

Review article

Acute cardiac failure in pregnancy: diagnosis and treatment

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ABSTRACT

Heart disease affects approximately 4% of pregnancies and is the leading non-obstetric cause of maternal death during pregnancy in many countries. The physiological hormonal and haemodynamic changes that occur during this period can unmask previously undiagnosed cardiac disease and destabilise pre-existing cardiovascular conditions. Acute heart failure during pregnancy is uncommon; however, it can lead to severe maternal and fetal complications. Although the diagnostic criteria are the same as those used in the general population, diagnosis can be challenging because the cardinal signs and symptoms of heart failure overlap with several symptoms and clinical findings that commonly occur during pregnancy. A comprehensive clinical history therefore remains fundamental to the diagnostic approach, enabling the rational use of complementary investigations and, in many cases, guiding treatment. Management of acute heart failure during pregnancy must be highly individualised and may involve decisions regarding continuation of the pregnancy and the optimal mode of delivery. Care should therefore be led by an experienced multidisciplinary team with expertise in the diagnosis and management of cardiovascular disease during pregnancy, with the aim of achieving the best possible outcomes for both the mother and the fetus.

Keywords: Heart Failure; Pregnancy; Pregnancy Complications; Cardiovascular; Peripartum Cardiomyopathy (Source: MeSH-NLM)

RESUMEN

Insuficiencia cardíaca aguda en el embarazo: diagnóstico y tratamiento

La enfermedad cardíaca afecta aproximadamente al 4% de los embarazos y es la principal causa de muerte no obstétrica durante el embarazo en muchos países. Los cambios hormonales y hemodinámicos que fisiológicamente se presentan durante este periodo precipitan la aparición de síntomas de la enfermedad cardíaca no conocida con anterioridad y pueden inestabilizar condiciones cardiovasculares ya diagnosticadas. La falla cardíaca aguda durante el embarazo es una entidad poco frecuente; sin embargo, puede conducir a complicaciones graves materno-fetales. Su diagnóstico no cambia con relación a la población general; no obstante, puede ser un gran desafío, puesto que los síntomas y signos cardinales de la enfermedad son similares a varias molestias y hallazgos propios de la gestación. Sin embargo, la historia clínica será la herramienta fundamental para el enfoque diagnóstico, haciendo un uso racional de las ayudas paraclínicas y, en muchas ocasiones, dirigiendo la terapia. El tratamiento de la gestante con falla cardíaca aguda es altamente individualizado e incluye decisiones sobre la continuidad del embarazo y la vía final del parto. Por tal razón, su abordaje debe estar comandado por un grupo interdisciplinario con experiencia en el diagnóstico y tratamiento de la enfermedad, buscando siempre los mejores resultados para la madre y el producto de la gestación.

Palabras clave: Insuficiencia Cardíaca; Embarazo; Complicaciones Cardiovasculares del Embarazo; Cardiopatía Periparto (Fuente: DeCS-Bireme)

Introduction

Pregnancy is associated with substantial haemodynamic changes, including a 40% increase in blood volume, an increase in heart rate, and a 25-30% reduction in systemic vascular resistance, allowing the increased blood volume to be accommodated without causing hypertension ⁽¹⁾. During labour, uterine activity increases blood flow requirements, resulting in a 15-50% increase in cardiac output in women with structurally normal hearts. In women with structural heart disease, these changes can precipitate symptoms of heart failure (HF). These haemodynamic changes reverse during the puerperium, over 6-12 weeks postpartum. However, placental delivery, with the subsequent loss of the uteroplacental circulation, and relief of uterine compression of the inferior vena cava cause a substantial increase in preload. This period therefore carries the greatest risk of HF, making close clinical follow-up and careful monitoring of changes in symptoms essential for early intervention ⁽¹⁻⁴⁾.

Acute HF may occur in women with pre-existing HF who poorly tolerate the haemodynamic changes of pregnancy, or it may present *de novo*, as in peripartum cardiomyopathy (PPCM), with *de novo* cases accounting for the largest proportion of fatal events ^(5,6). Cardiovascular disease complicates up to 4% of pregnancies in developed countries ⁽⁷⁾. In developing countries, its frequency may be higher because of the greater prevalence of rheumatic heart disease, uncorrected congenital heart disease, and uncontrolled cardiovascular risk factors among women of reproductive age ⁽⁷⁻¹⁰⁾. Cardiac disease is the leading cause of indirect maternal death, with a mortality rate of 1.6 per 100,000 maternities reported in UK records ⁽⁶⁾. Maternal heart disease is also associated with adverse fetal outcomes, depending on the complexity of the underlying condition. Recognising HF symptoms during pregnancy is challenging. Its association with poorer maternal and fetal outcomes, together with restrictions on the use of some treatments, makes HF during pregnancy a complex condition requiring a coordinated, multidisciplinary approach to optimise outcomes ^(7,11).

These patients should be assessed and followed by a multidisciplinary cardio-obstetrics team, with regular reviews tailored to their risk profile to optimise outcomes ⁽¹²⁾. Risk stratification models have been developed for women with structural or functional heart disease to classify the risk of adverse maternal and perinatal outcomes. Among these, the latest version of the modified World Health Organization classification (mWHO 2.0) stratifies patients according to the severity of their cardiovascular condition into classes I-IV, reflecting the risk of disease-related adverse outcomes during pregnancy. Each class informs key recommendations regarding counselling, Pregnancy Heart Team involvement, the level of care required for medical management and delivery, and the

frequency of follow-up ⁽²⁾. A detailed description of the risk associated with each condition and its modifiers is beyond the scope of this review; readers are referred to the relevant guidelines for further information.

Methods

A narrative literature review was conducted to identify and synthesise the available evidence on the diagnosis and treatment of acute HF during pregnancy. A comprehensive search for English- and Spanish-language publications was performed in PubMed/MEDLINE, Embase, and LILACS, from database inception to February 2026, without date restrictions. The English-language search used the following strategy with MeSH terms: ("pregnancy" AND "heart failure"). In LILACS, the Health Sciences Descriptors (DeCS) were used: ("insuficiencia cardíaca" AND "embarazo").

Study selection proceeded in sequential stages. Eligible publications included clinical practice guidelines, randomised controlled trials, cohort studies, systematic reviews with meta-analyses, narrative reviews, reports from recognised international organisations, and case series considered informative by the investigators. Letters to the editor and animal studies were excluded. Four investigators (EMO, JAGR, MBGS, and YIAE) performed the initial screening of titles and abstracts. The reference lists of shortlisted articles were subsequently searched manually to minimise the omission of relevant literature. Each investigator assessed a variable number of potentially eligible articles in full text. The evidence was organised into five thematic categories: general considerations, epidemiology, pathophysiology, diagnosis, and treatment. Section drafts were prepared according to the authors' areas of expertise and subsequently integrated, with consensus reached on the final version.

Definition and epidemiology

The definition of HF in the general population applies equally to pregnant women. HF is a clinical syndrome characterised by typical symptoms (e.g., dyspnoea, oedema, and fatigue), which may be accompanied by signs (e.g., jugular venous distension, hepatomegaly, and pulmonary crackles), caused by a structural or functional cardiac abnormality and corroborated by elevated natriuretic peptide concentrations or objective evidence of pulmonary or systemic congestion on imaging or haemodynamic assessment, either at rest or during exercise ⁽¹³⁾. This definition highlights the importance of the clinical history as the starting point for assessment, complemented by appropriate diagnostic investigations to demonstrate a structural or functional cardiac abnormality. However, the physiological changes of pregnancy, the higher prevalence of certain cardiac conditions, and restrictions on

the use of some treatments require a distinct approach to HF during pregnancy.

HF during pregnancy is uncommon, and acute pulmonary oedema has been reported in 0.08-3% of pregnant women^(14,15). This presentation requires intensive care admission in most cases and endotracheal intubation in 22%⁽¹⁶⁾. According to the CARPREG study, congenital heart disease, whether repaired or unrepaired, is the most common cardiac condition complicating pregnancy, followed by acquired cardiovascular disease, predominantly valvular disease, and arrhythmias. These findings are consistent with data from the European Registry of Pregnancy and Cardiac Disease (ROPAC)⁽¹⁷⁾, in which cardiomyopathies, including idiopathic dilated, hypertrophic, and ischaemic cardiomyopathy, accounted for 7.9% of cases. Only 7.4% of patients included in the study had a left ventricular ejection fraction (LVEF) < 40% (11). The predominance of patients without ventricular dysfunction highlights the importance of HF with preserved ejection fraction (HFpEF) in pregnant women. Earlier registries, including ROPAC, dichotomised patients into groups with reduced or preserved LVEF using a 40% threshold^(11,17). Current evidence recognises HFpEF as a distinct pathophysiological entity in which hypertensive disorders of pregnancy and obesity are key determinants of myocardial remodelling and diastolic dysfunction^(2-4,13).

Aetiology

Unlike in the general population, coronary artery disease is not the leading cause of HF during pregnancy, being present before pregnancy in fewer than 2% of pregnancies complicated by heart disease⁽¹⁷⁾. Causes can be classified as pregnancy-specific conditions, including PPCM, hypertensive disorders of pregnancy, and amniotic fluid embolism, or conditions that are not exclusive to pregnancy but occur more frequently during this period. These include left-sided obstructive lesions (mitral or aortic stenosis), acute aortic syndrome, congenital heart disease, proximal vascular abnormalities such as aortic coarctation, pulmonary or fat embolism, pulmonary hypertension, and infective endocarditis⁽¹⁸⁾. HF is usually precipitated by the physiological changes of pregnancy; however, other potential triggers, including infections, pulmonary embolism, arrhythmias, and acute coronary syndromes, should be investigated.

Particular attention is given to PPCM, a condition exclusive to pregnancy and the puerperium, and mitral stenosis, which is frequently diagnosed during pregnancy in low- and middle-income countries because of the prevalence of rheumatic heart disease. PPCM is defined as an acute HF syndrome presenting in the final month of pregnancy or within five months postpartum, associated with an LVEF < 45% and no other identifiable cause⁽¹⁹⁾. Its pathophysiology remains

incompletely understood. Proposed mechanisms include myocardial injury induced by the 16-kDa prolactin fragment, dysregulated IL-6-mediated inflammation, environmental factors, and genetic susceptibility associated with variants in the gene encoding titin^(20,21). Most case series report that PPCM predominantly presents postpartum, although 17-24% of cases occur before delivery. The prognosis is generally favourable, with recovery of LVEF in a substantial proportion of patients. Mitral stenosis may present as acute HF during the second trimester; its prognosis depends on the severity of the lesion and the response to treatment⁽²²⁻²⁵⁾.

Clinical presentation

HF symptoms may develop over hours, days, or weeks and usually prompt patients to seek medical attention. Some patients with established HF experience decompensation, termed acute decompensated HF, whereas others develop manifestations for the first time during pregnancy, termed de novo acute HF.

Physiological changes during pregnancy can cause symptoms such as exertional dyspnoea and oedema, which are also cardinal symptoms of HF, making diagnosis challenging. Clinical assessment should therefore be systematic, with a thorough history addressing cardiovascular risk factors, pre-existing heart disease, a history of pre-eclampsia, symptom onset and progression, and the presence of palpitations or syncope. Particular attention should be paid to HF symptoms unlikely to be explained by pregnancy alone, such as paroxysmal nocturnal dyspnoea, orthopnoea, and bendopnoea⁽¹³⁾. A careful physical examination is also required to identify signs of congestion or HF, including abnormal vital signs, a narrow pulse pressure, chest retractions, jugular venous distension, a positive abdominojugular reflux, ascites or dependent oedema, a leftward-displaced point of maximal impulse, epigastric pulsations, and signs of low cardiac output. Cardiac auscultation may reveal a third heart sound (S3) or murmurs⁽²⁶⁾. Fetal wellbeing should also be assessed clinically.

Diagnostic investigations

Electrocardiography: pregnancy produces several changes in the electrocardiographic tracing. Expected variations include a 15-20° leftward axis shift, repolarisation changes involving the ST segment and T wave, a Q wave and inverted T waves in lead III, an attenuated Q wave in lead aVF, and inverted T waves in leads V1, V2, and V3. Electrocardiography is considered safe for both mother and fetus^(27,28).

Chest radiography: this should be reserved for patients in whom the cause of dyspnoea remains unclear after the initial clinical assessment. The fetal radiation dose from a chest radiograph is < 0.01 mGy; however, its use should be limited

where possible. If essential, the recommendation is to perform the examination with lead shielding after discussing the risks and benefits with the patient. The radiation dose used should be documented ⁽²⁹⁾.

Echocardiography: this is the investigation of choice during pregnancy because of its availability and absence of ionising radiation. It allows assessment of congenital defects, valvular function, the pericardium, the great vessels, and LVEF, measured using three-dimensional imaging or, if unavailable, the biplane Simpson method. LVEF allows HF to be classified as HF with reduced ejection fraction ($\leq 40\%$), mildly reduced ejection fraction (40-49%), preserved ejection fraction ($\geq 50\%$), or improved ejection fraction (an initial LVEF $\leq 40\%$, followed by an increase of 10 percentage points and a final LVEF $> 40\%$). This classification is essential to guide treatment. According to European guidelines, echocardiography should be prioritised within the first 48 hours in all pregnant patients with suspected acute HF and performed emergently in cases of acute pulmonary oedema or cardiogenic shock ^(2,30).

A comprehensive echocardiographic assessment should quantify LVEF and ventricular volumes and assess diastolic function to evaluate ventricular stiffness and relaxation using tissue Doppler and transmitral flow measurements, including E and e' velocities and the E/e' ratio. Pulmonary artery systolic pressure should also be estimated from the tricuspid regurgitation velocity to assess for pulmonary hypertension, and pericardial effusion should be excluded ^(2,30-33). Transoesophageal echocardiography and submaximal exercise echocardiography are safe during pregnancy and are not associated with an increased risk of miscarriage ⁽²⁾.

In recent years, point-of-care ultrasound (PoCUS) has been used to support clinical decision-making through protocols such as RUSH, BLUE, and VExUS; however, these protocols have not been validated in pregnant populations. Pregnancy-specific focused cardiac ultrasound (FoCUS) protocols, such as the MaatHRI protocol, have been developed to provide qualitative bedside assessment of cardiac function and expedite some clinical decisions ^(31,32). Nevertheless, broader population-based validation is required before their use can be generalised.

Natriuretic peptides: plasma concentrations are used as an initial diagnostic test when assessing dyspnoea of suspected cardiac origin, particularly when echocardiography is unavailable, to identify patients who would benefit from further investigations ⁽⁶⁾. For the assessment of acute HF in the emergency department, the European Society of Cardiology (ESC) recommends thresholds of < 100 pg/mL for B-type natriuretic peptide (BNP) or < 300 pg/mL for N-terminal pro-B-type natriuretic peptide (NT-proBNP) to rule out HF in patients older than 18 years ⁽³⁰⁾. The latest ESC guidelines on cardiovascular disease and pregnancy adopt these same thresholds for pregnant women, as pregnancy does not substantially alter these concentrations, preserving their negative predictive value ⁽²⁾. Labour and pre-eclampsia

may cause mild elevations. However, in an appropriate clinical context, markedly elevated concentrations support the diagnosis of HF. Studies derived from the CARPREG and ZAHARA cohorts have therefore proposed NT-proBNP concentrations > 200 pg/mL as an independent predictor of HF decompensation ⁽³³⁾. Measurement is not recommended in asymptomatic patients. By contrast, in patients with established heart disease, preconception measurement and periodic monitoring during pregnancy are recommended to identify the risk of decompensation ⁽²⁾.

Other biomarkers: biomarkers such as the 16-kDa prolactin fragment and sFlt-2 have been proposed for the diagnosis of PPCM; however, further studies are needed to establish their routine use ⁽³⁴⁾. HF can cause non-ischaemic troponin elevation; therefore, routine troponin measurement is not recommended in this setting, and results should be interpreted in the clinical context. Other diagnostic investigations should be requested at the clinician's discretion.

Treatment

The treating team should promptly assess and address the most important clinical issues in pregnant patients with acute HF.

1. Does the patient have cardiogenic shock or respiratory failure?

Cardiogenic shock and respiratory failure must be recognised promptly, as both threaten maternal and fetal wellbeing and require aggressive treatment. Management in an intensive care unit (ICU), with continuous monitoring, is necessary to achieve rapid stabilisation. Pregnancy-specific considerations regarding ventricular assist devices and extracorporeal membrane oxygenation are beyond the scope of this review; readers are referred to the relevant literature. Indications for ICU admission include endotracheal intubation, signs or symptoms of hypoperfusion, oxygen saturation (SpO_2) $< 90\%$ despite supplemental oxygen, a respiratory rate > 25 breaths/min, a heart rate < 40 or > 130 beats/min, systolic blood pressure < 90 mmHg, or any concomitant condition requiring close monitoring at the clinician's discretion. In the absence of these features, care could theoretically continue on a general ward. However, given the severity of potential outcomes and the high risk of sudden deterioration, the authors suggest that all pregnant patients with acute HF receive monitoring in at least a high-dependency unit under the care of a multidisciplinary team.

2. What is the precipitating cause of acute HF?

The initial assessment should identify precipitating factors requiring additional, specific treatment to achieve stabilisation. The ESC proposes the CHAMPIT mnemonic: acute Coronary syndrome, Hypertensive emergency, Arrhythmias, acute Mechanical complications, Pulmonary embolism, Infection, and cardiac Tamponade. Pregnancy increases the

risk of hypertensive disorders, arrhythmias, and thrombosis, in addition to the causes described in the aetiology section ⁽³⁰⁾.

3. What is the clinical presentation of acute HF?

The ESC HF guidelines classify acute presentations as follows:

- A. Acute decompensated HF.
- B. Acute pulmonary oedema.
- C. Isolated right ventricular failure.
- D. Cardiogenic shock.

The main characteristics and pregnancy-specific management considerations for each presentation are summarised in **Table 1** ⁽³⁰⁾.

Loop diuretics are the cornerstone of treatment in the first three scenarios.

For pregnant patients not previously receiving furosemide, the ESC guidelines, drawing on findings from the DOSE trial, recommend an initial bolus of 20–40 mg,

with subsequent treatment adjusted according to urine output, urinary sodium, and symptom relief, favouring bolus administration over continuous infusion ⁽³⁰⁾. If the response remains insufficient after furosemide titration, a thiazide-type diuretic may be added, as both classes have demonstrated safety during pregnancy. The ESC guidelines on cardiovascular disease and pregnancy recommend strictly avoiding “triple blockade” because of the harmful fetal effects of potassium-sparing diuretics ^(30,35).

Although diuretic use during pregnancy has been associated with fetal growth restriction (FGR) due to reduced perfusion, recent ROPAC data did not identify this association after adjustment for other variables. This finding suggests confounding by indication, whereby FGR may be attributable to the underlying condition prompting diuretic treatment rather than to the medication itself. These findings do not apply to mineralocorticoid receptor antagonists (MRAs), which are contraindicated during pregnancy, or to acetazolamide, whose safety during pregnancy is unknown ⁽³⁶⁾.

Table 1. Characteristics of acute heart failure presentations and pregnancy-specific considerations.

	Acute decompensated heart failure	Acute pulmonary oedema	Isolated right ventricular failure	Cardiogenic shock
Main mechanism	Left ventricular dysfunction and renal sodium and water retention. Exacerbating factors: physiological changes of pregnancy, including increased plasma volume and cardiac output requirements. Postpartum: autotransfusion.	Increased afterload or predominant diastolic dysfunction. Valvular heart disease. Increased capillary permeability during pregnancy.	Right ventricular dysfunction or precapillary pulmonary hypertension. Precipitating factor: increased risk of embolic events during pregnancy.	Severe cardiac dysfunction. Predisposing factors: increased afterload and reduced preload due to uterine compression of the aorta and inferior vena cava, respectively.
Main cause of symptoms	Fluid accumulation. Increased intravascular pressure.	Fluid redistribution to the lungs and acute respiratory failure.	Increased central venous pressure and systemic hypoperfusion.	Systemic hypoperfusion.
Onset	Gradual (days).	Rapid (hours).	Gradual or rapid.	Gradual or rapid.
Main clinical profile	Wet and warm or wet and cold.	Wet and warm.	Wet and cold.	Wet and cold.
Main treatment	Diuretics: furosemide and hydrochlorothiazide. Spironolactone is contraindicated. Inotropes, vasopressors, or circulatory support may be required if cardiogenic shock develops.	Diuretics: furosemide and hydrochlorothiazide. Spironolactone is contraindicated. Early non-invasive ventilation; assess the need for invasive ventilation. Vasodilators: nitroglycerin is not contraindicated. Avoid sustained hypotension.	Diuretics: furosemide and hydrochlorothiazide. Spironolactone is contraindicated. Inotropes: levosimendan is preferred. Withholding beta-blockers is suggested. Vasopressors: noradrenaline is preferred. Mechanical circulatory support. Renal replacement therapy.	Inotropes: levosimendan is preferred. Withholding beta-blockers is suggested. Vasopressors: noradrenaline is preferred. Mechanical circulatory support. Renal replacement therapy.

Adapted from the 2021 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure ⁽³²⁾, incorporating pregnancy-specific considerations ⁽¹⁻³⁾.

When vasopressors and/or inotropes are required, the ESC guidelines suggest temporarily withholding beta-blockers and favouring noradrenaline over dopamine, based on evidence from the general population; pregnancy-specific data are unavailable⁽³⁷⁾. The ESC also recommends using vasopressor support for the shortest possible duration, with monitoring of fetal wellbeing, given its potential effects on uteroplacental circulation. No differences in efficacy have been established between inotropes; however, the ESC suggests levosimendan

on the basis of expert opinion, given the lack of evidence during pregnancy^(2,38). The assessment and management of HF in these patients are summarised in **Figure 1**.

For chronic management, several components of guideline-directed medical therapy (GDMT) for HF are contraindicated during pregnancy, including angiotensin-converting enzyme inhibitors (ACE inhibitors), angiotensin II receptor blockers (ARBs), angiotensin receptor-neprilysin inhibitors (ARNIs), MRAs, and sodium-glucose cotransporter

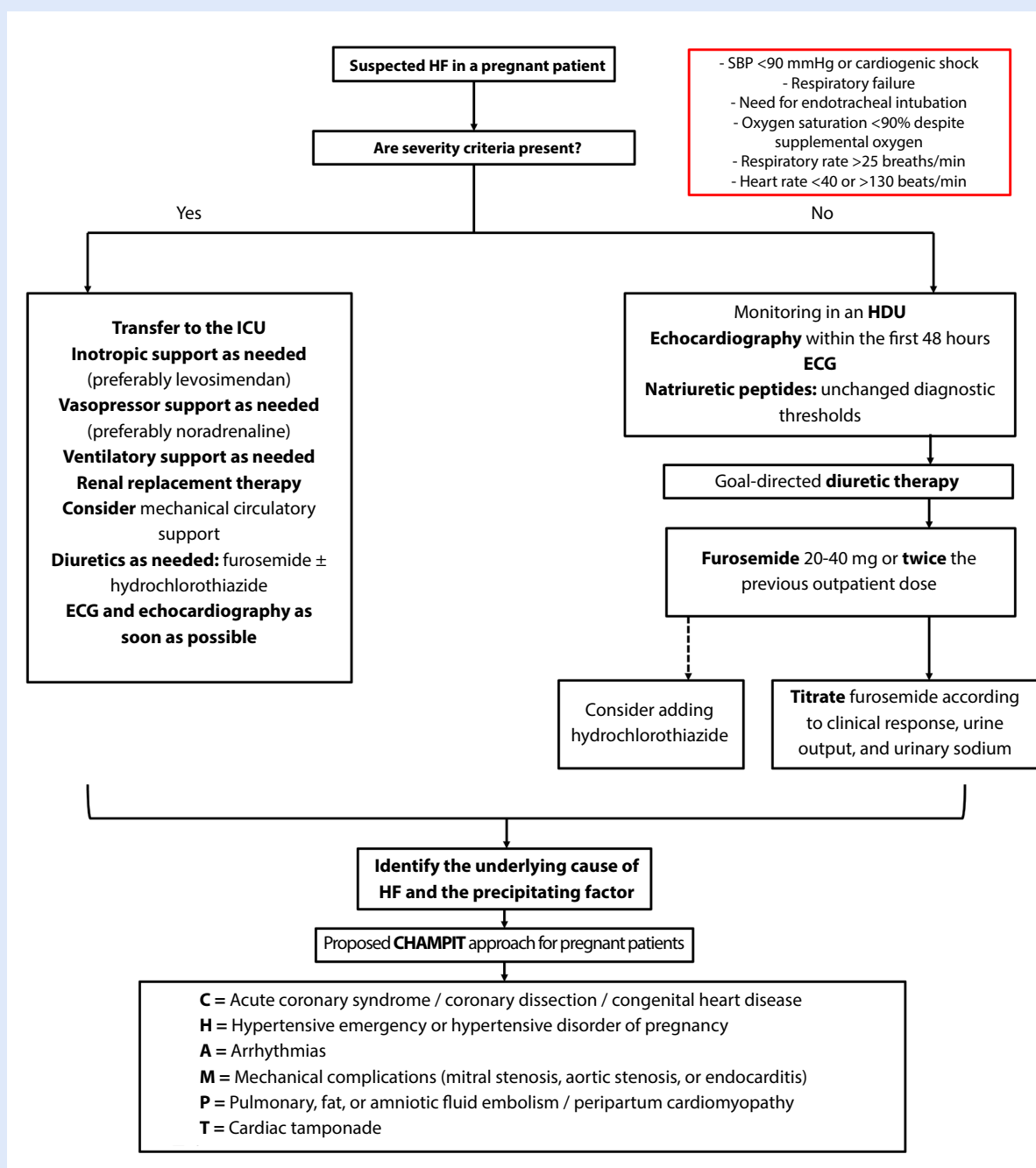


Figure 1. Algorithm for the initial assessment and management of heart failure in pregnant women.

HF: heart failure; SBP: systolic blood pressure; breaths/min: breaths per minute; beats/min: beats per minute; ICU: intensive care unit; HDU: high-dependency unit.

2 (SGLT2) inhibitors ⁽³⁵⁾. Although initial case reports linked beta-blocker use during pregnancy to FGR and neonatal hypoglycaemia, these associations have not been confirmed in studies with greater statistical power, and beta-blockers have been shown to be safe during pregnancy, with the most extensive evidence available for metoprolol succinate ^(2,35). ACE inhibitors and MRAs may be initiated during breastfeeding. However, the ESC recommends consulting medication safety and adverse-event reporting resources before prescribing any medication during lactation.

The guidelines recommend continuing GDMT after LVEF recovery because of the high risk of relapse ^(2,30). If the patient wishes to become pregnant again, clinical practice guidelines recommend maintaining GDMT at the maximum tolerated doses for at least one year, followed by gradual withdrawal of medications contraindicated during pregnancy ^(2,30).

For PPCM, given the important role of prolactin in its pathophysiology, prolactin antagonists have been proposed as treatment. However, their benefit has not been conclusively established because of the absence of statistically significant differences in primary outcomes and a substantial increase in embolic events. The 2025 ESC guidelines assign their use a class IIb recommendation and do not recommend routine administration ^(2,20). An ongoing randomised controlled trial (RCT) is investigating whether this intervention provides a benefit (NCT05180773). Based on the current evidence and the balance between embolic risk and uncertain benefits for clinically important outcomes, the authors suggest avoiding routine use. One RCT demonstrated that selenium reduced HF symptoms without improving other outcomes; its routine use is therefore not recommended ⁽³⁹⁾.

The safety profiles of medications commonly used during pregnancy and breastfeeding in patients with HF are described in **Table 2**.

4. What are the appropriate timing and mode of delivery?

Labour induction and subsequent monitoring require coordinated care involving cardiologists, obstetricians, and specialist anaesthesiologists in experienced maternal-fetal medicine units. No RCTs have established the ideal gestational age for induction. Delivery at 37-39 weeks has therefore been proposed for patients with HF, although this decision depends on maternal clinical status and additional indications.

Vaginal delivery is preferred because it carries lower risks of bleeding, infection, and thromboembolic events than caesarean delivery ^(40,41). Ideally, labour should begin and progress spontaneously, with induction reserved for an unfavourable Bishop score to avoid prolonged induction ^(42,43). The patient may adopt a left lateral position to attenuate the haemodynamic effects of uterine contractions. Artificial rupture of membranes and oxytocin infusion are safe in this setting. HF is not an absolute contraindication to misoprostol use.

Caesarean delivery is reserved for obstetric indications. When acute HF itself is the indication, caesarean delivery

may be considered for persistent symptoms despite optimal treatment, New York Heart Association functional class III-IV, LVEF < 20%, or hypertrophic cardiomyopathy with a left ventricular outflow tract gradient > 50 mmHg ⁽²⁾. Oxytocin and prostaglandin F analogues may be used safely to manage postpartum haemorrhage unless the patient has pulmonary hypertension.

Given the haemodynamic changes described above, close monitoring during the first 12-24 hours postpartum is essential ^(2,18,21).

Conclusions

Heart disease complicates a substantial number of pregnancies and is the leading cause of non-obstetric maternal mortality. Caring for high-risk patients, including pregnant women with acute HF, requires a collaborative approach involving obstetricians, maternal-fetal medicine specialists, cardiologists, and anaesthesiologists. This teamwork supports optimal clinical decisions for both mother and child. Pregnancy is associated with cardiovascular changes that can substantially affect underlying heart disease or lead to its clinical manifestation. Understanding these adaptations is essential to interpret maternal symptoms and conventional cardiovascular investigations, anticipate the effects of pregnancy in women with underlying heart disease, and understand how maternal cardiac disease may affect the fetus. Overall, the diagnostic and therapeutic approach to acute HF during pregnancy is similar to that used in the general population, with specific considerations for clinical interpretation and the use of certain treatments.

Ethical aspects

This study was conducted in accordance with the ethical principles for research involving human participants set out in the Declaration of Helsinki. Additionally, the authors declare that artificial intelligence was not used in the preparation of the manuscript.

Author contributions

EMO and JAGR: conceptualisation, methodology, software, validation, formal analysis, investigation, resources, data curation, writing-original draft, visualisation, supervision, project administration. **JPFM:** conceptualisation, validation, investigation, writing-original draft, visualisation. **EHG:** conceptualisation, validation, investigation, writing-original draft. **JAVP:** conceptualisation, investigation, data curation, writing-original draft, visualisation. **MBGS:** conceptualisation, methodology, software, validation, formal analysis, investigation, resources, data curation, writing-original draft, visualisation, supervision, project administration, writing-review and editing. **YIAE:** conceptualisation, methodology, software, validation, formal analysis, investigation, resources, data curation, writing-original draft, visualisation, writing-review and editing.

Table 2. Safety profiles of commonly used medications in patients with heart failure during pregnancy and breastfeeding*

Pharmacological class	Commonly used medications	Safety during pregnancy	Safety during breastfeeding	Additional considerations
Beta-blockers	Metoprolol succinate, bisoprolol, carvedilol	Safe	Safe	Preferred agents for chronic heart failure management during pregnancy. Atenolol is contraindicated during pregnancy because of the risk of fetal growth restriction (FGR). Recent studies have not confirmed the risks of FGR and neonatal hypoglycaemia associated with other beta-blockers.
Angiotensin-converting enzyme inhibitors	Enalapril, lisinopril, ramipril	Absolute contraindication	Safe	Severe fetotoxicity has been documented across all trimesters. Associated with oligohydramnios, pulmonary hypoplasia, irreversible neonatal renal injury, and fetal death. Minimal excretion into breast milk.
Angiotensin II receptor blockers	Losartan, valsartan, candesartan	Absolute contraindication	Safe	Toxicity profile similar to that of angiotensin-converting enzyme inhibitors. Minimal excretion into breast milk. Most available data concern candesartan and valsartan.
Angiotensin receptor-neprilysin inhibitors	Sacubitril/valsartan	Absolute contraindication	Limited evidence	Because the combination contains valsartan, its toxicity profile during pregnancy is similar. Current evidence is insufficient to recommend its use during breastfeeding; it appears safe, but this requires confirmation in further studies.
Sodium-glucose cotransporter 2 inhibitors	Empagliflozin, dapagliflozin	Not recommended	Not recommended	Human data during breastfeeding are limited. Safety during pregnancy has not been established; use is not recommended.
Mineralocorticoid receptor antagonists	Spirolactone, eplerenone, finerenone	Absolute contraindication	Spirolactone is safe. Eplerenone and finerenone are not recommended.	Fetal antiandrogenic effects have been demonstrated; use is contraindicated during pregnancy. Spirolactone is minimally excreted into breast milk and may be used during breastfeeding. Insufficient information is available on the safety of eplerenone and finerenone during breastfeeding; their use is not recommended.
Loop diuretics	Furosemide	Safe	Safe	Agent of choice for decongestion during pregnancy and breastfeeding. Recent studies have not demonstrated an association with FGR, as suggested by earlier studies. Nevertheless, use requires caution and medical supervision.
Thiazide diuretics	Hydrochlorothiazide, chlortalidone, indapamide	Safe	Safe	Second-line agents for decongestion during pregnancy. Recent studies have not demonstrated an association with FGR. Nevertheless, use requires caution and medical supervision.
Carbonic anhydrase inhibitors	Acetazolamide	Not recommended	Not recommended	Human data during breastfeeding are limited. Safety during pregnancy has not been established; use is not recommended.
Dopamine agonists-prolactin inhibitors	Bromocriptine, cabergoline	Routine use for peripartum cardiomyopathy (PPCM) is not recommended.	Contraindicated	Current evidence suggests that use during pregnancy for PPCM is not associated with differences in clinically important outcomes. Increased embolic events have been reported during pregnancy and the puerperium. The 2025 ESC guidelines do not recommend routine use in this setting. Contraindicated during breastfeeding because these agents suppress lactation.
Mineral supplements	Selenium	Safe	Safe	Safe during pregnancy and breastfeeding. Use in PPCM has been associated with improvement in heart failure symptoms without benefits for other clinically relevant outcomes. The 2025 ESC guidelines do not recommend routine use in this setting.
Oxytocics	Oxytocin	Safe	Safe	Use is not contraindicated when required. Bolus administration is suggested over infusion to avoid hypotension and reflex tachycardia. Continuous monitoring and assessment of fetal wellbeing are required.
Ergot alkaloids	Methylergometrine	Absolute contraindication	Absolute contraindication	Not recommended in patients with heart failure because of the risk of decompensation due to increased afterload, coronary vasospasm, and pulmonary oedema.
Prostaglandin analogues	Misoprostol	Safe	Safe	Use is not contraindicated when required. Continuous monitoring and assessment of fetal wellbeing are required.

Prepared by the authors using data from references 2, 4, 20, and 32. *The table was compiled using information available from <https://www.e-lactancia.org/> and LactMed®, accessed on 3 June 2026. The 2025 ESC guidelines on cardiovascular disease and pregnancy recommend consulting an up-to-date resource to confirm medication safety before prescribing during pregnancy or breastfeeding.

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