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Review

Pregnancy Management and Outcomes with Hypertrophic Cardiomyopathy

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ABSTRACT

Hypertrophic cardiomyopathy (HCM) is a common inherited genetic cardiac disorder that increasingly is being diagnosed in people of childbearing age, due to advancements in genetic testing and the evolution of screening guidelines. Many women may first present with HCM during pregnancy, which can lead to adverse maternal and fetal outcomes if not appropriately managed. Although most women with HCM tolerate pregnancy well, it can be associated with increased cardiovascular risks, including heart failure, arrhythmias, and adverse fetal outcomes, particularly among those with suboptimal baseline functional status. Medical therapy remains the mainstay of management of HCM in pregnancy, as only a limited number of studies have been done on the safety and effectiveness of invasive procedures, such as alcohol septal ablation and cardiac myomectomy, in pregnant women. Large cohort studies of women with HCM are very

RÉSUMÉ

La cardiomyopathie hypertrophique (CMH) est une maladie génétique cardiaque héréditaire fréquente, de plus en plus diagnostiquée chez les femmes en âge de procréer en raison de l'amélioration des tests génétiques et de l'évolution des directives de dépistage. Chez de nombreuses femmes, la CMH peut se déclarer durant la grossesse et avoir des conséquences néfastes pour la mère et le fœtus si elle n'est pas prise en charge de manière appropriée. Alors que la plupart des femmes atteintes d'une CMH tolèrent bien la grossesse, celle-ci peut être associée à des risques cardiovasculaires accrus, dont l'insuffisance cardiaque et les arythmies, et à des effets défavorables pour le fœtus, en particulier chez les femmes dont le statut fonctionnel initial est sous-optimal. Le traitement pharmacologique demeure le pilier de la prise en charge de la CMH pendant la grossesse, car les études sur l'innocuité et l'efficacité des interventions invasives comme l'ab-

Hypertrophic cardiomyopathy (HCM) is the most common genetic cardiomyopathy, with a prevalence of 1 in 500 worldwide, as determined by clinical criteria including echocardiography.[1,2] Advances in cardiac imaging, genetic testing, and the screening of asymptomatic family members have revealed that the estimated prevalence is likely closer to 1 in every 200 people.[3] Similarly, prevalence is expected to be higher in pregnancy than previously thought. However, HCM is generally underdiagnosed in pregnancy, reported to occur in less than 1 in 1000.[4] In more than 50% of HCM patients, a disease-causing mutation can be identified,

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limited, and management practices vary within centres, with no protocolized care. This review aims to summarize the unique challenges faced by pregnant women with HCM, highlighting the need for large multicentric studies and more standardized care protocols to optimize maternal and fetal outcomes.

lation septale à l'alcool et la myomectomie cardiaque chez la femme enceinte sont limitées. Les études de cohorte de grande envergure menées chez des femmes atteintes d'une CMH sont rares, et les pratiques de prise en charge varient d'un centre à l'autre, faute de protocoles de soins standardisés. Cet article vise à résumer les défis particuliers auxquels les femmes enceintes atteintes d'une CMH sont confrontées, en soulignant la nécessité de mener des études multicentriques de grande envergure et de mettre en place des protocoles de soins davantage standardisés afin d'optimiser les résultats pour la mère et le fœtus.

commonly involving genes encoding sarcomere proteins.[5] HCM refers to the thickening of the ventricular wall (> 15 mm), which is not explained by a physiological adaptation to abnormal loading states. Wall thickening is generally asymmetric and commonly involves proximal interventricular septum, but concentric hypertrophy of the left ventricle, and right ventricular involvement, also can be seen.[6] Left ventricular (LV) hypertrophy can cause either dynamic or static LV outflow tract (LVOT) obstruction, found in ~70% of patients.[7] These structural and functional abnormalities can cause fatigue, chest pain, palpitations, presyncope and/or syncope, and symptoms of heart failure, such as shortness of breath and exercise intolerance.

HCM can initially present in younger individuals during pregnancy. Pregnancy is a physiologically stressful state characterized by multiple hemodynamic adaptations. It can potentially exacerbate underlying cardiac pathology and increase the risk of complications. Mortality among pregnant women with HCM has been reported to be 0.2%-0.5%, which is higher than the overall maternal mortality rate in the US.[8,9] Increases in maternal morbidity and adverse fetal outcomes also have been reported.[9] Preconception risk assessment, genetic counselling, multidisciplinary management, close monitoring, and follow-up care are vital for improving maternal and fetal outcomes.[10-12] This narrative review seeks to outline the distinct challenges faced by pregnant individuals with HCM, while highlighting the available evidence, acknowledging the knowledge gaps, and underscoring the importance of preconception assessment and comprehensive management strategies to enhance both maternal and fetal outcomes. The important changes during pregnancy, high risk features, and management strategies have been summarized in the Central Illustration.

Physiology of HCM in Pregnancy

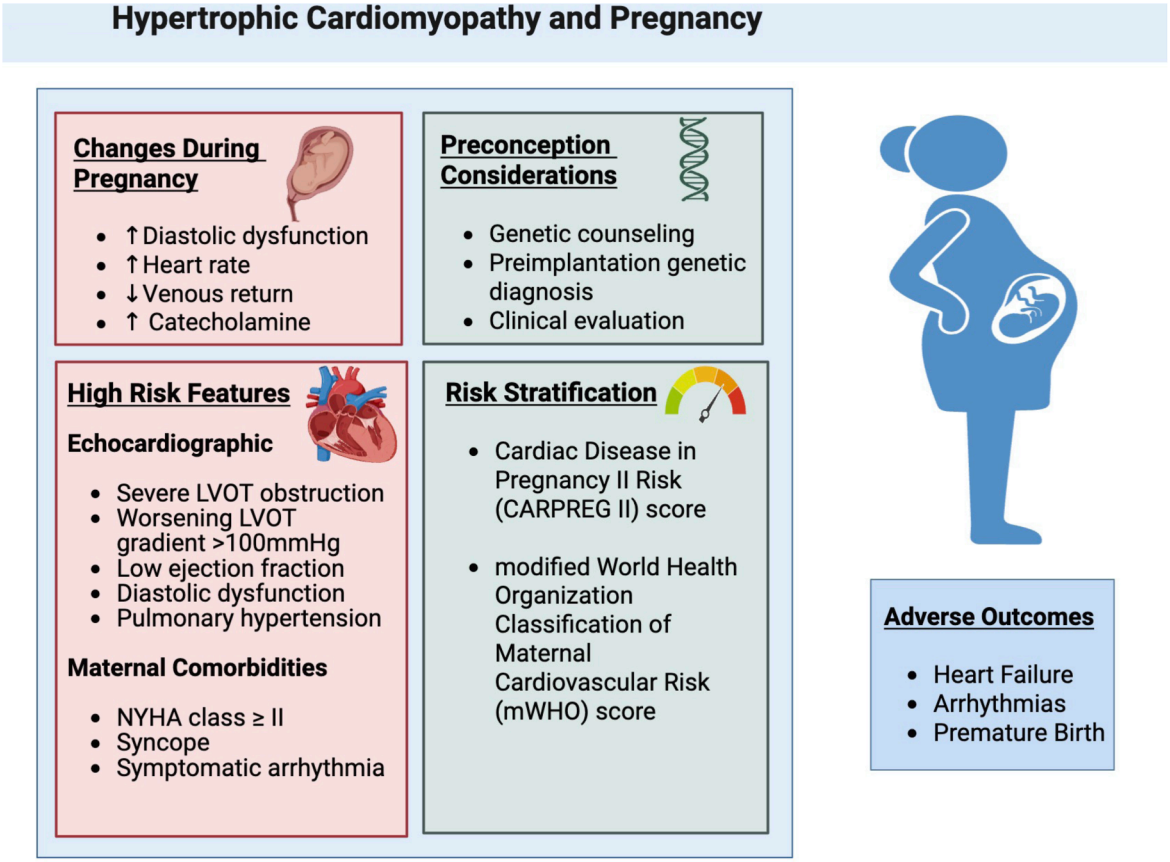
Pregnancy is associated with multiple hemodynamic changes that cause an increased demand on the maternal cardiovascular (CV) system. A significant increase in circulating plasma volume occurs, by around 40%-50%, as well as an increase in cardiac output, and a decrease in systemic vascular resistance. An increase in plasma volume expands the size of the LV cavity, theoretically decreasing the LVOT obstruction.[13] However, the left ventricle becomes hyperdynamic, increasing the contractility to generate more cardiac output, which can potentially worsen the LVOT obstruction. As the pregnancy progresses, a rise in heart rate during the third trimester also drives the increase in cardiac output, resulting in a progressive increase in the LVOT gradient.[14] Hemodynamic changes of increased plasma volume expansion with a smaller ventricle in the setting of HCM may increase the risk of congestive heart failure and adverse maternal outcomes during pregnancy. During labor, LVOT obstruction is further exacerbated by an increase in heart rate from the catecholamine surge secondary to pain, uterine contraction, blood loss, and stress. These acute changes in hemodynamics and volume shifts during labor and in the immediate postpartum period create a highly vulnerable time for clinical deterioration of patients.[15] Finally, stretch and expansion of cardiac chambers throughout pregnancy in the setting of volume expansion may be contributors to supraventricular arrhythmias, such as atrial fibrillation, which can result in decompensation among women with HCM.

Adverse Maternal and Fetal Outcomes in Pregnancy

HCM is usually well tolerated in pregnancy, and most pregnancies are uneventful, with mothers remaining asymptomatic or having mild symptoms during the pregnancy course.[8,13] However, some individuals may face significant morbidity and mortality, especially those with severe LVOT obstruction or pre-pregnancy symptoms.[4,8,13] The high-risk features of HCM associated with adverse maternal and fetal outcomes are summarized in Table 1.

Adverse maternal outcomes

A pooled analysis of 9 studies including 408 pregnancies in 237 women with HCM reported a maternal mortality rate of 0.5%, and around 30% of the women experienced some complication or worsening of symptoms during pregnancy.[8] Another systematic review, including 18 studies with 1624 pregnancies, reported that the maternal mortality rate was 0.2% and that 43% of the pregnancies were complicated with cesarean section, with a 14% incidence of urgent cesarean section due to CV conditions. Although the maternal mortality reported is not exceedingly high, compared to that in the general population, an increased risk of CV complications, such as ventricular tachycardia (6% overall with sustained in 1%), atrial fibrillation (4%), heart failure (5%), and adverse fetal outcomes such as preterm births (22%), has been reported.[9] A higher incidence of arrhythmias was reported in the third trimester, particularly in women with early trimester symptoms or preexisting arrhythmias, highlighting the need for early prenatal care and risk stratification. The commonly



Central Illustration. Hypertrophic cardiomyopathy and pregnancy. LVOT, left ventricular outflow tract; NYHA, New York Heart Association.

reported CV events related to HCM in pregnancy include heart failure, arrhythmias such as supraventricular arrhythmias (atrial fibrillation), ventricular tachycardia (sustained or nonsustained), and thromboembolic events, with heart failure reported in 5%-15% of pregnancies[8,9,18]; the risk of clinical deterioration was twice as high in patients with LVOT obstruction.[18] A prospective multicentric cohort study using the Registry of Pregnancy and Cardiac Disease (ROPAC) reported that 23% of 60 pregnant women with HCM experienced a major adverse cardiovascular event (MACE), most commonly heart failure (15%) and ventricular tachyarrhythmia (10%), primarily in the last trimester or postpartum.[17] A strong association between MACE and New York Heart Association functional class of $\geq$ II and signs of heart failure before pregnancy was reported, with no significant differences in outcomes between obstructive vs nonobstructive cardiomyopathy.[17]

Adverse fetal outcomes

Adverse fetal outcomes are also an important consideration in mothers with HCM. In the ROPAC data analysis by Goland et al., and in a systemic analysis by Moolla et al., the incidence of fetal mortality was comparable to that in the general population.[9,17] Fetal mortality occurred mostly in patients who also experienced MACE, highlighting the importance of enhanced fetal monitoring in this high-risk population. Multiple studies also have reported an increased incidence of preterm birth compared to that in the general population. Moolla et al., Goland et al., and Schinkel reported preterm birth in 22%, 26%, and 26%, respectively, in comparison to 10.4% in all birthing individuals in the US.[8,9,17] However, a likely contributor to this finding is precautionary early pregnancy delivery, due to a MACE.[17]

Preconception Considerations

Risk assessment and counselling for individuals with HCM planning for pregnancy should start as soon as possible with specialized pre-pregnancy counselling to optimize pregnancy planning and outcomes. For preconception assessment of women with HCM, adopting a multidisciplinary approach that integrates clinical, genetic, and imaging tools to individually customize management strategies is crucial for achieving optimal maternal and fetal outcomes. Ongoing monitoring and collaboration between cardiology and high-risk obstetric teams are important.[19] A comprehensive examination, including a detailed medical history, physical examination, drug history, and detailed record review, along with a 3-generational family history covering structural, vascular, and rhythm disorders and instances of sudden unexpected death, is important for appropriate preconception care and risk stratification.

Table 1. High-risk features of hypertrophic cardiomyopathy associated with adverse maternal and fetal outcomes

Echocardiographic features
Severe LVOT obstruction or worsening LVOT gradient > 100 mm Hg[8,16]
Low ejection fraction[10,17]
Diastolic dysfunction[9]
Pulmonary hypertension[10,17]
Maternal comorbidities/functional status
NYHA class ≥ II[8,11]
Syncope[8,9]
Symptomatic arrhythmia[17]

LVOT, left ventricular outflow tract; NYHA, New York Heart Association.

Genetic counselling and testing

Preconception genetic counselling and testing is vital for individuals with first-degree relatives with HCM and symptomatic individuals. A majority of the patients with HCM follow an autosomal dominant pattern of inheritance with an identifiable mutation of the sarcomere protein.[5] However, these genetic mutations have variable penetrance and phenotype expressivity, leading to variable disease severity among affected family members. The polygenic nature of HCM involves multiple mutations in sarcomere-associated protein genes such as *MYH7* and *MYBPC3*, which are 2 of the most common genes involved in encoding for β-myosin heavy chain and myosin-binding protein C, respectively.[6] With the new advanced genomic sequencing methods, many new genes with variable penetrance have been identified, including *TTN* (titin), *TCAP* (telethonin), *MYOZ2* (myozenin 2), and *FHL1* (four and a half LIM domains 1), which may be responsible for sporadic cases.[6] The genetic heterogeneity with the identification of multiple mutations in responsible genes necessitates the utilization of advanced methods such as whole-genome sequencing for a more comprehensive examination. This process requires greater expertise and collaborative interpretation.[20] Moreover, next-generation sequencing provides fast and accurate analysis of a large number of genes.[20] Genetic counselling and/or testing, depending on individual preference, has become a vital part of diagnosis of HCM and reproductive counselling.

Prenatal reproductive and genetic testing for HCM is a class I recommendation by the European Society of Cardiology, the American College of Cardiology (ACC), and the American Heart Association (AHA).[10-12] Cascade genetic testing, which refers to genetic testing of family members of patients who have been identified to have a likely pathogenic or pathogenic genetic variant, is recommended.[11] The risk of adverse maternal events is 2-fold higher in those with pathogenic or likely pathogenic variants, compared with those without.[21] The screening guidelines for relatives are summarized in Table 2.

Preimplantation genetic diagnosis

The advancement in genetic testing and precision medicine has opened a field of opportunities for genetic therapeutics and the prevention of genetic illnesses. Preimplantation genetic testing (PGT) identifies causal mutations and aids in family planning decisions by providing the parents an opportunity to prevent the transmission of genetic illness to their children.[23-25] This technique is employed during the *in vitro* fertilization process, in which the embryo cells are checked for mutation before implantation.[23] However, this technique has certain challenges, such as amplification failure, financial constraints, and ethical concerns. Its use is prohibited by certain countries, and it is not widely available.[26] These challenges underscore the importance of genetic counselling of patients and their family members in a multidisciplinary centre to provide support, discuss possible outcomes, manage strategies, and address any psychological and ethical concerns.

Clinical evaluation and risk stratification

Clinical evaluation and risk stratification are crucial for managing HCM during the preconception period and pregnancy. Multiple risk-stratification tools are available; 2 of the common ones used in pregnant women with cardiomyopathy are the cardiac disease in pregnancy II risk (CARPREG II) score[27] and the modified World Health Organization Classification of Maternal Cardiovascular Risk (mWHO) score.[28] The mWHO classification categorizes pregnancy into low risk (classes I and II) and higher risk (classes III and IV), with class IV considered to be extremely high risk for pregnancy, in which case pregnancy might be contraindicated.[28,29] Patients with HCM typically fall into class II or III, necessitating a close cardiology follow-up evaluation, every trimester or monthly and bimonthly, respectively. Patients in the class IV category require monthly follow-up evaluations.[30] Complications such as severe LV dysfunction with LV ejection fraction < 30%, and those with New York Heart Association functional class III or IV, severe LVOT obstruction, or symptomatic arrhythmias place patients in class IV, with advice against pregnancy.[28,31,32]

Echocardiography is essential during preconception risk stratification and during pregnancy to assess the cardiac structure and function monitoring. Cardiopulmonary tests such as exercise tolerance testing are informative for assessment of functional capacity, heart rate response, and presence of arrhythmias. Parameters such as LV wall thickness, LVOT obstruction, diastolic function, and degree of mitral regurgitation need to be monitored regularly to guide management decisions, especially for high-risk patients.[33]

Diagnostic Considerations

Cardiac imaging

Cardiac imaging is vital for making diagnosis and management decisions during pregnancy. Echocardiography is the main modality for imaging, and a transthoracic echocardiogram should suffice in the majority of patients. The potential risks of ionizing radiation during pregnancy are primarily related to the cumulative fetal radiation dose. Deterministic effects, such as growth restriction, microcephaly, or intellectual disability, have not been reported with fetal exposures below a threshold of 50 mGy (5 rad), which is widely accepted as the safe limit for diagnostic imaging during pregnancy.[10,34] Most diagnostic procedures—including computed tomography of the chest (0.01-0.66 mGy), computed tomography coronary angiography (1-3 mGy), and cardiac catheterization/

Table 2. Screening guidelines for first-degree family members

Genetic variant identified	Examples	Recommendations
Pathogenic/ likely pathogenic	*MYBPC3* truncating variants (eg, p.Arg502Trp), *MYH7* missense variants (eg, p.Arg403Gln), *TNNT2*, *TNNI3* mutations—well-established pathogenic variants in familial HCM[11,22]	Cascade genetic testing (informing family members followed by genetic testing by those who request it) Clinical screening: electrocardiogram and transthoracic echocardiogram
Benign/likely benign	*MYBPC3* p.Glu542Gln, *MYH7* p.Arg870His—variants with high-population frequency or functional data suggesting no disease association[11,22]	Clinical screening: electrocardiogram and transthoracic echocardiogram
Variants of unknown significance	*MYH7* p.Val404Met—variants with limited or conflicting evidence for pathogenicity[11,22]	Clinical screening: electrocardiogram and transthoracic echocardiogram

HCM, hypertrophic cardiomyopathy.

fluoroscopy (typically 1-=10 mGy depending on duration and shielding)—will fall below this threshold when appropriate dose-reduction techniques, avoidance of direct abdominal radiation, and abdominal shielding are employed.[34] Nonetheless, imaging should be performed only when it is clinically necessary and optimized to minimize exposure. The preferred imaging modalities and their advantages and disadvantages are summarized in Table 3.

Cardiac biomarkers

Studies on the utilization of cardiac biomarkers for diagnosing and monitoring cardiac disease in pregnancy are scarce. N-terminal pro-B-type natriuretic peptide (NT-proBNP) has been reported to be an independent predictor of mortality and heart failure events in patients with HCM outside of pregnancy.[39] Its levels are reported to remain stable throughout pregnancy, with spikes during labor and delivery, with higher levels in stable cardiac patients.[40,41] An NT-proBNP level of 200 pg/mL has been proposed as a probable cutoff for diagnosis of heart failure in pregnant mothers.[40] However, cardiac biomarkers during pregnancy have not been evaluated rigorously , and their association with maternal outcomes in those with HCM has not been well studied, limiting their utilization.

Management Considerations

Management focuses on symptom control and strategies to avoid complications and teratogenic drugs.

Medical management

A list of teratogenic drugs can be accessed on Teratogenic Information System (TERIS) (https://terisweb.deohs.washington.edu/).

Table 3. Imaging modalities in pregnant individuals with hypertrophic cardiomyopathy

Imaging modality	Advantages	Disadvantages
Echocardiography	○ Free of radiation ○ Readily available ○ Inexpensive ○ Can provide details of chamber size, hemodynamics of heart ○ Safe in pregnancy ○ Consider transesophageal echocardiography—determine pathology of obstruction, delineate left ventricular outflow tract and differentiate from potential mimickers—sub-aortic membrane	○ Skill dependent ○ Limited by the quality of acoustic windows[35] ○ Gravid uterus can hamper the image window ○ Interobserver variation
Magnetic resonance imaging	○ Free of radiation ○ Low interobserver variation ○ Not skill dependent ○ High-resolution images ○ Superior diagnosis of left ventricular apical abnormalities[36] ○ Identification of other coexisting lesions such as aortopathy ○ Safe in pregnancy	○ Not readily available ○ Expensive ○ Longer duration ○ Poorly tolerated in individuals with claustrophobia ○ Controversial evidence on safety of gadolinium contrast during pregnancy. Preferentially avoided unless necessary. Use of gadolinium in majority of patients can be deferred to postpartum period.[10,34,37,38]
CT scan	○ Shorter duration ○ Better tolerated in individuals with claustrophobia ○ No added benefit in patient with HCM	○ Radiation ○ Radioiodine contrast-associated risks such as allergy, contrast-induced nephropathy, etc. Limited data on fetal toxicity. ○ Inferior temporal resolution compared to echocardiography ○ Use is not recommended, unless other modalities such as echocardiography and MRI are insufficient or unavailable[38]

CT, computed tomography; HCM, hypertrophic cardiomyopathy; MRI, magnetic resonance imaging.

Beta-blockers. The use of beta-blockers is critical in managing HCM and typically should be continued in pregnancy. Pregnant patients with symptoms related to LVOT obstruction should be started on maximally tolerated beta-blockers for symptom control and improved outcomes.[11] Although data have been conflicting regarding the use of beta-blockers during pregnancy and their association with congenital malformations, multiple large cohort studies, after adjusting for maternal comorbidities, have not reported an increased risk of congenital malformations.[42,43] All beta-blockers except for atenolol are considered safe during pregnancy. However, beta-blockers may cross the placenta, potentially causing hypoglycemia, neonatal bradycardia, and intrauterine growth retardation, making careful monitoring essential if these medications are used.[44,45] ACC/AHA guidelines recommend use of selected beta-blockers for symptoms related to outflow tract obstruction or arrhythmias, with monitoring of fetal growth (class I).[10,12] Metoprolol and propranolol are the preferred beta-blockers in pregnancy for HCM management.[45] Metoprolol, being cardioselective, is preferred to propranolol and more commonly used. Usage of atenolol is related to a significant risk of intrauterine growth restriction and hence should be avoided.[46] Notably, beta-blockers should be up-titrated throughout pregnancy, based on maternal heart rate. An abrupt cessation of beta-blockers can cause reflex tachycardia, posing a risk to both the mother and the fetus. Therefore, any decision to discontinue beta-blockers should be made under close medical supervision, with careful consideration of potential consequences.

Calcium-channel blockers. Calcium-channel blockers, such as verapamil and diltiazem, also play a role in managing HCM in mothers when beta-blockers are contraindicated or not tolerated.[10] Evidence has been conflicting regarding the potential side effects. Verapamil use has been linked previously to an increased risk of neonatal seizures, likely secondary to hypocalcemia[47]; more recent studies, however, do not report any such risk.[48] So they can be considered as an alternative to beta-blockers if necessary, with close monitoring of fetal heart rate to mitigate the risk of atrioventricular block.[13,29]

Myosin inhibitors. Use of a myosin inhibitor, such as mavacamten, is not recommended in pregnant individuals due to potential teratogenic effects (class III, level of evidence C: Expert Opinion).[12] Individuals should be advised to be off mavacamten for at least 6 months before conception.[38] During mavacamten therapy, combined hormonal contraceptives containing a specific combination of ethinyl estradiol and norethindrone are preferred due to potential drug-drug interactions that may reduce the effectiveness of other types of hormonal contraceptives. Due to the potential interaction, the addition of intrauterine or barrier contraception to hormonal contraception should be considered.[38] The safety of mavacamten use during lactation is unknown.[49]

Invasive procedures

Medical management remains the primary course of management of HCM during pregnancy. Only a paucity of literature is available on the utilization of invasive procedures during pregnancy; hence, these rarely are considered. These can be considered when symptoms are refractory to medical management, when evidence of severe LVOT obstruction and worsening symptoms are present. Per a few case reports, alcohol septal ablation has shown successful symptomatic improvement during pregnancy. The procedure was performed in a limited fluoroscopy time and with radiation shielding to minimize the risk to the fetus. The procedure was well tolerated by the patient and the fetus, with an improvement of the obstructive pathology.[50,51] Therefore, if needed, it should be carried out in a multidisciplinary centre with cardiologists, obstetricians, and anesthesiologists, by a skilled interventionalist or a surgeon with expertise in performing the procedure.

Surgical myomectomy is another invasive procedure, preferred over alcohol septal ablation in young individuals with concomitant mitral valve pathology.[29] However, its safety and efficacy in pregnancy have not been evaluated thoroughly. Before proceeding with any invasive cardiac procedure, a thorough risk-benefit discussion is essential to consider potential postprocedure complications, such as heart block and tachyarrhythmias, which may require a pacemaker or implantable cardiac defibrillator (ICD). Whenever necessary, these procedures preferentially should be performed in the second trimester, avoiding the first trimester due to potential fetal developmental risks.

Anticoagulation therapy

Pregnancy is considered a prothrombotic state; however, no concrete data are available on stroke risks or validated stroke risk scores for pregnant patients with atrial fibrillation. Anticoagulation therapy is recommended for all pregnant patients with HCM with paroxysmal or persistent atrial fibrillation.[10,12,30] The risk of embryopathy by warfarin is dose-dependent, and evidence with low-dose warfarin (< 5 mg daily) remains limited.[10] Low-molecular-weight (LMW) heparin is especially important if the daily warfarin dose is $>$ 5 mg/d.[52] The mother can be transitioned to a therapeutic dose of LMW heparin before conception. The timing of LMW heparin near the time of delivery should be coordinated carefully among cardiology, obstetrics, and anesthesia teams to ensure adequate predelivery interruption, allowing for safe administration of neuraxial anesthesia and minimizing the risk of postpartum hemorrhage.[10] Due to limited evidence on the use of direct oral anticoagulants in pregnancy, and a higher rate of fetal complications compared with LMW heparin in a metanalysis, ACC/AHA guidelines for the management of HCM recommend against their use during pregnancy.[11,12] During the postpartum period, warfarin is considered safe during lactation; however, only limited safety data are available regarding the use of direct oral anticoagulants during lactation.

Role of ICD

A previously implanted ICD is not a contraindication to pregnancy and has not been associated with increased maternal or fetal risks.[53] ICDs are indicated during the preconception period if the mother is deemed during risk stratification to be at high risk of sudden cardiac death.[54] The device also can be implanted during pregnancy under limited fluoroscopy and echocardiography-guided mapping

techniques, and no increase in the risk of ICD-related complications has been reported.[55] If a concern for radiation exposure during pregnancy is present, patients may opt for a wearable cardioverter defibrillator initially, followed by ICD insertion in the postpartum period. The clinical effectiveness of this device in pregnancy is yet to be explored fully.[56]

Fetal echocardiography

Utilization of routine fetal echocardiography to diagnose HCM in a fetus is not recommended, as the disease is unlikely to be clinically expressed in the fetus. However, the disease warrants consideration in patients with a history of pediatric-onset disease, severe manifestations in parents or other family members, or a strong suspicion of metabolic or syndromic disorders.[11,29]

Delivery Planning

Despite the relatively low risk of severe complications for patients with HCM, guidelines recommend delivery planning with a multidisciplinary team.[10,11] A plan for the mode of delivery and the timing should be made by the end of the second trimester, when maximal maternal blood-volume expansion occurs. Factors to be considered include symptomatology, ejection fraction, history of arrhythmias, comorbidities, and severity and progression of LVOT obstruction through pregnancy.

Vaginal delivery vs cesarean section generally is recommended for most patients with HCM, due to the lower associated risks of blood loss, infection, and thromboembolism with vaginal delivery.[10] Continuous blood pressure and heart rate monitoring is advised during labour—due to increased arrhythmia risk with hemodynamic stress—to detect early signs of decompensation; delivery should take place in high-risk labour and delivery units where monitoring and urgent cardiac intervention are available.[10] Careful administration of induction medications, such as oxytocin, and judicious use of intravenous fluids to prevent arrhythmias and volume overload are needed.[10,30]

During delivery, women with ICDs require multidisciplinary coordination among cardiology, obstetrics, anesthesia, and electrophysiology teams. To reduce electromagnetic interference from electrocautery during cesarean delivery, bipolar cautery is preferred. If monopolar cautery is necessary, short bursts should be used to minimize the electromagnetic interference.[57,58] When significant interference is expected, ICD therapies should be suspended temporarily using a magnet or reprogramming, with external defibrillator pads available. For patients undergoing vaginal delivery, deactivation of the device is not needed.[57,58]

Patients with severe LVOT obstruction, severe heart failure, or preterm labor while on therapeutic anticoagulation face a heightened risk of severe complications, warranting consideration for a cesarean section with close circulatory and heart rhythm monitoring.[10,30] Bhave et al. exemplified the importance of interdisciplinary management for patients with severe risk factors by detailing the successful management of an HCM patient, complicated by an outflow tract gradient > 100 mm Hg, with a cesarean section.[16] Important in this case is the exercise of caution, to prevent hypotension during the administration of epidural and spinal anesthesia in patients with severe LVOT obstruction; therefore, single-shot anesthesia is not recommended in these patients.[10]

The close monitoring of hemodynamic status should continue in the postpartum period as well. Any rapid mobilization of extravascular fluid causes considerable hemodynamic shifts, raising the risk for pulmonary edema and arrhythmia, warranting close monitoring in the immediate postpartum phase.[9,10]

Conclusion

HCM is generally well tolerated in pregnancy and carries a low likelihood of adverse maternal and fetal outcomes. Adverse outcomes generally are associated with poor pre-pregnancy functional status and clinical features, such as severe LV systolic function, severe symptomatic LVOT obstruction, and symptomatic arrhythmias. Patients planning for pregnancy should undergo a thorough preconception clinical evaluation, genetic counselling, and risk stratification. This process helps assess pregnancy risks, facilitates shared decision-making, and aids in family planning. Maternal complications during pregnancy are related primarily to arrhythmias and heart failure, and preterm delivery represents a major adverse fetal outcome. For patients whose symptoms are well controlled on beta-blockers, these should be continued during pregnancy. Beta-blockers also may be initiated during pregnancy if new symptoms related to LVOT obstruction develop. Invasive management, such as alcohol septal ablation, is reserved for a small subset of patients with severe obstructive symptoms despite maximally tolerated medical therapy. Most women with HCM can safely deliver vaginally, with cesarean section reserved for only high-risk obstetric or emergent cardiac indications. Women with HCM have an increased risk of pulmonary edema during the postpartum period, due to fluid shifts, and should be monitored closely for at least 24-48 hours post-delivery. A multidisciplinary approach with close and frequent follow-up evaluation is essential for favourable outcomes during and after pregnancy.

Ethics Statement

The research reported in this paper adhered to relevant ethical guidelines. This study is a review of previously published literature and did not involve human participants, animals, ro new experimental guidelines.

Patient Consent

As this is a review article, the authors confirm that patient consent is not applicable.

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