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Current management of patients with severe acute peripartum cardiomyopathy: practical guidance from the Heart Failure Association of the European Society of Cardiology Study Group on peripartum cardiomyopathy

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Introduction

Acute heart failure (AHF) due to peripartum cardiomyopathy (PPCM) provides a challenge for treating physicians. Moreover, in patients still pregnant, therapeutic interventions need always to consider the health of both the mother and the foetus. Especially challenging are severe forms of PPCM, as the mortality of these women is quite high. The use of inotropic drugs and mechanical circulatory support devices may be necessary in the initial phase of severe forms of acute PPCM. Many patients, after initial stabilization, recover LV function.[1-3] Unfortunately, some patients need further mechanical circulatory support or urgent heart transplantation despite maximal therapy. In addition, the time frame and extent of recovery are unpredictable, and patients may suffer from cardiac arrest due to ventricular fibrillation in the first months after diagnosis.[4] The clinical course may be further aggravated by atrial and/or ventricular thrombus formation with subsequent cardio-embolic complications.

As evidence-based data from randomized clinical trials are scarce, in this practical guidance we summarize recent data and clinical experience in the treatment of patients with severe acute PPCM to help physicians in the diagnosis, acute treatment, and long-term management of these young critically ill patients.

Definition and pathophysiology

The Working Group on PPCM of the Heart Failure Association (HFA) of the European Society of Cardiology (ESC) recently proposed a new simplified definition of PPCM as an idiopathic cardiomyopathy frequently presenting with heart failure secondary to LV systolic dysfunction (LVEF <45%) towards the end of pregnancy or in the months following delivery, if no other cause of heart failure is found.[1] Since no specific test to confirm PPCM exists, it remains a diagnosis of exclusion. In particular, aggravation of pre-existing heart disease by pregnancy-mediated haemodynamic changes should be differentiated from PPCM.

The pathophysiology of PPCM remains poorly understood. The current status of knowledge of the pathophysiological mechanisms of PPCM has been published elsewhere.[3] A ‘two-hit’ model of angiogenic imbalance in the heart during the peripartal period has recently been proposed, combining systemic antiangiogenic signals during late pregnancy and host susceptibility through insufficient local proangiogenic defences in the heart.[1-3, 5] Angiogenic imbalance can further be triggered by oxidative stress activating cathepsin D, a protease responsible for the cleavage of the nursing hormone prolactin into the angiostatic and proapoptotic 16 kDa subfragment.[4, 6]

Clinical presentation of acute peripartum cardiomyopathy

Most patients admitted with PPCM present typical symptoms of AHF associated with signs of congestion. Because early signs and symptoms of heart failure in PPCM patients may mimic physiological changes occurring during/after pregnancy, delayed diagnosis may occur. The differential diagnosis of acute PPCM includes myocarditis, pre-existing cardiomyopathy, valve disease, or congenital heart disease. In the case of cardiogenic shock, pregnancy-associated myocardial infarction, pulmonary embolism, and amniotic liquid embolism should be immediately ruled out to provide adequate care (*Table* 1).

Table 1. Peripartal acute dyspnoea: differential diagnosis of acute peripartum cardiomyopathy

	PPCM	Pre-existing CMP,valve disease orcongenital heartdisease	Pregnancy-associatedmyocardial infarction	Pulmonary embolism/amniotic liquid embolism	Myocarditis
History	Most commonly post-partal onset of dyspnoea	Earlier onset (during second trimester) Sometimes family history	Retrosternal chest pain, abdominal discomfort, nausea	Pleuritic chest pain	Infection
Biomarkers	Elevated natriuretic peptides	Elevated natriuretic peptides	Elevated troponin	Elevated D-dimer, troponin, natriuretic peptides	Elevated troponin Possibly. elevated natriuretic peptides
Echocardiography	Left and/or right ventricular dysfunction	Evidence of pre-existing valve disease or congenital defect	Regional hypokinesis/akinesis	RV dysfunction, elevated RV pressure, McConnell's sign	Regional or general hypokinesis
Additional tests	Consider MRI	Consider MRI Consider genetic test	Coronary angiography	CT-scan or. V/Q scintigraphy; consider angiography	MRI Consider myocardial biopsy

CMP, cardiomyopathy; MRI, magnetic resonance imaging; PPCM, peripartum cardiomyopathy; RV, right ventricular.

Evaluation of acute peripartum cardiomyopathy

As for any AHF, initial evaluation of patients with suspected acute PPCM includes two parts, which should be performed simultaneously to allow timely diagnosis and treatment delivery: evaluation of cardiopulmonary distress; and confirmation of the diagnosis with additional tests.

Evaluation of cardiopulmonary distress

Evaluation of cardiopulmonary distress is crucial because it will influence subsequent treatment and patients' allocation. The presence of criteria defining cardiopulmonary distress should lead to intensive cardiac care unit admission: haemodynamic instability (systolic blood pressure <90 mmHg, heart rate >130 b.p.m. or <45 b.p.m.), respiratory distress (respiratory rate >25/min; peripheral oxygen saturation <90%), or signs of tissue hypoperfusion with abnormal cellular oxygen metabolism (increased blood lactate >2.0 mmol/L; low central–venous oxygen saturation <60%, if available; altered mental state; cold, clammy, mottled skin; oliguria <0.5 mL/kg/h).[1, 7]

Confirmation of the diagnosis

Since PPCM is a diagnosis of exclusion, several additional tests should be performed (see below). This should not delay the start of treatment, which should be instituted as soon as AHF is confirmed.

An ECG should be performed in all patients with suspected PPCM as it has high negative predictive value and might help in identifying the cardiac origin of dyspnoea. Indeed, despite the fact that no specific ECG pattern for PPCM seems to exist, at initial evaluation, the ECG is rarely normal and repolarization abnormalities are common.[4, 8, 9] Patients with acute PPCM usually have elevated plasma concentrations of natriuretic peptides.[10, 11] Measurement of natriuretic peptides may help during screening

for identifying a cardiac origin of dyspnoea, although it does not help in the differentiation of PPCM from other cardiomyopathies. More specific biomarkers would be helpful to allow a faster and more reliable diagnosis of PPCM, but these are yet to be adequately defined. Echocardiography is indicated as soon as possible, in all cases of suspected PPCM to confirm the diagnosis, assess concomitant or pre-existing cardiac disease, exclude complications of PPCM (e.g. LV thrombus), and obtain prognostic information.

Cardiac magnetic resonance imaging (MRI) is not routinely needed, but can be performed after stabilization in cases where additional information, not available with echocardiography, is needed. However, administration of gadolinium to assess late enhancement should be avoided until after delivery, unless absolutely necessary. Endomyocardial biopsy does not add any diagnostic or prognostic information in the case of PPCM but can be used to exclude acute myocarditis after delivery. Only a few PPCM cases have been related to myocarditis so far,[12] but myocarditis may underlie cases of dilated cardiomyopathies and AHF that can occur or worsen during pregnancy, and specific immunosuppressive or immune-modulatory treatments may be helpful for some forms of myocarditis.[13]

Management of acute peripartum cardiomyopathy

The management of heart failure around pregnancy is challenging (Box 1), and, in the absence of evidence-based data, the initial management of patients with PPCM is similar to the treatment of AHF of other aetiologies.[14, 15] Interdisciplinary approaches of cardiologists, intensivists, obstetricians, neonatologists, anaesthetists, and cardiac surgeons are necessary in cases of severe AHF. Pre-specified protocols of interdisciplinary work-up of these patients are helpful (*Figure* 1).[16] Timely diagnosis and treatment delivery are crucial. *Figure* 2 summarizes the recommended treatment algorithm for patients with acute PPCM. Of note, the initial treatment of patients with severe forms of acute PPCM is significantly different from that of stable patients.

BOX 1. Peculiarities in the management of acute heart failure caused by peripartum cardiomyopathy

- Multidisciplinary approach with focus on health of mother and foetus.
- Avoidance of heart failure (HF) drugs with foetal toxicity during pregnancy (i.e. ACE inhibitors/ARBs, mineralocorticoid receptor antagonists) and breastfeeding; thereafter standard HF therapy.
- Consideration of bromocriptine (2.5 mg twice daily for 2 weeks, followed by 2.5 mg per day for 6 weeks) in addition to standard HF therapy.
- Anticoagulation with heparin to avoid cardio-embolic complications in patients with LVEF ≤35% or treated with bromocriptine (if no contraindication exists).
- In the case of cardiogenic shock, consideration of levosimendan (0.1 µg/kg/min for 24 h) instead of catecholamines as first-line inotropic drug. Early transfer to experienced centre. Early evaluation of mechanical circulatory support according to the centre's experience.
- Prevention of sudden cardiac death, early consideration of wearable cardioverter-defibrillator devices in patients with LVEF ≤35%.

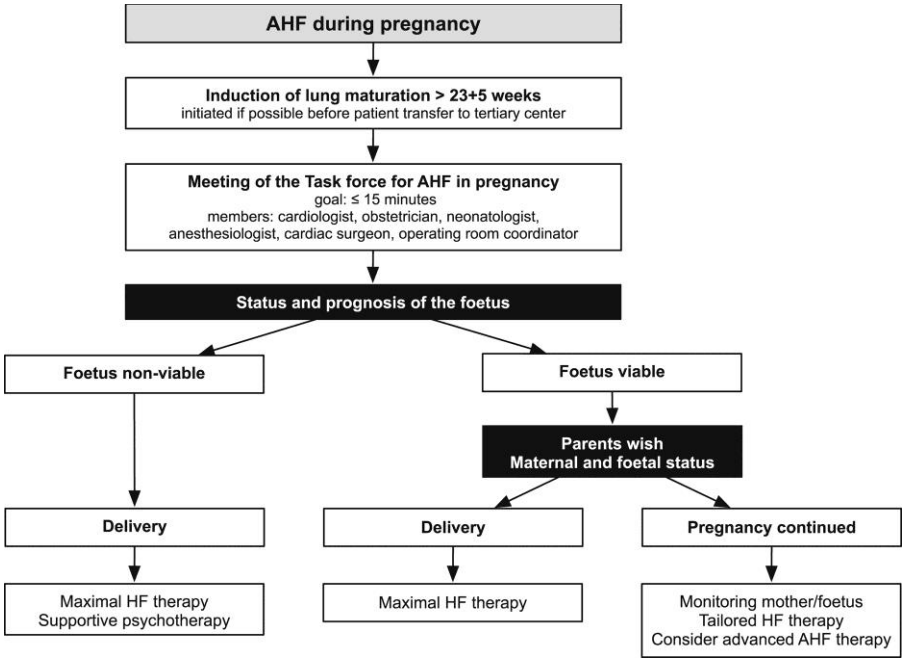

Figure 1. Example of prespecified protocol of interdisciplinary work-up for acute heart failure (AHF) during pregnancy (modified from the protocol of the Medical School Hannover).

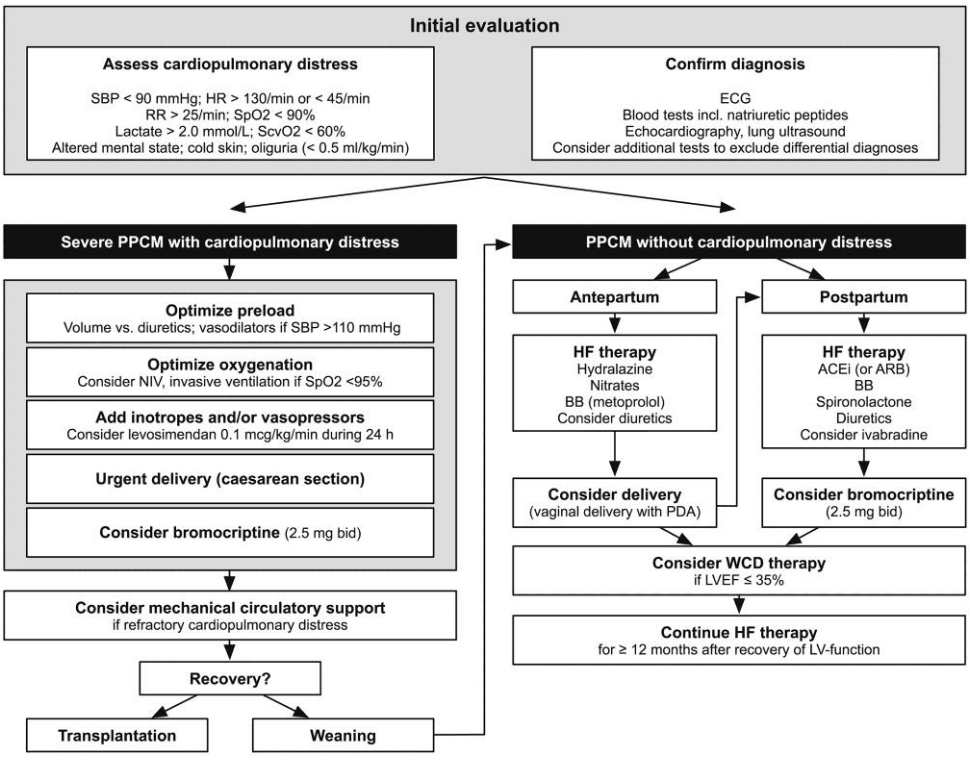

Figure 2. Algorithm for initial management. BB, beta-blocker; HF, heart failure; HR, heart rate; NIV, non-invasive ventilation; PDA, peridural anaesthesia; RR, respiratory rate; SBP, systolic blood pressure; SpO2, peripheral oxygen saturation; WCD, wearable cardioverter-defibrillator.

Initial management of acute peripartum cardiomyopathy with cardiopulmonary distress and/or haemodynamic instability

Patients with signs of cardiopulmonary distress and/or circulatory shock need rapid and more aggressive therapy and should be admitted to the intensive cardiac care unit. Initial therapy includes five main elements: optimization of the preload; optimization of oxygenation; restoration of haemodynamics with inotropes and/or vasopressors; urgent delivery if heart failure occurs during pre-partum; and consideration of adjunctive therapies with bromocriptine (2.5 mg twice daily for 2 weeks followed by 2.5 mg per day for 6 weeks).

Optimization of preload includes, depending on the clinical scenario, administration of fluids or diuretics. If there is no sign of overt fluid overload, a fluid challenge (250–500 mL over 15–30 min) is recommended, especially in patients with intravascular depletion secondary to peripartal blood loss or overaggressive diuretic therapy. In the presence of signs of congestion, intravenous diuretics should be administered. In patients with systolic blood pressure >110 mmHg, intravenous vasodilators (e.g. nitrates) should be started.

At the same time, oxygenation should be optimized (target peripheral oxygen saturation, $SpO_2 > 95\%$). Non-invasive ventilation (NIV) reduces respiratory distress and may decrease intubation and mortality rates.[17] Intubation with mechanical ventilation should be considered in the case of altered mental state or persistent hypoxaemia.

In the presence of signs of cardiogenic shock, haemodynamics should be rapidly restored to avoid irreversible organ damage. Inotropes and vasopressors may be considered, although the use of catecholamines is associated with adverse effects in patients with advanced heart failure or cardiogenic shock.[18, 19] Experimental evidence and clinical experience suggest that catecholamines such as dobutamine are less favourable in PPCM patients due to metabolic compromise.[20] Therefore, catecholamines should be avoided whenever possible or used only with extreme caution. Levosimendan, in contrast to dobutamine and adrenaline, does not increase myocardial oxygen demand and may be considered as the preferred inotropic agent as continuous infusion of 0.1 µg/kg/h for 24 h without an initial loading dose (bolus) for patients with severe PPCM.[21] A recent small study including 28 patients showed that the use of levosimendan in patients with PPCM induced rapid haemodynamic recovery and profound decongestive effects.[22] In case levosimendan is unavailable, dobutamine is the other option, while adrenaline should be avoided. As for other causes of shock, noradrenaline should be the first-line vasopressor.

Patients with haemodynamic instability despite treatment should undergo urgent delivery irrespective of gestation duration. Caesarean section with combined spinal and epidural analgesia and involvement of an experienced interdisciplinary team are recommended.

The administration of adjunctive therapies with the prolactin blocker bromocriptine has shown promising results in several case series and in a small proof-of-concept study,[23] and should be considered for patients with cardiopulmonary distress. The starting dose of bromocriptine is usually 2.5 mg twice daily, but an increased dose may be necessary to lower prolactin levels in selected cases (see below).

As thrombo-embolic events have been reported during the use of bromocriptine (albeit mostly at higher dosages), bromocriptine treatment should always be accompanied by at least prophylactic anticoagulation with heparin.[24] Anticoagulation with heparin should also be started in all patients with acute PPCM and severely reduced LV systolic function (LVEF $\leq 35\%$). Indeed, the combination of reduced EF and the procoagulant activity during the peripartal phase exposes patients to a clinically important risk of cardio-embolic events.

In general, patients with severe distress should be transferred early to an experienced centre whenever possible. For patients with persistent haemodynamic instability despite medical treatment, mechanical circulatory support should be considered (see below).

Advanced management of severe acute peripartum cardiomyopathy

Implantation of a mechanical circulatory support should be considered early as a rescue therapy in patients who cannot be stabilized with medical therapy alone.

If necessary, a device for temporary support should be implanted in the acute phase, either as 'bridge-to-recovery', if ventricular function improves during the subsequent days and weaning can be achieved, or as 'bridge-to-bridge', if haemodynamic impairment persists and circulatory support has to be ensured by switching to a more durable (and usually more invasive) device. Because of the higher proportion of patients with at least partial recovery of ventricular function compared with other cardiomyopathies, an initial 'bridge-to-transplantation' strategy is seldom necessary.

Since several devices exist, and there is little evidence about which device should be preferred;[25] we provide here an overview of some devices based on experts' opinion.

For the choice of the initial device, several factors should be taken into account (needed haemodynamic support, periprocedural risks, costs), but the oxygenation status of the patient plays a central role. If the patient is adequately oxygenated, percutaneous [e.g. intra-aortic balloon pump (IABP), Impella®] or surgical (e.g. CentriMag®, AbiomedBVS 5000®) devices can be used to restore circulation. In contrast, in the presence of impaired oxygenation, other devices with integrated oxygenation should be used [e.g. TandemHeart®, veno-arterial extracorporeal membrane oxygenation (ECMO)]. Most importantly, as the treatment of patients on mechanical circulatory support is very challenging, the choice of the device should also consider the local availability and the experience of the involved care team (physicians, nurses, and perfusionists).

Percutaneous devices offer the advantage of fast and easier placement and removal without the need for open surgery, but complications related to the access site (bleeding, infection, ischaemic limbs) are not uncommon. An IABP provides less haemodynamic support compared with other devices, but on the other hand is easily placed and needs less strict anticoagulation. Given the negative results of the IABP-SHOCK II trial and the lack of data in PPCM, the value of this device in patients with severe PPCM is uncertain, although it is used in selected cases by some centres.[26] The Impella® rotary pump is an alternative percutaneous device for temporary support. It is inserted percutaneously from the femoral artery and is placed in the left ventricle through the aortic valve. Depending on the model, it provides a higher degree of haemodynamic support compared with IABP (up to 5 L/min) but is associated with haemolysis and, especially in the context of PPCM, where a procoagulant state is frequent, a stricter anticoagulation regime than for IABP is needed.[27] In a small trial in patients with cardiogenic shock complicating myocardial infarction, there was no difference in terms of survival between IABP and Impella®.[28] Clinical experience in several PPCM patients with the Impella 3.5 device suggests effective LV support over up to 7–10 days when used as bridge-to-recovery in most patients. The marked decrease in the need for catecholamines may importantly contribute to the beneficial outcome observed in several patients (*Figure* 3). The TandemHeart® device offers similar haemodynamic support to Impella® (up to 5 L/min) with additional improvement in oxygenation. The placement of this device is performed percutaneously in the catheterization laboratory but it requires a more complex placement with atrial trans-septal puncture. No evidence of improved outcomes in patients with cardiogenic shock receiving TandemHeart® compared with IABP exists.[29, 30]

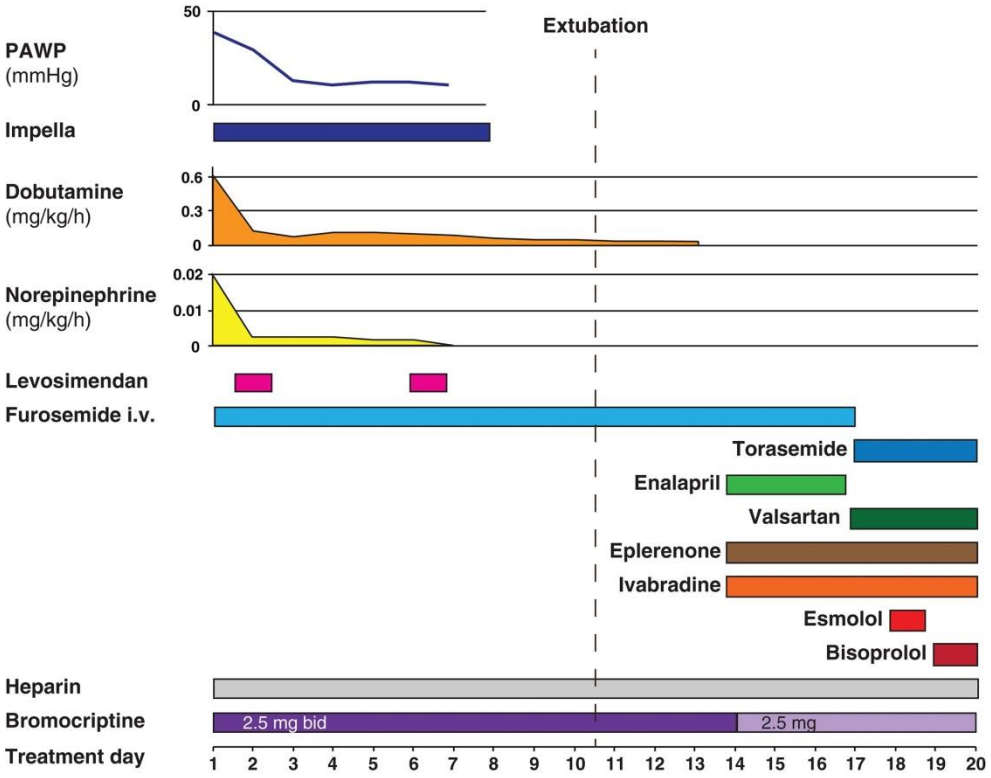

Figure 3. Example of the management of a peripartum cardiomyopathy (PPCM) patient with cardiogenic shock in the intensive care unit (ICU). The figure depicts the management of a patient with newly diagnosed PPCM in cardiogenic shock with severely reduced EF requiring mechanical ventilation and high dose vasopressor (norepinephrine), and inotropic support (dobutamine) at the acute presentation. Upon referral to the Acute and Advanced Heart Failure Unit of Medical School Hannover, temporary circulatory support with the Impella CP® rotary pump was initiated with concurrent invasive haemodynamic monitoring including pulmonary artery wedge pressure (PAWP) by means of a Swan–Ganz catheter. Note the decline of catecholamine dosage and PAWP after insertion of the Impella CP® rotary pump. Additionally, two cycles of 24-h infusion of levosimendan were administered with an interval of 1 week, and diuretic therapy was given throughout the intensive care. After 10 days, when haemodynamic stabilization was achieved, the patient could be extubated and standard heart failure therapy including an ACE inhibitor (later replaced by an ARB due to cough), mineralocorticoid receptor antagonist, and beta-blocker was established. Heart rate reduction with ivabradine was initiated early during ICU course. The patient was treated with a wearable cardioverter-defibrillator for 6 months and had an uneventful course; EF showed a partial recovery during the following 6 months.

Extracorporeal membrane oxygenation with veno-arterial cannulation offers the maximal available haemodynamic effect with biventricular support and additional improvement in oxygenation.[31] As increased prolactin levels during ECMO treatment have been reported, which may be specifically detrimental in patients with PPCM,[32] effective suppression of prolactin under sequential measurements of prolactin levels could be considered in this particular situation with bromocriptine doses up to 10 mg twice daily.

After the initial phase, if no weaning from mechanical circulatory support can be achieved after a maximum of 7–10 days, a switch to a durable device should be planned. As for temporary support, several devices exist and little evidence is available to guide the choice of the optimal device. Special attention should be paid to right ventricular function. In the presence of impaired right ventricular function, a biventricular assist device (BiVAD or total artificial heart) may be chosen (e.g. Berlin Heart EXCOR®). Alternatively, several strategies of transient right ventricular support in patients after left ventricular assist device (LVAD) implantation have been adopted in different centres (e.g. veno-arterial ECMO, Impella® RP, or similar). In patients with preserved right ventricular function, LVADs should be preferred. The most commonly used devices are the continuous-flow axial (HeartMate II®) and

centrifugal (HeartWare®) LVADs which have shown promising results in patients with end-stage heart failure.[33-35] Given the high likelihood of at least partial recovery of ventricular function in PPCM, temporary devices should always be the preferred initial strategy. Cardiac transplantation is reserved for patients where mechanical circulatory support is not possible or satisfactory ventricular recovery after 6–12 months is not achieved. Post-transplant outcomes in women with PPCM appear to be worse than in other recipients: in particular, women with PPCM show higher mortality, a higher incidence of rejection with shorter graft survival, and higher rates of re-transplantation.[36]

Management of acute peripartum cardiomyopathy without cardiopulmonary distress

The initial treatment of patients with confirmed PPCM without cardiopulmonary distress depends on the time point of onset. Patients who present after delivery should be treated according to the ESC guidelines for heart failure.[37] For patients presenting during pregnancy, joint cardiac and obstetric care in observance of the ESC guidelines for management of cardiovascular diseases in pregnancy is recommended.[38]

During pregnancy, ACE inhibitors, ARBs, and renin inhibitors are contraindicated because of foetal toxicity. Hydralazine and nitrates can be used instead. After delivery, ACE inhibitors can be started, but during breastfeeding captopril or enalapril should be preferred. Despite an increased risk of foetal growth restriction, beta-blockers are indicated in all patients in stable condition, with metoprolol succinate being the preferred agent.[38,39] Mineralocorticoid receptor antagonists (MRAs) should be avoided during pregnancy and lactation, but should be started afterwards in stable patients. Diuretics should be administered with caution during pregnancy as they may impair perfusion of the placenta. Recommendations for drug use during pregnancy and breastfeeding are summarized in table 21 of the ESC guidelines for management of cardiovascular diseases in pregnancy.[38]

Bromocriptine in addition to heart failure therapy should be considered because it has shown promising results with improved LV systolic function and clinical outcomes in several case series and in a small prospective proof-of-concept study.[23] In the retrospective non-randomized German PPCM Registry, treatment with beta-blockers, ACE inhibitors, and bromocriptine (2.5 mg twice daily for 2 weeks followed by 2.5 mg per day for 6 weeks) was associated with favourable outcomes.[40] A German study with 60 patients randomized to either short-term or long-term treatment with bromocriptine has terminated patient enrolment, and results will be available in the near future.[41] Anticoagulation with heparin should be started in all patients with acute PPCM treated with bromocriptine and in those with severely reduced LV systolic function (LVEF $\leq$35%).[24]

While diuretics should be tapered when possible after stabilization and when LVEF improves, ACE inhibitors, beta-blockers, and MRAs should probably be given in guideline-based dosages and not discontinued during the first 12 months after complete recovery of LV dimensions and systolic function. Earlier, stepwise discontinuation of heart failure therapy might be considered if both complete recovery of ventricular function and normal exercise response are achieved. Ivabradine should be given according to established indications. Furthermore, early treatment with ivabradine even before or in parallel with beta-blockers may be considered, as it appears to be safe and effective (*Figure 3*).[42]

As relapses have been observed after recovery, tapering of the disease-modifying heart failure drugs should be performed under close assessment of systolic function.[3]

Joint cardiologic and obstetric management including counselling on the potential risk of PPCM recurrence with futures pregnancies is recommended.

Prevention of sudden cardiac death

Despite increasing knowledge about the epidemiology and pathophysiology of PPCM, mortality rates are not well described and may range from <5% up to 50%. It is assumed that about a quarter of deaths are caused by ventricular tachyarrhythmia, mostly occurring during the first 6 months, and therefore optimal management may prevent a substantial number of deaths.[4]

Severely impaired ventricular function is associated with increased risk of life-threatening arrhythmias. Current ESC guidelines for the treatment of heart failure recommend implantation of an ICD (implantable cardioverter defibrillator) for primary prevention in patients with symptomatic heart failure and LVEF ≤35% despite optimal pharmacological treatment or for secondary prevention in patients with documented ventricular arrhythmia causing haemodynamic instability.[37]

In the context of PPCM, where young women with the potential for complete recovery of ventricular function are involved, decisions about implantation of an ICD should be taken with caution. After diagnosis of PPCM, clinicians are faced with the uncertainty about the subsequent evolution of ventricular function. Therefore, the related decision of whether to implant an ICD or not may be very challenging.

Several publications reported recovery of LV function in at least 50% of patients within 6 months after diagnosis.[43,44] However, a Turkish study reported delayed recovery (after 6 months) in a significant proportion of patients.[45] A recent retrospective study from the USA showed complete recovery of LV function in 23% of patients and partial recovery in another 19% over a mean duration of 33 ± 21 months, confirming frequent delayed recovery over 6 months (83%).[46] In this study, Afro-American women showed lower rates of recovery compared with Caucasians, and post-partum diagnosis was a predictor of good recovery. In a South African study, age and low LV end-diastolic diameter were predictors of recovery, whereas LVEF was not.[47]

In light of these data, early implantation of an ICD in patients with newly diagnosed PPCM is not appropriate. However, postponement of ICD implantation beyond the time point when further recovery of ventricular function is unlikely (6–12 months) exposes young mothers to an unacceptable risk of sudden cardiac death.

Novel therapies, such as the wearable cardioverter-defibrillator (WCD) (LifeVest®, Zoll, Pittsburgh, PA, USA) are an interesting alternative for the prevention of sudden cardiac death in the first months after diagnosis, until a definitive decision about ICD implantation can be made. A German study reported their experience with the WCD in patients with PPCM and severely reduced LV function. Seven out of nine women with PPCM and LVEF ≤35% received the WCD early after diagnosis. During a cumulative wearing period of 932 days, four adequate shocks were delivered for ventricular fibrillation in three patients without any inappropriate shock delivery within the first months of diagnosis.[4] These results may suggest the prescription of the WCD due to the relevant risk for ventricular tachyarrhythmias for at least 3–6 months after diagnosis to allow 'protected' recovery from severely reduced LV function.

In patients without recovery despite 3–6 months on optimized heart failure therapy, a conventional recommendation for the primary prophylactic implantation of an ICD applies.[37,48] In patients without LBBB or symptomatic sick sinus syndrome, single-chamber ICDs are recommended. Subcutaneous ICDs (S-ICDs) represent an alternative to transvenous systems in these young patients. Subcutaneous systems avoid intravascular leads and thus the potential complication of infections leading to endocarditis and lead extractions. On the other hand, subcutaneous systems can provide neither antitachycardia pacing (ATP) nor post-shock pacing and therefore might not be the optimal choice for patients with recurrent ventricular tachycardia successfully terminated by ATP.

In patients with heart failure, LVEF ≤35% despite optimal medical therapy for at least 3–6 months and LBBB, CRT is indicated, although no large studies have evaluated the value of CRT in patients with PPCM. Significant improvement of LV function in two PPCM patients undergoing CRT device implantation because of persistent symptomatic LV dysfunction was reported.[49]

According to the current ESC guidelines on CRT, in patients with symptomatic heart failure, persistent LVEF ≤35%, and complete LBBB (QRS duration >130 ms), CRT should be offered; in patients with wide QRS complex with non-LBBB morphology (QRS duration >150 ms), CRT may be considered.[48] Although no data on device therapy in patients with PPCM exist, recommendations for the device therapy may be applied as in patients with dilated cardiomyopathy.

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