Long-term prognosis, subsequent pregnancy, contraception and overall management of peripartum cardiomyopathy: practical guidance paper from the Heart Failure Association of the European Society of Cardiology Study Group on Peripartum Cardiomyopathy

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Peripartum cardiomyopathy is an idiopathic cardiomyopathy presenting with heart failure secondary to left ventricular systolic dysfunction towards the end of pregnancy or in the months following delivery, where no other cause for heart failure is identified. Outcome varies from full recovery to residual left ventricular systolic dysfunction and even death. Many women return to their physician to acquire information on their long-term prognosis, to seek medical advice regarding contraception, or when planning a subsequent pregnancy. This position paper summarizes current evidence for long-term outcome, risk stratification of further pregnancies and overall management. Based on the best available evidence, as well as the clinical experience of the European Society of Cardiology Study Group on Peripartum Cardiomyopathy members, a consensus on pre- and postpartum management algorithms for women undergoing a subsequent pregnancy is presented.

Keywords Peripartum cardiomyopathy • Subsequent pregnancy • Mortality

Introduction

Peripartum cardiomyopathy (PPCM) is an idiopathic form of cardiomyopathy presenting with heart failure secondary to left ventricular (LV) dysfunction towards the end of pregnancy or in the months following delivery, where no other cause of heart failure is identified. Patients can present with severe acute heart failure with high morbidity and mortality requiring a multi-disciplinary approach. Even those who present with more subtle symptoms can still have long-term impaired cardiac function. Until recently, data on suspected or confirmed cases of PPCM from Europe and most other regions of the world were limited. However, data from...
the ongoing EURObservational Research Registry on PPCM have shown that this condition occurs globally.4,5 Patients with PPCM are often young, have just started their families and, often, wish to fall pregnant again. The impact of a subsequent pregnancy (SSP) on clinical outcome is crucial,6 as it will not only affect the pregnant woman, but also other family members such as her partner and other children under their care. Moreover, after the traumatic experience of PPCM, patients may also develop psychological disorders that need treatment.

What is known about the long-term outcome of women with peripartum cardiomyopathy?

When a woman is diagnosed with PPCM, questions relating to the long-term consequences of the condition commonly arise. Women have described feeling terrified and devastated following a diagnosis of PPCM.7 They often struggle with recommendations to avoid a further pregnancy and report damaging effects on their marriage and relationships with other family members.7 In a review of comments from a PPCM support website, several messages were from users searching for information related to recovery, its length of time, the impact of SSPs and the duration of medical therapy.8 Many studies on PPCM have investigated the clinical course over 6 months or a year, but few have investigated outcomes over several years or decades. Women are understandably interested not only in outcomes during the early period following a diagnosis, but also in the long-term prognosis. Risk prediction of normalization of cardiac function and survival beyond 5 years cannot be provided based on solid prospective data. There are also no prospective long-term outcome data on large cohorts of patients with PPCM that have received a LV assist device or cardiac transplantation to make clear recommendations on long-term benefit. Much of what we do know comes from small studies of selected populations and from restricted geographical areas (Table 1)9−52.

Long-term mortality

1−6 months postpartum

Outcomes for patients with PPCM appear to be more favourable than for those with other forms of cardiomyopathy. In a comparison of mortality, over a mean follow-up period of 4.4 years in a large single-centre study of 1230 patients with cardiomyopathy, survival was greater in patients with PPCM than in those with idiopathic cardiomyopathy (adjusted hazard ratio 0.31, 95% confidence interval 0.09−0.98).33 Nonetheless, despite increasing recognition and understanding of PPCM, mortality rates have only been studied in a small number of countries and most available data come from the USA, South Africa, Haiti, Turkey and Pakistan. There are very few studies from Europe which report outcomes for women with PPCM and these are mainly small case series of no more than 12 patients.14,41,49,53 Most studies to date have concentrated on mortality at 6 months, with a wide variation in reported rates, ranging from 2% in Germany14 to 12.6% in a large cohort of 206 patients with PPCM from South Africa.15 Our current focus is on outcomes beyond 6 months.

6−12 months postpartum

Mortality up to 12 months is 4−14%, with the majority towards the lower end of this range (Table 1). The highest rates of mortality at 6−12 months are seen in African women (12−14%).10,11 This racial variation is evident elsewhere. For example, in the Investigations of Pregnancy-Associated Cardiomyopathy (IPAC) study in the USA (30% self-designated black), mortality at 12 months was 4%22 while in Detroit (96% African-American women) 11% died at a median follow-up of 12.5 months.23 A recent trial adding the prolactin blocker bromocriptine on top of standard therapy for heart failure reported an excellent 6-month follow-up outcome in severely diseased patients having over 60% full recovery and 0% mortality, heart transplantation and/or use of assist device.54

1−5 years postpartum

Of 182 women from the USA (56% Caucasian, 29% African-American, 10% Hispanic, 3% Asian), mortality was 7% at a mean follow-up of 19 months.19 A significantly larger proportion of the group who died or underwent cardiac transplantation were non-Caucasian, compared with the rest of the group (76% vs. 39%, P = 0.0001). At around 2 years, studies of black populations report mortality of 28% in South Africa,30 16% in Louisiana, USA23 and 15% in Haiti.27 Similarly, in a small case series of 13 women from New York, of which 69% were non-Caucasian, 23% of women had died at 2.1 years.25 In other studies from the USA, outcomes for women with PPCM are more favourable, with 2-year mortality of 0−9%.26,31 Mortality is also lower in women with PPCM from Brazil (8% at 2.1 years),28 and China (4% at 2.3 years).32 Mortality between 2−5 years varies even more considerably, ranging from 0−6% in French and American women33−37,39,43 to 15−30% in women from China,43 Brazil,34 Turkey,38,40,43 South Africa,55 and the Philippines.44

Beyond 5 years postpartum

There are few data beyond 5 years for women with PPCM. In three studies from the USA, mortality ranges from 7−16% at between 7 and 8.6 years.33,30,52 In India (n = 56), mortality was 23% at 6.1 years48 and in Malaysia (n = 12) 8.3% at 6.4 years.51 In a prospective study of 181 Nigerian women with PPCM from 1989, 26% had died at 10 years.46 There are no more recent studies of mortality beyond 5 years in African women.

Predictors of late mortality and mode of death

As recently highlighted by Sliwa and Anthony,56 late maternal death (>42 days postpartum) is globally poorly reported despite being an ICD-10 coding recommendation. Few studies on PPCM with follow-up extending beyond 2 years have sought to identify prognostic indicators in women with PPCM. In Turkey, baseline LV ejection fraction (LVEF) and LV end-systolic diameter were identified...
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Table 1 continued

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NA, not available.
as significant predictors of mortality due to heart failure.40 These
results conflict with other long-term follow-up studies, in which
there were no differences in baseline echocardiographic param-
eters between deceased and surviving study participants.1,29

While much of the existing data suggest that the risk of death
is greatest in the early postpartum period, late deaths occurring
either due to deterioration of cardiac function or arrhythmia have
been noted. Only 36% of deaths occurred within 6 months in a
group of 80 women with PPCM from South Africa, with a fur-
ther 36% at 6–12 months and 27% at 12–24 months.30 Notably,
in this cohort, recovery of LV function had occurred in 29% of
patients who died between 6–24 months, suggesting that even in
those with early recovery, a risk of mortality persisted beyond
this.30 In a retrospective two-centre study of 100 women from the
USA, 2 out of 11 women who died had recovered LV function by a
mean of 23 months and death occurred at a mean of 83 months.52

In fact, more recent data using wearable cardioverter-defibrillator
clearly demonstrate a high risk for ventricular tachyarrhythmias,
and sudden death in patients with PPCM is more common in the
acute phase of the disease but may even occur during recovery.37,58
In general, the recommendations of the European Society of Cardiology (ESC) guidelines for acute and chronic heart
failure on the use of wearable cardioverter-defibrillators should be
followed.39

However, clear markers to identify patients at risk for arrhythmic
death or non-sudden death do not exist. Consequently, although the
time at which death occurs following a diagnosis of PPCM is
clearly important with regard to informing patients and also to
decide whether or not continued pharmacological or device
therapy may be warranted, more research is needed to identify
prognostic factors or markers associated with late death in women
with PPCM.

Recovery at 6 months

Studies of recovery of cardiac function with an echocardiographic
LVEF >45% have largely focused on the trajectory over the first
6 months postpartum and rates vary markedly from one coun-
try to another. Six-month recovery rates between 46–63% have
been reported in Japan,12 China,60 Turkey,40 Germany,14 and the
USA.31 Six-month recovery is worse (21–36%) in South Africa,61,62
Nigeria,63 Pakistan,64 and the Philippines.44

Recovery beyond 6 months

Although data on early recovery of LV function are more frequently
reported, the concept of myocardial recovery beyond 6 months is increasingly recognized. In the recent IPAC study, a prospective
multi-centre American study of 100 women with PPCM, recovery
(LVEF >50%) was seen in 72% of women at 12 months.29 Rate of
recovery between 6 and 12 months is unavailable. In a prospective
analysis of a group of 42 Turkish women with PPCM, recovery of
LV function beyond 6 months was seen in 44%.40 Of those
who recovered, 60% did so beyond 12 months, with recovery
seen up to 42 months postpartum. Similarly, a median time to
recovery of 54 months was reported in a retrospective analysis of
44 women with PPCM in Louisiana, USA, with a 30% non-recovery
rate reported up to 9 years after diagnosis.29 Recovery at 6-month
intervals, up to 36 months, was also illustrated in a cohort of 116
women from Haiti, with 28% of all women regaining good cardiac
function.65 At each follow-up interval, there was a steady increase
in the proportion of women who demonstrated recovery of LV
function; 53% of recovered patients did so beyond 18 months.

These data illustrate that recovery can occur after 6 months and
continuing recovery can be seen, at least in some countries, after
several years. However, the current literature is far from comprehen-
sive. More prospective, multi-centre studies of myocardial
recovery in unselected populations with long-term follow-up are
required in order to describe the natural history of cardiac func-
tion in women with PPCM. More sophisticated echocardiographic
investigation, including strain measurements and cardiac magnetic
resonance imaging, would allow more detailed assessment of car-
diac function and structure following a diagnosis. To date, recovery
of cardiac function has focused on LVEF and the long-term effects
on right ventricular function have not been studied. There is a need
to investigate the long-term effects on mitral incompetence and if
PPCM are left with a residual myocardial scar.

Does myocardial function
deteriorate after stopping
medical therapy for peripartum
cardiomyopathy with recovered
cardiac function?

A common clinical question when myocardial recovery occurs in
PPCM is: ‘Should medical therapy be stopped?’ There are few
data to guide this decision and, therefore, no recommendations
within current guidelines exist. In a prospective, two-centre study
from Turkey investigating recovery in 42 patients with PPCM, four
patients (two who had full recovery of LV function and two who
had partial recovery of LV function) showed delayed deteriora-
tion at 12, 24, 26 and 34 months after diagnosis.40 In the two
patients who had fully recovered, medication was discontinued
when cardiac function improved, although the time at which medi-
cal therapy was stopped is not clear. The other two patients were
still receiving heart failure therapy. The only other observational
study is a cohort of women from North Carolina with recov-
ered myocardial function.15 Of the five patients who had discontinue-
td treatment with both beta-blocker and angiotensin-converting
enzyme (ACE) inhibitor, none demonstrated deterioration in car-
diac function after a mean follow-up of 29 months. There is one
further case series describing three women from the USA with
recovers LV function who had a subsequent deterioration of
myocardial function (unrelated to a further pregnancy) ‘several
months or years’ later.19 Whether these women were still receiv-
ing medical therapy is not reported. Therefore, studies evaluat-
ing criteria for treatment duration (clinical signs, biomarkers)
to determine the time and composition of long-term treatment
are needed.
What can be recommended for the long-term management of women with peripartum cardiomyopathy?

The current recommendation, based on consensus of the Heart Failure Association (HFA) PPCM Study Group members, is a 6-month visit including echocardiography in all women until they recover to an LVEF >50%. In women with LV recovery who remain stable after tapering of heart failure drug therapy, an annual visit is recommended for up to 10 years.

Patients with persistently reduced LVEF should continue treatment with ACE inhibitors, beta-blockade and with mineralocorticoid receptor antagonist (MRA), as well as ivabradine and sacubitril/valsartan according to the current ESC guidelines for acute and chronic heart failure.59 There is no consensus as to whether heart failure medication can be stopped in women with a recovered LV function or subclinical dysfunction. However, weaning from medication should be performed sequentially with careful and close monitoring of patients’ clinical and cardiac performance.3

Some of the PPCM Study Group members recommend lifelong heart failure therapy at the highest tolerated dose based on the fact that deterioration of LV function has been observed in women with normalized cardiac function. If patients display signs of incomplete recovery despite recovered LVEF such as persistent LV dilatation or reduced myocardial strain, continuation of heart failure drugs (ACE inhibitors, beta-blocker, MRA) should be considered. Research in this field is urgently needed via carefully conducted studies. Genetic testing may be considered in patients with a family history of cardiomyopathy, as recent studies showed that 15% of PPCM patients carry cardiomyopathy-causing mutations. The presence of TTN truncating variants was significantly correlated with a lower LVEF at 1-year follow-up,66 which features important for long-term therapy concepts.

How can patients be best advised about the risks for a subsequent pregnancy?

Data on SSPs in women with a previously diagnosed PPCM are limited. Table 2 summarizes the studies of women who had a SSP after being diagnosed with PPCM.6,29,34,37,48,64,67–78 However, the mode of data collection is highly variable with some studies not reporting the LVEF of the index pregnancy, nor the cardiac dimensions, left and right ventricular function at onset of the SSP. Also the date of the last assessment post-SSP is highly variable. Therefore, there are enormous gaps in our knowledge. Elkayam recently summarized the available information related to SSP in women with a history of PPCM.73 One of the largest studies to date, published in the New England Journal of Medicine in 2001, was based on information of 44 women with PPCM and SSP, collected via a retrospective survey of members of the American College of Cardiology.70 Twenty-eight of these women had a recovered cardiac function, while 16 pregnancies occurred in women with persistent LV dysfunction. All pregnancies were associated with a reduction in mean LVEF (from 49.9 ± 12% to 42 ± 13%, P < 0.001), with no mortality in women with normalized LVEF at onset of a SSP vs. 19% in those women who had impaired systolic function (LVEF 32.0 ± 2%) at onset of SSP (P = 0.06). In the same study, frequency of premature delivery (11% vs. 37%) and therapeutic abortions (4% vs. 25%) was also greater in women with impaired vs. recovered cardiac function at onset of SSP. As these data were collected via a retrospective survey, information on medical therapy provided during pregnancy was not available.70

A recently published prospective study reported the management and outcome of SSPs in 34 PPCM patients in Germany, Scotland and South Africa.6 Persistently reduced LVEF (<50%) prior to entering SSP was present in 47%, while full recovery (LVEF ≥50%) was present in 53% of patients. The majority of these patients were of African ethnicity (75%). Overall relapse rate (LVEF <50% or death after at least 6-month follow-up) was 56% with 12% (4/34) mortality. All four deaths occurred in the women with persistently reduced LVEF prior to SSP. Patients obtaining standard therapy for heart failure, and bromocriptine immediately after delivery, displayed significantly better LVEF at follow-up and a higher rate of full recovery, with no patient dying, compared to patients obtaining standard therapy for heart failure alone. This was independent of African or Caucasian race. Bromocriptine was provided to a number of patients, based on previous research suggesting that the nursing hormone, prolactin, which is highly elevated during delivery, and periodically in nursing mothers, is a key player in the pathophysiology of PPCM.80 A number of factors, such as oxidative stress, promote the cleavage of prolactin in an anti-angiogenic 16 kDa-PRL fragment being causally involved in PPCM.75 Furthermore, experimental studies in mice and small clinical pilot studies suggest a beneficial effect of the prolactin blocker bromocriptine to improve outcome of acute PPCM81 or prevent relapse after a SSP.6 Recent data from the EURObservational PPCM global registry have demonstrated that of 411 patients, 21.1% received bromocriptine in their index pregnancy.5 However, breastfeeding should be encouraged in women with mild cardiac dysfunction, particularly in areas of poor sanitation and unsafe water supply.

Based on published data6,79 and consensus by the HFA PPCM Study Group, our position is that full recovery of LVEF before a SSP is associated with lower mortality and better cardiac function at follow-up. However, all patients have a risk of deterioration of cardiac function. Women with impaired LV function at the onset of a SSP have a high risk of relapse, heart failure and death, and pregnancy should be avoided.

Data have shown that patients with a history of PPCM could have subclinical cardiac dysfunction82,83 and demonstrated reduced myocardial strain after recovery in women with PPCM at least 12 months after the diagnosis. There are no published data on the impact on SSP in this group of patients.

Addition of bromocriptine to standard therapy for heart failure immediately after delivery was found to be safe and seemed to be associated with a better outcome of SSPs in African and Caucasian patients.6

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The current recommendation based on published data and consensus of the HFA PPCM Study Group members on the management of pregnancy and postpartum period in women with a previous PPCM is summarized in Figure 1. Patients can be stratified according to impaired or recovered LV function at onset of SSP. However, each case would need to be assessed by a multi-disciplinary team, which includes a cardiologist, obstetrician, neonatologist, anaesthetist, and possibly other specialists. Neonatologists could provide advice on neonatal outcome if delivery needs to occur prematurely. Anaesthetists need to provide input in patients delivering with symptoms and signs of heart failure. In addition, overall health system factors and maternal factors such as age and sub-clinical thyroid disease need to be considered.

In pregnant women, renin–angiotensin receptor inhibitors need to be terminated because of foetal toxicity. In addition, hydralazine–nitrate combination should be used instead of angiotensin receptor blockers for management of heart failure as well prevention of further deterioration. Anticoagulation with low molecular weight heparin during pregnancy in PPCM women with LV dysfunction should be considered.

In terms of pregnancy from an obstetric standpoint, scans for foetal growth should be performed every 4 weeks from week 24. Timing of the delivery, for obstetric reasons, should be driven by the usual parameters including foetal growth restriction or pre-eclampsia. For cardiac reasons, early delivery should be considered with deteriorating cardiac function and particularly with heart failure. Ideally the target would be to reach 37 weeks. However, the best compromise will be achieved by discussion with the multi-disciplinary team to balance maternal health and foetal maturity.

Figure 2 provides a check box for all women with a diagnosis of PPCM—either newly diagnosed or with PPCM and SSP.

Table 2  Studies of peripartum cardiomyopathy patients undergoing a subsequent pregnancy

<table>
<thead>
<tr>
<th>First author</th>
<th>Year</th>
<th>Number</th>
<th>Pregnancies*</th>
<th>Post-index pregnancy LV function</th>
<th>Persistent LVSD post-subsequent pregnancy</th>
<th>Maternal death</th>
<th>Miscarriage/ foetal death n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Recovered n (%)</td>
<td>Unrecovered n (%)</td>
<td>Total No. death n (%)</td>
<td>in unrecovered LV function (% of total deaths)</td>
</tr>
<tr>
<td>Sutton67a</td>
<td>1991</td>
<td>4</td>
<td>4</td>
<td>4 (100)</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Witlin68a</td>
<td>1997</td>
<td>6</td>
<td>7</td>
<td>NA</td>
<td>NA</td>
<td>1 (17)</td>
<td>1 (100)</td>
</tr>
<tr>
<td>Albanesi Filho69b</td>
<td>1999</td>
<td>12</td>
<td>16</td>
<td>6 (50)</td>
<td>6 (50)</td>
<td>1 (8)</td>
<td>1 (100)</td>
</tr>
<tr>
<td>de Souza74a</td>
<td>2001</td>
<td>7</td>
<td>7</td>
<td>NA</td>
<td>NA</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Elkayam70c</td>
<td>2001</td>
<td>44</td>
<td>35</td>
<td>28 (64)</td>
<td>16 (36)</td>
<td>3 (7)</td>
<td>3 (100)</td>
</tr>
<tr>
<td>Avila71b</td>
<td>2002</td>
<td>18</td>
<td>19</td>
<td>7 (39)</td>
<td>11 (61)</td>
<td>1 (6)</td>
<td>1 (100)</td>
</tr>
<tr>
<td>Sharieff72b</td>
<td>2003</td>
<td>9</td>
<td>NA</td>
<td>2 (22)</td>
<td>7 (78)</td>
<td>2 (22)</td>
<td>NA</td>
</tr>
<tr>
<td>Sliva73a</td>
<td>2004</td>
<td>6</td>
<td>6</td>
<td>2 (33)</td>
<td>4 (67)</td>
<td>2 (33)</td>
<td>2 (100)</td>
</tr>
<tr>
<td>Chapa73a</td>
<td>2005</td>
<td>6</td>
<td>8</td>
<td>4 (67)</td>
<td>2 (33)</td>
<td>0</td>
<td>NA</td>
</tr>
<tr>
<td>Fett74b</td>
<td>2006</td>
<td>15</td>
<td>16</td>
<td>1 (7)</td>
<td>14 (93)</td>
<td>1 (7)</td>
<td>NA</td>
</tr>
<tr>
<td>Mishra48</td>
<td>2006</td>
<td>9</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>5 (56)</td>
<td>NA</td>
</tr>
<tr>
<td>Hilfiker-Kleiner75b</td>
<td>2007</td>
<td>12</td>
<td>12</td>
<td>12 (100)</td>
<td>0</td>
<td>3 (25)</td>
<td>0</td>
</tr>
<tr>
<td>Habi37</td>
<td>2008</td>
<td>37</td>
<td>21</td>
<td>NA</td>
<td>NA</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Modi29c</td>
<td>2009</td>
<td>15</td>
<td>4</td>
<td>27 (73)</td>
<td>11 (73)</td>
<td>0</td>
<td>6 (40)</td>
</tr>
<tr>
<td>Chee30c</td>
<td>2009</td>
<td>2</td>
<td>1</td>
<td>2 (100)</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Fett77e</td>
<td>2010</td>
<td>56</td>
<td>61</td>
<td>29 (52)</td>
<td>27 (48)</td>
<td>1 (2)</td>
<td>1 (100)</td>
</tr>
<tr>
<td>Mandal78f</td>
<td>2011</td>
<td>6</td>
<td>6</td>
<td>5 (83)</td>
<td>1 (17)</td>
<td>1 (17)</td>
<td>1 (100)</td>
</tr>
<tr>
<td>Hilfiker-Kleiner66</td>
<td>2017</td>
<td>34</td>
<td>31</td>
<td>18 (53)</td>
<td>16 (47)</td>
<td>4 (12)</td>
<td>4 (100)</td>
</tr>
</tbody>
</table>

LV, left ventricular; LVSD, left ventricular systolic dysfunction; NA, not available.

*Number without therapeutic abortion.
†At last follow-up.
‡Fractional shortening 30% used as cut-off.
§Unknown cut-off.
aEjection fraction 50% used as cut-off.
bUnknown cut-off.

cUnknown cut-off.

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Anticoagulation for 6–8 weeks after the delivery could be considered because of the hypercoagulability state during this period of time.

**Contraception for women with recent peripartum cardiomyopathy and for patients at high risk in a subsequent pregnancy**

Contraceptive counselling should begin early—i.e. as soon as PPCM has been diagnosed. A review on the use of contraceptives in women with heart disease has been published recently by Roos-Hesselink and colleagues.85 Types of the most commonly used contraceptives are summarized in Figure 3. Appropriate advice may be complex and will require the input of both a cardiologist and an obstetrician to identify the optimal approach. As no studies have been performed in women with heart disease, and in particular in women with PPCM, the relative risks and benefits of different contraceptive methods are based on consensus only. As women with PPCM with LV dysfunction are at a substantial risk of thrombo-embolic events,5 hormonal contraceptives with a pro-thrombotic effect should be avoided. The risk of venous thrombosis is significantly increased (up to seven-fold) by the...
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Figure 3  Sketch illustrating different types of contraceptives.

Contraception (Female)
- Intrauterine contraceptive device (e.g. Mirena or copper IUCDs)
- Oral contraceptive (combined or progestin only pills)
- Depomedroxyprogesterone acetate (DMPA) injections
- Tubal ligation
- Diaphragm
- Contraceptive implants
- Contraceptive patch
- Hysteroscopic tubal occlusion (HTO)
- Vaginal ring
- Safe period

Contraception (Male)
- Condom
- Vasectomy

Oestrogen component in oral contraceptives—irrespective of type of progestin used. However, the risk in the general population is small in absolute numbers (8–10 000 women-years exposure). The risks of using a combined oral contraceptive must be weighed against that of an unplanned pregnancy. However, since oestrogen-containing oral contraceptives not only increase the risk of venous thrombosis, but also of arterial thrombosis and hypertension, they are contraindicated in most forms of cardiac disease, particularly those associated with increased venous or arterial thrombotic risk, hypertension or ischaemic heart disease. Further, given that the most effective types of contraception are the long-acting reversible forms (intrauterine contraceptive devices or progesterone cutaneous implants) and that they have no pro-thrombotic effects, this group of contraceptives should be advised in most cases. They are at least as effective as sterilization, the finality of which some women struggle to accept. The progestogen (etonogestrel) implant, known as Implanon, has no cardiac effects, is effective and has fewer side-effects, such as irregular bleeding, than other implants. The progesterone-releasing intrauterine system, Mirena, is preferred to the older copper intrauterine contraceptive devices as the majority of users have no periods. Due to high failure rates, barrier methods should only be recommended in addition to other contraceptive methods.

In reaching a decision about type of contraception given the significant maternal morbidity and mortality risk of a SSP, the partners of the women should be involved in reaching the decision about the type of contraception. In decision-making, the following issues should be considered:

1. The risk of pregnancy for the mother and the consequences of an unplanned pregnancy.
2. The impact of any pregnancy on the entire family, which may include hospitalization due to heart failure, embolic events and death.
3. The risks and benefits of the type of contraception, in particular pro-thrombotic effects.
4. Failure rates of the type of contraception.
5. The availability and affordability of different types of contraception.
6. The individual’s preferences, which may include the option of sterilization for women or their partners.
7. For the majority of women, a long-acting reversible form such as an intrauterine contraceptive device will be most favourable.

Conclusion and way forward
Current evidence for long-term outcome is based mostly on single-centre studies or small registries. All patients with a previously diagnosed PPCM and their partners should receive careful counselling about long-term prognosis and undergo risk
stratification if further pregnancies are considered. Patients who undergo a SSP should be monitored by an experienced multidisciplinary team throughout the pregnancy and for at least 1 year postpartum. Based on recently published data, women undergoing a SSP with an impaired systolic function are at substantial risk for relapse and death and should therefore be advised against pregnancy. Breastfeeding is not advisable in cases with severely impaired systolic function. In those patients, inhibition of prolactin with bromocriptine should be considered. The ongoing EURObservational Programme on PPCM will provide much needed longer-term outcome data.5

Acknowledgements

The authors would like to acknowledge to overall support of the Heart Failure Association of the European Society of Cardiology. We also acknowledge the support of Ms Sylvia Dennis, Hatter Institute for Cardiovascular Research in Africa, in preparing the manuscript.

Conflict of interest: none declared.

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