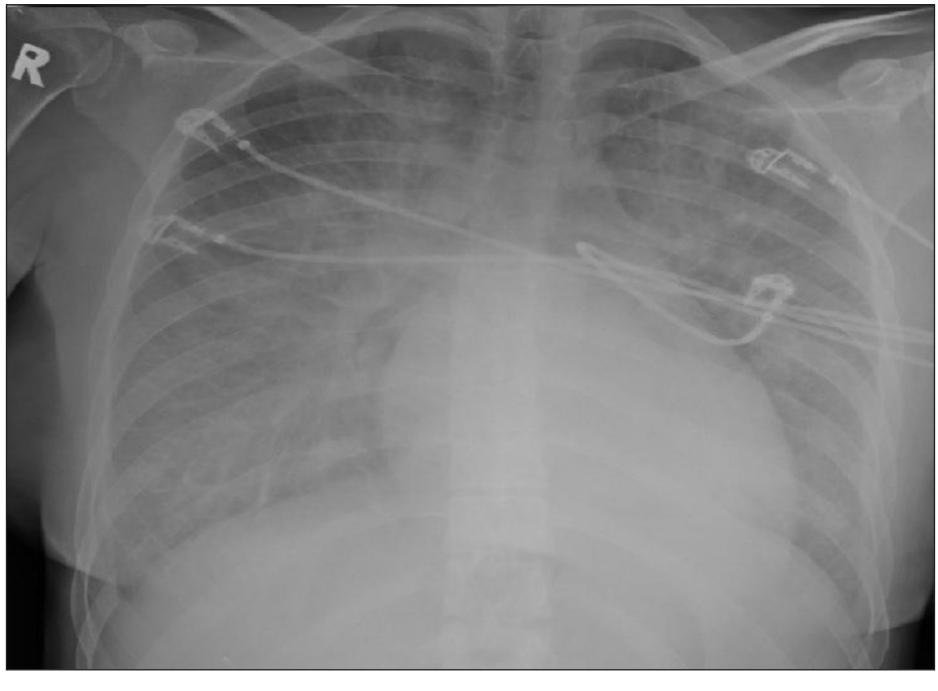

Fig. 3. Chest radiograph demonstrating pulmonary oedema and pleural effusions

SpO_2 – 100%. Cardio-pulmonary ultrasound examination was performed to the patient and increased fluid in the right pleural cavity, fluid in the left pleural cavity and pericardium were found. A multidisciplinary discussion was held and it was decided to end the pregnancy by an urgent Caesarean section due to vital maternal indications: rapid worsening of the general condition and acute cardiopulmonary insufficiency.

At 33 weeks and 6 days of pregnancy, a Caesarean section was performed through a lower midline incision as it had been recommended in order to avoid venous collateral damage.The patient was given perioperative antibiotic prophylaxis of Cefasolin 2.0 g intravenously. A female newborn weighing 1700 grams and 46 cm tall was born, Apgar score of 9 at 1 minute and 10 at 5 minutes. After the operation the patient was transferred to the intensive care unit (ICU). The patient was treated with antibiotics Cefasolin 1.0 g intravenously three times daily, diuretics, deep veins thrombosis prophylaxis. Diuresis was stimulated by furosemide bolus, the response was adequate. One day later the patient was transferred to the Obstetrics Department. She was treated with Metoprolol tablets 25 mg once or twice daily (depending on the arterial blood pressure and pulse), Furosemid tablets 20 mg/d, Spironolactontablets 25 mg/day, Lasix 40 mg intravenously (depending on diuresis), Fraxiparin 0.3 ml/d subcutaneously. The postoperative period was normal with positive dynamics. After taking into account the improvement of the woman's general condition, absence of complaints, normalised airiness of the lungs, and decreasing levels of fluid in pleural and pericardial spaces, the patient and the newborn were discharged from the hospital six days after delivery. Breastfeeding, pharmacotherapy (β-blockers, diuretics, LMWH), surgical wound care and follow-up with a cardiologist one month after the Caesarean section were recommended.

LITERATURE REVIEW AND DISCUSSION

Congenital heart diseases are becoming a significant problem for women of reproductive age as a result of improved diagnostic and reparative techniques which leads us to increasing survival rates and a growing population of people with congenital heart disease. Literature suggests that around 85% of these people live to adulthood (6). In addition, more and more pregnant women are being diagnosed with an acquired heart disease because of the current tendency to delay childbearing and increasing age-related risk of developing complications of hypertension, diabetes, obesity and a wide variety of other diseases (4). The incidence of cardiovascular disease in pregnancy is increasing because more women with congenital or acquired heart disease are reaching childbearing age due to improved medical and surgical care, and they desire children. According to the Lithuanian Health Information Centre, the incidence of cardiovascular diseases in pregnancy is decreasing in Lithuania, from 1.4% in 2014 to 1% in 2016 (1).

The most common causes of valvular heart disease in young women are rheumatic heart disease, congenital heart defects and history of endocarditis in childhood (7). Heart diseases can aggravate maternal adaptive capabilities and complications to mother and foetus can occur. The risk of maternal and foetal complications depends on the degree of valve damage (stenosis/regurgitation), left ventricular ejection fraction (LVEF), and a grade of pulmonary edema (8, 9).

During normal pregnancy the woman undergoes many cardiovascular and hemodynamic alterations, such as increased LVEF, interstitial fluid volume, and decreased arterial blood pressure and systemic vascular resistance (10). Cardiovascular changes occur in order to improve uterine blood circulation which provides growing needs for the developing foetus and the growing placenta (11–13). Decrease in peripheral vascular resistance is usually observed from the 5th week of gestation as a result of developing utero-placental circulation and higher levels of oestrogen, progesterone, and relaxin. In the middle of the third trimester of pregnancy (around the 32nd gestational week) vascular resistance drops to 40–70% of its pre-pregnancy values. Due to reduced arterial blood pressure and vascular resistance, activation of renin-angiotensin-aldosterone system and mild decrease of atrial natriuretic peptide (ANP) plasma concentration occurs and plasma volume expands about 30–50%. This mechanism helps to ensure relatively stable arterial blood pressure during pregnancy-associated cardiovascular changes.

Expansion of blood volume remains in the plateau phase throughout the pregnancy (14). In the presented clinical case, the patient's condition significantly declined at exactly the 32nd week of pregnancy, when, according to literature, rapid increase in circulating blood volume occurs.

Despite the fact that cardiovascular disease complicates less than 1% of all pregnancies (15), it is scientifically proven that around 23.5% of women with a diagnosed heart disease undergo various cardiovascular complications; maternal mortality goes up to 2.7% and the risk of miscarriage, foetal death, or stillbirth reaches 7.7% (16). Tachycardia during pregnancy shortens left ventricular diastolic time and increases the mitral valve gradient (diastolic mitral flow) which can lead to maternal hemodynamic instability. Tachycardia can be managed using β-blockers or calcium channel blockers. It is not recommended to reduce the preload in order to maintain normal stroke volume and cardiac output (8). In our clinical case, the patient was administered β-blocker with constant pulse monitoring three times per day. It was recommended to discontinue pharmacotherapy in case of heart rate lower than 50 bpm. However, few days after the patient had stopped taking medications, her condition rapidly worsened. The effects of β-adrenergic blockers on the foetus are not well understood but the newest evidence shows the connection between usage of β-blockers and foetal growth restriction (17). Besides, β-blockers can reduce foetal heart rate and there are no well-controlled studies in humans analysing its possible adverse effects in foetus, therefore accurate evaluation of risk-benefit ratio should be performed before prescribing these medications to a pregnant woman.

Heart failure and pregnancy itself are associated with a hypercoagulable state. The risk of thrombosis increases 3–4 times during pregnancy (18). Some evidence suggests that the risk to develop deep venous thrombosis is even six times greater during gestational period (19). Moreover, our patient was diagnosed with congenital left iliac vein aplasia and chronic *v. saphena magna* disease on the left which makes the risk of thrombotic events even higher.

Unfractionated heparin or low-molecular-weight heparins (LMWH) are the first choice antithrombotic medications during pregnancy (4). Warfarin should not be administered and used during pregnancy because of its proven teratogenicity (4). Use of warfarin during pregnancy is associated with specific embryopathy called Foetal Warfarin Syndrome (FWS). FWS usually occurs when warfarin is administered at the 6th–12th week of gestation (20, 21). In our case, the patient had been diagnosed with chronic venous disease and the blood test showed high levels of D-dimers, and treatment with LMWH (Fraxiparin) was administered. Evidence states that LMWHs are safe to use during pregnancy (22). Delayed-type hypersensitivity is the most common adverse reaction of LMWHs and affects about 20% of pregnant women (23).

To assess the maternal risk of cardiac complications during pregnancy, the condition of the woman should be evaluated taking into account her medical history, functional class, natriuretic peptide levels, and echocardiographic assessment of ventricular and valvular function. Disease-specific risk should be assessed using the modified World Health Organization (mWHO) classification according to the 2018 ESC Guidelines for the Management of Cardiovascular Diseases during Pregnancy dealing with specific diseases. Maternal morbidity risk assessment is carried out according to the modified World Health Organization (WHO) risk classification (Table). This risk classification integrates all known maternal cardiovascular risk factors including the underlying heart disease. It includes contraindications for pregnancy. The general principles of this classification and its practical application are given in Table.

In women of WHO class I, the risk is very low, cardiologic follow-up during pregnancy may be limited to one or two visits. Those of WHO class II are at a low or moderate risk, follow-up every trimester is recommended. For women of WHO class III, there is a high risk of complications and frequent (monthly or bimonthly) cardiology and obstetric review during pregnancy is recommended. Women in WHO class IV should be advised against pregnancy but, if they become pregnant and will not consider termination, monthly or bimonthly follow-up is needed.

Moderate and severe mitral stenosis is poorly tolerated during pregnancy. Heart failure occurs in pregnant women with moderate or severe MS

Table. Modified WHO classification of maternal cardiovascular risk (2018)

	mWHO I	mWHO II	mWHO II–III	mWHO III	mWHO IV
Diagnosis (if otherwise well and uncomplicated)	Small or mild – pulmonary stenosis, patent ductus arteriosus, mitral valve prolapse Successfully repaired simple lesions (atrial or ventricular septal defect, patent ductus arteriosus, anomalous pulmonary venous drainage) Atrial or ventricular ectopic beats, isolated	Unoperated atrial or ventricular septal defect. Repaired tetralogy of Fallot Most arrhythmias (supraventriculararrhythmias) Turner syndrome without aortic dilatation	Mild left ventricular impairment (EF >45%) Hypertrophic cardiomyopathy Native or tissue valve disease not considered WHO I or IV (mild mitral stenosis, moderate aortic stenosis) Marfan or other HTAD syndrome without aortic dilatation Aorta <45 mm in bicuspid aortic valve pathology Repaired coarctation Atrioventricular septal defect	Moderate left ventricular impairment (EF 30–45%) Previous peripartum cardiomyopathywithout any residual left ventricular impairment Mechanical valve Systemic right ventricle with good or mildly decreased ventricular function Fontan circulation. If otherwise the patient is well and the cardiac condition uncomplicate Unrepaired cyanotic heart disease. Other complex heart disease Moderate mitral stenosis Severe asymptomatic aortic stenosis Moderate aortic dilatation (40–45 mm in Marfan syndrome or other HTAD; 45–50 mm in bicuspid aortic valve, Turner syndrome ASI 20–25 mm/m^2, tetralogy of Fallot <50 mm) Ventricular tachycardia	Pulmonary arterial hypertension Severe systemic ventricular dysfunction (EF <30% or NYHA class III–IV) Previous peripartum cardiomyopathy with any residual left ventricular impairment Severe mitral stenosis Severe symptomatic aortic stenosis Systemic right ventricle with moderate or severely decreased ventricular function Severe aortic dilatation (>45 mm in Marfan syndrome or other HTAD, >50 mm in bicuspid aortic valve, Turner syndrome ASI >25 mm/m^2, tetralogy of Fallot >50 mm) Vascular Ehlers–Danlos Severe (re)coarctation Fontan with any complication

Table. (continued)

	mWHO I	mWHO II	mWHO II–III	mWHO III	mWHO IV
Risk	No detectable increased risk of maternal mortality and no/mild increased risk in morbidity	Small increased risk of maternal mortality or moderate increase in morbidity	Intermediate increased risk of maternal mortality or moderate to severe increase in morbidity	Significantly increased risk of maternal mortality or severe morbidity	Extremely high risk of maternal mortality or severe morbidity
Maternal cardiac event rate	2.5–5%	5.7–10.5%	10–19%	19–27%	40–100%
Counselling	Yes	Yes	Yes	Yes: expert counselling required	Yes: pregnancy contraindicated: if pregnancy occurs, termination should be discussed
Care during pregnancy	Local hospital	Local hospital	Referral hospital	Expert centre for pregnancy and cardiac disease	Expert centre for pregnancy and cardiac disease
Minimal follow-up visits during pregnancy	Once or twice	Once per trimester	Bimonthly	Monthly or bimonthly	Monthly
Location of delivery	Loca hospital	Local hospital	Referral hospital	Expert centre for pregnancy and cardiac disease	Expert centre for pregnancy and cardiac disease

ASI – aortic size index; EF – ejection fraction; HTAD – heritable thoracic aortic disease; mWHO – modified World Health Organization classification; NYHA – New York

Heart Association; WHO – World Health Organization.

during the second and third trimesters, even in previously asymptomatic women. Heart failure is often progressive. Obstetric complications are related to the risk of acute heart failure during or just after delivery.

Prematurity rates are 20–30%, intrauterine growth retardation 5–20%, stillbirth 1–3%. Offspring risk is higher in women in NYHA class III/IV during pregnancy (24).

All patients with moderate or severe MS (even when asymptomatic) should be counselled against pregnancy and pre-pregnancy intervention should be performed. Clinical and echocardiographic follow-up is indicated monthly or bimonthly depending on haemodynamic parameters. In mild MS, evaluation is recommended every trimester and prior to delivery. It should also be considered in women with moderate or severe MS or congestive heart failure, because these women are at a very high thrombo-embolic risk.

According to scientific literature, percutaneous mitral commissurotomy is preferably performed after 20 weeks of gestation. Closed commissurotomy remains an alternative in low-middle-income countries when percutaneous commissurotomy is not available. Open heart surgery should be reserved for cases in which all other measures have failed and mother's life is threatened (24).

When there is an indication for anticoagulation therapy before pregnancy, anticoagulation

should also be maintained during pregnancy. The type of anticoagulation during pregnancy (UFH vs. LMWH) needs to be decided on individual basis. Because of the increased risk of bleeding in these patients, subcutaneous application of LMWH or UFH is an advantage over oral anticoagulation during pregnancy. Careful monitoring of blood coagulation parameters is necessary (INR, activated partial thromboplastin time).

According to the ESC Guidelines on the management of cardiovascular diseases during pregnancy, restricted activities and beta-1-selective blockers are recommended to patients with symptoms of pulmonary hypertension. Diuretics are recommended when congestive symptoms persist despite beta-blockers. Therapeutic anticoagulation is recommended in the case of paroxysmal or permanent AF, left atrial thrombosis or embolism. Intervention should be considered before pregnancy in patients with MS and valve area <1.5 cm^2.

Induction, management of labour, delivery and post-partum surveillance require specific expertise and collaborative management by skilled cardiologists, obstetricians, and anaesthesiologists in experienced maternal–foetal medicine units.

The mode of delivery should be individualized. Planned caesarean delivery and vaginal delivery are an advantage over emergency caesarean delivery.

Vaginal delivery should be considered in patients with mild MS and in patients with moderate or severe MS in NYHA class I/II without pulmonary hypertension. A Caesarean section is considered in patients with moderate or severe MS, who are in NYHA class III/IV or have pulmonary hypertension despite medical therapy, to whom percutaneous mitral commissurotomy cannot be performed or has failed (24).

CONCLUSIONS

1. More women with congenital or acquired heart disease are reaching childbearing age due to improved medical and surgical care, hence the incidence of cardiovascular disease in pregnancy is increasing. In Lithuania, the incidence of cardiovascular disease in pregnancy is decreasing.

2. Pregnancy in conjunction with heart disease is a complicated condition and presents a serious therapeutic challenge. The third trimester appears to be the most susceptible period when maternal and foetal status can decline rapidly, therefore, multidisciplinary prenatal care (consisting of an obstetrician gynaecologist, a cardiologist, and an anaesthesiologist) is necessary.

3. Taking into account the fact that pregnancy itself is associated with a higher risk of thrombotic events, prophylaxis is recommended if there are any additional risk factors for a hypercoagulable state. Prophylaxis includes pharmacotherapy with unfractionated heparin or LMWH and compression stockings.

4. Tachycardia during pregnancy can be managed using β-blockers or calcium channel blockers but it is essential to maintain a normal stroke volume and cardiac output in order to achieve the best outcome for both mother and foetus.

5. Pregnancy can aggravate heart disorders which are the most common non-obstetric cause of maternal and foetal mortality and morbidity, therefore preconception counselling and evaluation of the heart function are recommended for women with a diagnosed cardiovascular disease.

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References

1. Lithuanian Health Information Centre. Health Statistics of Lithuania. 2016.
2. Marelli AJ, Mackie AS, Ionescu-Ittu R, Rahme E, Pilote L. Congenital Heart Disease in the General Population Changing Prevalence and Age Distribution. Circulation. 2007 Jan 16; 115(2): 163–72.
3. Thornburg KL, Jacobson SL, Giraud GD, Morton MJ. Hemodynamic changes in pregnancy. Semin Perinatol. 2000 Feb; 24(1): 11–4.
4. Hall ME, George EM, Granger JP. The heart during pregnancy. Rev Esp Cardiol. 2011 Nov; 64(11): 1045–50.
5. Kuklina E, Callaghan W. Chronic heart disease and severe obstetric morbidity among hospitalisations for pregnancy in the USA: 1995–006. BJOG Int J Obstet Gynaecol. 2011 Feb; 118(3): 345–52.
6. Fernandes SM, Pearson DD, Rzeszut A, Mitchell SJ, Landzberg MJ, Martin GR. Adult congenital heart disease incidence and consultation: a survey

of general adult cardiologists. J Am Coll Cardiol. 2013 Mar 26; 61(12): 1303–4.
7. Nanna M, Stergiopoulos K. Pregnancy complicated by valvular heart disease: an update. J Am Heart Assoc Cardiovasc Cerebrovasc Dis [Internet]. 2014 Jun 5 [cited 2015 Dec 1]; 3(3). Available from: http://www.ncbi.nlm.nih.gov/pmc/articles/PMC4309052/
8. Bonow RO, Carabello BA, Chatterjee K, de Leon Jr AC, Faxon DP, Freed MD, et al. 2008 Focused Update Incorporated Into the ACC/AHA 2006 Guidelines for the management of patients with valvular heart disease: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Revise the 1998 Guidelines for the Management of Patients With Valvular Heart Disease). Endorsed by the Society of Cardiovascular Anesthesiologists, Society for Cardiovascular Angiography and Interventions, and Society of Thoracic Surgeons. J Am Coll Cardiol. 2008 Sep 23; 52(13): e1–142.
9. Reimold SC, Rutherford JD. Valvular heart disease in pregnancy. N Engl J Med. 2003 Jul; 349(1): 52–9.
10. Christianson RE. Studies on blood pressure during pregnancy. I. Influence of parity and age. Am J Obstet Gynecol. 1976 Jun 15; 125(4): 509–13.
11. Ueland K, Metcalfe J. Circulatory changes in pregnancy. Clin Obstet Gynecol. 1975 Sep; 18(3): 41–50.
12. Petch MC. Cardiac disease in pregnancy. Postgrad Med J. 1979 May; 55(643): 315–7.
13. Longo LD. Maternal blood volume and cardiac output during pregnancy: a hypothesis of endocrinologic control. Am J Physiol. 1983 Nov; 245(5 Pt 1): R720–9.
14. May L. Cardiac physiology of pregnancy. Compr Physiol. 2015 Jul 1; 5(3): 1325–44.
15. Hameed AB, Sklansky MS. Pregnancy: maternal and fetal heart disease. Curr Probl Cardiol. 2007 Aug; 32(8): 419–94.
16. Avila WS, Rossi EG, Ramires JAF, Grinberg M, Bortolotto MRL, Zugaib M, et al. Pregnancy in patients with heart disease: Experience with 1,000 cases. Clin Cardiol. 2003 March; 26(3): 135–42.
17. Briggs GG, Freeman RK, Yaffe SJ. Drugs in pregnancy and lactation: a reference guide to fetal and neonatal risk. Lippincott Williams & Wilkins; 2011. 1728 p.
18. James AH. Pregnancy and thrombotic risk. Crit Care Med. 2010 Feb; 38(2 Suppl): S57–63.
19. Andersen AS, Bergholt T, Salvig JD, Hvas A-M. [Pregnancy predispose to higher incidence of venous thromboembolism]. Ugeskr Laeger. 2015 Nov 30; 177(49): V06150557. Danish.
20. The Turkish Journal of Pediatrics [Internet]. [cited 2016 May 2]. Available from: http://www.turkishjournalpediatrics.org/?fullTextId=1052&lang=eng
21. Wainwright H, Beighton P. Warfarin embryopathy: fetal manifestations. Virchows Arch Int J Pathol. 2010 Dec; 457(6): 735–9.
22. Galambosi PJ, Kaaja RJ, Stefanovic V, Ulander V-M. Safety of low-molecular-weight heparin during pregnancy: a retrospective controlled cohort study. Eur J Obstet Gynecol Reprod Biol. 2012 Aug; 163(2): 154–9.
23. Schindewolf M, Gobst C, Kroll H, Recke A, Louwen F, Wolter M, et al. High incidence of heparin-induced allergic delayed-type hypersensitivity reactions in pregnancy. J Allergy Clin Immunol. 2013 Jul; 132(1): 131–9.
24. ESC Guidelines on the management of cardiovascular diseases during pregnancy. The Task Force on the Management of Cardiovascular Diseases during Pregnancy of the European Society of Cardiology (ESC). Endorsed by the International Society of Gender Medicine (IGM), the German Institute of Gender in Medicine (DGesGM), the European Society of Anaesthesiology (ESA), and the European Society of Gynecology (ESG). European Heart Journal. 2018; 39: 3165–3241.

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ŠIRDIES VOŽTUVINĖ LIGA NĖŠTUMO METU: KLINIKINIS ATVEJIS IR LITERATŪROS APŽVALGA

Santrauka

Tikslas. Širdies ydų diagnostika ir gydymo metodai tobulėja, todėl įgimtos širdies ydos tampa vis aktualesne vaisingo amžiaus moterų problema. Įgytų širdies ligų tikimybė didėja ir dėl to, kad gimdo vyresnio amžiaus moterys, kai gali pasireikšti hipertenzijos, cukrinio diabeto ir kitų ligų komplikacijos. Lietuvoje atvirkščiai – nėščiųjų sergamumas širdies ir kraujagyslių ligomis mažėja: nuo 1,4 % 2014 m. iki 1,0 % 2016 m. (Lietuvos sveikatos informacijos Centro, Lietuvos sveikatos statistikos duomenys 2016). Širdies ligos gali sumažinti organizmo adaptacines galimybes ir sukelti komplikacijų, keliančių grėsmę motinos ir vaisiaus sveikatai ar gyvybei. Tokių būklių gydymas yra iššūkis gydytojų komandai. Straipsnio tikslas – remiantis konkretaus klinikinio atvejo pavyzdžiu aptarti nėštumo metu kylančius diagnostikos ir nėščiosios priežiūros ypatumus, esant hemodinamiškai reikšmingai širdies ydai.

Medžiaga ir metodai. Analizuojamas 30-ies metų amžiaus pacientės klinikinis atvejis, kuriai nėštumo metu diagnozuota sudėtinė mitralinio vožtuvo yda. Pacientei jau buvo diagnozuota vyraujanti kritinė stenozė, II–III laipsnio mitralinio vožtuvo nepakankamumas, vidutinio ir sunkaus laipsnio plautinė hipertenzija, širdies nepakankamumo C stadija, NYHA II funkcinė klasė.

Rezultatai ir išvados. Nėštumo, komplikuoto kardiologine patologija, eiga yra sudėtinga, todėl reikalinga multidisciplininė nėščiosios priežiūra (dalyvaujant gydytojų komandai: akušeriui ginekologui, kardiologui, anesteziologui). Skiriant trombozių profilaktinį gydymą, pirmuoju nėštumo trečdaliu reikėtų rinktis mažos molekulinės masės heparinus. Nėštumas gali pabloginti širdies ligų gydymą, todėl moterims patariama nėštumą planuoti tik po nuoseklaus širdies funkcijos ištyrimo ir gydytojo kardiologo konsultacijos.

Raktažodžiai: nėštumas, širdies ydos, vožtuvinė širdies liga, mitralinio vožtuvo nesandarumas, širdies nepakankamumas