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REVIEW ARTICLE

Cardiogenic shock in pregnancy

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Abstract

Cardiac disease complicates 1%–4% of pregnancies globally, with a predominance in low and middle-income countries (LMICs). Increasing maternal age, rates of obesity, cardiovascular comorbidities, pre-eclampsia and gestational diabetes all contribute to acquired cardiovascular disease in pregnancy. Additionally, improved survival in congenital heart disease (CHD) has led to increasing numbers of women with CHD undergoing pregnancy. Implementation of individualised care plans formulated through pre-conception counselling and based on national and international guidance have contributed to improved clinical outcomes. However, there remains a significant proportion of women of reproductive age with no apparent comorbidities or risk factors that develop heart disease during pregnancy, with no indication for pre-conception counselling. The most extreme manifestation of cardiac disease is cardiogenic shock (CS), where the primary cardiac pathology results in inadequate cardiac output and hypoperfusion, and is associated with significant mortality and morbidity. Key to management is early recognition, intervention to treat any potentially reversible underlying pathology and supportive measures, up to and including mechanical circulatory support (MCS). In this narrative review we discuss recent developments in the classification of CS, and how these may be adapted to improve outcomes of pregnant women with, or at risk of developing, this potentially lethal condition.

KEYWORDS

cardiac disease, cardiogenic shock, extracorporeal membrane oxygenation (ECMO), maternal mortality, mechanical circulatory support, peripartum cardiomyopathy, pre-conception counselling, pregnancy, spontaneous coronary artery dissection, The Society of Cardiovascular Angiography and Interventions (SCAI) classification

1 | INTRODUCTION: CARDIAC DISEASE IN PREGNANCY

The improvement in maternal mortality rates seen in some developed countries in recent years may be attributable to the implementation of clinical guidance developed with feedback from outcomes observed from national obstetric surveillance systems.[1–6] Although the proportions of women dying or seriously injured from obstetric conditions such as pre-eclampsia and postpartum haemorrhage has steadily fallen, the mortality and morbidity from non-obstetric pathologies, such as cardiac disease, has not seen the same pattern of decline.[6]

In the UK, cardiac disease is a leading cause of maternal mortality, with 23% of all maternal deaths between 2016 and 2018 caused by maternal cardiac conditions.[7] In the past, this demographic was dominated by women with congenital heart disease (CHD). However, as advances have been made, both in corrective cardiac surgery and in multidisciplinary preconception and antenatal management planning, successful outcomes for mother and baby are now more common, coinciding with increased support from

KEY CLINICAL POINTS

- Cardiac disease is a prominent cause of maternal mortality globally and can lead to cardiogenic shock.
- The majority of cardiac deaths in pregnancy are due to acquired heart disease – vigilance is required for "at risk women" which includes those with advanced maternal age, obesity, diabetes mellitus and hypertensive disorders, including pre-eclampsia.
- Evaluation of cardiorespiratory symptoms in women without known cardiovascular risk factors is just as critical - 3 out of 4 women who suffered cardiac deaths in the UK between 2015 and 2017 had no known risk factors.
- In women presenting with cardiorespiratory symptoms, exclusion of a narrow differential (e.g., venous thromboembolism, arrhythmia or acute myocardial infarction) alone is inadequate and appropriate clinical assessment, investigation and specialist referral should be undertaken in order to reach a definitive diagnosis.
- Adapted for pregnancy, the SCAI classification is a clinical tool that can be used for the identification and management of the pregnant woman at risk of developing, or who develops, cardiogenic shock.
- The SCAI classification can be used to facilitate:
  - o Timely escalation to optimal intervention
  - o Effective communication with cardiologists and cardiac intensivists

FUTURE RESEARCH

- Implementation of a national registry of women in pregnancy managed with mechanical circulatory support.
- An observational study of timing of delivery and clinical outcomes in pregnant women and their babies in women receiving mechanical circulatory support.
- An observational study of ECMO management protocols for women in pregnancy: clinical outcomes and complications.
- National reporting system via "app" and audit of cardiorespiratory symptoms in pregnancy: subsequent referral, investigation, management and outcomes or diagnosis.
- Retrospective and prospective validation studies to evaluate the performance of modified SCAI parameters in pregnancy, recruiting from UK and international ECMO centres.
- Longitudinal observational study of spontaneous coronary artery dissection (SCAD) in women of reproductive age: Referral to pre-conception counselling &/ or to the maternal medicine multi-disciplinary team during pregnancy as well as clinical and pregnancy outcomes.

specialist multidisciplinary teams in tertiary centres in developed countries.[8,9] Indeed, maternal mortality data from the United Kingdom Obstetric Surveillance System (UKOSS) reveals that the majority of maternal deaths due to cardiac disorders are now associated with acquired cardiac disease.[7]

2 | CARDIOGENIC SHOCK

Cardiogenic shock (CS) is a clinical syndrome characterised by inadequate cardiac output caused by left, right or biventricular failure that results in organ hypoperfusion, and is associated with up to a 40% 30-day in-hospital mortality rate.[10,11]

3 | CARDIOGENIC SHOCK IN PREGNANCY

The precise pathophysiology of CS is poorly understood, and this is further compounded by the heterogeneity of the underlying aetiologies.[12] In pregnancy, precipitants of CS may fall into four broad categories of predictable risk (Figure 1). Unpredictable cases may arise without warning (de novo), because of the absence of known cardiovascular risks, or in those with known cardiovascular (CV) comorbidities; cases are more predictable in those with pre-existing cardiac disease. Those with pre-existing cardiac pathology may be further categorised as acute decompensated disease or those with a predictable deterioration in CV function (such as women with dilated cardiomyopathy or pulmonary arterial hypertension). Underlying aetiologies, however, are diverse, and include peripartum cardiomyopathy (PPCM), amniotic fluid embolism (AFE), spontaneous coronary artery dissection (SCAD) and arrhythmias (Figure 2).

4 | UNPREDICTABLE CS IN PREGNANCY: de novo

The 'Mothers and babies: reducing risk through audits and confidential enquiries' (MBRRACE) report of 2015–2017 revealed that three-quarters of women who died had no prior knowledge of having underlying heart disease. Notably, a number of the women that suffered cardiac death in pregnancy presented with symptoms that were either attributed to pregnancy or were not fully investigated. Finally,

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Cardiogenic shock risk category	UNPREDICTABLE		PREDICTABLE	
	Unknown	Known	Known	Known
Cardiogenic shock category	De novo	Cardiovascular comorbidities	Acute decompensated	Expected deterioration in cardiovascular function
Pathology	Pulmonary embolism Amniotic fluid embolism Venous air embolism Arrhythmias Peripartum cardiomyopathy	High BMI Hypertension Coronary heart disease Diabetes mellitus Pre-eclampsia Family history of cardiovascular disease Ethnicity	Ischaemic heart disease Corrected congenital cardiac disease	Acute coronary heart disease Pulmonary hypertension Dilated cardiomyopathy with reduced LV function

FIGURE 1 Categories of predictability of risk associated with the underlying cause of cardiogenic shock in pregnant and recently pregnant women (created with BioRender.com).

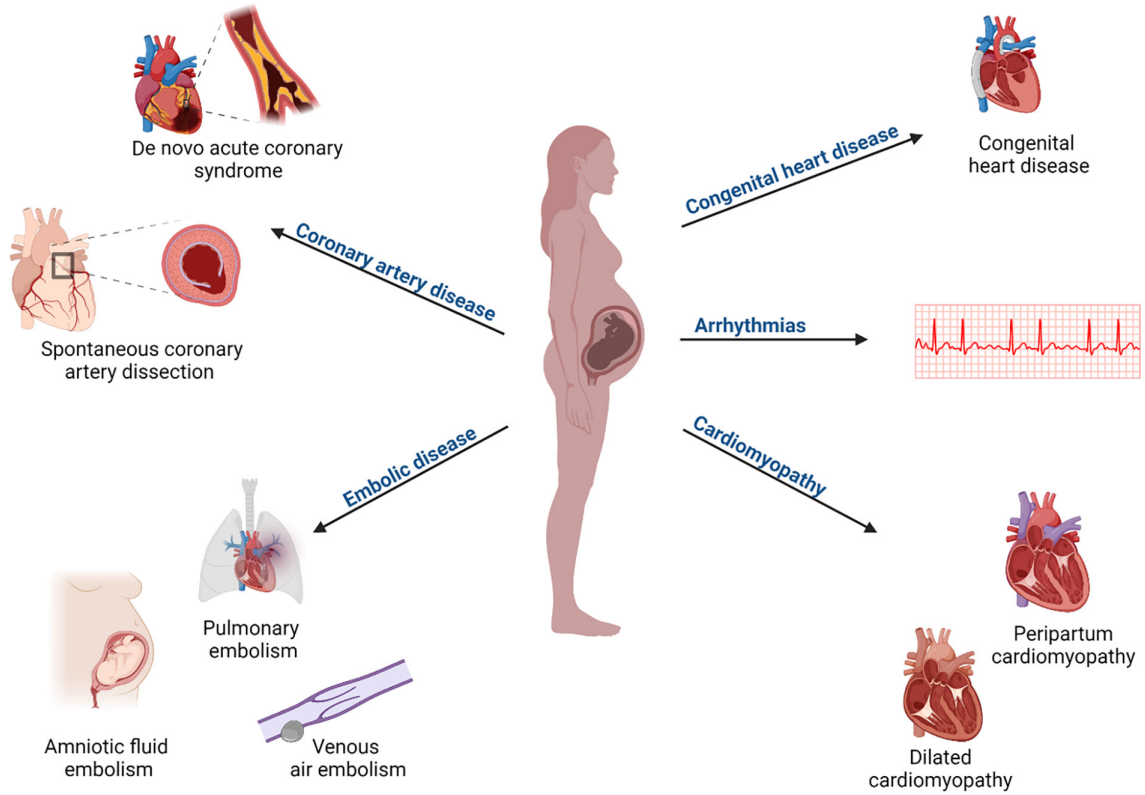

FIGURE 2 Aetiology of cardiogenic shock affecting women during pregnancy and the puerperium (created with BioRender.com).

the report highlighted that, when investigations were undertaken, there was a focus on the exclusion of high-risk diagnoses rather than on attaining a definitive diagnosis. Here, we emphasise the importance of acting on the consequent recommendations of the report, which included implementing processes to improve the recognition of cardiac disorders in pregnant women as well as counselling women to escalate their care to an appropriate clinician if they experience chest pain, palpitations or have a family history of cardiac disease, particularly that of sudden cardiac death before the age of

40 years.[13] In this review, we aim to provide a clinical toolkit for the initial management and escalation of acute decompensation of such cardiac disorders, which may arise in this group of women.

4.1 | Peripartum cardiomyopathy

The incidence of PPCM is estimated to be 1–100 in 10 000 live births, although significant geographical variation exists, compounded by differences in disease definition.[14,15] Reports of recent increases in incidence, in association with a mortality rate at 1 year of 5%–25%, emphasise the importance of raising awareness of the condition.[16,17] A multifactorial aetiology with genetic and environmental (including hormonal) components is likely, with risk factors including advanced maternal age, multiparity, multiple pregnancy and hypertensive disorders of pregnancy, such as pre-eclampsia. Although women of all ethnicities are affected, the worst maternal and neonatal outcomes have been observed in women in the Middle East.[14–16,18–20]

A diagnosis of exclusion, PPCM typically occurs in the latter part of pregnancy and up to 6 months postpartum.[21] It is categorised as a dilated cardiomyopathy leading to left ventricular (LV) systolic dysfunction in association with an LV ejection fraction of <45%–50%, depending on the clinical picture.[22] It can result in CS when the LV function is inadequate to perfuse the organs. The pathophysiology remains an area of continuing research, but the potential for an underlying genetic predisposition has involved analysis for associations with truncating loss-of-function variants in the *TTN* gene (*tTTN*), coding for titin.[23,24] This protein is fundamental for myofibrillar stability in cardiac myocytes, and has been associated with familial primary dilated cardiomyopathies.[25,26] Ware et al. sequenced 43 genes associated with dilated cardiomyopathy, including *tTTN*, and mutations in this gene were detected in 10% of cases of PPCM, compared with 1.4% of cases in the control reference data of more than 60 000 population samples.[27] Despite this association, a causal relationship remains to be determined. It is possible that, in some pregnant women, a combination of a genetic predisposition (such as a mutation in the *TTN* gene) alongside the cardiovascular effects of certain pregnancy hormones may increase the risk of LV dysfunction. For example, in variable concentrations, prolactin can regulate the contraction and relaxation of vascular smooth muscle, thus modulating vascular tone. In animal models, excess oxidative stress is postulated to mediate the upregulation of prolactin cleavage factors, leading to an increase in the concentration of a prolactin isoform with anti-angiogenic and apoptotic properties.[28,29] This isoform has been associated with dysfunctional cardiometabolism, destruction of the cardiac microvasculature and impaired cardiac muscle contraction.[30,31] The inhibition of anti-angiogenic molecules can be achieved by the administration of bromocriptine, a dopamine D2 agonist that inhibits prolactin synthesis and exocytosis from the anterior pituitary lactotrophic cells. In a cardiomyocyte-specific *STAT3* gene knockout mouse model, the administration of bromocriptine demonstrated lower mortality ($p < 0.01$) in association with the prevention of cardiac fibrosis.[29] Bromocriptine has been used therapeutically in selected cases and in small studies in humans.[29,32–34] In one small randomised study (10 cases and 10 controls), a greater recovery of the LV ejection fraction ($p = 0.012$) and a trend towards lower mortality was demonstrated by 6 months in women treated with bromocriptine in addition to standard care for PPCM, compared with standard care alone.[30] Further large randomised controlled trials evaluating clinical outcomes with bromocriptine therapy, in addition to standard care for PPCM, might provide additional evidence of benefit.[35,36] Anti-angiogenic imbalance, also implicated in the pathophysiology of pre-eclampsia, may underlie the susceptibility of women with pre-eclampsia to develop PPCM. In pre-eclamptic pregnancies, elevated levels of placentally derived soluble fms-like tyrosine kinase-1 (sFlt-1) are present in the circulation. As an inhibitor of vascular endothelial growth factor (VEGF), sFlt-1 is anti-angiogenic and cardiotoxic (promoting endothelial cell dysfunction and apoptosis), and although potentially sufficient to cause heart failure alone, sFlt-1 may contribute to the multifactorial pathogenesis of PPCM, as seen with the elevated levels of sFlt-1 achieved in an anti-angiogenic mouse model, using gene knockout of the pro-angiogenic molecule peroxisome proliferator-activated receptor-γ coactivator 1α (PGC-1α), which were associated with cardiac failure to a greater extent in the knockout than in the wild-type mice.[20,37]

4.2 | Amniotic fluid embolism

In cases of AFE, CS arises from an anaphylactoid reaction to amniotic fluid, lanugo, vernix or meconium in the maternal circulation, which is purported to trigger a cascade of events that can culminate in the acute onset of cardiorespiratory arrest and disseminated intravascular coagulopathy.[38,39] Pulmonary arterial vasospasm is understood to lead to right ventricular dysfunction and dilatation, which impinges on the left ventricle, affecting systolic function and cardiac output.

Amniotic fluid embolism (AFE) is rare and was the cause of death in six women (0.26 in 100 000 maternities) during 2015–2017 in the UK.[6] Less frequently seen, venous air embolism (VAE) may present with chest pain and breathlessness and has been associated in the past with 1% of maternal deaths. VAE results from the inadvertent introduction of air into the venous circulation, in association with a negative pressure gradient, and large volumes of gas may lead to pulmonary arterial hypertension and right-sided cardiac strain.

The diagnosis is often clinical, although precordial Doppler studies or transoesophageal echocardiogram may identify intracardiac VAE. Alternatively, end-tidal volume or carbon dioxide/nitrogen level may support the diagnosis. Uterine exteriorisation, the Trendelenburg position, uterine rupture and positive pressure ventilation have been postulated as risk factors.[40,41]

Some CS aetiologies that occur in the general population can be expected to be notable causes of CS in pregnancy.[12] Thromboembolism risk is increased four- to five-fold in pregnancy (physiologically a prothrombotic state), and this is further increased in the postpartum period.[42] In a similar fashion to the mechanism of action leading to CS in AFE, a massive thrombus within the pulmonary vasculature triggers the release of vasoconstrictive mediators that lead to an increase in right ventricular afterload.[43] Increased right ventricular pressures impinge on the left ventricle, diminishing output.

5 | UNPREDICTABLE CS IN PREGNANCY: KNOWN COMORBIDITIES

Detailed antenatal plans are common amongst women with pre-existing comorbidities and, in the absence of routine pre-conception counselling, antenatal clinics provide an opportunity to reinforce lifestyle advice for those with cardiovascular risk factors. With the combination of pregnancy at advanced ages and the use of assisted reproductive techniques, women with existing comorbidities such as hypertension, type 2 diabetes mellitus and obesity are conceiving more commonly. Equally, comorbidities such as hypertension, diabetes and cardiovascular disorders in older mothers may be unmasked during pregnancy. It is rare for women to enter pregnancy with a history of ischaemic cardiovascular disease, but more commonly women will have known CV risk factors for ischaemic pathology, such as obesity and advanced maternal age.[44] Globally, obesity rates are rising in the general population, including amongst women of reproductive age.[45] This trend has also been seen in the UK, with a reported prevalence of 21% amongst pregnant women.[46,47] Obesity, associated with the development of metabolic disorders such as dyslipidaemia and diabetes, predisposes women to CV disease and premature death. In a UK study, a three-fold increased risk of myocardial infarction (MI) in pregnant women with a BMI greater than 30 kg/m^2 has been reported.[48–50] Likewise, this trend was seen in advanced maternal age, and there was an increased association of MI in women carrying multiple pregnancies and in women that had developed pre-eclampsia.

Establishing patient pathways for this 'at risk' group of women (with known CV risk factors) is required to improve identification and the timely referral to an appropriate clinician; the objective being to assess and mitigate for the risks that could result in cardiovascular disease in pregnancy.

6 | PREDICTABLE RISK FACTORS FOR CS IN PREGNANCY: ACUTE DECOMPENSATED/ EXPECTED DETERIORATION

For patients known to be at risk of developing CS during pregnancy, established risk stratification models, such as the CARdiac disease in PREGnancy risk scores I and II (CARPREG I and II), Zwangerschap bij Aangeboren HARtAfwijkingen I (ZAHARA) and the Modified World Health Organization Classification of Maternal Cardiovascular Risk (mWHO), have been developed to aid pregnancy counselling and management in women with known congenital and acquired cardiac disease. All these tools are useful for predicting cardiac events in these women.[51–54] As a result, few women are advised to absolutely avoid pregnancy, and instead they are supported in optimising their pre-pregnancy cardiac function and receive regular clinical review during pregnancy to guide medical management and delivery.[55] However, despite an improvement in overall clinical outcomes in pregnancy for patients with known heart disease in the last decade, specific cardiac conditions still pose significant risk. Pulmonary arterial hypertension is associated with the highest maternal risk, with mortality rates of 16%–30%, and planned pregnancy is not currently recommended by the European Society of Cardiology.[2,56] Pre-existing dilated cardiomyopathy (DCM) also carries significant risk in pregnancy, with an increased risk of clinical deterioration associated with New York Heart Association (NYHA) functional classes III and IV and an ejection fraction of <40%.[2]

6.1 | Coronary artery disease

It would appear that even in acute MI-related pathology, much is to be determined regarding the underlying mechanisms that lead to CS.[57] In non-pregnant human studies, up to 50% of LV myocardial loss has been demonstrated to occur in a self-perpetuating cardiac insult that happens in the absence of medical intervention.[57] The initial cardiac insult results in reduced cardiac performance, cardiac output and organ perfusion. Cardiac performance is further impacted by an exacerbation of cardiac hypoperfusion, and this leads to an extension of the area of MI. Further decompensation may be caused by an inappropriate host response to organ hypoperfusion, with catecholamine release triggering vasoconstriction and an increase in heart rate and force of cardiac contraction, thus increasing the work of the heart.[57] Additionally, a systemic pro-inflammatory response and release of cytokines may lead to myocardial depression, compounding tissue hypoperfusion by triggering vascular dysfunction, endothelial

injury, increased capillary permeability and, conversely, vasodilation.[57]

The UKOSS data from 2005 to 2010, reviewing outcomes of women with acute coronary syndrome in pregnancy and the immediate postpartum period (1 week after delivery), reported an estimated incidence of 0.7 in 100 000 maternities (95% CI 0.5–1.1). Remarkably, of the 25 cases experiencing myocardial infarction, no women died. Although two women had no cardiac pathology, the remaining women had coronary atheromatous plaques (that required stenting), coronary thrombus or coronary artery dissection. Of note, the UKOSS report also described the variable management of acute coronary syndrome (ACS) in pregnant women, suggesting the need to improve education and the consistency of clinical care offered to these women.[50]

6.2 | Spontaneous coronary artery dissection

Among the data presented by UKOSS, it is also important to highlight the growing number of young women presenting with a history of acute CS prior to pregnancy, including SCAD. This group of women are at particularly high risk of subsequent acute cardiac decompensation during pregnancy. We propose the development of consensus guidelines and referral pathways for women of reproductive age who have had a diagnosis of SCAD, with the objective of reducing the subsequent risk of fatality during pregnancy.

It appears that SCAD occurs more commonly in women of reproductive age and is specifically associated with pregnancy. However, our understanding is limited, and the relevant literature is predominantly composed of case reports. Although the actual prevalence is uncertain, rates are reported to be between 1% and 4% of all cases of ACS.[58,59] However, SCAD may account for upto 35% of ACS in women less than 50 years of age and 43% of MIs in pregnancy.[59–62] Not surprisingly, hormonal factors are thought to be one of many risk factors that predispose individuals to the condition.

Pathways need to be put in place to ensure that these women have long-term follow-up and receive appropriate pre-pregnancy counselling.

6.3 | Pulmonary hypertension and dilated cardiomyopathy

Although there is variation in clinical outcome associated with the underlying subset aetiology, maternal mortality as a result of pulmonary hypertension is significant and has been reported to be 16%–30% (rising to 50% in the context of Eisenmenger's syndrome). Accordingly, pregnancy is contraindicated clinically and women of reproductive age are counselled regarding this risk and advised to use safe and effective contraception. In the event of conception, or where women exhibit the clinical features of pulmonary hypertension for the first time in pregnancy, termination of the pregnancy (which also requires specialist planning and input) must be discussed with the woman and her family. For continuing pregnancies, a detailed and individualised multidisciplinary plan together with close surveillance and monitoring is essential in improving outcomes, and should continue into the puerperium, the period of greatest risk.[2,63]

Dilated cardiomyopathy (DCM) can occur de novo in pregnancy but may be a pre-existing condition. The highest risk of decompensation is in patients with an ejection fraction of <20%, mitral regurgitation, right ventricular failure,

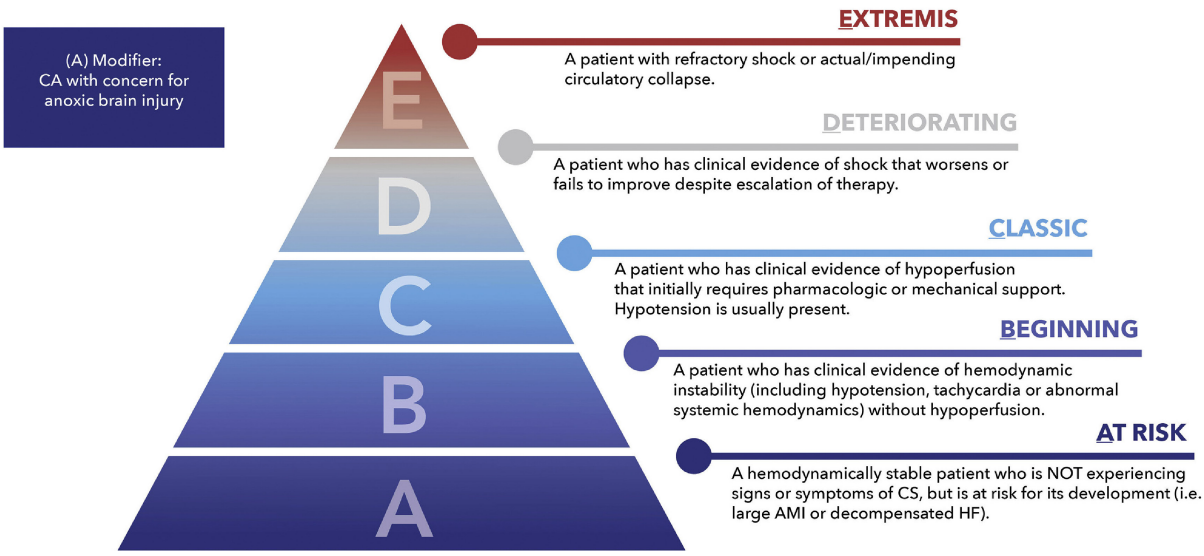

FIGURE 3 The Society of Cardiovascular Angiography and Interventions (SCAI) SHOCK classification pyramid.[65] A, arrest; AMI, acute myocardial infarction; CA, cardiac arrest; CS, cardiogenic shock; HF, heart failure.

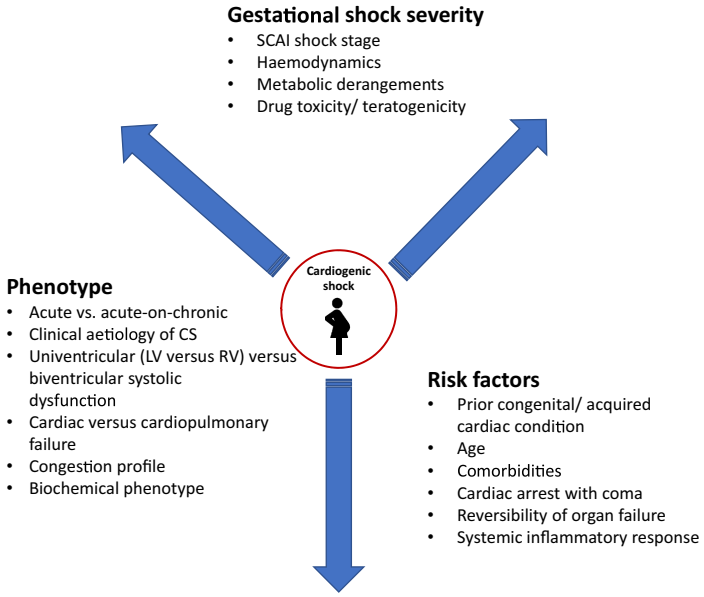

FIGURE 4 Modified aide for the evaluation and prognosis of cardiogenic shock (CS) in pregnancy (adapted from Naidu et al.[65]). LV, left ventricular; RV, right ventricular.

atrial fibrillation or hypotension, and clinical deterioration can be anticipated. Delivery of the fetus may be required to optimise haemodynamic stability.[2]

7 | CARDIOGENIC SHOCK: THE SCAI CLASSIFICATION AND ITS APPLICATION IN PREGNANT WOMEN

Patient outcomes following CS are influenced by a number of factors, including aetiology and the individual patient clinical characteristics.[64] Recently, the Society of Cardiovascular Angiography and Interventions (SCAI) classification has been incorporated into clinical practice to facilitate the evaluation of the clinical severity of CS. It can be used as a staging and communication tool to 'risk assess' patients and guide the clinical team from a stage of high vigilance and preparatory measures, for those at risk of CS, through to increasing degrees of intervention and escalation, for those in CS.[65]

The SCAI framework comprises a pyramid of incremental phases, termed A–E, associated with increasing degrees of organ dysfunction and death (Figure 3). SCAI-A identifies patients '*a*t risk' of CS, SCAI-B is attributed to early shock or the '*b*eginning' phase, SCAI-C is considered '*c*lassic' shock, and is associated with organ hypoperfusion, and SCAI-D classifies a patient who is entering a '*d*eteriorating' phase, which (if after at least 30 minutes of early resuscitative measures have been ineffective) leads to a state of SCAI-E, in '*e*xtremis'.[66] More recently, validation studies using the framework have reinforced the advantages of using the tool at the bedside, and have facilitated the development of a consensus update (published in 2021), refining mortality risk aligned with CS phenotype and presumed aetiology (Figure 4).[65] The tool can also be used in pregnancy to identify patients 'at risk' of developing CS, as well as those with evolving/established CS (Figure 1).

8 | ARGUMENT SUPPORTING THE SCAI CLASSIFICATION FOR CS IN PREGNANCY

The cardiac risk stratification tools for evaluation in pregnancy, highlighted previously, facilitate the early identification of 'at risk' patients with comorbidities or pre-existing conditions. This provides the opportunity for management planning for the antenatal, peripartum and postpartum periods, and (as appropriate) cardiology referral and investigations. In pregnancy, the SCAI classification can be modified using pregnancy-specific reference ranges for haemodynamic parameters and biomarkers to determine the SCAI stage (Table 1).

Initial management for mild to moderate hypotension in CS (SCAI-B) is probably more frequently encountered in smaller units, with most pregnant women with pre-existing cardiac disorders being referred to tertiary centres for their care. Using established criteria for CS, such patients may subsequently develop hypoperfusion associated with hypotension and elevated LV filling pressures, and will likely require careful fluid management, vasopressors and potentially careful and transient inotropes, supported by cardiac and intensivist teams (SCAI-C).[67] In pregnancy the general principles of management of CS are the same as for non-pregnant patients, with the focus being on the restoration of perfusion.[31] In PPCM, one of the most common

TABLE 1 Determination of SCAI classification by clinical assessment, biomarkers and haemodynamic parameters (adapted from Naidu et al.[65]).

		Physical examination		Biochemical markers[a]		Haemodynamics	
Stage	**Description**	**Typically includes**	**May include**	**Typically includes**	**May include**	**Typically includes**	**May include**
A. At risk	A patient who is not currently experiencing signs or symptoms of CS, but is at risk for its development (e.g. prior acute coronary event or SCAD)	Normal JVP Warm and well-perfused Strong distal pulses Normal mental state	Clear lung sounds	Normal lactate[a] (consider context, as can be elevated in normal labour)[75,76] (<2 mmol/L outside of labour; commonly, ≥2 mmol/L in labour and can be ≥4 mmol/L)	Normal laboratory results[74,a] Normal (or equivalent to baseline) renal function[74]	Normotensive (SBP ≥ 90 mmHg or at baseline)	If invasive haemodynamics assessed: Cardiac index ≥ 2.5 L/min/m^2 (if acute) CVP ≤ 10 mmHg PCWP ≤ 15 mmHg PA saturation ≥ 65%
B. Beginning CS	A patient who has clinical evidence of haemodynamic instability (including relative hypotension or tachycardia) without hypoperfusion	Elevated JVP Warm and well-perfused Strong distal pulses Normal mental state	Crepitations in lung fields	Normal lactate (as above)	Minimal acute renal functional impairment, i.e. creatinine >77 μmol/L in pregnancy (0.87 mg/dL),[77,78] but <1.2 increase from baseline[65,b] (for further details, see below for trimester-specific levels[c]) Elevated BNP[79,a]	Hypotension SBP <90 mmHg (individualise for patient and clinical context) MAP <65 mmHg >30 mmHg drop from baseline Tachycardia Heart rate ≥ 100 bpm	
C. Classic CS	A patient who manifests with hypoperfusion and who requires one intervention (pharmacological or mechanical) beyond volume resuscitation These patients typically present with relative hypotension and individualised care is therefore essential (but hypotension is not required)	Volume overload	Looks unwell Acute alteration in mental status Feeling of impending doom Cold and clammy Extensive crepitations Ashen, mottled, dusky or cool extremities Delayed capillary refill Urine output <30 mL/h	Lactate ≥ 2 mmol/L (consider clinical context, as above)	Creatinine increase, ≥1.2 baseline[65,b] Increased LFTs[74,80] Elevated BNP[79]	If invasive haemodynamics assessed (strongly recommended): Cardiac index < 2.2 L/min/m^2 PCWP > 15 mmHg	

TABLE 1 (Continued)

		Physical examination		Biochemical markers[a]		Haemodynamics	
Stage	**Description**	**Typically includes**	**May include**	**Typically includes**	**May include**	**Typically includes**	**May include**
D. Deteriorating	Clinical deterioration despite initial support strategy to restore perfusion, as evidenced by worsening haemodynamics or rising lactate	Any of stage C and worsening (or not improving) signs/ symptoms of hypoperfusion, despite initial therapy		Any of stage C and lactate rising and persistently >2 mmol/L (consider clinical context, as above)	Deteriorating renal function,[77,78] Worsening LFTs[74,80] Rising BNP[79]	Any of stage C and requiring escalating doses or increasing numbers of pressors or the addition of a mechanical circulatory support device to maintain perfusion	
E. Extremis	Actual or impending circulatory collapse (ongoing cardiopulmonary resuscitation)	Typically, unconscious	Near pulselessness Cardiac collapse Multiple defibrillations	Lactate ≥ 8 mmol/L	CPR (A-modifier) Severe acidosis pH < 7.2 Base deficit > 10 mEq/L	Profound hypotension despite maximal haemodynamic support	Need for bolus doses of vasopressors

Abbreviations: BNP, B-type natriuretic peptide; CPR, cardiopulmonary resuscitation; CS, cardiogenic shock; CVP, central venous pressure; JVP, jugular venous pressure; LFTs, liver function tests; MAP, mean arterial pressure; PA, pulmonary artery; PCWP, pulmonary capillary wedge pressure; SBP, systolic blood pressure; SCAD, spontaneous coronary artery dissection.

[a]Use of gestation-dependent reference ranges.

[b]This is an arbitrary value set at a level to minimise harm and has been determined using current evidence, which suggests that renal parameters in pregnancy may be 80% of those seen in the non-pregnant population, and used in association with SCAI non-pregnant guidance for the assessment of abnormal renal function. Validation studies are recommended, evaluating clinical outcome and renal function where increases between 1.2 and 1.4 above baseline occur, to determine further clinical application.[74,77]

[c]Trimester-specific creatinine levels: upper limit of normal for first, second and third trimesters are 76, 72 and 77 μmol/L, respectively.[74]

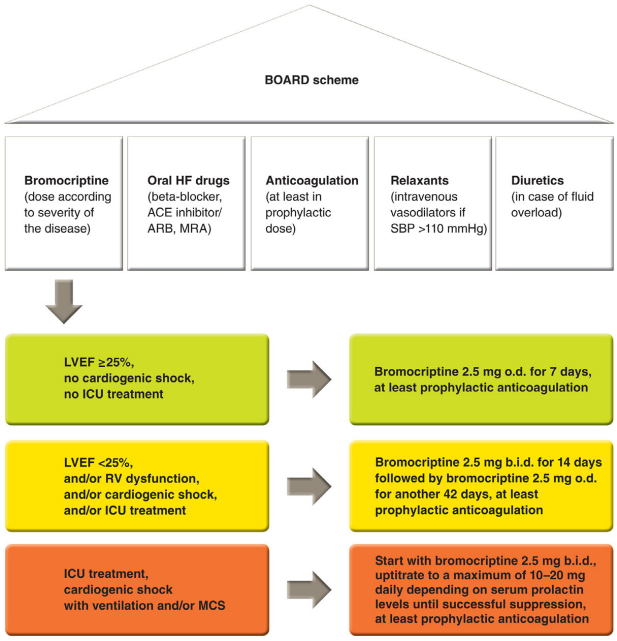

FIGURE 5 BOARD scheme outlining the medical management options for the postnatal patient with acute peripartum cardiomyopathy (not breastfeeding), with patient-specific therapeutic modifications individualised to the clinical severity.[21] ACE, angiotensin-converting enzyme; ARB, angiotensin receptor blocker; b.i.d., twice a day; HF, heart failure; ICU, intensive care unit; LVEF, left ventricular ejection fraction; MCS, mechanical circulatory support; MRA, mineralocorticoid receptor antagonist; o.d., once a day; RV, right ventricular; SBP, systolic blood pressure.

cardiac causes for CS in pregnancy, and the puerperium, there is recommended guidance for management and the mnemonic 'BOARD' has been proposed for the postnatal patient (Bromocriptine, Oral heart failure therapies, Anticoagulation, vasoRelaxing agents, and Diuretics, with non-invasive ventilation added in patients with pulmonary congestion) (Figure 5).[2,68] Although initial steps are likely to be managed appropriately at local intensive care units, patients falling into the category of SCAI-B and beyond require expert intensive care and cardiology input. Critical decompensation (SCAI C-E) will be seen more rarely, and in these cases local expertise will be limited and should be transferred immediately to a tertiary centre. Here the SCAI classification can be a valuable tool to utilise for the initial assessment and management of the pregnant patient with a known cardiac condition or who develops acute CS, and should be incorporated into the obstetrician's lexicon.

The SCAI classification can be broadly applied, initially, to manage CS in pregnancy irrespective of aetiology, with modifications for pregnancy-specific reference ranges for haemodynamics and biochemistry (Table 1), guided by clinical judgement. Biochemical criteria for derangement requires careful consideration, as gestation-dependent values can be lower in pregnancy (e.g. for alanine aminotransferase (ALT), albumin, aspartate aminotransferase (AST), and creatinine). Similarly, potential therapies will need to be adjusted to exclude those with adverse effects on the developing fetus. We propose an adapted algorithm to include pathologies either more common in or unique to pregnancy. These changes would create a clinically relevant tool for the management of CS in obstetrics, optimising the management of this increasingly common emergency. With the lack of consistent 'hands-on' exposure to patients with CS, we consider that there is a need to improve education and training in obstetrics and gynaecology to heighten awareness and improve recognition and initial resuscitation. We consider that the SCAI algorithm provides a platform to support this.

9 | CS IN PREGNANCY: THE ROLE OF MECHANICAL CIRCULATORY SUPPORT

For those that do not respond to medical management, mechanical circulatory support (MCS) has been used across a range of diagnoses.[69] The early recognition of CS, appropriate initial acute management and prompt escalation are key factors determining the long-term prognosis of a patient requiring MCS. MCS survival rates vary depending on several factors, including patient comorbidity, frailty, age and underlying aetiology. For example, ischaemic pathologies associated with significant myocardial tissue loss or other pre-MCS organ dysfunction have a poorer prognosis. Individual sites practising MCS often apply their preferred protocols, and site expertise is associated with improved patient outcomes. Furthermore, it has been proposed that sites that apply stringent criteria to patient selection demonstrate lower mortality rates.[70]

The current data are limited, but maternal and fetal survival rates following extracorporeal membrane oxygenation (ECMO) for all pathologies are encouraging, with a reported 74%–80% maternal survival rate, which is considered comparable with non-pregnant women of reproductive age, and with a 65%–70% survival rate for the fetus.[69,71] Fetal survival data are also likely to be gestation dependent. The optimal time of delivery for the fetus in women managed with ECMO is uncertain, with limited evidence to guide decision-making. Currently, a multidisciplinary approach is used to make birth decisions, and these are therefore very much individualised, with a view of balancing the risks and benefits for both mother and baby. In the context of respiratory failure related to COVID-19 in the general population, although initial data showed mortality rates of up to 60%, more recent data have demonstrated significantly improved outcomes for patients when strict criteria for the initiation of ECMO were used. Key factors for lower rates of mortality included early escalation and referral compared with patients who were referred in advanced stages during the height of the pandemic.[70,72]

The MCS survival rates in the general population, including non-pregnant women of reproductive age, vary considerably and depend on aetiology. Further studies are required to determine ECMO survival rates in this group as well as to compare this with their pregnant counterparts.

The ECMO survival rate significantly drops if referral occurs at late stages of the SCAI classification, emphasising

the importance of early referral for expert input.[70,73] We propose early consideration for gaining advice from an ECMO centre in the context of pregnant and puerperal patients exhibiting signs of CS, as well as regarding admission for both the venovenous (VV) and venoarterial (VA) ECMO service.

We consider that the adoption of SCAI encourages the development of a shared vocabulary with acute cardiology and intensive care teams that can only improve the effective management of these cases. As obstetricians become more familiar with the use of the algorithm, they will become confident in the assessment and acute management of CS, recognising it as a potential diagnosis sooner and expediting the initiation of life-saving treatment and referral to cardiac intensivists and ECMO centres.

10 | CONCLUSION

In this review article, we emphasise that cardiac disease remains the leading indirect cause of maternal mortality and, therefore, identifying those at risk of CS and managing such cases efficiently and effectively must be prioritised. CS can occur in patients with known risk factors, as well as in those without. The causes range from medical to obstetric-specific aetiologies. However, despite such varied pathology, the use of the SCAI algorithm facilitates a universal approach to identification, resuscitation and acute management. Furthermore, it provides a framework for the recognition of the deteriorating patient, and thus is a key communication aid between specialties for the escalation to units with diverse resources and expertise, to enable optimal care for pregnant women with CS.

AUTHOR CONTRIBUTIONS

SP and MRJ jointly conceived the article topic and review objectives. OYOG undertook the primary literature search, reviewed and summarised the current evidence, was responsible for writing the initial and revised drafts based on comments and suggestions from co-authors and submitted the final version. RA, SP, NMS and MRJ reviewed the current literature and incorporated this into the article, reviewed drafts with critical revision of intellectual content, observing current areas of debate, and advised on the content and structure of the article. All authors read and approved the final version for publication.

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SP is a board member of the European Society of Cardiology and receives no financial reward in this role.

DATA AVAILABILITY STATEMENT

Data sharing not applicable to this article as no data sets were generated or analysed during the current study.

ETHICS STATEMENT

Ethical approval was not required for conducting this review.

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