Utility of Cardiac Magnetic Resonance Imaging Versus Cardiac Positron Emission Tomography for Risk Stratification for Ventricular Arrhythmias in Patients With Cardiac Sarcoidosis

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Abnormalities on cardiac magnetic resonance imaging (CMR) and positron emission tomography (PET) predict ventricular arrhythmias (VA) in patients with cardiac sarcoidosis (CS). Little is known whether concurrent abnormalities on CMR and PET increases the risk of developing VA. Our aim was to compare the additive utility of CMR and PET in predicting VA in patients with CS. We included all patients treated at our institution from 2000 to 2018 who (1) had probable or definite CS and (2) had undergone both CMR and PET. The primary endpoint was VA at follow up, which was defined as sustained ventricular tachycardia, sudden cardiac death, or any appropriate device tachytherapy. Fifty patients were included, 88% of whom had a left ventricular ejection fraction >35%. During a mean follow-up 4.1 years, 7/50 (14%) patients had VA. The negative predictive value of LGE for VA was 100% and the negative predictive value of FDG for VA was 79%. Among groups, VA occurred in 4/21 (19%) subjects in the LGE+/FDG+ group, 3/14 (21%) in the LGE+/FDG− group, and 0/15 (0%) in the FDG+/LGE− group. There were no LGE−/FDG− patients. In conclusion, CMR may be the preferred initial clinical risk stratification tool in patients with CS. FDG uptake without LGE on initial imaging may not add additional prognostic information regarding VA risk. **(Am J Cardiol 2020;134:123−129)**

In patients with sarcoidosis, the prevalence of cardiac sarcoidosis (CS) ranges from 5% to 17% and brings increased risk for ventricular arrhythmias (VA) and sudden cardiac death.[1−4] The predisposition for VA in CS may occur in the absenc of traditional risk factors including despite preservation in left ventricular ejection fraction (LVEF) and may manifest as the presenting clinical feature of CS.[5−8] Late gadolinium enhancement (LGE) on cardiac magnetic resonance imaging (CMR) and 18-flourodeoxyglucose (FDG) uptake on cardiac positron emission tomography (PET) have both shown promise for risk stratification in VA and are often used in complement to one another in clinical practice.[6,8−10] However, the relationships that govern inflammation, fibrosis, and arrhythmogencity as well as the ability to evaluate these using cardiac imaging in CS remain incompletely understood.[4] As a result, when and how to utilize each modality remains a topic of debate.[11] The aim of our study was to compare the utility of PET and CMR to predict VA in patients with CS.

Methods

The electronic medical record (EMR) was queried for patients treated at Johns Hopkins Hospital between 2000 and 2018 who(1) had a diagnosis of probable or definite CS according to the criteria set forth in the 2014 Heart Rhythm Society (HRS) Expert Consensus Statement[10]; (2) had undergone both CMR and PET.

The study was approved by our Institutional Review Board and conducted in accordance with institution guidelines.

1.5-T magnetic resonance imaging units (GE Medical Systems, Waukesha, Wis; or Avanto, Siemens, Erlangen, Germany) were utilized to obtain CMR images using previously described techniques of electrocardiographic gating and breath holding.[12] Late gadolinium enhancement (LGE) imaging was performed 10 to 18 minutes after injection of 0.2 mmol/kg of gadolinium (gadopentetate dimeglumine; Bayer Healthcare Pharmaceuticals, Montville, New Jersey). We utilized phase sensitive inversion recovery gradient recall echo sequences (repetition time of 2.5 to 5.5 ms, echo time of 1.52 ms, flip angle at 10°, in-plane resolution of 1.3 × 1.3, slice thickness of 8.0 mm, and inversion time selected for maximal myocardial nulling, typically 240 to 290 ms) for the assessment of focal myocardial fibrosis.

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Financial Support: This study was supported by Robert E. Meyerhoff Professorship to Jonathan Chrispin, MD. All authors have reviewed and signed the confidentiality agreement.

See page 128 for disclosure information.

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0002-9149/
https://doi.org/10.1016/j.amjcard.2020.08.007

Metabolic imaging was performed with cardiac PET/Computed tomography (CT; Discovery Rx VCT PET/CT [GE Healthcare, Milwaukee, Wisconsin]). Preceding PET imaging, patients were instructed to follow a previously described dietary pattern (24 hours of high fat, low carbohydrate intake followed by 12-hour fast) for optimal suppression of FDG uptake by normal myocardium through preferential fatty acid metabolism.[14] Cardiac and whole-body FDG PET/CT scans were performed 60 minutes after 18F-FDG was administered intravenously 0.135 mCi/kg as previously described.[13] PET perfusion imaging was not performed. Only studies used and interpreted for clinical examination were included, excluding those with poor PET preparation or FDG myocardial suppression.

Experienced clinical radiology and nuclear medicine physicians interpreted CMR and PET images respectively as a part of clinical care, and these interpretations were accessed in the EMR. CMR and PET images were categorized as either positive or negative for presence of LGE or focal FDG uptake respectively based on clinical interpretation.

The EMR was queried by natural language search capability and confirmed in a structured fashion for clinical VA occurring after the time of CMR or PET, which included sustained VT, ventricular fibrillation, sudden cardiac death, or any appropriate device tachytherapy. Repeat imaging was not included. Device tachytherapies without a documentation of underlying rhythm were included. Follow-up time was defined as the time from initial CMR until the last documented clinical encounter in the EMR.

Study data were collected and managed using REDCap (Research Electronic Data Capture) electronic data capture tools housed at Johns Hopkins University and statistical analyses were performed using the STATA software (Version 14.1, StataCorp, College Station, Texas). Continuous data is presented as mean ± standard deviations, while categorical variables are presented as percentages. Comparisons between patients were performed using the Student *t* test or Analysis of variance (continuous variables) and the Chi-square test or Fisher's exact test (categorical variables, based on expected frequency 5+ or <5, respectively). Cox proportional hazards model was used to determine the association of imaging variables and incident VA. Long-rank test was used to compare patients categorized by their cardiac imaging findings for VA-free survival. A p-value of ≤0.05 was considered statistically significant.

Results

A total of 402 consecutive patients were referred for evaluation of known or suspected CS by CMR at Johns Hopkins Hospital (Baltimore, MD) between January 1, 2000 and December 30, 2018. Overall, 92 patients met HRS criteria for probable or definite CS, 50 of which underwent both CMR and PET imaging, which was the analyzed study cohort.[10] Table 1 summarizes the baseline characteristics of the study cohort. The mean age was 52 ± 10 years, 42% were female, and 40% were black. The mean LVEF was 53 ± 14%, and 88% had LVEF >35%. 96% had biopsy-proven extra-CS. Seventy percent were on some form of immunosuppression prior to CMR imaging.

CMR revealed LGE in 35 (70%) patients. PET revealed FDG uptake in 36 patients (72%). Among these, 21 (42%) had both LGE and FDG uptake (Figure 1). There were 0 patients without PET and CMR imaging. Median time between CMR and PET was 259.5 days. The average time of clinical follow-up was 1,714 days. Figure 2 provides a flow chart stratifying VA events first by presence or absence of imaging findings and second by positive or negative VA.

VA occurred in a total of 7 (14%) patients. In total, there were 4 (8%) device shocks, 4 (8%) episodes of antitachycardia pacing, 3 sustained ventricular tachycardia (VT) (6%) including 1 VT storm (2%), and 1 ventricular fibrillation (2%). Four patients had recurrent VA events.

In patients with VA, 4 (57%) had FDG uptake and 7 (100%) had LGE. Among FDG+/LGE-, FDG+/LGE+, and FDG−/LGE+ patients, there was no difference in VA event rates (Figure 3). Due to the absence of VA in the FDG +/LGE- group, VA-free survival is reported as log-rank rather than hazard ratios. There was a statistically significant difference in VA-free survival between CS patients with LGE versus those without (p = 0.05) (Figure 4). There was no difference in VA-free survival between CS patients with FDG uptake versus those without (p = 0.18) (Figure 5). Among groups, VA occurred in 4/21 (19%) subjects in the

Table 1
Baseline characteristics of study population

	All subjects	+FDG/+LGE	+FDG/−LGE	−FDG/+LGE	p-Value
Total number	50	21	15	14	
Age (years)	53 ± 14	53 ± 11	50 ± 9	52 ± 9	0.66
Men	29 (58%)	13 (62%)	7 (47%)	9 (64%)	0.62
Caucasian	28 (56%)	13 (62%)	7 (47%)	8 (57%)	0.72
Left ventricular ejection fraction (%)	53 ± 14	52 ± 13	57 ± 11	52 ± 17	0.56
Left ventricular ejection fraction >35%	44 (88%)	18 (86%)	15 (100%)	11 (79%)	0.18
History of ventricular arrhythmia	10 (20%)	8 (38%)	0 (0%)	2 (14%)	0.01
Coronary artery disease	3 (6%)	3 (14%)	0 (0%)	0 (0%)	NA
Congestive heart failure	23 (46%)	8 (38%)	7 (47%)	8 (57%)	0.54
Immunosuppression	30 (60%)	13 (62%)	9 (60%)	8 (57%)	1.0
Late gadolinium enhancement	35 (70)				
18-Flurodeoxyglucose	36 (72)				

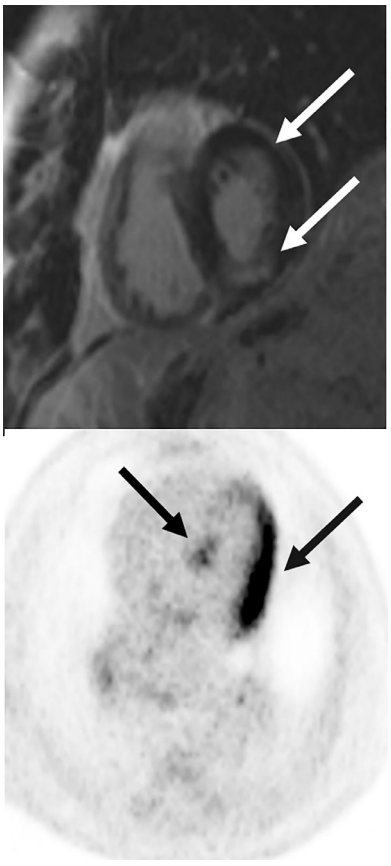

Figure 1. Characteristic Imaging of CS Patients. (*A*) Cardiac magnetic resonance imaging. Short axis slice at the basal level showing midmyocardial inferior scar and anterolateral midmyocardial and subendocardial scar (blue arrows). (*B*) PET imaging. Short axis slice at the basal level showing FDG uptake in the lateral wall and septum (blue arrows). CS = Cardiac sarcoidosis; PET = positron emission tomography; FDG = 18-flurodeoxyglucose.

LGE+/FDG+ group, 3/14 (21%) in the LGE+/FDG− group, and 0/15 (0%) in the FDG+/LGE− group. There were no LGE−/FDG- patients.

Those with VA were significantly more likely to have had prior VA (71% vs 12%, p = 0.005). There was a significant difference in history of VA among FDG+/LGE−, FDG+/LGE+, and FDG-/LGE+ patients (p = 0.01) (Table 1). Those with VA were significantly more likely to have LVEF <35% (28% vs 9%, p = 0.025). There were no significant differences between those with and without VA with respect to age, sex, race, or use of immunosuppression. Univariate analysis of these characteristics with respect to VA is summarized in Table 2.

The test characteristics for CMR and PET for predicting VA are summarized in Table 3. The sensitivity of FDG for VA was 57% and the sensitivity of LGE for VA was 100%. The specificity of FDG for VA was 26% and the specificity of LGE for VA was 35%.

Discussion

The purpose of this study was to identify the combined utility of CMR and PET in identifying risk of VA in patients with CS. In this cohort of HRS guideline defined CS patients having undergone both CMR and PET there were 3 major findings. First, all patients with VA had LGE on CMR, resulting in excellent negative predictive value (NPV) for VA; NPV for FDG was modest in comparison. Second, the addition of FDG uptake did not result in an increase in risk for VA in CS patients who are LGE positive. Third, a history of prior VA or LVEF <35% comprised substantial risk factors for future VA.

In our cohort, FDG uptake on PET was not predictive of VA in CS patients. The literature is mixed on the impact of FDG uptake in predicting VA in CS patients. Blankstein et al showed an increased likelihood of VA in 118 patients with suspected CS who had FDG uptake on PET, particularly when located in the RV or when combined with perfusion defects, although the majority of patients with imaging abnormalities do not develop VA (hazard ratios 2.6 to 4.2).[6] However, in a study by Bravo et al of 56 patients with suspected CS who underwent PET and CMR, FDG uptake was associated with a hazard ratio of 3.3 but not found to be an independent predictor of VA or death when adjusting for LGE.[14] This finding is congruent with our results: most CS patients with VA who had FDG uptake also had LGE on CMR. However, given the construction of the studies and varying follow-up event times, neither study was designed to assess the temporal relation between active inflammation identified by FDG uptake and VA. Moreover, information regarding dose and duration of

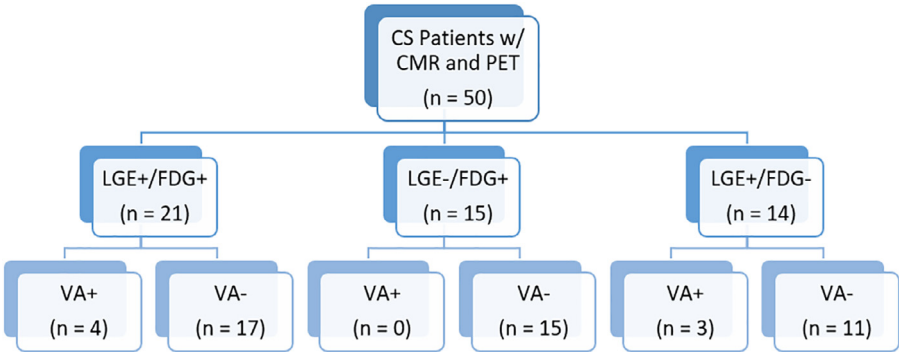

Figure 2. Flow chart of study population. CS patients were stratified first by presence or absence of LGE/FDG and second by VA on follow-up. CS = Cardiac sarcoidosis; CMR = cardiac magenetic resonance imaging; FDG = 18-flurodeoxyglucose; LGE = late gadolinium enhancement; PET = positron emission tomography; VA = ventricular arrhythmia.

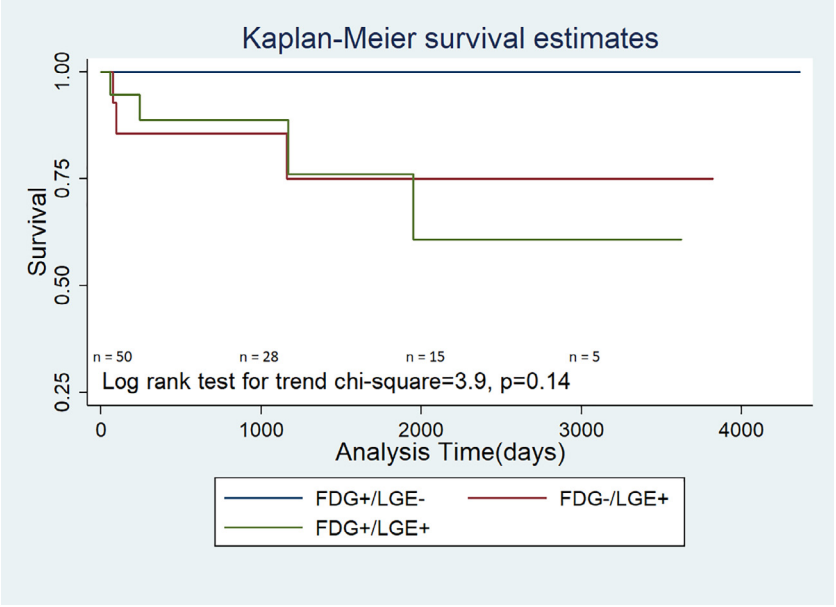

Figure 3. Kaplan-Meier curves for VA-free survival based on the presence and absence of LGE and FDG. FDG = 18-flurodeoxyglucose; LGE = late gadolinium enhancement; VA = ventricular arrhythmia.

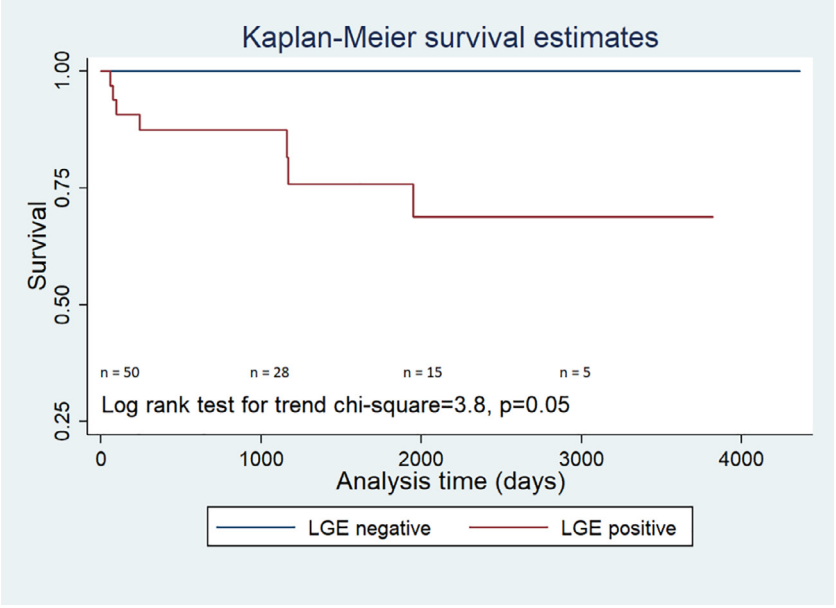

Figure 4. Kaplan-Meier curves for VA-free survival based on the presence and absence of LGE. LGE = late gadolinium enhancement; VA = ventricular arrhythmia.

immunosuppression was limited; inflammation on PET may have been suppressed with immunosuppressants before scar formation in the FDG+/LGE- group.

In our cohort, LGE on CMR had excellent negative predictive value and moderate-high hazard for VA in CS patients. In addition, all patients with a history of VA had LGE on CMR. This supports the current body of literature on the prognostic value of LGE for VA. Our study resulted in a comparable association between LGE and risk of VA as found in the literature. Hulten et al reviewed 7 studies of 694 suspected CS patients who underwent CMR, and found a cumulative relative risk of 19.5 for VA in patients with LGE.[15] Coleman et al reviewed 10 studies of 760 patients with suspected CS who underwent CMR, and found a cumulative odds of 10.7 for VA in patients with LGE, primarily driven by patients with LVEF >50%.[8] Both studies suggested LGE as an independent prognostic factor for VA in meta-analyses of cohorts of suspected CS undergoing CMR only. The study by Bravo et al included a cohort of suspected CS patients with higher event rates, found that only LGE was associated with VA risk in multivariate analysis, and suggested that LGE may be the preferred imaging utility for stratification of VA risk.[14] However, the study did not contain a cohort of patients with FDG uptake without LGE; moreover, only 36% of patients (n = 20) met HRS criteria for CS in the study, suggesting arrhythmogenic risk

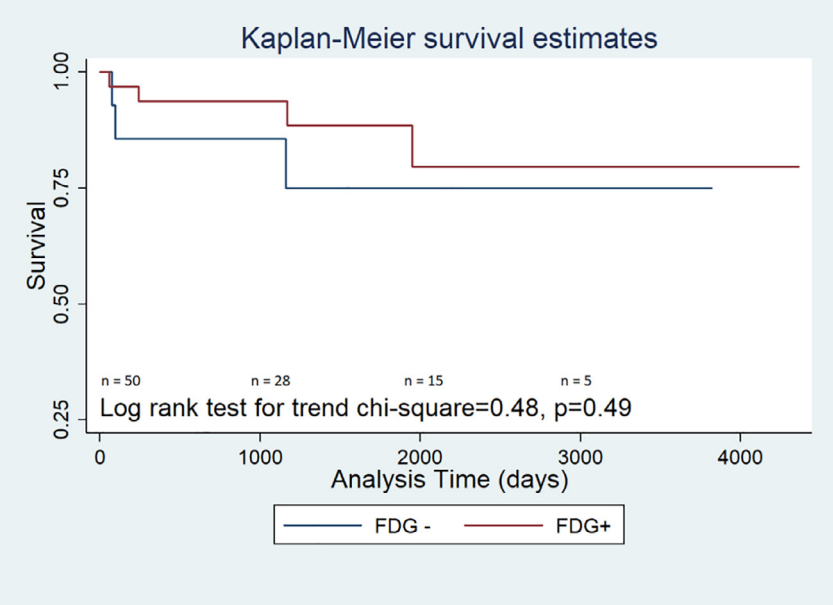

Figure 5. Kaplan-Meier curves for VA-free survival based on the presence and absence of FDG. FDG = 18-flurodeoxyglucose; VA = ventricular arrhythmia.

Table 2
Univariate analysis of ventricular arrythmia events in study population

Variable	VA+ (7)	VA- (43)	HR	p-Value
Age (years)	50 +- 11	52 +- 10	.99	0.72
Men	6 (86%)	23 (53%)	1.4	0.58
Black	1 (14%)	19 (44%)	0.21	0.16
Ventricular arrythmia	5 (71%)	5 (12%)	10.5	0.005
Coronary artery disease	4 (57%)	19 (44%)	1.4	0.65
Congestive heart failure	1 (14%)	2 (5%)	4.2	0.19
Left ventricular ejection fraction (%)	52 +- 17	54 +- 13	0.97	0.22
Left ventricular ejection fraction >35%	2 (28%)	4 (9%)	5.6	0.025
Late gadolinium enhancement	7 (100%)	28 (65%)	3.8*	0.05*
18-Flurodeoxyglucose	4 (57%)	32 (74%)	0.59	0.492
Immunosuppression	4 (57%)	26 (60%)	3.9	0.2

* The reported number and p-value are calculated for Log Rank test.

Table 3
Test characteristics of FDG and LGE alone and together for predicting VA

	Sensitivity	Specificity	PPV	NPV	Odds ratio	Confidence interval
18-Flurodeoxyglucose+	57%	26%	11%	79%	0.46	(0.09−2.4)
Late Gadolinium Enhancement+	100%	35%	20%	100%	8.2	(0.4−152.6)
18-Flurodeoxyglucose+ and Late Gadolinium Enhancement+	57%	60%	19%	90%	2.0	(0.4−10.3)

attributed to LGE may be confounded by inclusion of other cardiomyopathies with high VA risk.[14,16]

In our cohort, CS patients with both LGE and FDG uptake on CMR and PET, respectively, were not shown to have increased risk for VA in comparison to those with LGE uptake alone. This supports the current body of literature on the prognostic value of both LGE and FDG uptake for VA. In the aforementioned study by Bravo et al, no additional risk was seen in patients with FDG, although the authors continued to recommend PET as a complementary method for diagnostic and medical management purposes.[14] In a slightly different population, Vita et al retrospectively reviewed 107 patients referred for evaluation of CS who underwent both PET and CMR, and found similar rates of composite adverse events of ventricular tachycardia and all-cause mortality in patients with LGE and patients with both LGE and FDG uptake as compared with those without LGE.[17] Only 31% (n = 33) of these patients met HRS or Japanese Ministry of Health and Welfare (JMHW) criteria for CRS, predominantly in the subgroup with both LGE and FDG uptake.[17] Wicks et al recently compared imaging data from 51 patients with suspected CS undergoing hybrid

PET/CMR. In their cohort, 65% met JMHW criteria for CS, and they found hazard ratios of 8.0 for LGE and 5.8 for RV-FDG uptake after both were adjusted for LVEF for a primary composite outcome which included complete heart block and hospital admission for acute decompensated heart failure combined with mortality, sustained VA, and sudden cardiac death.[18]

The present study is the largest study to compare prognostic utility of CMR and PET for VA in CS patients meeting HRS criteria undergoing both imaging modalities. The findings of our study build on the existing literature by affirming previous findings in a HRS-positive cohort. In comparison to prior studies, this study was a larger CS cohort with a lower event rate using a narrower arrhythmia-related composite outcome. These findings suggest scar as the dominant substrate for VA in patients with CS and support CMR as the primary imaging modality for risk stratification of future VA in CS patients based on its negative predictive value. Although history of VA and LVEF <35% conferred independent risk in this cohort, the majority of patients with VA had LVEF >35%, supporting CMR as a primary risk stratification tool in VA-free CS patients not otherwise meeting a class I indication for an implantable cardiac defibrillator.[9]

This study had several limitations. The modest sample size is a function of disease prevalence and supports the study of larger multicenter cohorts. Its retrospective design is limited by the clinical indications for CMR and PET imaging, which may portend for unintended selection bias. CMR and PET imaging and their familiarity have both advanced during the time frame of the study in which clinical interpretations were utilized. Dietary indiscretion and resultant confounding may not always be clear from FDG-PET pattern recognition alone. CMR and PET did not occur simultaneously and subsequent imaging studies that may have been performed were not included. These may not have accounted for the dynamic nature of myocardial inflammation in this cohort of CS patients. There were no FDG-/LGE- patients in our cohort, likely related to limited use of endomyocardial biopsies at our institution, which may impact results. The event rate was more modest than in some other studies, although this may be related to our use of stricter inclusion criteria, specifically meeting the definition of CS, to avoid inclusion of other nonischemic cardiomyopathies that may have been included in previous studies. We cannot exclude the possibility of undocumented VA events despite robust follow-up (84% with >12 months follow-up). Additionally, risk of VA in CS patients was most strongly associated with previous history of VA, necessitating further studies to help generalize these results to a VA-free CS patient population when considering the impact of imaging-based risk stratification for device implantation.

In conclusion, CMR may be the preferred clinical VA risk stratification tool in patients with CS, including those with LVEF >35% and those without conventional indications for ICD therapy. Presence of FDG uptake on initial imaging may not provide additional prognostic information regarding VA risk if CMR is available, but PET remains an important tool for diagnosis and guiding immunosuppression in CS. Additional studies are required to help define the role of imaging in risk stratification for VA in CS.

Author Statement

Gowani: Conceptualization, Validation, Formal Analysis, Investigation, Data Curation, Visualization, Writing – Original Draft and Review and Editing. **Habibi**: Validation, Formal Analysis, Supervision. **Okada**: Conceptualization, Validation, Methodology, Investigation, Data Curation, Visualization, Project Administration, Supervision. **Smith:** Conceptualization, Validation, Investigation, Data Curation. **Derakhshan**: Conceptualization, Validation, Investigation, Data Curation: **Misra:** Writing – Review and Editing. **Gilotra:** Writing – Review and Editing. **Berger:** Conceptualization. Writing – Review and Editing. Supervision. **Caulkins:** Writing – Review and Editing. Supervision. **Tandri:** Conceptualization. Writing – Review and Editing. Supervision. **Chrispin:** Conceptualization, Validation, Methodology, Data Curation, Visualization, Project Administration, Funding Acquisition, Resources, Supervision. Writing – Original Draft and Review and Editing.

Disclosures

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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