The role of cardiac magnetic resonance in identifying appropriate candidates for cardiac resynchronisation therapy – A systematic review of the literature.

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Abstract

Introduction

Despite the strict indications for cardiac resynchronisation therapy (CRT) implantation, a significant proportion of patients will fail to adequately respond to the treatment. This systematic review aims to present the existing evidence about the role of cardiac magnetic resonance (CMR) in identifying patients who are likely to respond better to the CRT.

Methods

A systematic search in the MedLine database and Cochrane Library from their inception to August 2021 was performed, without any limitations, by two independent investigators. We considered eligible observational studies or randomised clinical trials (RCTs) that enrolled patients >18 years old with HF of ischemic or non-ischemic etiology and provided data about the association of baseline CMR variables with clinical or echocardiographic response to CRT for at least 3 months. This systematic review was performed in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA Statement).

Results

Following our search strategy, 47 studies were finally included in our review. CMR appears to have an additive role in identifying the subgroup of patients who will respond better to CRT. Specifically, the presence and the extent of myocardial scar associatewith increased non-response rates, whilst those with no scar respond better. Furthermore, existing data show that scar location can be associated with CRT response rates. CMR-derived markers of mechanical desynchrony can also be used as predictors of CRT response. CMR data can be used to optimise the position of the left ventricular lead during the CRT implantation procedure. Specifically, positioning the left ventricular lead in a branch of the coronary sinus that feeds an area with transmural scar associate with poorer response to CRT.

Conclusions

CMR can be used as a non-invasive optimisation tool to identify patients who are more likely to achieve better clinical and echocardiographic response following CRT implantation.

Keywords: cardiac resynchronisation therapy; Cardiac magnetic resonance; Late gadolinium enhancement; myocardial fibrosis; CRT response

Introduction

According to current guidelines, cardiac resynchronisation therapy (CRT) is recommended in patients with advanced heart failure (HF), impaired left ventricular ejection fraction (LVEF), and a wide QRS complex [1,2]. However, a significant proportion of CRT patients will fail to respond to the treatment with the rates of success varying according to the definition of the response criteria [3]. Specifically, the CRT response rates can range from 32% to 91% [4]. The identification of baseline characteristics that are associated with a higher probability of CRT response is therefore of great importance.

Cardiac magnetic resonance (CMR) is an imaging modality that can provide valuable clinical data, and therefore its use has been expanded in the current clinical practice [5]. In the field of HF, CMR can provide the "gold standard" method for measurements of biventricular ejection fraction, characterise myocardial tissue and mainly identify presence, location and burden of myocardial fibrosis, assess myocardial viability, and thus help diagnose specific cardiomyopathies [6]. Furthermore, the presence and degree of myocardial scar on CMR has been associated with an adverse prognosis, including arrhythmias in conditions like aortic stenosis [7] and dilated cardiomyopathy [8,9]. It is therefore possible, that the presence and extent of myocardial scar can adversely affect the benefit following CRT and indeed the use of CMR in the pre-procedural evaluation of CRT candidates in combination with other diagnostic tools is able to identify patients unlikely to respond to CRT [10]. This systematic review aims to present the existing evidence about the role of CMR in identifying patients who are likely to respond to the CRT.

Myocardial Tissue Characterisation

CMR is the only radiation-free modality that enables accurate assessment of myocardial tissue characterization. Various methods exists for this as well as different commercial packages. However, the mainstay of tissue characterization relies on the administration of the paramagnetic agent gadolinium and imaging the myocardium some 10-20 minutes after the administration. This delayed (or late) phase of imaging after the administration of gadolinium, often referred to as "late gadolinium enhancement, LGE" enables to identify scarred (or dead) myocardium from alive myocardium (Figure 1). The white areas following gadolinium administration represent muscle that has died, often referred to as replacement (or focal) fibrosis and there has been extensive validation of the CMR findings with histology. This type of fibrosis/ scar is irreversible and forms the mainstay of assessment for viability by CMR. The

black areas represent healthy myocardium. This also allows the location of the scar to be identified, with subendocardial or transmural scar relating to myocardial infarction and ischaemic cardiomyopathy. Midwall or subepicardial scar in the context of cardiomyopathy would relate to the non-ischaemic cardiomyopathy. Non-ischemic cardiomyopathy is an umbrella term, capturing a plethora of conditions, which include cardiomyoapathy secondary to dilated cardiomyopathy, myocarditis, cancer (either cancer itself or chemotherapy-related), hypertension, infiltrative and autoimmune processes. When it comes to the quantification of the scar there are both visual and semi-automatic/ artificial-intelligence guided methods. A simple, but well recognized and valuable method, is to simply use the 16 AHA segment model and for each segment allocate 0 if there is not scar, 1 if there is subendocardial non-transmural scar and 2 if there is transmular scar. This will therefore give a total score of 32, or each of the 16 segments representing 6% of the myocardium. More recent methods such as those provided by Circle CVI (Calgary, Canada) or MASS (Leiden, Netherlands) allow the operator to select a region of interest of normal (or abnormal) myocardium and the software will then automatically select and quantify the total scar for each ventricular slice. The software will then add the scar in all the slices giving an overall percentage both as mass and as a percentage. The quantification relies either on the Full Width Half Maxium method using regions defined above 50% of maximal signal intensity of the enhanced area, or the standard deviations method, where LGE can be quantified using a threshold above 2-7Standard deviations above a remote reference region [11].

More recently, different CMR sequences based on T1 mapping, have been histologically validated [12] to show diffuse (or interstitial) fibrosis, effectively a "finer" form of fibrosis that might not be irreversible. This relies on expansion of the extracellular matrix, which is one of the initial hallmarks of pathology and if the underlying aetiology is left untreated, is thought that it will result to myocardial scar. The two most common forms of measuring this diffuse fibrosis are Native T1 mapping (which does not require administration of gadolinium) and Extracellular Volume Fraction (ECV), which relies on measuring myocardial and blood T1 values before and after the administration of gadolinium. Both LGE and T1 mapping are therefore methods that provide myocardial tissue characterization and will be reviewed in this paper.

Methods

This systematic review was performed in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA Statement) [13].

Search strategy

Two independent investigators performed a systematic search in the MedLine database and Cochrane Library from inception to August 2021 without any limitations. In addition, we manually searched the reference lists of the relevant review and research studies. The following algorithm was used to retrieve all relevant studies: "(cardiac magnetic resonance) AND (cardiac resynchronisation therapy OR CRT)". We first screened the titles and abstracts of each study and when a study was judged as relevant, we reviewed through the full text. Disagreements were resolved by a third investigator.

Eligibility criteria

We considered eligible observational studies or randomised clinical trials (RCTs) that enrolled patients >18 years old with HF of ischemic or non-ischemic etiology and provided data about the association of baseline CMR variables with clinical or echocardiographic response to CRT for at least 3 months. We excluded studies that provided data about acute CRT response only, studies that did not offer a definition of CRT response, and studies written in a different language than English.

Data collection process

The following data were extracted for each included study: Publication data (First author, Year of publication), Patient characteristics [number of patients in each group (responders/nonresponders), mean age, gender, type of cardiomyopathy, left ventricular ejection fraction, LBBB, sinus rhythm] and the crude data of the reported outcomes. Two independent investigators performed the data extraction.

Results

Search results

Our search strategy in electronic databases returned 489 possible relevant studies. Of them, 19 studies were excluded as duplicate records, 373 studies were excluded at the title/abstract, level while 50 studies were excluded at the full-text level. As a result, 47 studies were finally included in the review (Figure 2, Table 1).

Association of CMR indices with CRT response

Impact of the myocardial scar on CRT response

The association of myocardial scar with CRT response has been studied in several studies. Both the presence of scar and scar extent/scar burden has been associated with increased non-response rates [14-16]. Also, the location of LV scar has been associated with CRT response [17,18].

In the setting of ischemic cardiomyopathy, a scar size ≥33%, a transmurality ≥51%, and pacing over a posterolateral scar have been associated with a suboptimal response to CRT [19]. By CMR imaging in the DCM group, the percentage of regional scar segments and percentage of regional scar score in the left ventricular inferior wall were significantly higher in the nonresponders than in the responders [20]. However, no statistical difference was found in the ICM, mainly because of the small sample size [20]. Midwall fibrosis seems to have a different impact on outcomes depending on the cause of cardiomyopathy. Specifically, it has been found that left ventricular reverse remodeling was observed in DCM without midwall hyperenhancement and in ICM but not in DCM with midwall hyperenhancement [21]. The same study showed that there were no differences between groups regarding the clinical response [21].

Focal scar burden detected by LGE CMR has been associated with a poor echocardiographic response to CRT, while diffuse interstitial fibrosis assessment by T1 mapping was not associated with CRT response [22]. A high scar tissue burden is more pronounced in nonresponders [23]. Specifically, in an observational study, scar burden as indicated by the total scar burden score and the number of transmurally infarcted segments was significantly higher in nonresponders [24]. Additionally, another study including ischemic and non-ischemic patients, defined scar burden on a 5-point scale, whereby 0 had no scar, and each incremental point corresponded to a group of 25% of transmurality, 1=1-25%, 2=26=50%, 3=51-75%, 4=76-100%. It was found that all patients with high scar burden score of >1.2 failed to respond to CRT [24].

In a prospective, multicenter study, the septal scar was found to be a significant predictor of reverse remodeling defined as at least 15% reduction in LVESV indexed to body surface area at six months follow-up. In comparison, any scar in the septum showed a

sensitivity of 81% for non-response to CRT [25]. In the same study, the combined assessment of septal viability and lateral wall to septal work difference performed better in predicting CRT response than work difference alone [25]. In a small cohort study of both ischemic and non-ischemic patients, both global and lateral wall scar burden were significantly associated with reverse remodeling [26]. Specifically, a cutoff value of 36.5% for global LV scar burden showed a sensitivity of 81.8% and specificity of 68.4% for predicting non-response. In comparison, a cutoff for lateral wall scar burden 40.5% of the whole lateral wall had a sensitivity of 72.7% and specificity of 68.4% [26]. Similarly, another study showed that patients with a transmural posterolateral scar had low clinical response rates [17,18]. A significant positive correlation was found between myocardial scar mass and the evolution of LVESV at 12-month follow-up [27]. On the other hand, in a small observational study, the absence of scar in the posterolateral area (in the region of LV lead placement) was associated with CRT response only in univariate analysis butnot in the multivariate analysis [28]. Interestingly, LV dyssynchrony remained the most critical determinant of response to CRT, even in the presence of posterolateral scar.

LGE$\geq$ 14% is associated with non-response, defined as no death or hospitalisation for a major cardiovascular event, and a significant decrease in left ventricular end-systolic volume of 15% or more in a study with mixed ischemic and non-ischemic patients [29]. The percent of LGE was higher, regional vector of circumferential strain variance was lower and uniformity of radial strain was higher in nonresponders vs. responders.[30] The same study showed that transmurality of LGE was an important predictor of lack of response to CRT in ischemic HF [30]. In a prospective database of patients, it was found that the presence of subendocardial scar on CMR predicted CRT clinical non-response but was not associated with echocardiographic response [10]. Another study which aimed to investigate the role of the delayed enhancement MRI in predicting CRT response showed that a total scar of 15% or septal scar of 40% accurately identified patients with clinical response to CRT [31]. Similarly, in another observational study, the percentage of responders tended to be lower among patients with identifiable areas of delayed enhancement on CMR while scar mass and scar as a percentage of LV myocardium were not significantly different between responders and nonresponders [32].

Scar quantification can have an incremental value in the prediction of CRT response. In this setting, incorporating scar quantification to QRS duration and presence of LBBB was found to improve the prediction of CRT response [33]. The same study also found that an

increase in scar burden was associated with worse outcomes in any location [33]. Native T1 mapping and extracellular volume have also been associated with poor response, while Scar2SD and Gray2SD, markers of focal scar core were substantially better at predicting CRT response [34]. By combining QRSarea and CMR focal scar assessment, CRT response prediction improves beyond that by either vectorcardiography or scar parameters alone [34].

The impact of scar in the left ventricular pacing site on CRT response

Scar in the left ventricular pacing site has been associated with adverse outcomes following CRT implantation [15]. Interestingly, the chronic response has been found to be significantly better in patients paced in a CMR target segment [35]. Pacing in the scar-free segments was found to lead to a greater reverse remodeling [16]. CMR guided lead placement has been associated with significantly better clinical response rates compared to electrophysiological-guided placement in patients with more advanced HF [36]. However, in another study, left ventricular reverse remodeling was less pronounced in the CMR guided and pacing scar group than in the non-CMR guided group [37]. In the CMR guided and non-pacing scar group, a scar burden of < 10% was not associated with a better LV reverse remodeling or clinical response compared with a scar burden of ≥10% [37].

Scar presence, total scar extent, transmural scar, and position the left ventricular lead in an branch of the coronary sinus that correspoinds to an area with transmural scar were associated with non-response to CRT. Interestingly, the non-transmural scar was not associated with CRT response [38]. Another study showed that the presence of septal scar was associated with a poor acute and chronic response to CRT in ischemic cardiomyopathy patients. At the same time, this finding may be related to the inability to achieve a right ventricular septal lead placement [39]. Beyond the impact on CRT response, a myocardial scar in the region of the left ventricular pacing lead has been associated with worse long-term survival in ischemic heart failure patients treated with CRT [40]. A left ventricular lead position over a segment with scar emerged has also been found to be a strong predictor of cardiac mortality and cardiac mortality or HF hospitalisations [41].

On the other hand, results of another prospective study showed that pacing outside of scar compared to pacing within scar did not result in a significant improvement in clinical composite score or reduction in left ventricular end-systolic volume [10]. However, it should be noted that most patients in this study had a subendocardial scar.

A multimodality cardiac imaging using speckle tracking echocardiography and CMR imaging to guide CRT implantation was found to increase the response rates [42]. These findings highlight the potential use of CMR in guiding the LV lead placement during CRT implantation procedure.

Other variables

Combined CMR scar and dyssynchrony imaging prior to CRT identified those patients who subsequently had a clinical response to CRT [43]. A systolic dyssynchrony index derived from volume-change can predict reverse remodeling following CRT whilst a 16-segment systolic dyssynchrony index of regional strain did not [44]. Measures of dyssynchrony (SD-TTP$_{LV}$) and discoordination (ISF$_{LV}$) were strongly related to CRT response when using myocardial tagging [45]. The end-systolic septal strain parameter showed a consistent high correlation with LVESV change for all techniques [45]. Magnetic resonance myocardial tagging (MR-MT) assessment of circumferential mechanical dyssynchrony was found to achieve an excellent predictive value for clinical response following CRT. At the same time, its accuracy could be further improved by combining MR-MT with scar imaging by delayed enhancement magnetic response imaging [46]. Circumferential uniformity ratio estimate, delayed circumferential contraction onset at left ventricular lead position, absent left ventricular lead position scar, and time from QRS onset to left ventricular lead position electrogram have been associated with echocardiographic response [47]. Recently, circumferential uniformity ratio estimated with singular value decomposition with Displacement Encoding with Stimulated Echoes strain imaging was significantly correlated with echocardiographic CRT response [48]. Septal flash predicted increased response to CRT while the presence of septal flash with no scar was a highly specific predictor of CRT response [49]. The left ventricular dyssynchrony and interventricular dyssynchrony were significantly longer in responders, while it was found that the use of the optimal cutoff of left ventricular dyssynchrony ≥ 65 ms can differentiate CRT responders from nonresponders with a sensitivity and a specificity of 100% [50].

On the other hand, internal stretch fraction, defined as the ratio of stretch to shortening during ejection, has been found to differ significantly between responders and non-responders [46] significantly. The findings of this study show that discoordination rather than dyssynchrony can predict the reserve contractile capacity recruited by CRT [51]. An observational study showed that LV shape might play a role in selecting CRT patients [52].

Specifically, it was revealed that nonresponders had a relatively shorter septal wall and longer lateral wall, while in responders, thicker walls in the lateral and basal regions were revealed compared with nonresponders [52]. Time to the onset of circumferential shortening (TOS)/QRS, scar at the LV pacing site, and circumferential uniformity ratio estimate calculated using singular value decomposition have been found to be significantly associated with the change in LVESV 6 months after CRT [53]. Furthermore, the regional vector of strain variance and uniformity of radial strain was found to be lower in nonresponders than responders [30]. In patients with non-ischemic cardiomyopathy, a wide pattern of systolic left ventricular volume/time curves measured using CMR was significantly associated with CRT response compared to patients with a narrow pattern [54]. Summed maximum vortex flow can predict CRT response in DCM, while circumferential temporal delay and longitudinal temporal delay were not associated with CRT response [55]. Furthermore, a standard deviation of 16 segment time-to-peak radial thickness was significantly larger in responders compared with nonresponders [38]. Segment length in cine strain analysis can be used for the prediction of CRT response [56]. Specifically, of all parameters, the end-systolic septal strain showed the strongest correlation with reverse remodeling after CRT [56]. In patients with strict LBBB and with an indication of CRT implantation, a "U-shaped" contraction pattern was found to be strongly predictive for reverse remodeling and super-response [57]. Additionally, a "U-shaped" wall motion pattern compared to a homogenous (type I) wall motion pattern and a concordant LV lead can predict CRT response [44,58]. These findings may help to improve patient selection by evaluating wall motion pattern and targeting LV lead placement.

The right ventricular function may have a significant role in predicting response following CRT implantation. Specifically, right ventricular dysfunction evaluated by CMR has been significantly associated with non-response to CRT. At the same time, the same study showed that late enhancement presence, lateral wall fibrosis, and the percentage of LV fibrosis were not associated with CRT response [59]. Likewise, another small retrospective study confirmed these results showing that except myocardial scar burden, CMR-derived right ventricular dysfunction was an independent predictor of non-response and adverse outcomes in patients on CRT [60].

Discussion

This systematic review shows the additive role of CMR in the selection of potential CRT candidates and the CMR role in guiding the left ventricular lead placement away from

areas of transmural scar, for achieving a better outcome. Furthermore, we confirm that for individuals with high burden of myocardial scar there is less likelihood of having a good response. CRT is mainly recommended in patients with HF and LVEF ≤ 35% in sinus rhythm who have a QRS duration ≥ 130 msec and remain symptomatic despite optimal medical therapy. Additionally, CRT may be considered in patients with HF and LVEF ≤ 35% in AF who have a QRS duration ≥ 130 msec and remain in New York Heart Association (NYHA) class III or IV despite optimal medical therapy, provided biventricular capture can be ensured or the patient is expected to return to sinus rhythm [61]. Occasionally, CRT may be used as an upgrade from a conventional pacemaker or an ICD in patients with HFrEF who develop worsening HF attributable to a high rate of right ventricular pacing [61]. Although it has been shown that CRT improves cardiac function, symptoms, and quality of life, as well as it reduces morbidity and mortality in appropriately selected patients with HFrEF, not all patients who have an indication for CRT according to current guidelines respond favorably to this therapeutic modality [61]. Therefore, novel imaging techniques such as CMR that can assess additional factors to LVEF including myocardial scar, cardiac dyssynchrony, and site of latest activation of the LV are increasingly being studied as utilities for the better selection of patients appropriate for CRT, as well as for optimal lead placement [61]. Whilst identification of areas of myocardial scar can guide the branch positioning of the left ventricular lead in the coronary sinus, it should also be appreciated that complex venous anatomy might prevent pacing the optimal scar-free area [62].

It is already known that the type of cardiomyopathy is a significant predictor of CRT response. Most of the included studies in this review consisted mainly of a mixed population. However, six studies [17,18,16,14,23,19] included ischemic cardiomyopathy patients and one study [55] included only dilated cardiomyopathy patients. In ischemic cardiomyopathy patients, a posterolateral scar imaged using LGE-CMR, was an independent predictor of cardiovascular death or hospitalisations for HF [18]. Furthermore, scar size of 33%, transmurality of 51%, and pacing over a posterolateral scar were associated with a suboptimal response to CRT in ischemic cardiomyopathy patients [17,19]. Scar burden has also been associated with higher non-response rates in the setting of ischemic HF [14,23]. Additionally, LV reverse remodelling following CRT implantation was affected by the total left ventricular scar burden [16]. In the setting of dilated cardiomyopathy, summed maximum vortex flow has been found to predict CRT response [55].

According to our systematic literature review, both the presence and burden of scar as identified by CMR, as well as scar in certain locations, may be useful markers of non-response to CRT. Focal scar burden identified by LGE in CMR, numerically represented by the total scar burden score and/or the number of transmurally infarcted segments, has been shown to be a good marker of poor CRT response. Furthermore, the presence of either any septal scar or increased lateral wall scar burden were associated with less induction of reverse ventricular remodeling, while reports regarding scar in the posterolateral area are mixed, and LV dyssynchrony seems to remain the most important determinant of response to CRT, even in the presence of posterolateral scar. Of note, the combined assessment of septal viability and lateral wall to septal work difference has been reported to be a better marker of CRT response than work difference alone, and regional vector of circumferential strain variance was lower, and uniformity of radial strain was higher in nonresponders. By combining scar quantification and QRS duration in the setting of LBBB, or QRS area and CMR focal scar assessment, CRT response prediction significantly improved. Even further, the available evidence summarised in this review suggests that CMR may have an ancillary role in guiding the LV lead placement during CRT implantation procedure. Indeed, pacing in the scar-free segments has been shown to induce reverse remodeling to a greater extent, and CMR guided lead placement has been associated with a better chronic response and higher BNP response rates. Additionally, pacing over a transmural scar has been associated with non-response to CRT, and pacing over a myocardial scar in the region of the left ventricle has been associated with worse long-term survival in ischemic heart failure patients treated with CRT, and may portend a worse prognosis with regard to cardiac mortality and/or HF hospitalisations. However, CMR guided lead placement has not been found to be superior to electrophysiologically-guided procedures in some studies, and therefore further research is needed before reaching definitive conclusions. Finally, other CMR modalities such as dyssynchrony and strain imaging, as well as discoordination measures may also have and additive role on CRT candidate selection.

Except for chronic CRT response outcome, acute response following CRT implantation is another outcome of interest. CMR identified scar had been found to predict acute CRT response adversely [63]. Specifically, septum-to-lateral wall myocardial work ratio at baseline is significantly related to acute response to CRT defined as acute left ventricular pump function improvement [64]. Furthermore, it has been found that pacing within scar on electroanatomic mapping and CMR (both epicardial and endocardial positions) resulted in failure to capture and a poor acute hemodynamic response [65]. Interestingly, patients with a postero-lateral

scar as identified on CMR can be benefited from a multi-site left ventricular pacing as it can increase acute response by 16% compared to single-site pacing [65].

Beyond the role of CMR in predicting CRT response, it can be used to predict clinically important outcomes during follow-up. Specifically, the presence and size of myocardial scar have been associated with malignant arrhythmic events irrespective of CRT response [27]. Additionally, the presence of myocardial scar has been associated with all-cause mortality [27], while myocardial dyssynchrony assessed by CMR-tissue synchronisation index was an independent predictor of mortality and morbidity after CRT [66]. An index derived by a combination of dyssynchrony, posterolateral scar location, and creatinine has been significantly associated with cardiovascular mortality [67].

As already discussed, pacing in the scar-free segments has led to a greater reverse remodeling and better long-term survival [40,16]. Overlaying the CMR and CT dataset onto live fluoroscopy during left ventricular lead placement is a feasible technique that may lead to a greater reverse remodeling during follow-up [68]. A double-blind, randomised controlled trial showed the clinical benefit of multimodality imaging-guided left ventricular lead placement in CRT [69]. Specifically, it was found that imaging-guided LV lead placement using cardiac computed tomography venography, 99mTechnetium myocardial perfusion imaging, and speckle-tracking echocardiography radial strain reduced the number of clinical nonresponders [69]. However, a recent randomised study failed to demonstrate the benefit of the evaluation of delayed activation on echocardiography combined with anatomic information from computed tomography (coronary sinus tributary anatomy) and CMR (large scar preventing lead placement) in terms of clinical, echocardiographic response or in a significant reduction of death or heart failure hospitalisation [70].

Several echocardiographic and clinical definitions of CRT response have been used to assess the efficacy of CRT [71]. CMR has also been used to evaluate CRT response, providing very high-quality data on the right/left ventricular function and strain/synchrony before and after CRT [72].

This systematic literature review has several potential limitations. Firstly, our results were derived from observational studies, which are subject to selection biases and have a limited ability to determine causality. In addition, populations were heterogenous between the studies which prevented reaching definitive conclusions. Furthermore, as all the studies included patients who fulfilled the clinical guideline criteria for a CRT, we were unable to review non-traditional criteria such as pacing-induced cardiomyopathy. Finally, the included

studies provided dissimilar CMR data and how the scar was quantified and therefore a quantitative synthesis was not feasible.

Conclusions

The clinical significance of CMR is to identify scar regions that should be avoided during the coronary sinus LV lead implantation. Furthermore, CMR can be used as an optimisation tool for identifying those patients with extensive myocardial scar who might achieve a sub-optimal clinical and echocardiographic response following CRT implantation. However, further research is needed to elucidate the role of CMR in predicting important outcomes in CRT patients.

Acknowledgments

None

Conflicts of interest

None

Figures

Figure 1. Imaging in the late phase following gadolinium administration of the mid ventricular level. The white part of the myocardium in the septal and anteroseptal areas, indicated by the yellow arrows, correspond to hyperenhancement of the myocardium, and effectively dead muscle. This was as a result of a left anterior descending artery myocardial infarction. The remaining myocardium which is black in colour is healthy.

Figure 2. Flowchart of the search strategy

Tables

Table 1. Baseline characteristics and main findings of the included studies.

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